

# Overestimation of pulmonary artery occlusion pressure in pulmonary hypertension due to partial occlusion

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**Objective:** To evaluate partial occlusion in patients with pulmonary hypertension with regard to a) the degree to which it leads to overestimation of pulmonary artery occlusion pressure (Ppao) and b) identification of factors that could enhance its recognition.

**Design:** Observational descriptive study.

**Setting:** Medical intensive care unit.

**Patients:** Fourteen patients with pulmonary hypertension and an increased pulmonary artery diastolic pressure (Ppad) – Ppao gradient ( $\geq 10$  mm Hg).

**Interventions:** None.

**Measurements and Main Results:** The Ppao was recorded during partial occlusion (partial Ppao) and after catheter repositioning to obtain a lower, more accurate value (best Ppao). The error due to partial occlusion, defined as the difference between the partial Ppao and the best Ppao, was  $13 \pm 5$  mm Hg (range,

6–21 mm Hg). The previously widened Ppad – Ppao gradient invariably narrowed during partial occlusion and then increased by  $13 \pm 5$  mm Hg (range, 5–23) during the best Ppao measurement. There was a moderate correlation between the error due to partial occlusion (partial Ppao – best Ppao) and both the mean pulmonary artery pressure ( $r = .77, p < .01$ ) and the Ppad – Ppao gradient ( $r = .79, p < .01$ ).

**Conclusions:** Partial occlusion in patients with pulmonary hypertension may lead to significant overestimation of the Ppao and should be suspected when there is a substantial increase in the Ppao without a concomitant increase in the Ppad, as reflected by a marked narrowing of a previously widened Ppad – Ppao gradient. (Crit Care Med 2003; 31:93–97)

**KEY WORDS:** catheterization; pulmonary artery flotation catheter; pulmonary occlusion pressure; hypertension; pulmonary; hemodynamics; physiologic monitoring; critical care

The pulmonary artery occlusion pressure (Ppao) often has considerable influence on the diagnosis and management of critically ill patients who undergo right heart catheterization. It is therefore crucial that the Ppao be measured accurately. In the intensive care unit, the two principle criteria used to confirm transition from a pulmonary artery pressure (Ppa) to a Ppao tracing after balloon inflation are the appearance of a low-amplitude atrial (venous) waveform and a decrease in pressure (1). In the absence of prominent left atrial *a* or *v* waves or pulmonary valve insufficiency, the mean Ppao should be equal to or less than the pulmonary artery end-diastolic pressure (Ppad), with the difference between the Ppad and Ppao (Ppad – Ppao gradient) reflecting pulmonary vascular resistance (2). In the normal pulmonary circulation, there is little or no forward blood flow within the lung by end-diastole, and the

Ppad – Ppao gradient is minimal (0–5 mm Hg) (3). A widened Ppad – Ppao gradient is seen in pulmonary hypertension due to increased pulmonary vascular resistance (4).

One problem that can interfere with accurate measurement of the Ppao is partial occlusion or “partial wedging” (5). This occurs when the catheter’s inflated balloon does not completely occlude the pulmonary artery, resulting in a measured pressure that is intermediate between the mean Ppa and left atrial pressure (5, 6). If pulmonary vascular resistance is normal, partial occlusion will be evidenced by a measured Ppao that unexpectedly exceeds the Ppad (7). In contrast, with increased pulmonary vascular resistance and a widened Ppad – Ppao gradient, partial occlusion may be less apparent since the Ppao may remain less than or equal to the Ppad (8). A schematic depiction of partial occlusion with a normal and widened Ppad – Ppao gradient is shown in Figure 1.

In this article we examine the problem of partial occlusion in patients with pulmonary hypertension. The purpose of our report is to increase awareness of this problem, to determine the magnitude of

error in measurement of the Ppao, and to evaluate potential factors that could enhance bedside recognition.

## METHODS

We documented partial occlusion in 14 patients with pulmonary hypertension (mean Ppa  $> 25$  mm Hg) and a widened Ppad – Ppao gradient ( $\geq 10$  mm Hg) who had undergone pulmonary artery catheterization in our medical intensive care unit between June 1997 and July 2001. Episodes of partial occlusion were identified prospectively by one of the authors and were typically suspected when a routinely scheduled hemodynamic assessment revealed a Ppao that was significantly (and unexpectedly) higher than earlier values. In a few instances, partial occlusion occurred at the time of initial catheter insertion and was suspected because the measured Ppao was much higher than anticipated. Partial occlusion was confirmed when catheter manipulation, without any other changes, resulted in a significantly lower Ppao upon repeat balloon inflation. The Ppao measured during partial occlusion was termed the “partial Ppao,” and the “best Ppao” was defined as the lowest Ppao accompanied by a tracing whose overall appearance was consistent with an atrial waveform (although the individual components of the atrial waveform could not be identified in every case).

