

Pulmonary Artery Catheters State of the Controversy

Susan K. Frazier, PhD, RN; Glenda J. Skinner, MS, RN, CPHQ

Since 1970, pulmonary artery catheters (PACs) have been used in clinical practice to monitor the hemodynamic status of critically ill and injured patients. This technology was introduced and commercialized without considerable testing to determine safety and efficacy. After years of common clinical use, investigators identified potential increases in mortality associated with PAC use. For the past decade, investigators have studied various patient populations to elucidate the safety and efficacy of the PAC. This article reviews the historical context of PAC use, findings from recent clinical trials intended to determine safety and efficacy, issues with reliability and validity of PAC use, and complications associated with PAC use. Data from recent clinical trials do not support routine use of PACs, and the authors suggest that PAC-guided therapy should be the focus of study in future trials.

KEY WORDS: hemodynamic monitoring, pulmonary artery catheter, Swan-Ganz catheter

Experts estimate that 1.5 million pulmonary artery catheters (PACs) are used each year in the United States to monitor the hemodynamic status of critically ill and injured patients.¹ Thirty percent of these catheters are placed in cardiac surgical patients, 30% in patients in cardiac care units and catheterization laboratories, 25% in trauma and high risk surgery patients other than cardiac surgery, and 15% in medical intensive care patients. An ongoing controversy about the safety of and benefit associated with the use of PACs, despite their common use, exists. A majority of the recent clinical trials considered placement of the PAC as the independent variable. However, the PAC is intended to provide information to guide therapy. Thus, placement of the PAC alone cannot be expected to improve patient outcomes. This article will review the historical context of this controversy, recent clinical trials intended to determine safety and efficacy, issues with reliability and validity of PAC use, and complications associated with PAC use.

Historical Perspective

In 1929, the first right heart catheterization was performed in Germany by Werner Forssmann, a physician with an interest in the circulatory system.² This groundbreaking event was even more unusual because after animal study, Dr Forssmann became the first human to experience a right heart catheterization when he performed this technique on himself. Dr Forssmann did not receive support to continue his research, but others built on his foundational work. In the 1940s, in New York, Drs Cournand and Richard continued to develop the technique and used the PAC to elucidate important mechanisms inherent to cardiac physiology. In 1956, Drs Forssmann, Cournand, and Richard jointly accepted the Nobel Prize in Physiology or Medicine “for their discoveries concerning heart catheterization and pathologic changes in the circulatory system.”² After this international recognition, right heart catheterization became more widely used over the next decade and a half but remained restricted to the catheterization laboratory, primarily for diagnosis of intracardiac and valvular defects.

In 1970, Swan and colleagues³ published the first reported use of a flow-directed PAC. The innovative balloon at the tip of the catheter supported placement of the catheter in the pulmonary artery without the use of fluoroscopy. Bedside placement of the PAC was instantly embraced by clinicians and touted as a “reliable early objective measure of left ventricular failure...an excellent guide to therapy.”⁴ The later addition of a thermistor to the distal catheter and the development of the thermodilution cardiac output technique in 1971 by the same group⁵ led to

Susan K. Frazier, PhD, RN

Associate Professor, University of Kentucky College of Nursing, Lexington, Kentucky.

Glenda J. Skinner, MS, RN, CPHQ

Director of Performance Improvement and Patient Safety, Holzer Medical Center, Gallipolis, Ohio.

The authors of this article have no significant ties, financial or otherwise, to any company that might have an interest in the publication of this educational activity.

Corresponding author

Susan K. Frazier, PhD, RN, University of Kentucky College of Nursing, 523 CON Building, 760 Rose Street, Lexington, KY 40536-0232 (skfraz2@email.uky.edu).

considerable commercial development and an explosion of advanced monitoring of hemodynamics at the bedside. Pulmonary artery pressure and cardiac output measurement became routine for cardiac surgery patients in particular and ubiquitous in critical care units throughout the United States.

In 1976, after the report of more than 10,000 injuries related to medical devices like the Dalkon Shield intrauterine device, the Medical Device Amendment to the Food, Drug, and Cosmetic Act of 1938 was enacted.⁶ This amendment charged the Food and Drug Administration with ensuring the safety and efficacy of medical devices. A grandfather clause excluded PACs from mandatory clinical testing because they were assumed to provide an obvious benefit to patients, were already in wide use, and were determined to be Category II devices, which were considered nonlife supporting. Thus, PAC safety and their actual effect on outcome were not scientifically investigated.

