Cardiovascular monitoring tools: use and misuse
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Purpose of review
To review important areas of current and novel hemodynamic monitoring practice in the intensive care unit and to highlight potential areas of physiologic and clinical use or misuse, as well as areas of uncertainty and ongoing controversy.

Recent findings
To truly determine when hemodynamic monitoring tools are misused would require randomized controlled evidence of a measurable improvement in relevant clinical (as opposed to physiologic) outcomes. Unfortunately, little evidence of this kind exists, and that which does exist is highly controversial in nature. Because of the limited evidence of an effect of hemodynamic monitoring on clinical outcomes, the use and misuse of hemodynamic monitoring tools is typically judged on physiologic grounds (Does it improve physiology? Does it predict physiology? Is it physiologically rational?). The relation between physiologic gain and final clinical outcome, however, is tenuous. Recent investigations confirm this lack of a clear link. They also suggest that new technology that is now emerging to less invasively measure cardiac output and intrathoracic fluid compartments is ready for formal evaluations of efficacy and effectiveness.

Summary
The effectiveness of hemodynamic monitoring in the intensive care unit remains inadequately tested and unproven. New tools are now rapidly emerging to challenge established technologies. Formal assessment of their efficacy and effectiveness is needed to avoid a repeat of the pulmonary artery catheter experience.

Keywords
blood pressure, cardiac output, monitoring, outcome, pulse contour analysis

Efficacy of monitoring
Although we do not have direct evidence of any clinical benefits from invasive hemodynamic monitoring, we believe that more intensive monitoring (invasive and noninvasive) is needed to ensure the safety of acutely ill patients. If we did not, we would not have ICUs. Let’s now, therefore, for a moment, take such safety for granted (even though the insertion of intravascular catheters is actually associated with a defined risk of infection, pneumothorax, inadvertent arterial puncture, and bleeding), and let’s simply ask ourselves whether advanced monitoring is efficacious. This question is difficult to answer because the concept of efficacy indicates that an agent is capable of producing

The evils of controversy are transitory, while its benefits are permanent.

—R. Hall (1830)
Cardiopulmonary monitoring

the desired effect. This may be obvious and easy to define for an antihypertensive agent: the drug decreases blood pressure compared with placebo. What should be the measure of efficacy for a monitoring tool? A possible answer is that it should be able to measure that which it is designed to measure. Thus, a pulmonary artery catheter (PAC) designed to measure the cardiac output should be able to do so with accuracy and precision. Using such criteria, most currently available monitoring tools perform to an imperfect but seemingly reasonable degree. Whether that is “good enough” remains controversial yet practically unimportant because clinically applicable more precise and accurate tools do not exist. If they did, they would be used instead. More importantly, however, such measurements cannot bring about physiologic changes by themselves because they are not therapeutic tools. In the field of hemodynamics, the therapeutic tools are intravenous fluids, vasopressors, inotropic agents, vasodilators, diuretics, and so on. Thus, the effect of hemodynamic tools on physiology depends on how the signals they provide are used by clinicians to alter the use of therapeutic tools. There is ample evidence that (1) some signals are not correctly interpreted by physicians and (2) for a given signal, there can be extremely variable responses, which will inevitably lead to different physiologic outcomes and perhaps different clinical outcomes [1].

Faced with such uncertainty and variability, many articles have been written, debates conducted, and guidelines issued about what kind of responses represent physiologically correct or physiologically rational actions given a certain signal [1]. However, although physiologically rational behavior seems desirable, there is no evidence that applying it makes any difference in terms of patient outcome, that experts consistently agree on what such “correct” approaches are, or that some of the signals that we have used and continue to use to guide hemodynamic management are the ones that we should be measuring [1].

Furthermore, although there is much evidence that nurses’ and physicians’ knowledge of the PAC is limited [2–4], there is no evidence that clinicians who err in their interpretation of, for example, PAC waves, would then administer “wrong” therapies to patients, which would result in worse clinical outcomes. Thus, establishing use and misuse is very difficult indeed. Nonetheless, some physiologic facts have emerged over the last 25 years that are worth emphasizing.

**Facts, not opinions**

Despite the aforementioned controversies, some important facts related to hemodynamic monitoring have been convincingly demonstrated by many investigators [5,6,7•,8•,9–11,12•,13,14•] in different groups of patients as outlined in Table 1.