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Our definition of best Ppao was based on the principle that while the pressure recorded by a partially occluded catheter could overestimate left atrial pressure by varying degrees, the measured Ppao should never underestimate left atrial pressure. Maneuvers used to correct partial occlusion included slight advancement of the fully inflated catheter, withdrawal of the deflated catheter to a more proximal position after which it was inflated and readvanced, and use only 1–1.2 mL of air to obtain the best Ppao. The particular method chosen to achieve a best Ppao was sometimes influenced by factors such as catheter insertion distance and the position on chest radiographs but was often determined by trial-and-error. The best Ppao was always obtained within a few minutes of the episode of partial occlusion, during which time there no changes in drug therapy or clinical status.

All pressure measurements were made at end-expiration with the patient relaxed and without change in ventilator settings. Pressures were read from paper strip recordings of pressure tracings and a simultaneous electrocardiographic lead. To avoid underestimation of the Ppad due to catheter ringing, the Ppad was recorded just before the systolic increase in pressure, as suggested previously (9). All pressure tracings were recorded and analyzed by the authors. Data are expressed as mean  $\pm$  sd. Our institutional review board approved data collection and analysis without need for informed consent.

## RESULTS

Demographic and baseline hemodynamic data for the 14 patients are shown in Table 1.

The difference between the partial Ppao and best Ppao was  $13 \pm 5$  mm Hg (range, 6–21 mm Hg). The partial Ppao and best Ppao for individual patients are given in Table 2 and shown in Figure 2A.

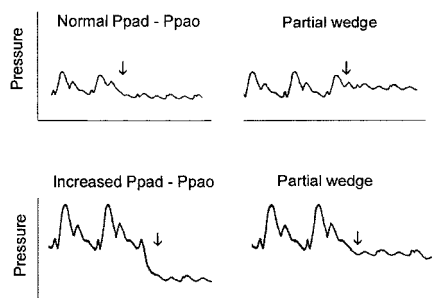


Figure 1. Schematic depiction of partial occlusion when the gradient between the pulmonary artery systolic pressure (Ppas) and pulmonary artery occlusion pressure (Ppao) is normal (top) or is increased (bottom). Arrow denotes balloon inflation.

The change in the Ppad – Ppao gradient between the partial Ppao and best Ppao measurements was  $13 \pm 5$  mm Hg (range, 5–23 mm Hg). The Ppad – Ppao gradients for individual patients during partial Ppao and best Ppao measurements are shown in Figure 2B.

The relationships between the error due to partial occlusion (partial Ppao – best Ppao) and indexes of pulmonary hypertension are shown in Figure 3. There

Table 1. Patient demographic and baseline hemodynamic data

Total subjects, n	14
Female, n	5
Age, yrs	58 (15)
Primary diagnosis, n	
ARDS	7
Pulmonary fibrosis	3
Scleroderma	1
Cirrhosis	2
Heat stroke	1
Hemodynamic variables	
MAP, mm Hg	77 (14)
HR, beats/min	100 (19)
CI, L/min	3.02 (0.94)
Pra, mm Hg	13 (4)
Mean Ppa, mm Hg	36 (10)
Ppao, mm Hg	14 (4)
Ppad – Ppao, mm Hg	14 (4)
SVRI, dynes·sec·cm <sup>–5</sup>	1840 (691)
PVRI, dynes·sec·cm <sup>–5</sup>	646 (315)

ARDS, acute respiratory distress syndrome; MAP, mean systemic arterial pressure; HR, heart rate; CI, cardiac index; Pra, right atrial pressure; Ppa, pulmonary artery pressure; Ppao, pulmonary artery occlusion pressure; Ppad, pulmonary artery diastolic pressure; SVRI, systemic vascular resistance index; PVRI, pulmonary vascular resistance index.

Values are mean (SD) unless otherwise noted.