Even though complications related to PAC use were reported soon after the introduction of PACs,⁷⁻⁹ PAC safety was not clearly questioned until near the end of the 1980s and into the 1990s when the mortality rate was reported to be greater in those patients who received a PAC after acute myocardial infarction.^{10,11} However, a majority of scientists and clinicians discounted these concerns. Studies that suggested PAC use was associated with greater morbidity and mortality were criticized for poor study design, and study samples were considered biased because patients who received PACs purportedly had greater severity of illness, so would naturally have greater morbidity and mortality.¹²

In 1996, a large-scale, multisite, case-matched study of critically ill medical and surgical patients ($n = 5,735$) by Connors and colleagues¹³ indicated that PAC use was associated with greater morbidity and mortality. Serious questions about the safety and efficacy of PAC were soon expressed by both clinicians and the general public. In response, the National Institutes of Health, National Heart, Lung and Blood Institute, in conjunction with the Federal Drug Administration, convened the Pulmonary Artery Catheterization and Clinical Outcomes Workshop in 1997.¹ This group was charged with developing recommendations to improve the utility and safety of the PAC. Four recommendations arose from a consensus process. These included the following: (1) standardization of education for physicians and nurses with regular measurement and monitoring of knowledge to improve the quality and use of data obtained and ensure safety; (2) conduct of randomized clinical trials in specific patient populations (refractory heart failure, low-risk coronary artery bypass surgery, hypoxic pulmonary disease, and sepsis) to determine the safety and efficacy of PAC use; (3) systematic evaluation of any new

technology developed for use with critically ill populations to ensure safety and efficacy; and (4) the use of international collaborative efforts in this research.

Although the original structure of the PAC has remained unchanged since its inception, these catheters evolved with the addition of lumens for measurement of venous pressure, right ventricular volumes, and ejection fraction and cardiac pacing. The addition of fiber optics and the use of spectrophotometry for mixed venous oxygen saturation measurement and the integration of a thermistor coil for continuous cardiac output and right ventricular volume measurements also increased the complexity of the original PAC. Currently, there are a wide variety of catheters used internationally.

Recent Research Findings

Studies That Compare the Use of PAC Versus No PAC

Several recently published studies compared clinical outcomes of patients managed using a PAC to those managed without a PAC (Table 1). Many of these studies focused on the patient populations identified by the consensus workshop group.¹⁴⁻¹⁹ Mortality and morbidity,^{14-17,19-22} intensive care and hospital days,^{15,16,19,22} intervention use (mechanical ventilation, renal support),^{16,19} and medication use^{14-16,19} were the most commonly evaluated outcomes of these studies.

In one of the first studies after the Pulmonary Artery Catheterization and Clinical Outcomes Workshop, Rapoport and colleagues²³ analyzed retrospective data from 34 critical care units located at 27 sites to evaluate PAC use and found important variations in practice related to PAC use across sites. Admission to a surgical unit doubled the likelihood of PAC use (odds ratio [OR], 2.17), whereas care provided by an intensivist reduced the likelihood of catheter use by two-thirds (OR, 0.36). White race (OR, 1.38) and private insurance coverage (OR, 1.33) were also positively associated with PAC use. These data demonstrated that organizational characteristics, as well as patient characteristics, influenced the use of PACs.

Several observational studies that provided little support for routine PAC use were published early in this decade. In a comparison of patients managed with a PAC to those managed without, Vieillard-Baron and colleagues¹⁴ found that PAC use was not associated with either beneficial or adverse effects in acute respiratory distress syndrome (ARDS) patients. Polanczyk and colleagues²¹ evaluated the use of PAC in noncardiac, elective surgery patients and found that PAC use did not improve outcomes in these patients but was actually associated with a tripling of the likelihood of postoperative heart failure (OR,

TABLE 1 Findings From Recent Randomized Clinical Trials of PAC Safety and Efficacy

Authors	Study n	Patient Population	Clinical Setting	Outcomes (Control vs PAC)
Sandham et al ²⁰	n = 1,994 997 control 997 PAC	Surgical patients: elective or emergent major surgery Age ≥ 60 y, high risk	19 Canadian centers	Mortality: 7.7% vs 7.8%, <i>P</i> = .93 LOS: 10 vs 10 d, <i>P</i> = .411
Richard et al ¹⁶	n = 676 341 control 335 PAC	Patients with shock, ARDS or the combination of both	36 French centers	14-d mortality: 51.3% vs 49.9%, <i>P</i> = .70 90-d mortality: 72% vs 70.7%, <i>P</i> = .71 LOS hospital: 14.4 vs 14 d, <i>P</i> = .67 LOS ICU: 11.9 vs 11.6 d, <i>P</i> = .72
ESCAPE investigators ¹⁷	n = 433 206 control 207 PAC	Patients with severe, symptomatic heart failure	26 US centers	Mortality: 19% vs 21%, <i>P</i> = .35 Hospital LOS: 8.3 vs 8.7 days, <i>P</i> = .67
Harvey et al ²²	n = 1,014 522 control 519 PAC	Adult ICU patients	65 centers in the United Kingdom	Mortality: 65.7% vs 68.4%, <i>P</i> = .39 ICU LOS: 15.7 vs 16.8 d, <i>P</i> = .43
ARDS Clinical Trials Network ¹⁹	n = 1,000 487 CVC 513 PAC	Patients with acute lung injury for <48 h	20 North American centers	Mortality: 26.3% vs 27.4%, <i>P</i> = .69 Ventilator-free days: 13.5 vs 13.2 d, <i>P</i> = .58

Abbreviations: ARDS, acute respiratory distress syndrome; CABG, coronary artery bypass graft; CVC, central venous catheter; ESCAPE, Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheter Effectiveness; ICU, intensive care unit; LOS, length of stay; PAC, pulmonary artery catheter; PE, pulmonary embolism.