<table>
<thead>
<tr>
<th>Table 1. Factual observations about hemodynamic monitoring</th>
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<tr>
<td>1. The central venous pressure does not reliably predict the right ventricular end-diastolic volume [5].</td>
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<tr>
<td>2. The pulmonary artery occlusion pressure does not reliably predict left (or right) end-diastolic volume [5].</td>
</tr>
<tr>
<td>3. Neither the central venous pressure nor the pulmonary artery occlusion pressure reliably predicts whether the administration of a fluid bolus will or will not significantly increase cardiac output [6,7•,8•].</td>
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<td>4. The cardiac output cannot be reliably predicted by physical examination [9].</td>
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<tr>
<td>5. Neither the central venous pressure nor the pulmonary artery occlusion pressure reliably predicts the likelihood of developing or having just developed pulmonary edema [10].</td>
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<tr>
<td>6. A mean arterial blood pressure within normal limits does not reliably indicate an adequate cardiac output [9].</td>
</tr>
<tr>
<td>8. A normal mixed venous oxygen saturation does not reliably indicate adequate organ perfusion [12].</td>
</tr>
<tr>
<td>9. A change in oxygen consumption in response to a change in calculated oxygen delivery does not reliably indicate the presence of an “oxygen debt” [13].</td>
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<tr>
<td>10. The pulmonary artery occlusion pressure is not the pressure in the pulmonary capillaries [14•].</td>
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It should be reasonably assumed that these facts are well known to critical care physicians, as they have been discussed, presented, published, and disseminated over many years. However, we do not have epidemiologic evidence that such knowledge does exist. We also have no evidence that clinicians who are aware of these hemodynamic facts achieve better clinical outcomes than physicians who are not. We would like to think so, but we do not know. More importantly, daily observation in any ICU would immediately indicate to any educated observer that clinicians do not typically use any of these hemodynamic signals in isolation. If they measure the central venous pressure (CVP) to help them assess the need for intravenous fluids, they use this information in a context in which the blood pressure is known; skin perfusion is assessed; urinary output, serum creatinine, and blood lactate are measured; the fluid balance for the last 24 hours or last few days is known in detail; and so on. Thus, although the CVP, for example, is by itself a rather inaccurate tool to predict a given cardiac output response to intravenous fluids or to predict the right ventricular end-diastolic volume, such deficiencies may not matter in the clinical context. In such a situation it is the CVP, lactate concentration, physical examination, urine output, noninvasive echocardiographic data, serum creatinine concentration, blood pressure, skin perfusion, ventilator data, chest radiograph, arterial blood gases, fluid balance, and more, all integrated with knowledge of the patient’s illness and pre-illness status, that determine hemodynamic management. No studies have been conducted to test either the efficacy or the predictive value or effectiveness of this approach. Thus, in 2003, we do not know what the precision, accuracy, or physiologic relevance of integrated hemodynamic monitoring (which is what is used in ICUs in the developed world every day) really is. This lack of knowledge is a problem. If we
do not know the physiologic relevance of integrated data from hemodynamic monitoring tools, how can we possibly know their clinical relevance? If we know neither physiologic nor clinical relevance, how can we establish what is correct use and what is misuse of such tools?

We can probably say that using a single tool in isolation is prone to a high likelihood of error, but we cannot say much more than that. Furthermore, the meaning of such statements is unclear. Investigators, if they so wish (and they do seem to do so), can take a dozen single hemodynamic signals and use each one in isolation to predict an increment in cardiac output in response to fluid therapy [5,6,7•,8•,9–11,12•]. They can statistically prove that hemodynamic variable $x$ correlates better than hemodynamic variable $y$ with a given increment in cardiac output. Yet what is the clinical relevance of such information? How many clinicians practice that way? Even if thousands of physicians did, and even if variable $x$ was 100% accurate in predicting a cardiac output response to an intravenous fluid bolus, we still do not know whether that fluid should be given in the first place. We still do not know whether the clinical cost of 1 additional liter of fluid in the body is worth the clinical gain of a 15% increase in cardiac output. The cost–benefit ratio may be low in a low cardiac output state (fluid should be given), limited in the presence of a normal cardiac output (it does not matter), and perhaps high in the setting of a high cardiac output state (fluid makes things worse).

We can predict with an area under the receiver operating characteristic curve of 1 that the blood pressure will increase if norepinephrine is infused intravenously? This is much better than the reported area under the receiver operating characteristic curve for blood pressure variation’s ability to predict an increment in cardiac output with intravenous fluids during mechanical ventilation [15]. Does that mean that we should therefore administer norepinephrine? We think not. Is all of this hemodynamic manipulation “much ado about nothing”? Is it beneficial for the patient? Is it actually deleterious? In modern ICUs, how often does organ failure occur because of overt or covert hemodynamic insufficiency? Or is organ failure immunologic in nature [16•], cytopathic [17], or a result of mitochondrial dysfunction [18]? Does increasing cardiac output in a septic patient simply lead to an extra surge in cytotoxic plasma delivery to several vital organs, thereby increasing rather than decreasing organ injury? Until these fundamental questions have been more clearly answered, it is impossible to define use and misuse of hemodynamic tools.

