Commentary on the ASA’s Revised Practice Guidelines for Pulmonary Artery Catheterization

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Over the last 10 years, few medical devices have suffered a greater diminution in status than the pulmonary artery catheter (PAC). Once a mainstay in the intensive care unit (ICU) and cardiac operating rooms, the PAC now suffers from a growing consensus of investigators who consider it to be both obsolete and possibly dangerous. Partly in response to this, the ASA re-convened a Task Force to update its 10-year-old Practice Guidelines for Pulmonary Artery Catheterization. Dutifully, the Task Force reviewed the published literature, relied on expert opinion, and even polled the membership in an effort to justify its recommendations. The sobering conclusion of this effort is that after 10 years of data gathering, we still don’t know whether a PAC is a benefit or a hazard, when and on whom to use it, or even whether it’s worth the considerable cost.

Widely popular since its introduction in the 1970s, the PAC quickly found acceptance as a more accurate measure of hemodynamic status in critically ill patients than could be derived from clinical assessment alone. In contrast to a central venous pressure catheter (CVPC), the PAC could continuously and reliably measure cardiac output and pulmonary artery occlusion pressure (PAOP) which provided a direct estimate of left ventricular pre-load and function. The saturation of mixed venous blood (SvO₂), which could only be obtained from a PAC, allowed calculation of systemic oxygen consumption (VO₂). This information allowed physicians to draw conclusions about the adequacy of perfusion to the vital organs well before they were apparent in the clinical exam. The PAC helped sort out confusing hemodynamic derangements and facilitated earlier and more precisely targeted interventions. In addition, the effect of interventions to improve organ perfusion could be observed over time.
Mostly un-blinded early studies suggested that PAC derived data changed therapy in 30-62% of cases. Anesthesiologists routinely inserted PACs pre-operatively and used the data to guide intra-operative fluid and pressor management on the assumption that optimization of hemodynamic variables would optimize patient outcomes. In both medical and surgical ICUs, PAC data was used to guide management of devastating illnesses such as acute coronary syndromes, sepsis, and Adult Respiratory Distress Syndrome. In patients with chronic congestive heart failure, cardiologists tailored afterload reduction and diuretic therapy to data derived from PACs. The prospect of knowing in real time the effect of interventions on cardiac output, oxygen delivery and extraction, and stroke volume quickly made PACs highly appealing to clinicians, so much so that by 1996, two billion dollars were spent annually on PACs. The assumption underlying this enthusiasm was that appropriate use of PAC data would translate into better care for the sickest patients. That assumption turned out mostly to be wrong.

Undeniably, much of what is known about cardiac physiology and oxygen transport during critical illness was learned using a PAC, but surprisingly, little data has been published that has been able to demonstrate a direct benefit of the device to the patient in whom it was placed. In 1988, outcomes were compared in critically ill general surgical patients monitored with a CVPC, or a PAC used to normalize hemodynamic variables, or a PAC used to achieve supra-normal values of cardiac index (CI), oxygen delivery (DO₂) and VO₂.¹ The patients whose PAC was used only to normalize the patient’s hemodynamics did no better than the CVPC group, but when supra-normal values were achieved, mortality dropped from 33% to 4%. This finding generated an interest in the potential benefits of PAC guided “goal-directed therapy” for surgical patients at high risk. “Confirmatory” studies followed doxapamine infused into surgical patients both pre- and post-operatively to achieve DO₂ of 600 mL/min/m² decreased 28-day mortality from 22% to 5.7% over controls.² However, other investigations were unable to show a statistically significant difference in mortality, rate of organ failure, and ICU or hospital stay for patients who underwent interventions to enhance DO₂.³ Nonetheless, they did report that survival among a subgroup of patients with supra-normal DO₂—either induced or spontaneous—was better than those with normal values. Enhanced DO₂, it was suggested, might be a therapy for an otherwise undetectable tissue hypoxia, and measurement and treatment of low DO₂ might be life saving in a subset of patients who, though unable to enhance DO₂ on their own, would respond to therapy. This data failed to clarify whether the hypo-
dynamic characteristics of these survivors of critical illness were just a surrogate for a better reserve or really represented a therapeutic target. By increasing the \( \text{DO}_2 \), \( \text{VO}_2 \) and CI, were physicians only revealing patients that would have survived anyway, or were they rescuing non-survivors?

In an effort to answer this question, a study was designed for 100 critically ill patients who had failed to meet pre-established goals for CI, \( \text{DO}_2 \) and \( \text{VO}_2 \) with volume expansion alone.\(^4\) Half of the patients were randomized to receive titrated doses of dobutamine, and then their mortality was compared with controls. Treated patients who were able to significantly increase their CI and \( \text{DO}_2 \) seemed no better able to extract oxygen at the tissue level than did their controls. More worrisome, however, was that treated patients had a significantly higher mortality than controls. This suggested that achieving certain physiologic targets was a proxy for physiologic reserve, and not therapeutic success, and that the therapy itself might prove detrimental. With the failure to establish a clear benefit of “goal directed therapy,” the value of a PAC in the ICU was less clear cut.