2.9) and a doubling of the likelihood for noncardiac morbid events (OR, 2.2). Rhodes and colleagues²⁴ demonstrated no difference in mortality between a heterogeneous group of critically ill patients managed with a PAC from those managed without; however, those managed with a PAC demonstrated a significantly greater prevalence of thrombocytopenia and renal failure and received significantly greater fluid support in the 24 hours after PAC insertion (in all of these findings, *P* < .03).

In the first published clinical trial related to PAC use in this decade, Sandham and colleagues²⁰ randomized high-risk adult surgical patients (*n* = 1,994) to either goal-directed therapy with a PAC or standard perioperative care and found no difference in mortality or hospital days. They concluded that there was no benefit to PAC-guided therapy in this population, but did identify a significantly higher prevalence of pulmonary embolus (*P* < .004) in the group that received goal-directed therapy with PAC.

In another randomized clinical trial, Richard and colleagues¹⁶ studied adult patients (*n* = 676) who met standard criteria for shock, ARDS, or both. Patients at 36 centers were randomized to care managed with a PAC or standard care. Although patient management was decided by each physician, all sites agreed to optimize circulating blood volume, to use vasoactive support to maintain a mean arterial pressure of 60 mm Hg or more when fluid volume was adequate, and to administer low-molecular-weight heparin to prevent thromboembolism unless contraindicated. Data analysis demonstrated no differences between the groups in mortality at days 14, 28, or 90. At day 14, there were also no significant differences in vasoactive support, the need

for renal support, or the number of organ system failures, and at day 28, there were no differences in the number of intensive care or hospital days and no difference in mechanical ventilation use between these groups. These investigators concluded that PAC use was safe in the ARDS and shock populations and that the lack of a consistent treatment protocol may have been responsible for the lack of beneficial results in the PAC group.

An observational case-control study of patients with sepsis (*n* = 1,010) demonstrated a slight but not statistically significant lower mortality in patients managed with a PAC (41% vs 47%, not significant), but this difference was eliminated by adjusting for comorbidities and severity of illness.¹⁵ Resource utilization was equivalent between the groups with a slight trend toward lower costs in PAC patients. However, the investigators concluded that randomized trials were necessary to evaluate efficacy.

In 2005, 3 important publications demonstrated a lack of data-based support for PAC use. Shah and colleagues²⁵ reported a meta-analysis of data from 13 clinical trials published between 1985 and 2005, which established that use of a PAC to manage patients did not improve patient outcomes, did not influence mortality, and did not reduce hospital days. Two large-scale clinical trials were also published in that year.

Harvey and colleagues²² reported the results of a large multisite randomized clinical trial in the United Kingdom. Both medical (respiratory failure, multi-organ dysfunction, decompensated heart failure, or other) and surgical (elective or emergent) intensive care patients (*n* = 1,041) were included, and patient management was not controlled but was at the discretion of the individual physicians. No harm or

benefit with the use of PAC-guided care was demonstrated. Hospital mortality, 90-day survival, length of intensive care unit stay, and days requiring organ support were similar between those patients managed with and without a PAC. These investigators concluded that the true benefit of PAC use would not be evident without clinical trials testing management protocols guided by PAC data.

The second publication reported a study that arose from the PAC Clinical Outcomes Workshop. The Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheter Effectiveness (ESCAPE) clinical trial¹⁷ found that in patients with severe, symptomatic, recurrent heart failure (n = 433), PAC-managed care provided no survival benefit. Pulmonary artery catheter-guided care was associated with greater adverse events (22% vs 12%); however, there was a trend for greater improvement in exercise capacity and quality of life compared with patients managed without PAC. The ESCAPE investigators concluded that PAC use in refractory heart failure is safe but does not confer major beneficial effects based on these data. More recently, 2 of the ESCAPE investigators provided guidelines for the use of a PAC in patients with advanced heart failure (Table 2).²⁶

Several studies reporting the safety and efficacy of the PAC were published in 2006. Friese and colleagues²⁷ performed a retrospective analysis of data from the National Trauma Data Bank. Patients who were managed with a PAC (n = 1,933) were compared with those managed without (n = 51,379). Their initial analysis identified a significant increase in mortality in patients managed by PAC ($P < .001$). However, once severity of injury was controlled, investigators found a survival benefit with PAC-guided management of shock upon arrival to the hospital in patients 61 to 90 years old, those with an arrival base deficit worse than -11 and those with an Injury Severity Score 25 to 75. Thus, PAC-guided therapy for shock reduced mortality in patients with severe shock at hospital arrival and in elderly patients with moderate shock.

TABLE 2 Indications for Use of a PAC in Patients With Advanced Heart Failure

Patients with: Failure of initial therapeutic management Unclear volume and perfusion state Worsening renal function during therapeutic intervention Significant hypotension during therapeutic intervention Requirement for escalating doses of inotropic or vasoactive medications Requirement for chronic outpatient infusion of medication Need for preoperative evaluation for cardiac transplantation
--

Abbreviation: PAC, pulmonary artery catheters.