What’s the point of measuring anything?

Based on the previous discussion, one could reasonably conclude that (1) we have no adequate data to convincingly show that hemodynamic monitoring tools are clinically useful and (2) we have no data on how they are being used in ICUs around the world. Given such observations, perhaps we should call for a moratorium on all invasive hemodynamic monitoring, just as some investigators recently did for the PAC [1].

We believe that such an approach would be irrational. In patients with shock who need vasoactive drugs, their administration requires the presence of a central venous catheter to avoid local tissue injury and to ensure reliable delivery. Why not obtain continuous measurement of CVP to add yet more data to help assess the patient’s hemodynamic status?

If the patient needs an arterial line to help guide such potent vasopressor drug administration in the same setting, why not make the arterial catheter a thermistor-tipped one and thus obtain continuous pulse contour measurement of cardiac output and intermittent measurement of intrathoracic blood volume and extravascular lung water [8•]? Given that such data can be so easily collected, why not do so?

At this point it is important to ask oneself how many patients would have to be randomized to detect evidence of an effect on clinical outcome of one kind of monitoring versus another. Such thoughts would help clinicians realize why we still know so little of what constitutes use or misuse of these tools.

Trial size, if mortality rate is the primary outcome measure, depends on the expected mortality rate of the control group. Assuming a mortality rate of 15% in the control group, it would take a study population of more than 10,000 patients to detect a 10% relative decrease in mortality rate. Assuming a minimal cost of $300 per patient recruited, this study would cost more than $3 million. If the baseline mortality rate were 7.7%, as in the recent largest study of the PAC in surgical patients [19•], even with 1994 patients one would only have a 20% power of detecting a 20% relative decrease in mortality rate. To change the power to 90%, 14,240 patients would have to be randomized!

What should we measure? Pressures or volumes?

Critically ill patients who require fluid resuscitation and vasoactive drug therapy typically have their CVP and arterial pressure monitored. With the arrival of the PAC, the availability of cardiac output measurement created further goals for manipulation. Because of the perceived need to augment cardiac output (When is a given cardiac output ever enough?), various investigations have been conducted to allow physicians to predict when the administration of an intravenous bolus of fluids would increase the cardiac output. It has become clear that static pressure measurements are not very good at predicting such changes and that, in fact, phasic changes in blood pressure induced by mechanical ventilation might be the best pressure-based measurements to use in predicting
such a response [15]. Should we be going back to just measuring the blood pressure then? Probably not. We need the cardiac output to confirm such a response and perhaps filling pressures to help us decide when to stop. All of this might still require a PAC, with all of its risks [20,21]. More recently, however, technology that provides a simple and easier alternative has been developed and applied (pulse contour cardiac output [PiCCO] system; Pulsion Medical Systems, Munich, Germany). Such new technology means that any patient who needs a central venous catheter and an arterial catheter for drug infusion and pressure monitoring can also have three other variables measured to help guide therapy: continuous cardiac output, intrathoracic blood volume, and extravascular lung water [22,23,24,25,26]. In this setting, the PiCCO system offers a whole new set of data at no additional risk to the patient. Will such technology deliver better patient outcomes? Once again, it will take a long time to arrive at the answer given that, almost 30 years later, we still do not know whether the PAC can deliver better patient outcomes. Nonetheless, evidence is emerging that volume-based assessment of intravascular filling associated with continuous cardiac output can deliver levels of prediction of cardiac output changes that might be superior to those obtained with older technology [8•]. As practitioners familiar with both technologies, we find ourselves increasingly abandoning the PAC in favor of the PiCCO system because (1) the data on volume and extravascular lung water seem more physiologically and clinically relevant [24,25,26] in defining the variables we wish to modulate and (2) there is no additional risk to a patient who has a central venous catheter and needs an arterial catheter for continuous blood pressure monitoring anyway (Table 2). Thus, the PAC may soon become obsolete.

### Table 2. Comparative advantages and disadvantages of the pulmonary artery catheter and pulse contour cardiac output technology

<table>
<thead>
<tr>
<th>Aspect/variable</th>
<th>PAC</th>
<th>PiCCO</th>
</tr>
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<tbody>
<tr>
<td>Cardiac output</td>
<td>Yes (continuous with special technology)</td>
<td>Yes (always continuous)</td>
</tr>
<tr>
<td>PAOP</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Pulmonary pressures</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>RAP</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>RVEDV</td>
<td>Yes, with special technology</td>
<td>No</td>
</tr>
<tr>
<td>End-diastolic intrathoracic volume</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Extravascular lung water index</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Risk of pneumothorax</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Risk of infection</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Risk of arterial puncture</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Risk of pulmonary artery rupture</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Risk of air embolus</td>
<td>Yes</td>
<td>No</td>
</tr>
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PAC, pulmonary artery catheter; PAOP, pulmonary artery occlusion pressure; PiCCO, pulse contour cardiac output; RAP, right atrial pressure; RVEDV, right ventricular end-diastolic volume.

