

# Let us use the pulmonary artery catheter correctly and only when we need it

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**Objective:** To clarify the issues related to the use of the pulmonary artery catheter within a rational clinical perspective.

**Results:** Barriers include a) increased patient risk of pulmonary artery catheter placement; b) ability to measure similar variables via central venous catheterization, echocardiography, or other less invasive techniques; c) increased cost; d) inaccurate measurements; e) incorrect interpretation and application of pulmonary artery catheter-derived variables; and f) lack of proven benefit of pulmonary artery catheter use in the overall management of patients.

**Interpretation:** a) The risks are mainly due to insertion of a central catheter, not a pulmonary artery catheter; b) continuous monitoring of left ventricular filling pressures, pulmonary vascu-

lar pressures, and mixed venous oxygen saturation is a unique feature; c) additional costs are minimal relative to the cost of intensive care; d) measurement errors require ongoing programmatic educational efforts; e) pulmonary artery catheter-derived data need to be used within the context of a defined treatment protocol; and f) no monitoring device, no matter how simple or sophisticated, will improve patient-centered outcomes unless coupled with a treatment that, itself, improves outcome.

**Conclusion:** A treatment protocol for the use of pulmonary artery catheter-derived variables is proposed that could serve as a basis for a prospective clinical trial. (Crit Care Med 2005; 33:1119-1122)

It is the general impression of opinion leaders that the use of the pulmonary artery catheter (PAC) in acute care management has reduced markedly over the past 10 yrs, and indeed sales of the PAC in Europe, the United States, and Japan have fallen by almost 9% since 2002 (data on file, Edwards LifeSciences, Irvine, CA). In large part, this reduction in PAC use has been driven by the few highly publicized studies showing either no benefit of PAC-associated therapies (1-3) or a reduction in defined outcome measures when compared with subjects treated without a PAC (4). However, the current critical care medicine culture has swung this preference so far that presently catheter companies are promoting the use of central venous catheters (sometimes in combina-

tion with arterial catheters), which can derive many of the variables normally acquired only with the PAC, as viable substitutes for PAC insertion. Generally, if a more invasive form of catheterization can be avoided without loss of useful hemodynamic information needed to guide therapy, then we applaud these decisions. The excessive and indiscriminate use of any monitoring technology, whether it be the routine use of the PAC or measures of blood lactate, hemoglobin, ionized calcium, or troponin, will prove less effective, at best, and detrimental, in the extreme, if acted on inappropriately. However, the indiscriminate decline in PAC use, in our opinion, has a very profound and detrimental aspect that must itself negatively affect patient care, outcomes, and overall costs.

The reasons usually cited for the decision not to use the PAC include the following: a) increased risk to the patient of PAC placement; b) the ability to measure similar variables via central venous catheterization, echocardiography, or other less invasive techniques; c) increased cost; d) misuse of the PAC-derived variables by their inaccurate measurement; e) incorrect interpretation and application of the PAC-derived data to clinical care; and f) lack of proven benefit of PAC use in the overall management of patients. Although certain patient sub-

groups readily lend themselves to PAC monitoring, such as high-risk surgery patients and those with combined cardiopulmonary disease or preexisting heart failure, perhaps the most effective use of the PAC will be in the identification of occult tissue hypoperfusion. Thus, subjects with an increased base deficit, lactic acidosis, tachycardia, continuing poor mentation, and low urine output following initial resuscitation should be considered candidates for PAC placement.

Regrettably, all these arguments have significant defects that obscure the primary concept, which is that no monitoring device, no matter how simple or sophisticated, will improve patient-centered outcomes unless coupled with a treatment that, itself, improves outcome. For example, it would be difficult to document in prospective randomized clinical trials that the measurement of arterial blood gases improves outcome. Hypercapnia may not be deleterious in the absence of intracranial hypertension, and metabolic acidosis may not require bicarbonate administration, yet would any critical care physician not measure these routinely despite the increase in cost that they add to the overall health care bill? Let us consider the preceding arguments in turn.