In an observational clinical trial, Djaiani and colleagues¹⁸ investigated data requirements for clinical management of patients during and after elective coronary artery bypass surgery. All patients had a PAC, but data were blinded unless patients exhibited specific objective criteria; PAC data were then made available and used to manage therapy. Only 23% of patients studied (n = 200) demonstrated an actual need for the PAC data, and treatment was altered based on these data in only 9% of patients. This study did not support routine use of the PAC in elective coronary artery bypass surgery patients. The investigators concluded that patients receiving elective coronary artery bypass surgery do not require routine placement of a PAC, and PAC insertion should be delayed until clinical need is apparent.

Harvey and colleagues²² published a systematic review of studies reported between 1995 and 2003 to determine the effects of PAC-guided care on mortality and costs. These investigators found only 11 studies that met their inclusion criteria, and pooled data indicated that neither mortality nor costs were different for patients with PAC-guided therapy. Because only 2 of the included studies were clinical trials, this group followed this review with a multicenter, randomized clinical trial with 65 centers in the United Kingdom participating (n = 1,041) and found no difference in mortality, intensive care or hospital days, or number of days of organ support when patients managed with PAC were compared with those managed without. These investigators suggested that because the PAC has been demonstrated to produce no improvement in survival, clinical trials should be focused on testing protocols guided by PAC data in selected homogeneous groups of critically ill patients to more clearly evaluate efficacy.

Protocol-Driven Therapy Using PAC Versus Central Venous Catheter

Only one study has been published recently that reported the results from a multicenter factorial, randomized clinical trial that included the testing of a systematic treatment protocol based on hemodynamic data. The ARDS Clinical Trials Network¹⁹ randomized patients with acute lung injury to a treatment protocol guided by PAC data (n = 513) or one guided by central venous catheter (CVC) data (n = 487). Patients were further randomized to either liberal or conservative fluid management. A specific hemodynamic protocol guided the management of all patients; ranges for measures obtained from the catheter (PAC or CVC) and for blood pressure, urinary output, and physical findings were imbedded in the protocol. Patients with acute lung injury managed with a PAC demonstrated no difference in mortality, ventilator-free

days, intensive care days, lung and kidney function, hypotension rates, ventilator settings, or use of dialysis or vasopressors when compared with those managed with a CVC. Pulmonary artery catheter use was associated with more complications compared with CVC use. Although none were related to mortality, these included cardiac dysrhythmias, difficult placement of catheter, catheter malfunction, air embolus, arterial puncture, and local infection. To date, this is the only clinical trial to evaluate the use of PAC information to guide therapy without the unspoken assumption of other studies that placement of the PAC alone will improve patient outcome.

Issues With Reliability and Validity

There are a number of variables that may influence the validity and reliability of measures made with a PAC. These can be categorized as patient or clinician factors. Control of these variables has not typically been described in studies of PAC use, but lack of control may considerably influence data obtained by PAC and study outcomes; thus, consideration of these is vital.

Patient Factors

Patient variables that influence PAC measures include the position of the catheter within the pulmonary vascular system, the extent of pulmonary artery waveform excursion with ventilation, the degree of pulmonary and chest wall compliance, the amount and transmission of pleural pressure to pulmonary vessels, and the application of mechanical ventilation and positive end-expiratory pressure. Valid measures require the PAC tip be located in a West zone III lung area, a vascular area where pulmonary arterial pressure is greater than venous and alveolar pressure.²⁸ This location ensures a column of blood from the distal tip of the catheter to the left heart and measurement of pressures that reflect end-diastolic pressure. West zone III areas are typically located at or below the level of the left atrium. Distal catheter tip position may be evaluated by radiograph, inspection of the pulmonary artery occlusion pressure waveform, and comparison of the pulmonary artery end-diastolic pressure to the pulmonary artery occlusion pressure.²⁹

Ventilatory variation in the pulmonary artery pressure waveform is common. Changes in intrathoracic pressure produced with ventilation are reflected on pulmonary vessels within the closed thoracic cavity. During normal spontaneous ventilation, pulmonary artery pressure decreases with inspiration and increases during expiration. In this instance, pulmonary artery pressures are measured at end-expiration when intrathoracic and atmospheric pressures are equal. In patients with a 10- to 15-mm Hg fluctuation in

pulmonary artery pressure with ventilation, pulmonary artery occlusion pressure may be overestimated by as much as 10 mm Hg.³⁰ When substantial ventilatory excursions occur, measures made at the point midway between end-inspiration and end-expiration are more valid.³¹

To further complicate PAC measures, the extent of intrathoracic pressure transmitted to the pulmonary vessels is influenced by the degree of lung and chest wall compliance.^{32,33} Less pressure is transmitted to the pulmonary vessels in a patient with reduced lung compliance, as with ARDS, and/or increased chest wall compliance found with conditions like flail chest. Thus, pathophysiological alterations must be considered in the measurement and evaluation of PAC data.

Mechanical ventilation strategies confound the measurement of pulmonary artery pressure. Auto-positive end-expiratory pressure (PEEP) with hyperinflation or the application of mechanical PEEP may produce apparent elevations in pulmonary pressures that are not a true reflection of hemodynamic state. When PEEP of 10 cm H₂O pressure or less is used with mechanical ventilation, the effect on pulmonary vascular pressures is negligible. Auto-positive end-expiratory pressure > 10 cm H₂O pressure has been demonstrated to be transmitted to pulmonary vessels by 40% to 60% in the presence of both normal and reduced lung compliance and to increase linearly as PEEP level is increased.³³ However, transmission of PEEP often exceeds 60%. It is currently not possible to reliably estimate the degree of pressure transmission in any given individual.