Table 2. Pulmonary artery and pulmonary artery occlusion pressures for individual patients

Patient	Ppas	Ppad	Partial Ppao	Best Ppao	Best Ppad – Ppao
1	56	36	32	17	19
2	55	30	28	12	18
3	37	28	25	15	13
4	75	42	30	18	24
5	40	26	25	15	11
6	48	28	28	16	12
7	54	37	37	16	21
8	37	24	22	12	12
9	59	26	24	10	16
10	32	17	15	7	10
11	72	38	32	11	27
12	42	20	17	10	10
13	35	23	18	12	11
14	80	29	29	12	17
Mean	52	29	26	13	16
SD	16	7	6	3	5

Ppas, pulmonary artery systolic pressure; Ppad, pulmonary artery diastolic pressure; Ppao, pulmonary artery occlusion pressure.

Values are in mm Hg.

was a moderate correlation between both the partial occlusion error and the mean Ppa ( $r = .77, p < .01$ ) and the partial occlusion error and the Ppad – Ppao gradient ( $r = .79, p < .01$ ).

Examples of pressure tracings are shown in Figures 4–6. Figure 4 shows four measurements of Ppao over a period of a few minutes, with the first three tracings representing varying degrees of partial occlusion. In Figure 5, the partial Ppao tracing contains a single positive pressure wave that occurs at precisely the same time (relative to the electrocardiogram) as the Ppa systolic pressure and before a left atrial *v* wave would be seen (10), indicating that the tracing does not represent a left atrial waveform. Figure 6 shows Ppao tracings resulting from inflation of the catheter balloon with different volumes of air, without other manipulation. The best Ppao was obtained with the lower balloon volume. Review of the chest roentgenogram in this patient revealed that the catheter tip was too peripheral, and the catheter was subsequently retracted.

## DISCUSSION

The principle finding of this study is that partial occlusion in patients with pulmonary hypertension may result in significant overestimation of the Ppao, with the error being greatest in patients who have the largest Ppad – Ppao gradients. When the Ppad – Ppao gradient is minimal, clinically relevant overestimation of the Ppao due to partial occlusion should be evidenced by a Ppao that ex-

ceeds the Ppad. In a previous study, Wilson et al. (7) reviewed more than 1900 simultaneous measurements of the Ppad and Ppao and found that the Ppao unexpectedly exceeded the Ppad in almost 20% of cases, and in 5% of cases the difference was >5 mm Hg. The authors concluded that greater attention to the relationship between the Ppad and Ppao might reduce Ppao measurement errors and that the accuracy of the Ppao should be questioned if it exceeds the Ppad (7). While we fully agree with this conclusion, the results of our study indicate that a positive Ppad – Ppao gradient does not necessarily ensure reliability of the measured Ppao in the setting of pulmonary hypertension.

As it was not possible to obtain independent measurements of left atrial pressure, the interpretation of our data hinges on our assumption that with catheter manipulation, the best Ppao was always the lowest Ppao accompanied by a tracing consistent with an atrial waveform. Essential to this assumption is the basic concept of what the Ppao actually represents. When the balloon of the pulmonary artery catheter is inflated and completely interrupts flow in a branch of the pulmonary artery, the catheter will measure the pressure in the medium-to-large pulmonary veins at the junction (j point) of the static column of blood created by arterial occlusion and free-flowing blood in the unobstructed pulmonary vasculature (11). Since there is rarely any pressure gradient between the medium-to-large pulmonary veins and the left atrium, the Ppao accurately reflects left atrial pressure (11). If the in-

flated catheter only partially obstructs the branch of the pulmonary artery, there may be some continued flow around the catheter tip, and the measured Ppao may consequently overestimate left atrial pressure by varying degrees. However, when the Ppao is measured at end-expiration and the catheter-transducer system is functioning optimally, we can think of no circumstance in which the measured Ppao should underestimate left atrial pressure. Hence, we defined best Ppao as the lowest Ppao that can be obtained.

The position of the j point, and thus the measured Ppao, may be influenced by the size artery that is obstructed. If the catheter is wedged into a small pulmonary artery without balloon inflation, the j point will move upstream toward the pulmonary capillaries and, due to resistance in the small pulmonary veins, the measured pressure may be higher than the Ppao measured by conventional balloon inflation (12). In one study of patients with chronic obstructive pulmonary disease, the mean difference in pressure measured with full balloon inflation and peripheral wedging was 3 mm Hg (13). In certain of our cases, the best Ppao could have been measured a few centimeters more proximally than the partial Ppao, but both values were obtained with the balloon inflated and the change in catheter position would have been much less than in the study comparing a fully inflated and peripherally wedged catheter (13). Furthermore, in several of our cases, the best Ppao was obtained by either slight advancement of the inflated catheter or use of a 1- to

1.2-mL balloon inflation volume, maneuvers that would have resulted in the best Ppao being measured from a more distal location. Therefore, it is extremely unlikely that the large difference between the partial Ppao and the best Ppao seen in our patients was influenced by movement of the j point with catheter repositioning.

