

Hemodynamic monitoring in acute heart failure

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Hemodynamic monitoring has moved in the last few years from being the holy grail of evaluating patients with acute heart failure to being all but extinct. Recent studies have not demonstrated any sustained benefits from right heart catheterization, and some studies have even suggested harm due to adverse events related to this invasive procedure. It is possible that this lack of efficacy is related to multiple inherent deficiencies in the design of these studies, including the inclusion of patients with chronic heart failure or mild acute heart failure, use of the reduction in pulmonary artery occlusion pressure as the main hemodynamic target for intervention, choice of treatment algorithms, and selection of ambitious long-term efficacy and safety end points.

This review discusses the role of hemodynamic monitoring in patients with acute heart failure. We suggest that right heart

catheterization should be reserved for patients with acute heart failure and impending respiratory or circulatory failure especially in the presence of a diagnostic or therapeutic dilemma or when encountering acute heart failure or hemodynamic lability refractory to conventional therapy.

Therapeutic algorithms emphasizing modern variables for cardiovascular performance and using safer and more efficacious individualized therapies and possibly noninvasive measurement of certain hemodynamic variables may enhance the likelihood of a beneficial effect for hemodynamic guided therapy. (Crit Care Med 2008; 36[Suppl.]:S40–S43)

KEY WORDS: right heart catheterization; impedance cardiography; right heart catheter; cardiac output; cardiac index; cardiac power; pulmonary artery occlusion pressure

Acute heart failure (AHF) presents a medical emergency and requires immediate intensive medical therapy to alleviate symptoms and prevent end-organ loss and death. The clinical value and safety of right heart catheterization (RHC) have been the subject of considerable debate. Gore et al. (1) (in acute myocardial infarction patients with hypotension, heart failure, or shock) and Connors et al. (2) (in intensive care unit patients with respiratory and multiorgan failure) demonstrated a neutral to negative effect of RHC on patient outcome. These two publications resulted in a call for a moratorium (3) on RHC. In 1997, a consensus conference (4) attempted to reassess indications for RHC. Conditions that could be considered to benefit from RHC included myocardial infarction complicated by hypotension, shock, or mechanical compli-

cations; acute or chronic heart failure; and pulmonary hypertension. Former meta-analyses assessing the effects of RHC morbidity (5) and mortality (6) in clinical trials showed that mortality was unaffected but morbidity increased with the use of RHC.

To address these issues, in recent years a few prospective randomized studies have attempted to explore the value of RHC in patients admitted to intensive care units (PAC-Man) (7) or patients with established severe or deteriorating chronic heart failure (ESCAPE) (8). The results of both of these studies were negative, showing no survival benefit related to RHC, with a slight increase in the incidence of procedure-related adverse events. However, these studies had significant methodological drawbacks. The ESCAPE study enrolled patients with significant heart failure who did not truly require RHC; to be enrolled patients had to be “sufficiently ill with advanced heart failure to make use of the PAC reasonable, but also sufficiently stable to make crossover to PAC for urgent management unlikely” (8). Moreover, the inclusion criteria did not mandate AHF exacerbation or specify heart failure severity threshold or criteria. The mere requirement for 160 mg/day of loop diuretics qualified patients to be enrolled in ESCAPE. Import-

tantly, a treatment guideline was incorporated into the study protocol emphasizing only occlusion pressure reduction as the main target for RHC monitoring and treatment. The results of the study demonstrated no benefit of RHC in the studies’ primary end point—days alive out of hospital during 6 months from randomization. This very ambitious end point demonstrated no difference between study arms. A retrospective subanalysis disclosed considerable heterogeneity in the results, with a beneficial effect in experienced sites along with reduction in renal impairment in the RHC-treated patients (V Hasseblat, personal communication, 2006). A meta-analysis of 13 randomized clinical trials assessing RHC for critically ill patients, published simultaneously with ESCAPE, also demonstrated lack of effect of RHC on clinical outcomes (mortality and hospital stay) (9).

When taken at face value these studies seem to be conclusive; however, a few limitations need to be discussed:

First, the inclusion criteria for ESCAPE allowed for enrollment of patients with severe chronic heart failure, but patients often did not have AHF and sometimes did not pose a clinical or therapeutic dilemma. Nine such patients were excluded based on an absolute requirement for RHC. Hence, ESCAPE results are less relevant or appli-

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cable to the profoundly sick AHF patients who have traditionally been the foremost candidates for RHC.

Second, the hemodynamic variable targeted presents a limitation. ESCAPE and other studies examining the role of RHC in patients with AHF have overemphasized the role of pulmonary artery occlusion pressure (PAOP) as the primary therapeutic target. Data gathered recently cast considerable doubt regarding the role of PAOP-guided algorithms. One issue is that the real preload of the left heart is muscle length and not pressure. Therefore, left ventricular end-diastolic volume and not pressure or its proxy (PAOP) should be related to preload. Numerous studies have shown no correlation between PAOP and left ventricular end-diastolic volume (10). Hence, PAOP does not closely estimate preload. Another issue is that the role of preload in determining contractility has also been questioned (11). Therefore, the hemodynamic variables commonly used in RHC studies and especially in ESCAPE may have been inappropriate.