In addition to mechanical ventilation and PEEP, intra-abdominal hypertension, a frequent complication of critical illness, also influences the degree of pleural pressure transmission, with as much as 60% to 70% of intra-abdominal pressure transmitted to the pleural space.^{34,35} In some institutions, calculations may be used to “correct” the pulmonary artery occlusion pressure values for the application of PEEP; however, this correction is valid only in patients with normal lung and chest wall compliance and does not take into account the effects of intra-abdominal pressure.³⁶ Thus, for most critically ill patients, use of this correction may not provide valid data.

Hemodynamic homeostasis is maintained by rapid, complex alterations in a number of cardiovascular variables like heart rate, contractility, and vascular resistance. Pathological states, drug therapy, and co-existing disease processes may affect response to alterations in homeostasis. Often, critically ill individuals may not respond as expected because of undetected physiological alterations or drug effects. Thus, isolated or infrequent measures of hemodynamic parameters may not reflect true hemodynamic

state. Catheter tip position, pleural pressure, and lung and chest wall compliance may additionally confound the true hemodynamic picture and produce invalid measures upon which clinical decisions are made.

Clinician Factors

There are several variables related to clinicians that influence the reliability and validity of PAC measures. Perhaps one of the most serious issues with PAC use is the degree of clinician technical skill and knowledge. The measurement of reliable and valid data from a PAC requires a thorough understanding of cardiovascular anatomy, physiology, pathophysiology, and hemodynamic monitoring principles. Insufficient and inaccurate knowledge in any of these areas can lead to errors in equipment preparation and use, collection of invalid hemodynamic data, and inaccurate interpretation of patient hemodynamic status. Unfortunately, seriously inadequate knowledge for both critical care physicians and nurses³⁷⁻⁴⁰ and misinterpretation of PAC data⁴¹ has been previously documented. The Pulmonary Artery Catheter Educational Program was established by the Pulmonary Artery Catheterization and Clinical Outcomes Workshop and is currently available for clinicians (www.pacep.org) to provide consistent, high-quality information to improve clinician knowledge; however, its impact has not been recently evaluated.

Valid PAC measures require a fluid-filled system with an appropriate frequency response that has been correctly zeroed (referenced to atmospheric air), calibrated (standardized and calibrated by manufacturers), and leveled or referenced to the left atrium.⁴² Incorrect transducer leveling generates inaccurate pressure measures.⁴³ Rice and colleagues⁴⁴ performed a systematic comparison of methods commonly used for transducer leveling and identified significant differences in transducer placement dependent on leveling method used ($P < .05$). In this study, errors in transducer placement ranged from 4.6 cm above the left atrium to 6.3 cm below. Errors were greatest using simple visual alignment and least using a laser level. Thus, a factor as simple as transducer placement may significantly alter PAC data. Appropriately releveling the transducer with a change in patient position provides accurate and reliable data when performed correctly.⁴⁵⁻⁵⁴ Unfortunately, clinicians have not consistently adopted research findings related to PAC measures and body position.

Another technical issue that influences validity of PAC data is the choice to use digital values provided by monitoring equipment rather than measurement of pressures from a printed graphic representation

of the waveform. Values obtained by clinician examination of graphic waveforms were found to be significantly more accurate compared with the digital value provided at the bedside in a study by Ahrens and Schallom ($P < .05$).⁵⁵ Critical care clinicians who rely on the digital hemodynamic values provided by a monitor may seriously compromise patient safety with erroneous clinical decisions based on inaccurate data.

A major clinician-based issue is the standard normative values against which critically ill patient values are compared. These norms were obtained from resting, normal adults.⁵⁶⁻⁵⁸ However, by the very nature of their illness or injury, critically ill individuals are not normal. This does not mean that all patients will have hemodynamic values that fall outside established normal ranges; however, values within the normal range may be inadequate for critically ill patients.⁵⁹ Currently, optimal hemodynamic ranges do not exist for critically ill patients. Thus, the set goal of PAC interventions may be inappropriate, whereas the true goal remains unknown.

Complications Associated With PAC

Complications related to PAC use were not systematically investigated after the introduction of this new technology. Early published reports of complications included single case reports, usually with the admonition to be prepared for a similar case. In the 1970s, a number of serious complications directly attributed to the PAC were reported. These included fatal pulmonary hemorrhage,^{60,61} the development of complete heart block,⁶² pulmonary infarction,⁸ aseptic, thrombotic, endocardial vegetation,⁷ ventricular tachycardia requiring cardioversion,⁶³ and tangling of the PAC in intracardiac sutures.⁹ In response to a report of a patient death from pulmonary hemorrhage,⁶⁰ Drs Swan and Ganz⁶⁴ wrote a letter to the editors of *Annals of Internal Medicine* and said "Considering that between 1 and 2 million flow-directed catheterizations have been done since the introduction of balloon flotation catheters, the real incidence of this complication is *probably* low (although many cases have gone unreported); nevertheless, we feel that a majority of such events are avoidable by strict adherence to appropriate techniques." Swan and Ganz attributed complications to the lack of knowledge and training of physicians and nurses and lack of adherence to the published guidelines for PAC use.⁶⁵