Recognition of partial occlusion in patients with pulmonary hypertension may be difficult, because the reliability of the measured Ppao may not be questioned when it is equal to or less than the Ppad. Perhaps the most useful indicator of partial occlusion in this setting is a marked narrowing of a previously widened Ppad – Ppao gradient (Fig. 2B). Normalization of a markedly elevated Ppad – Ppao gradient would be unlikely in the absence of a therapeutic intervention that would dramatically reduce pulmonary vascular resistance. Of note, an increase in left atrial pressure does not narrow the Ppad – Ppao gradient (14). Therefore, unlike spurious elevations in the measured Ppao due to partial occlusion, true increases in the Ppao resulting from hypervolemia or a reduction in left ventricular compliance should be accompanied by an increase in the Ppad of similar magnitude (2, 14).

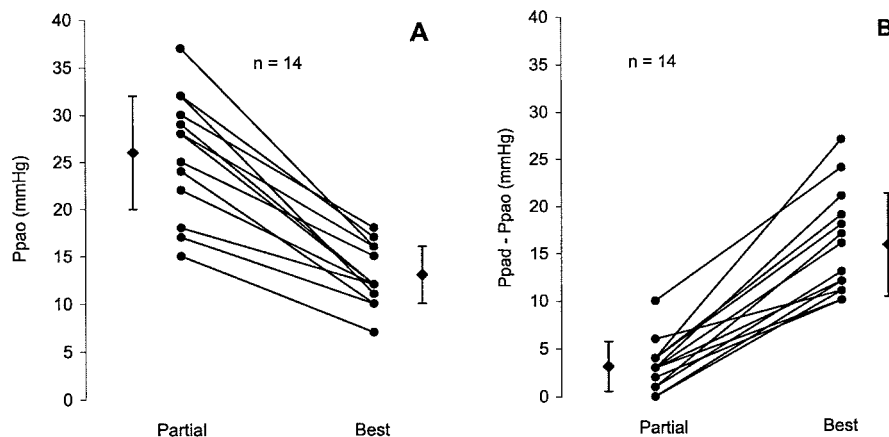


Figure 2. A, measured pulmonary artery occlusion pressure (Ppao) and B, pulmonary artery diastolic pressure (Ppad) – Ppao gradient during partial occlusion and after best Ppao obtained by catheter repositioning. Individual values for each of 14 patients, with mean  $\pm$  SD. See text for definition of partial and best Ppao.

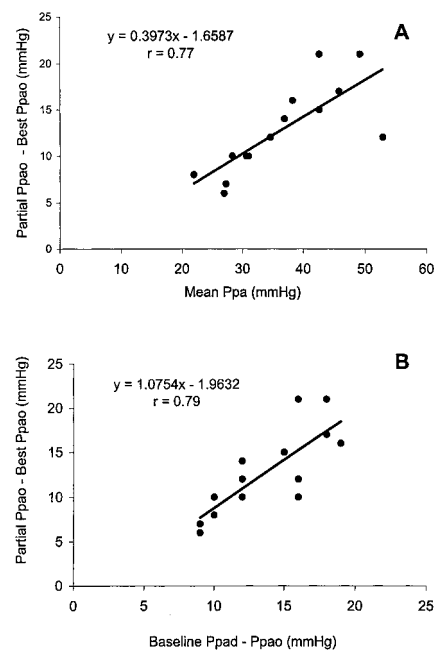


Figure 3. Correlation between error due to partial occlusion (partial pulmonary artery occlusion pressure [Ppao] – best Ppao) and A, the mean pulmonary artery pressure (Ppa) and B, the pulmonary artery diastolic pressure (Ppad) – Ppao gradient. (Ppad – Ppao gradient is during best Ppao measurement.) See text for definition of partial and best Ppao.