A much larger series (762 patients) treated patients to achieve either normal CI, supra-normal CI or normal \( \text{SvO}_2 \), and found no difference in mortality, number of dysfunctional organs or length of ICU stay.\(^5\) Patients whose CI and \( \text{SvO}_2 \) could be driven to supra-normal levels did no better than those with normal indices. Unlike the smaller study though, responders did no worse. A meta-analysis of seven randomized clinical trials followed and found no benefit to maximizing \( \text{DO}_2 \) in the critically ill.\(^6\) Goal directed therapy, at least for the moment, was sunk.

The demise of “goal directed therapy” did not, by itself, cast doubt on the value of the PAC. The editorial accompanying the larger series\(^7\) carefully pointed out that volume and inotropic management of critically ill patients can only be performed rationally when guided by a PAC. Still, the benefit of the PAC either in the ICU, operating room or elsewhere remained unproven. Some studies in medical patients found that patients monitored with a PAC had higher in-hospital mortality, longer hospital stay and worse survival than unmonitored controls. However, selection bias undermined the validity of many of these studies. Were patients in the PAC arm sicker to begin with, and did that fact-not the PAC-explain their worse outcome?
An important cohort study tried to control for selection bias by calculating a “propensity score” for each patient. The “propensity score” is a number derived from a logistical regression of all variables known to influence the decision to place the PAC. The argument was that if patients were stratified by the “propensity score,” then the effect of the PAC could be isolated from the reasons it was placed, and in this way the impact of the PAC on outcome could be independently assessed. Cases were matched to controls with similar “propensity scores.” The results were that survival of PAC patients was worse in the hospital at 30, 60 and 180 days. Moreover, PAC patients had longer stays in the ICU and generated substantially higher hospital costs. Few surgical patients were studied, but the impact of these results on the management of critically ill medical patients was substantial. Based on this data, an editorial recommended that the FDA should either support research to prove the catheter’s benefit or ban it.

Although the study using “propensity score” data included few surgical patients, it didn’t directly affect anesthesiologists. However, by 1985 studies had been published suggesting that the low risk cardiac surgery patient could be safely managed without a PAC. Even in higher risk coronary surgery patients, investigators failed to demonstrate that a PAC offered any benefit over a CVPC. More recently, a randomized study of nearly 2,000 high-risk surgical patients found no survival benefit in the hospital, or at 6 and 12 months. In fact, these results suggested that the PAC increased a patient’s risk of pulmonary embolism.

If the PAC’s dominance is dwindling in the ICU, its prospects in the operating room are threatened by the growing application of transesophageal echocardiography (TEE). According to a recent survey, TEE is available to 56% of practicing cardiovascular anesthesiologists and is widely used either in combination with—or in lieu of—a PAC. Measurements that once required a PAC can be obtained by TEE. Anesthesiologists also have used Doppler transducers to estimate stroke volume and cardiac output by measuring the outflow velocity along the LV outflow tract or even in the descending aorta. In addition, various investigators used TEE measurement of the inflow velocities in the pulmonary vein or across the mitral valve to estimate left atrial pressure (LAP). Comparison of these TEE results with simultaneous measurements of PAOP from a PAC found that the two different techniques estimated LAP with nearly equal accuracy. Pulmonary artery pressure has also been estimated from measurements of pulmonary artery flow velocities.
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Therefore, TEE is not only able to visualize the structure and performance of the ventricle and valves, but may reasonably estimate hemodynamic parameters.

Despite thin evidence supporting its use, the ASA Task Force did not support a PAC moratorium. In fact, among cardiac anesthesiologists, routine PAC utilization remains actively debated. Acknowledging inadequate data (especially in surgical patients), and insisting that physicians who use PACs be skilled in placing and interpreting them, the Task Force suggests that the decision to deploy a PAC depends on a hazy combination of risks derived from the patient, the surgery and the practice setting. Essentially, we are told, this decision should be made on a case-by-case basis.

Although these *Practice Guidelines for PA Catheterization* probably best reflect the quality of the accumulated data, these recommendations are so vague that they might even call into question the value of publishing recommendations at all. Indeed, this is what I have found to be the main difficulty with these *Practice Guidelines*. They are derived from a rigorously collected register of mostly non-supportive research (of widely varying quality). If these Guidelines fortify a reader’s skepticism about the utility of the PAC, they fail to provide any tangible avenues through which to channel it.

**References**


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In a democracy, your vote counts. In feudalism, your count votes.

Artificial intelligence is no match for natural stupidity.