Although recent data indicate that PAC is not associated with greater morbidity and mortality in several patient populations, the occurrence of serious adverse events continues to be anecdotally reported. Recent complications reported included pseudoaneurysm

formation,⁶⁶ arteriovenous fistula formation between carotid artery and internal jugular vein,⁶⁷ venous air embolus,⁶⁸ pulmonary artery rupture,^{69,70} and right ventricular perforation.⁷⁰ In a systematic investigation of PAC-related complications in patients with pulmonary hypertension, a particularly high-risk group, Hoepfer and colleagues⁷¹ found that 1.1% of patients (n = 7,218) receiving PAC experienced a serious adverse event, and only 0.06% experienced an adverse event that resulted in mortality. This low rate of serious adverse events and mortality is consistent with that reported in several of the clinical trials previously described.^{18,19,22} In addition, Bossert and colleagues⁷⁰ found only 4 serious complications of PAC use in perioperative cardiac surgery patients (n = 3,730), a prevalence of 0.1%. Several investigators purport that centers with a high volume of PAC use experience lower rates of serious adverse events potentially due to superior technical skills and knowledge;^{17,22} however, this relationship has not been systematically investigated.

Summary

Routine placement of PACs is not supported by studies published since the Pulmonary Artery Catheterization and Clinical Outcomes Workshop in 1997. Most of these studies found the PAC to be safe but did not identify any benefit from its use. Clearly, placement of the PAC alone should not be expected to improve outcomes because it is a diagnostic and monitoring device rather than a treatment. A therapy guided by a PAC should be the actual independent variable studied in future clinical trials.

REFERENCES

- Bernard GR, Sopko G, Cerra F, et al. Pulmonary artery catheterization and clinical outcomes. National Heart, Lung and Blood Institute and Food and Drug Administration Workshop Report. *JAMA*. 2000;283(19):2568–2572.
- Liljestrand G. Presentation speech. The Nobel Prize in physiology or medicine 1956. <http://nobelprize.org/nobel-prizes/medicine/laureates/1956/press.html>. Accessed July 18, 2007.
- Swan HJ, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *N Engl J Med*. 1970;283(9):447–451.
- Rutherford BD, McCann WD, O'Donovan PB. The value of monitoring pulmonary artery pressure for early detection of left ventricular failure following myocardial infarction. *Circulation*. 1971;XLIII:655–666.
- Ganz W, Donoso R, Marcus HS, Forrester JS, Swan HJC. A new technique for measurement of cardiac output by thermodilution in man. *Am J Cardiol*. 1971;27(4):392–396.
- Maisel WH. Medical device regulation: an introduction for the practicing physician. *Ann Intern Med*. 2004;140(8):296–302.
- Pace NL, Horton W. Indwelling pulmonary artery catheters: their relationship to aseptic thrombotic endocardial vegetations. *JAMA*. 1975;233(8):893–894.
- Reinke RT, Higgins CB. Pulmonary infarction complicating the use of Swan-Ganz catheters. *Br J Radiol*. 1975;48(575):885–888.
- Block PC. Snaring of a Swan-Ganz catheter. *J Thorac Cardiovasc Surg*. 1976;71(6):917–919.
- Gore JM, Goldberg RJ, Spodick DH, Alpert JS, Dalen JE. A community-wide assessment of the use of pulmonary artery catheters in patients with acute myocardial infarction. *Chest*. 1987;92(4):721–727.
- Zion MM, Balkin J, Rosenmann D, et al. Use of pulmonary artery catheters in patients with acute myocardial infarction: analysis of experience in 5841 patients in the SPRINT registry. *Chest*. 1990;98(6):1331–1335.
- Parsons PE. Progress in research on pulmonary-artery catheters. *N Engl J Med*. 2003;348(1):66–68.
- Connors AF, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. *JAMA*. 1996;276(11):889–897.
- Vieillard-Baron A, Girou E, Valente E, et al. Predictors of mortality in acute respiratory distress syndrome. *Am J Respir Crit Care Med*. 2000;161(5):1597–1601.
- Yu DT, Platt R, Lanken PN, et al. Relationship of pulmonary artery catheter use to mortality and resource utilization in patients with severe sepsis. *Crit Care Med*. 2003;31(12):2734–2741.
- Richard C, Warszawski J, Anguel N, et al. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized clinical trial. *JAMA*. 2003;290(20):2713–2720.
- Binanay C, Califf RM, Hasselblad V, et al. ESCAPE Investigators and ESCAPE Study Coordinators. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial. *JAMA*. 2005;294(13):1625–1633.
- Djaiani G, Karski J, Yudin M, et al. Clinical outcomes in patients undergoing elective coronary artery bypass graft surgery with and without utilization of pulmonary artery catheter-generated data. *J Cardiothorac Vasc Anesth*. 2006;20(3):307–310.
- ARDS Clinical Trials Network. Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. *N Engl J Med*. 2006;354(21):2213–2224.
- Sandham JD, Hull RD, Brant RF, et al. A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. *N Engl J Med*. 2003;348(1):5–14.
- Polanczyk CA, Rohde LE, Goldman L, et al. Right heart catheterization and cardiac complications in patients undergoing noncardiac surgery. *JAMA*. 2001;286(3):309–314.
- Harvey S, Stevens K, Harrison D, et al. An evaluation of the clinical and cost-effectiveness of pulmonary artery catheters in patient management in intensive care: a systematic review and a randomized controlled trial. *Health Technol Assess*. 2006;10(29):1–133.
- Rapoport J, Teres D, Steingrub J, Higgins T, McGee W, Lemeshow S. Patient characteristics and ICU organizational factors that influence frequency of pulmonary artery catheterization. *JAMA*. 2000;283(19):2559–2567.
- Rhodes A, Cusack RJ, Newman PJ, Grounds RM, Bennett ED. A randomised, controlled trial of the pulmonary artery catheter in critically ill patients. *Int Care Med*. 2002;28(3):256–264.