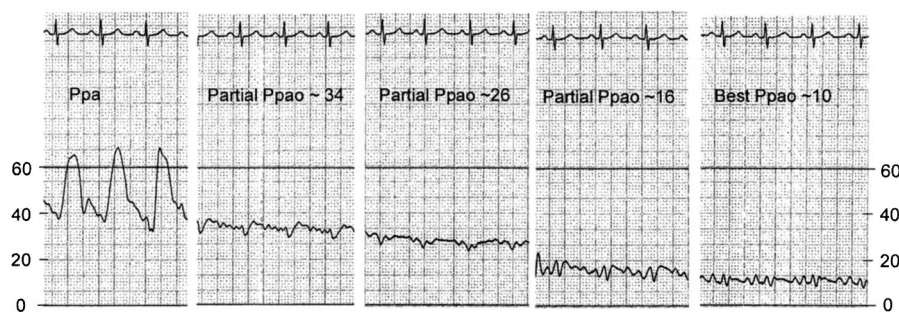


Figure 4. Serial measurements of pulmonary artery occlusion pressure ( $P_{pao}$ ) over several minutes in a patient with severe acute respiratory distress syndrome and pulmonary hypertension. The first three  $P_{pao}$  values reflect varying degrees of partial occlusion. See text for definition of partial and best  $P_{pao}$ . Scale in mm Hg.  $P_{pad}$ , pulmonary artery diastolic pressure.

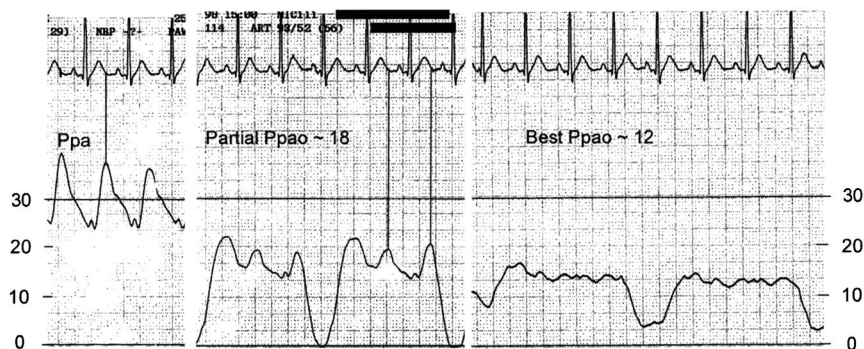


Figure 5. Partial occlusion in a patient with relatively mild pulmonary hypertension due to cirrhosis. Note that timing (relative to the electrocardiogram) of the single positive pressure wave of the partial pulmonary artery occlusion pressure ( $P_{pao}$ ) is identical to that of the pulmonary artery pressure ( $P_{pa}$ ) systolic wave. See text for definition of partial and best  $P_{pao}$ . Scale in mm Hg.

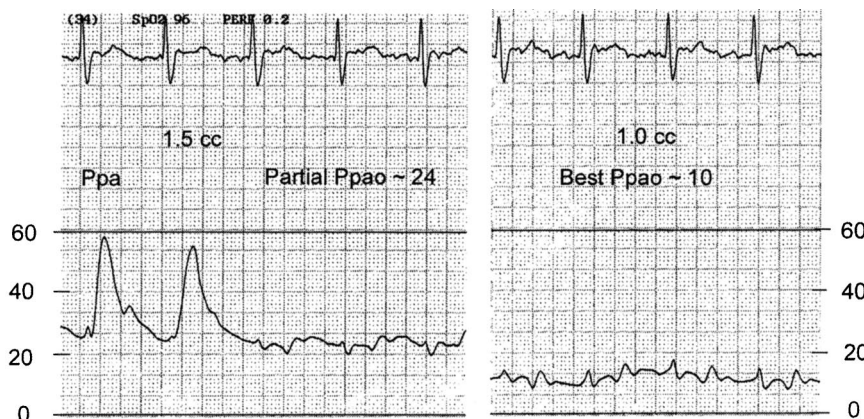


Figure 6. Partial pulmonary artery occlusion pressure ( $P_{pao}$ ) when 1.5 mL of air is used to inflate the catheter balloon; a much lower  $P_{pao}$  was obtained with a 1.0-mL inflation. Review of the chest roentgenogram revealed that the catheter tip was too peripheral, leading to retraction to a more proximal location.  $P_{pa}$ , pulmonary artery pressure. See text for definition of partial and best  $P_{pao}$ . Scale in mm Hg.