Third, the treatment algorithms incorporated into some of the studies evaluating RHC use drugs that are neither proven beneficial (high-dose loop diuretics) (12) nor shown to enhance long-term patient outcome (inotropes). Hence, the benefit of RHC could be potentially offset by the long-term harmful effects of such treatment. It is possible that if short-term therapeutic effects (dyspnea relief, oxygen saturation increase, prevention of persistent or worsening heart failure) had been examined in appropriately selected patients, a beneficial effect would have been demonstrated.

Fourth is a concern about medical staff (13, 14): Knowledge regarding RHC insertion, equipment assembly and use, and data acquisition and interpretation is suboptimal (15) and is not improved by abbreviated training sessions (16). Pitfalls and optimization of hemodynamic monitoring have been previously described (17). There are clearly serious issues with safety of insertion, operation, and timely removal of the device. RHC complications had reduced since 1980 (18), ranging from 0.1% to 0.5% in the coronary surgical literature (19). However, complications increased in ESCAPE (average duration of RHC <48 hrs) to 4.2%, with half of these being infectious complications. Moreover, the efficiency of RHC-guided therapy is heavily related to the team's experience. Indeed, the ESCAPE

investigators observed a significant heterogeneity in the response to hemodynamic monitoring with a trend toward benefit in patients treated in centers that have significant experience in the procedure. Hence, RHC is more beneficial and safer in centers with experience in its implementation.

Fifth, technical design of RHC is often suboptimal: New systems providing continuous pulmonary artery saturation (20), cardiac output (21, 22), and right ventricular ejection fraction (23) suffer from considerable limitations (24, 25) and consequently were never widely accepted (26). The Arrow "hands-off" RHC reduced systemic infections associated with RHC (27) but was never subjected to a meaningful efficacy and safety trial.

Who Should Be Hemodynamically Monitored?

The first question that one should ask when contemplating any method of hemodynamic monitoring is whether monitoring is truly indicated. Since most exacerbations of AHF are short-lived and respond favorably to conventional therapy (vasodilators and diuretics), initial clinical assessment is probably sufficient in most cases. We recently demonstrated that a short (2-min) assessment of respiratory failure (need for mechanical ventilation or low oxygen saturation) and circulatory failure (need for pressors or low systolic blood pressure) can predict adverse short-term outcome (28). The recently published European guidelines for the treatment of AHF (29) define the clinical, laboratory, and hemodynamic goals of therapy. These guidelines suggest that patients who do not respond to therapy or those with pulmonary congestion and hypotension or hypoperfusion should be considered for RHC. However, the criteria for hemodynamic monitoring in patients with AHF are not clearly defined.

Hence, we believe that hemodynamic monitoring should be restricted to patients with severe AHF: those with impending or full-blown respiratory or circulatory failure, especially when refractory to initial treatment; those with progressive end-organ failure (renal or hepatic impairment); or those considered for support devices or cardiac transplantation. We further believe that since RHC safety and efficacy are highly dependent on the operational capabilities of the medical team attending to the patient, this type of invasive monitoring should be performed

only by teams with both the know-how and capability to perform this procedure and follow the patients appropriately.

Methods for Hemodynamic Monitoring

The second issue is what method is best for hemodynamic assessment and monitoring. This depends on the clinical question being asked. Table 1 provides the variables that are obtained either directly or indirectly from the hemodynamic monitoring tools that are widely available. The current European guidelines suggest that the hemodynamic targets of therapy should be to increase cardiac output (CO) and stroke volume and to reduce occlusion pressure to <18 mm Hg. Thus, occlusion pressure and CO should be the main hemodynamic measures monitored. These measures and targets should be reevaluated in view of their deficiencies, detailed subsequently.

Clearly, for any patient admitted with new heart failure, a complete Doppler and echocardiography interrogation should be performed (recommendation class I, level of evidence C) (29), since it provides immediate data regarding the mechanisms contributing to heart failure. Moreover, it clarifies the severity and the relative contribution of each one of these processes to AHF.

However, echo-Doppler has a few shortcomings. First, it is difficult to obtain reliable measurements of right heart pressures in a significant proportion of patients. Second, although echo-Doppler can measure CO (30, 31), and hence cardiac index and cardiac power (CP) (32), these measurements by echo-Doppler are clearly time consuming, require adequate ultrasound window, demand expertise, and are subject to considerable variability. Third, echo-Doppler cannot provide online continuous real-time monitoring or recording, such as that offered by RHC and impedance cardiography. Fourth, CO, cardiac index, and CP obtained by echo-Doppler in its lower ranges may not be reliable enough to discriminate between low cardiac output (like AHF) and a very low CO, such as those that may be seen in cardiogenic shock. Fifth, standard echocardiographic measurements of right atrial pressure and other derived right-sided pressures can be difficult to interpret in patients on mechanical ventilation.