25. Shah M, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. *JAMA*. 2005; 294(13):1664–1670.
26. Shah MR, Miller L. Use of pulmonary artery catheters in advanced heart failure. *Curr Opin Cardiol*. 2007;22(3): 220–224.
27. Friese RS, Shafi S, Gentilello LM. Pulmonary artery catheter use is associated with reduced mortality in severely injured patients: a National Trauma Data Bank analysis of 53,312 patients. *Crit Care Med*. 2006; 34(6):1597–1601.
28. Glenny RW, Lamm WJ, Albert RK, Robertson HT. Gravity is a minor determinant of pulmonary blood flow distribution. *J Appl Physiol*. 1991;71(2):620–629.
29. Bridges EJ. Pulmonary artery pressure monitoring: when, how, and what else to use. *AACN Adv Crit Care*. 2006; 17(3):286–305.
30. Hoyt JD, Leatherman JW. Interpretations of the pulmonary artery occlusion pressure in mechanically ventilated patients with large respiratory excursions in intrathoracic pressure. *Intensive Care Med*. 1997;23(11): 1125–1131.
31. Rizvi K, Deboisblanc BP, Truwit JD, et al. Effect of airway pressure display on interobserver agreement in the assessment of vascular pressures in patients in acute lung injury and acute respiratory distress syndrome. *Crit Care Med*. 2005;33(1):98–103.
32. O'Quin RJ, Marini JJ, Culver BH, Butler J. Transmission of airway pressure to pleural space during lung edema and chest wall restriction. *J Appl Physiol*. 1985;59(4): 1171–1177.
33. Kallet RH, Katz JA, Pittet JF, et al. Measuring intraesophageal pressure to assess transmural pulmonary arterial occlusion pressure in patients with acute lung injury: a case series and review. *Respir Care*. 2000;45(9): 1072–1084.
34. Schachtraupp A, Graf J, Tons A, et al. Intravascular volume depletion in a 24-hour porcine model of intra-abdominal hypertension. *J Trauma*. 2003;55(4):734–740.
35. Malbrain M, Nieuwendijk R, Verbrugghe W, et al. Effect of intra-abdominal pressure on pleural and filling pressures. *Intensive Care Med*. 2003;29(suppl 1):S73.
36. Chapin JC, Downs JB, Douglas ME, Murphy EJ, Ruiz BC. Lung expansion, airway pressure transmission, and positive end-expiratory pressure. *Arch Surg*. 1979;114(10): 1193–1197.
37. Johnston IG, Jane R, Fraser JF, Kruger P, Hickling K. Survey of intensive care nurses' knowledge relating to the pulmonary artery catheter. *Anaesth Intensive Care*. 2004; 32(4):564–568.
38. Iberti TJ, Daily EK, Leibowitz AB, et al. Assessment of critical care nurses' knowledge of the pulmonary artery catheter: the Pulmonary Artery Catheter Study Group. *Crit Care Med*. 1994;22(10):1674–1678.
39. Burns S, Burns D, Shively M. Critical care nurses' knowledge of pulmonary artery catheters. *Am J Crit Care*. 1996; 5(1):49–54.
40. Trottier SJ, Taylor RW. Physician attitudes toward and knowledge of the pulmonary artery catheter: Society of Critical Care Medicine membership survey. *New Horiz*. 1997;5(3):201–206.
41. Nadeau S, Noble WH. Misinterpretation of pressure measurements from the pulmonary artery catheter. *Can Anesth Soc J*. 1986;33(3 pt 1):352–363.
42. Courtois M, Fattal P, Kovacs S, et al. Anatomically and physiologically based reference level for measurement of intracardiac pressures. *Circulation*. 1995;92(7): 1994–2000.
43. Bisnaire D, Robinson L. Accuracy of leveling hemodynamic transducer systems. *CACCN*. 1999;10(4):16–19.
44. Rice WP, Fernandez EG, Jarog D, Jensen A. A comparison of hydrostatic leveling methods in invasive pressure monitoring. *Crit Care Nurs*. 2000;20(6):20–30.
45. Dobbin K, Wallace S, Ahlberg J, Chulay M. Pulmonary artery pressure measurement in patients with elevated pressures: effect of backrest elevation and method of measurement. *Am J Crit Care*. 1992;1(2):61–69.
46. Wilson A, Bermingham-Mitchell K, Wells N, Zachary K. Effect of back position on hemodynamic and right ventricular measurements in critically ill adults. *Am J Crit Care*. 1996;5(4):264–270.
47. Woods S, Grose B, Laurent-Bopp D. Effect of backrest position on pulmonary artery pressures in critically ill patients. *Cardiovasc Nurs*. 1982;18(4):19–24.
48. Cason C, Lambert C. Backrest position and reference level in pulmonary artery pressure measurement. *Clin Nurs Spec*. 1987;2(1):159–165.
49. Ross C, Jones R. Comparisons of pulmonary artery pressure measurements in supine and 30 degree lateral positions. *Can J Cardiovasc Nurs*. 1995;6(3–4):4–8.
50. Bridges EJ, Woods SL, Brengelmann GL, Mitchell P, Laurent-Bopp D. Effect of the 30° lateral recumbent position on pulmonary artery and pulmonary artery wedge pressures in critically ill adult cardiac surgery patients. *Am J Crit Care*. 2000;9(4):262–275.
51. Briones T, Dickenson S, Bieberitz R. Effect of positioning on SVO₂ and hemodynamic measurements [abstract]. *Heart Lung*. 1991;20:297.
52. Cason CL, Holland CL, Lambert CW, Huntsman KT. Effects of backrest elevation and position on pulmonary artery pressures. *Cardiovasc Nurs*. 1990;26(1):1–6.
53. Groom L, Frisch S, Elliott M. Reproducibility and accuracy of pulmonary artery pressure measurements in supine and lateral positions. *Heart Lung*. 1990;19(2):147–151.
54. VanEtta D, Gibbons E, Woods S. Estimation of left atrial location in supine and 30° lateral position [abstract]. *Am J Crit Care*. 1993;2:264.
55. Ahrens TS, Schallom L. Comparison of pulmonary artery and central venous pressure waveform measurements via digital and graphic measurement methods. *Heart Lung*. 2001;30(1):26–38.
56. Pinsky MR. Hemodynamic monitoring in the intensive care unit. *Clin Chest Med*. 2003;24(4):549–560.
57. McKinley BA, Kozar RA, Cocanour CS, et al. Normal versus supranormal oxygen delivery goals in shock resuscitation: the response is the same. *J Trauma*. 2002; 53(5):825–832.
58. Velmahos GC, Demetriades D, Shoemaker WC, et al. Endpoints of resuscitation of critically injured patients: normal or supranormal? A prospective randomized trial. *Ann Surg*. 2000;232(3):409–418.
59. Murphy GS, Vender JS. Con: Is the pulmonary artery catheter dead? *J Cardiothorac Vasc Anesth*. 2007;21(1): 147–149.
60. Golden MS, Pinder T, Anderson WT, Cheitlin MD. Fatal pulmonary hemorrhage complicating use of a flow-directed balloon-tipped catheter in a patient receiving anticoagulant therapy. *Am J Cardiol*. 1973;32(6):865–867.
61. Pape LA, Haffajee CI, Markis JE, et al. Fatal pulmonary hemorrhage after use of the flow-directed balloon-tipped catheter. *Ann Intern Med*. 1979;90(3):344–347.
62. Abernathy WS. Complete heart block caused by the Swan-Ganz catheter. *Chest*. 1974;65(3):349.