**Increased Risk to the Patient of PAC Insertion.** The primary risks of PAC placement come during catheter inser-

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Table 1. Risks associated with pulmonary artery catheterization

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Complicated vascular access (pneumothorax, hematoma, arterial puncture)
Arrhythmias (heart block, ventricular tachycardia/fibrillation)
Catheter knotting and tricuspid/pulmonary valvular damage
Pulmonary thrombosis and infarction
Endothelial/valvular damage
Pulmonary artery rupture
Colonization and bacteremia

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tion and subsequent monitoring (Table 1). These risks include the risks of central vascular access and indwelling catheter placement. PAC insertion runs the risks of hemorrhage, pneumothorax, damage to large thoracic vessels, and transient arrhythmias. The occurrence of a knot in the catheter has become extremely rare with minimal precautions during catheter insertion. Only transient arrhythmias are unique to PAC insertion and have been shown to be both common and usually benign (5). The other risks of PAC insertion are identical to those for central venous catheter insertion, and they are much more common and cause much more morbidity than transient ventricular arrhythmias. The only other specific PAC complication is pulmonary artery rupture with balloon inflation, a catastrophic event. However, the frequency of pulmonary arterial rupture is vanishingly small (5). Finally, there is no evidence that catheter-associated infections are greater with the PAC than with central venous catheterization. Thus, we would argue that the risks of PAC insertion are similar to those for central venous catheterization, making the decision to insert a PAC in the setting of central venous catheterization primarily one concerning the need for PAC-specific data for the management of the patient.

*Ability to Measure Similar Variables Via Central Venous Catheterization.* The hemodynamic variables easily measured by a PAC include mixed venous oxygen saturation ( $SvO_2$ ), cardiac output, right ventricular ejection fraction (with some catheters), and intrapulmonary vascular pressures. Recent advances in catheter technology have produced impressive novel and innovative uses of monitoring devices requiring only central venous catheterization or central venous catheterization combined with arterial catheterization. Central venous fiberoptic technology also allows for the continuous measurement of superior vena caval oxygen saturation as a surrogate marker of  $SvO_2$ . Indeed, Rivers et al. (6) documented improved survival when resuscitation was

controlled to target superior vena caval oxygen saturation values, suggesting that occult tissue hypoperfusion often occurs in resuscitated patients treated only to more general hemodynamic end points, such as arterial pressure, heart rate, and sensorium. However, although superior vena caval oxygen saturation is physiologically lower than  $SvO_2$ , it is approximately 10% higher than  $SvO_2$  in stable acutely ill patients, and this difference may vary even more under septic conditions (7, 8). No study has really attempted to titrate vasoactive therapies to  $SvO_2$ -related measures in critically ill patients. However, it is difficult for us to imagine that the outcomes from such studies would be worse than those described by Rivers et al. Clearly,  $SvO_2$  is the gold standard for defining global adequacy of cardiovascular performance (9). Although echocardiographic techniques have evolved such that one can estimate mean pulmonary arterial pressure and left ventricular preload at the bedside (10), and PiCCO-derived measures can estimate intrathoracic and total cardiac blood volumes (11), the clinical utility of these measures in assessing both the determinants of cardiovascular insufficiency and their response to treatment has not been demonstrated. Moreover, pressure measurements may be more important than volume measurements to guide therapy if the major concern is the development of edema and pressure is the primary determinant of it, since hydrostatic and increased permeability pulmonary edema have different treatments. Furthermore, echocardiographic techniques are operator-dependent and their use longitudinally in patients is impractical. Finally, no indirect measure can accurately determine pulmonary artery occlusion pressure (PAOP). Although PAOP measures are fraught with inaccuracies in both their measurement and hemodynamic significance (12), only PAOP measures can separate out permeability pulmonary edema from hydrostatic edema (13). The one place where indirect measurement has equaled PAC-derived measures is in the estimation

of cardiac output. However, since there is no such thing as a normal cardiac output (14, 15), accurate measures of cardiac output are less important than measures of cardiac output changes in response to treatment and time. Accordingly, if one wants to accurately define the adequacy of cardiac output, measures of  $SvO_2$  are essential, whereas measures of pulmonary arterial pressure and PAOP are essential for the assessment of pulmonary hemodynamics.