**Testing hemodynamic monitoring tools: science and controversy**

To our knowledge, there are two important randomized controlled trials that have attempted to address the issue of whether hemodynamic monitoring can affect clinical outcomes. The first study was conducted by Rivers *et al.* [27•], who randomized 263 emergency department patients with severe sepsis or septic shock to receive 6 hours of goal-directed therapy guided by a new central venous catheter able to deliver continuous central venous oximetric data or standard care. Patients randomized to the intervention experienced a close to 30% decrease in mortality rate ($P = 0.009$).

Sandham *et al.* [19•] recently completed a large trial during which the investigators randomized 1994 American Society of Anesthesiologists physical status III or IV patients who were scheduled for surgery and postoperative ICU stay. Half of the patients were randomized to receive goal-directed therapy guided by PAC, while the other half received standard care without the use of the PAC [20]. Goal-directed therapy meant that, in this trial, those patients randomized to have a PAC should receive interventions whenever possible to achieve the following goals in order of priority: (1) an oxygen delivery of 550 to 600 mL/min/m$^2$, (2) a cardiac index of 3.5 to 4.5 L/min/m$^2$; (3) a mean arterial pressure of 70 mm Hg, (4) a pulmonary artery occlusion pressure of 18 mm Hg; (5) a heart rate less than 120 beats per minute; and (6) a hematoctrit greater than 27%. This trial showed that more patients in the goal-directed therapy group received inotropic agents ($P < 0.001$), vasodilators ($P < 0.001$), anti-hypertensive medication ($P < 0.001$), packed cells ($P < 0.001$), and colloids ($P = 0.002$). The mortality rate of control patients was 7.7% compared with 7.8% in the PAC group. Interestingly, there was a significant increase in the incidence of pulmonary embolism among the PAC patients ($P = 0.004$). The investigators concluded that there was “no benefit of therapy directed by PAC over standard care in elderly, high-risk surgical patients requiring intensive care” [19•].

Although each trial deserves detailed discussion, which cannot be done here, the recent evidence appears to overwhelmingly suggest that, in patients in the ICU or operating room, either (1) using the PAC to guide therapy is not helpful or (2) PAC monitoring might be unhelpful but only when “wrongly” used to achieve supranormal values of cardiac index or oxygen delivery. Things might be different in emergency room patients, but the study by Rivers *et al.* [27•] is a single-center investigation with a higher than expected mortality rate in the control group and huge potential for a “Hawthorne effect” of monitoring. Thus, more information is needed in this setting.
Conclusions
Much work needs to be done before we can decide how best to use hemodynamic tools and how to avoid their misuse. Such research is difficult and controversial in design. The so-called experts have spent almost 15 years trying to tell us that we should maximize oxygen delivery in ICU or operative patients [19•]. The data now stand in stark contrast to such claims and demonstrate a case of past and probably present misuse. The experts are now going to tell us how to use the PAC in acute respiratory distress syndrome with protocols (ARDSNet: http://bedwig.mgh.harvard.edu/ardsnet/ards05.html) that make no physiologic sense. With the proposed acute respiratory distress syndrome protocol, clinicians could give patients with acute respiratory distress syndrome randomized to so-called “fluid conservative therapy” who have a cardiac index of 4 L/min/m², a CVP of 14 mm Hg, a urine output of 0.6 mL/kg/h, a fraction of inspired oxygen of 0.65, an arterial oxygen tension of 58 mm Hg, and develop a mean arterial pressure of 55 mm Hg (vasodilatory shock) a bolus of 15 mL/kg of saline [sic] as possible therapy. How is this physiologically reasonable?

In our opinion, when it comes to such expert advice, the expression caveat emptor seems most appropriate. Thus, the debate will continue, physiologically irrational use of hemodynamic tools will also continue, and controversy will flourish. Nonetheless, we look forward to the day when trials of the PAC use this tool not to achieve supranormal values but to maintain homeostasis, when volume-based instead of pressure-based hemodynamic tools are used to guide therapy and prevent both inadequate diastolic filling and inappropriate surges in extravascular lung water, and when, in the field of hemodynamics, more clinical outcome-based studies become available to help us choose the right tool and the right use for the tool.

References and recommended reading
Papers of particular interest, published within the annual period of review, have been highlighted as:
** Of special interest
** Of outstanding interest


The first randomized controlled trial of mixed venous saturation monitoring in septic patients in the emergency department.

This article provides a clear explanation of the possible utility of functional dynamic monitoring instead of static pressure monitoring in ventilated patients.