63. Shimm DS, Rigsby L. Ventricular tachycardia associated with removal of a Swan-Ganz catheter. *Postgrad Med*. 1980; 67(3):291, 294.
64. Swan HJC, Ganz W. Complications with flow-directed balloon-tipped catheters. *Ann Intern Med*. 1979;91(3):494.
65. Swan HJC, Ganz W. Guidelines for use of balloon-tipped catheter. *Am J Cardiol*. 1974;34:119–120.
66. Poplasky MR, Rozenblit G, Rundback JH, et al. Swan-Ganz catheter-induced pulmonary artery pseudoaneurysm formation: Three case reports and a review of the literature. *Chest*. 2001;120(6):2105–2111.
67. Asteri T, Tsagaropoulou I, Vasiliadis K, et al. Beware Swan-Ganz complications: perioperative management. *J Cardiovasc Surg*. 2002;43(4):467–470.
68. Chakravarthy M. Yet another venous air embolism! *J Cardiothorac Vasc Anesth*. 2004;18(5):681–682.
69. Abreau AR, Campos MA, Krieger BP. Pulmonary artery rupture induced by a pulmonary artery catheter: a case report and review of the literature. *J Intensive Care Med*. 2004; 19(5):291–296.
70. Bossert T, Gummert JF, Hartmuth B, et al. Swan-Ganz catheter-induced severe complications in cardiac surgery: right ventricular perforation, knotting and rupture of a pulmonary artery. *J Card Surg*. 2006;21(3):292–295.
71. Hoepfer MM, Lee SH, Voswinckel R, et al. Complications of right heart catheterization procedures in patients with pulmonary hypertension in experienced centers. *J Am Coll Cardiol*. 2006;48(12):2546–2552.