*Increased Cost.* The one-time cost of PAC insertion includes the cost of the catheter and the time it takes to insert it. Considering that the average daily cost of a day in the intensive care unit in the United States is \$12,000 (16), the additional \$100 cost of the PAC and the additional 15 mins time it takes to insert relative to the overall time needed for central venous catheter insertion appear to these observers as minimal to nonexistent. Indeed, since all forms of central venous access usually require nursing care and a postprocedure chest radiograph, one cannot even evoke these additional costs in this comparison.

*Inaccurate Measurement of PAC-Derived Variables.* The problems with the accurate measurement of intrapulmonary pressures and flow in patients during ventilation are well known (11). However, in large part the inaccuracies come in the assessment of PAOP, not  $SvO_2$  or cardiac output. Significant strides in physician awareness and education on the methods to accurately measure PAOP are ongoing, including Web-based applications (e.g., <http://www.thoracic.org/criticalcare/ccpac.asp>), as previously suggested (17). Although these efforts are important and we applaud them, to a large extent the majority of the information gleaned from the PAC that these initiatives aid is the measurement of intrapulmonary vascular pressures, whereas the more fundamental hemodynamic measures needed to assess the adequacy of global blood flow are  $SvO_2$  and cardiac output changes, both of which are usually measured accurately.

*Incorrect Interpretation and Application of PAC-Derived Variables to Clinical Care.* Any monitoring technique can only be as good as the interpretation of the data derived, and this requires adequate and ongoing training. The fact that PAC-derived variables are incorrectly interpreted and applied does not mean that PAC-derived data are of no use but rather that in the wrong hands they are of less value: No one would argue that electro-

Resuscitate to a mean arterial pressure of > 65 mmHg

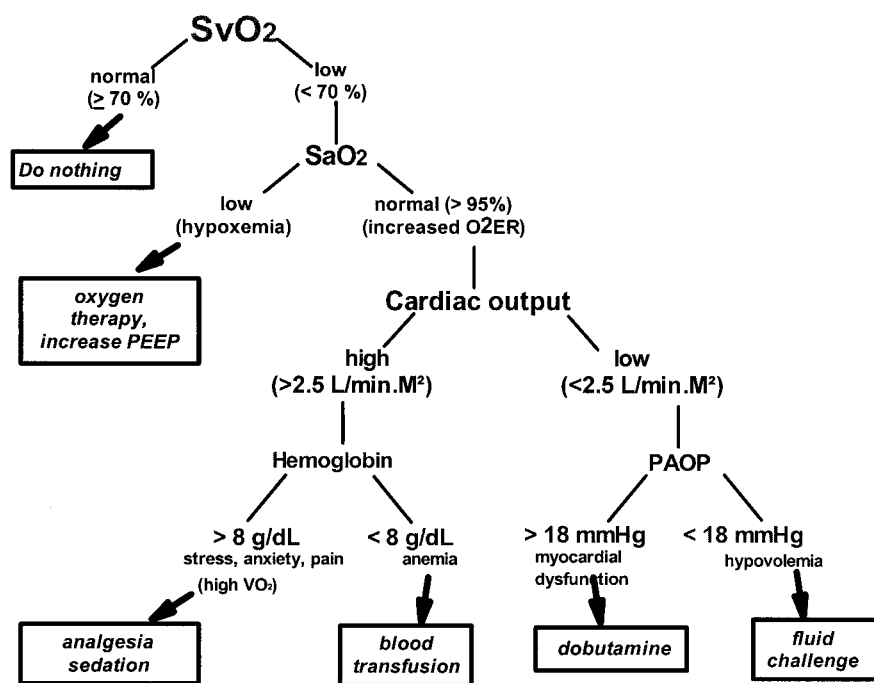


Figure 1. Diagnostic and therapeutic algorithm based on mixed venous oxygen saturation ( $SvO_2$ ) measurements: therapeutic options to be considered are presented in the rectangles.  $SaO_2$ , arterial oxygen saturation;  $O_2ER$ , oxygen extraction ratio;  $PEEP$ , positive end-expiratory pressure;  $PAOP$ , pulmonary artery occlusion pressure;  $VO_2$ , oxygen consumption.