Unless the catheter is placed fluoroscopically, partial occlusion that occurs at the time of initial insertion may be harder to detect, because a previously widened  $P_{pad} - P_{pao}$  gradient would not have been documented. Partial occlusion should be considered if the initial  $P_{pad} -$

$P_{pao}$  gradient is normal, but increased pulmonary vascular resistance would have been expected due to the underlying disease (e.g., severe chronic obstructive pulmonary disease, pulmonary fibrosis, or acute respiratory distress syndrome). Waveform analysis may also be helpful in

**P**artial occlusion in patients with pulmonary hypertension may lead to significant overestimation of the pulmonary artery occlusion pressure ( $P_{pao}$ ) and should be suspected when there is a substantial increase in the  $P_{pao}$  without a concomitant increase in the pulmonary artery diastolic pressure ( $P_{pad}$ ), as reflected by a marked narrowing of a previously widened  $P_{pad} - P_{pao}$  gradient.

detecting partial occlusion if the pressure waveform obtained after balloon inflation is more consistent with a  $P_{pa}$  tracing than a left atrial pressure waveform (Fig. 5) (8). Perhaps the single most important aspect of recognition is heightened vigilance when recording the  $P_{pao}$  in patients with pulmonary hypertension.

A potential limitation of our study is that we did not obtain occluded blood gas samples (15). This technique has been used to confirm the validity of the  $P_{pao}$  by documenting high oxygen content in blood withdrawn from the distal lumen when the catheter is appropriately occluded, and failure to obtain highly oxygenated blood might indicate partial occlusion. It should be appreciated, however, that occluded blood gas samples may sometimes be misleading due to occlusion in an area of atelectasis, withdrawal of blood faster than 3 mL/min, or failure to remove and discard at least 15–20 mL of vascular “dead space” blood before obtaining the blood gas sample (16).

After suspecting that the measured  $P_{pao}$  was spuriously elevated due to partial occlusion, we manipulated the catheter to obtain a more acceptable (lower)

Ppao. In certain cases, slight advancement of the fully inflated catheter yielded the best Ppao. Partial occlusion can also occur if the catheter balloon is not fully inflated (17). On the other hand, a catheter that is situated too peripherally may also encourage partial occlusion, in which case the best Ppao may be obtained with a balloon inflation volume of 1–1.2 mL (Fig. 6) (8). In patients with pulmonary hypertension, an acceptable Ppao tracing may be difficult to obtain (18) and may require a greater catheter insertion distance than when pulmonary artery pressure is normal (19). In addition, the catheter tip tends to migrate further toward the lung periphery upon balloon deflation when pulmonary artery pressure is elevated (19). Possible mechanisms of partial occlusion with a peripherally located catheter might include eccentric balloon inflation or location at an arterial branch point (6, 20). We believe that peripherally located catheters should be withdrawn to a more proximal position, even if this means that nurses will be unable to easily obtain a Ppao. The potential risk of pulmonary artery rupture due to repeated inflation of a peripherally placed catheter likely outweighs the benefit of having serial recordings of the Ppao. If the Ppao is desired for patient management, the physician may advance the fully inflated catheter until a reliable Ppao is obtained and then return the deflated catheter back to its more proximal (and safer) location.

The frequency with which partial occlusion occurs in patients with pulmonary hypertension is not known, nor are its clinical consequences. However, errors in measurement of the Ppao of the magnitude found in this study could lead to serious mistakes in management of critically ill patients, with the most likely consequence being that hypovolemic patients would be treated with vasopressors, inotropes, or diuretics instead of intravenous fluids. Although we limited this study to cases in which one of the authors personally documented the episode of partial occlusion, we have seen numerous additional patients with pulmonary hypertension whose recorded hemodynamic data revealed unexpected large increases in the measured Ppao without a concomitant change in the Ppad, leading us to believe that partial occlusion in pulmo-

nary hypertension may not be uncommon. The actual incidence of partial occlusion in pulmonary hypertension can only be determined by a prospective investigation.

## CONCLUSIONS

Partial occlusion in pulmonary hypertension may be difficult to recognize and can lead to significant overestimation of the Ppao. The most useful clue to partial occlusion in this setting is when a substantial increase in the Ppao occurs without a concomitant change in the Ppad, markedly narrowing a previously widened Ppad – Ppao gradient. Partial occlusion should also be suspected at the time of catheter insertion if the Ppad – Ppao gradient is normal and the underlying disease process would predict increased pulmonary vascular resistance. Partial occlusion may occur with a catheter that is either too proximal or too distal in the pulmonary artery, and appropriate repositioning may be corrective. If the problem is not easily corrected or continues to occur intermittently, foregoing routine Ppao determinations may avoid potentially serious mistakes in patient management based on erroneously high Ppao values.

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