Table 1. Relative ability of hemodynamic monitoring methods to measure selected variables

Variable	Pulmonary Artery Catheter	Bioimpedance Cardiography	Echo-Doppler	Left Heart Catheterization
Cardiac output index and power	+++	+++	+++	+
Right atrial pressures	+++	—	++	—
Right ventricular pressures	+++	—	++	—
Pulmonary artery pressures	+++	—	++	—
Left atrial pressures	++	—	+	+
Left ventricular pressures	—	—	+	+++
Systemic vascular resistance	+++	+++	+	+
Pulmonary vascular resistance	+++	—	+	—
Valvular disease	TS, PS	—	+++	AS
Diastolic dysfunction	—	—	+++	+
Systolic dyssynchrony	—	—	+++	—
Systolic function global and regional	—	—	+++	++
Thoracic fluid content	—	+++	—	—
Continuous online data monitoring and recording	++	+++	—	—
Shunt calculation	++ (±fluoroscopy guided)	—	++	—
Right heart saturations	+++ (±fluoroscopy guided)	—	—	—

TS, tricuspid stenosis; PS, pulmonic stenosis; AS, aortic stenosis; +++ extremely good; ++ very good; + good; — not good.

New Targets for Hemodynamic Monitoring

As suggested previously, the use of occlusion pressure as the main hemodynamic target in RHC may be suboptimal. In recent years, accumulated evidence has suggested that measurements of forward contractile power and flow are significant predictors of short- and long-term outcomes in patients with AHF. The calculation of cardiac power output (CPo), the product of simultaneously measured CO and mean arterial pressure (MAP) ($CPo = MAP \cdot CO$), and systemic vascular resistance (SVR) might be important in the diagnosis, risk stratification, and monitoring of patients with both chronic and acute heart failure (33). CPo integrates considerations of both CO and MAP, potentially providing an integrated means of assessing hemodynamic state in AHF. In a recent study, CPo and vascular resistance were invasively measured in 100 patients with AHF at baseline and for 30 hrs (34). Patients who experienced a recurrent AHF episode dur-

ing invasive hemodynamic monitoring had lower CPo at baseline and deterioration in CPo leading up to the acute event. Superimposed on this low and decreasing CPo was a steep increase in SVR immediately before the AHF event.

These core hemodynamic events lead to an acute mismatch between rapidly increasing afterload and impaired systolic performance, resulting in an acute increase in left ventricular end-diastolic pressure and a decrease in CO. Hence, monitoring of CPo and possibly SVR can be instrumental at least in diagnosing the different heart failure syndromes and can improve risk stratification of the patients. A striking example of the value of CPo in the risk stratification of hemodynamically unstable patients is the recent retrospective analysis of the SHOCK study, showing a very powerful predictive value of baseline CPo on hospital mortality (35).

Although currently no treatment algorithms are based on CPo, it is possible that the use of CPo and SVR in future

algorithms will provide a strategy superior to the one based on PAOP.

It seems that both thoracic (36) impedance and total body impedance (37, 38) can measure continuous CO and cardiac index. Moreover, impedance cardiography derived CO is less variable and more reproducible than thermodilution (39). Some bioimpedance systems, however, do not provide accurate CO (40) values when compared with the gold standard of thermodilution (41). Devices that can measure CO reliably may serve as tools for assessing pump performance by providing noninvasive online cardiac power (CPo) and cardiac power index. If indeed the cardiac power index becomes the prevailing hemodynamic descriptor for acute heart failure, impedance cardiography may provide some added value to bedside assessment and echocardiography (42). All these systems, however, do not provide us with right-sided pressures and pulmonary vascular resistance.

CONCLUSIONS

Hemodynamic monitoring using RHC in patients with moderate to severe, mostly stable heart failure, using current treatment algorithms, was not associated with clinical benefit in a few recently published prospective randomized studies. However, such benefit in high-risk AHF patients was not critically assessed. We believe that RHC is still a useful tool for the diagnosis and risk stratification of patients with severe AHF and impending or established respiratory or circulatory failure not responding to therapy, leading to end-organ dysfunction or requiring mechanical support. Efforts should focus on development of treatment algorithms based on measurements of cardiac contractility (cardiac power) and vascular resistance and development of new and efficacious treatments to be used as part of these algorithms. Noninvasive CO measurement, by providing hemodynamic trend, potentially may be used for the treatment of patients with severe AHF and may become a way to introduce hemodynamic monitoring to subacute and chronic heart failure cohorts.

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