cardiograms should not be performed because sometimes they are misread. In addition, with sufficient training, PAC monitoring could be considered to be less operator-dependent than the less invasive techniques such as echocardiography that are being touted as replacements for PAC-derived data. It is unlikely that most units will have 24-hr, 7-day/week access to a doctor experienced in echocardiography, but all units could have doctors available who are adequately trained in PAC insertion and data collection, interpretation, and application.

Regrettably, if any one fault can be levied on the critical care medicine establishment, it is the lack of a consistent and reasoned paradigm based on clinical outcomes studies to define specific treatment based on measured PAC variables. Although several scholarly reviews of this topic have been written, including a few by ourselves (18, 19), their implementation in clinical practice has been irregular at best. Clearly, we need to document clear and nonambiguous treatment protocols based on specific combinations of hemodynamic variables with documented benefits and see these recommendations followed before we can say that we have

accomplished our academic goal of defining optimal management of the patient in circulatory shock.

*Lack of Proven Benefit of PAC Use in the Overall Management of the Patients.* We agree that present data on PAC use have not documented its beneficial role in the management of the hemodynamically unstable patient. However, we argue that this lack of documented benefit likely reflects the lack of controlled clinical trials of treatment algorithms that include PAC-specific data in the algorithms. Of note, none of the studies examining outcome used the PAC-derived data in a defined treatment plan; they merely assessed whether the presence of a PAC altered outcome. However, lack of proof of benefit does not equate to proof of lack of benefit. Although a prospective clinical trial of PAC use as compared with central venous catheter-derived measures is presently ongoing, we believe that neither arm of that National Institutes of Health-funded study actually addresses the fundamental question about the utility of the PAC because it compares high vs. low PAOP and central venous pressure fluid management strategies, and neither PAOP nor central venous pressure pre-

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dicts preload responsiveness well. The question is not whether insertion of a PAC improves outcome but rather if PAC-derived data, as an integral part of the overall monitoring and management of the critically ill patient, improve outcome. No monitoring device, no matter how accurate or safe, will improve outcome unless it is coupled with a specific treatment plan known to improve outcome. In support of this argument, several investigators used PAC-guided aggressive preoperative resuscitation in high-risk surgical patients and compared outcomes to similar patients with or without a PAC in whom this "preoptimization" was not performed (20–22). These studies documented that PAC-guided preoptimization reduced mortality and morbidity and was cost-effective. Still, this approach is limited to high-risk surgical patients. Assuming the Rivers et al. (6) data are also applicable to patients who suddenly present with circulatory shock, then similar radical PAC-guided therapy should be possible in this very large patient group. It is surprising that comparison of PAC- to non-PAC-guided goal-directed therapy has not been done before. Accordingly, we propose the following simple diagnostic and treatment algorithm that uses PAC-derived data to answer sequential questions (Fig. 1). The trigger to use this protocol would be ongoing circulatory shock despite initial fluid resuscitation efforts or, in the setting of normotension, persistent tachycardia, metabolic acidosis, lactic acidosis, altered mental status, and decreased urine output, since all these signs are indirect markers of inadequate tissue perfusion. Clearly, some patient subsets may be more dependent on one form of treatment than other patient groups. However, the simple protocol described herein should differentiate between ma-

for pathophysiologic groups of patients. It would be interesting to see if by using such a treatment protocol patient outcome improved relative to using the same logic but without PAC-derived data. We would hope that this debate will spur physicians to develop multiple-center clinical trials using PAC-specific variable-defined treatment algorithms, along the lines of the treatment protocol we describe in Figure 1. Only after such a study is performed will we truly know if PAC use improves outcome in the critically ill.

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