Contemporary Review of Hemodynamic Monitoring in the Critical Care Setting

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Abstract

Hemodynamic assessment remains the most valuable adjunct to physical examination and laboratory assessment in the diagnosis and management of shock. Through the years, multiple modalities to measure and trend hemodynamic indices have evolved with varying degrees of invasiveness. Pulmonary artery catheter (PAC) has long been considered the gold standard of hemodynamic assessment in critically ill patients and in recent years has been shown to improve clinical outcomes among patients in cardiogenic shock. The invasive nature of PAC is often cited as its major limitation and has encouraged development of less invasive technologies. In this review, the authors summarize the literature on the mechanism and validation of several minimally invasive and noninvasive modalities available in the contemporary intensive care unit. They also provide an update on the use of focused bedside echocardiography.

Keywords

Hemodynamics, hemodynamic monitoring, shock, cardiogenic shock, intensive care unit, pulmonary artery catheter, Swan-Ganz catheter

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Hemodynamic assessment remains the cornerstone of accurate diagnosis of shock and the assessment of the response to therapy in critically ill patients. Contemporary cardiac intensive care units (CICU) manage patients with multiple co-morbidities along with an ailing heart.[1–3] An increasing number of patients with septic shock and undifferentiated shock are treated in the CICU in conjunction with patients with cardiogenic shock (CS).[4,5] For this reason, rapid and accurate hemodynamic assessment is essential for the differentiation of shock and subsequent guidance for treatment including escalation to pharmacological therapies or temporary mechanical circulatory support (MCS).[6]

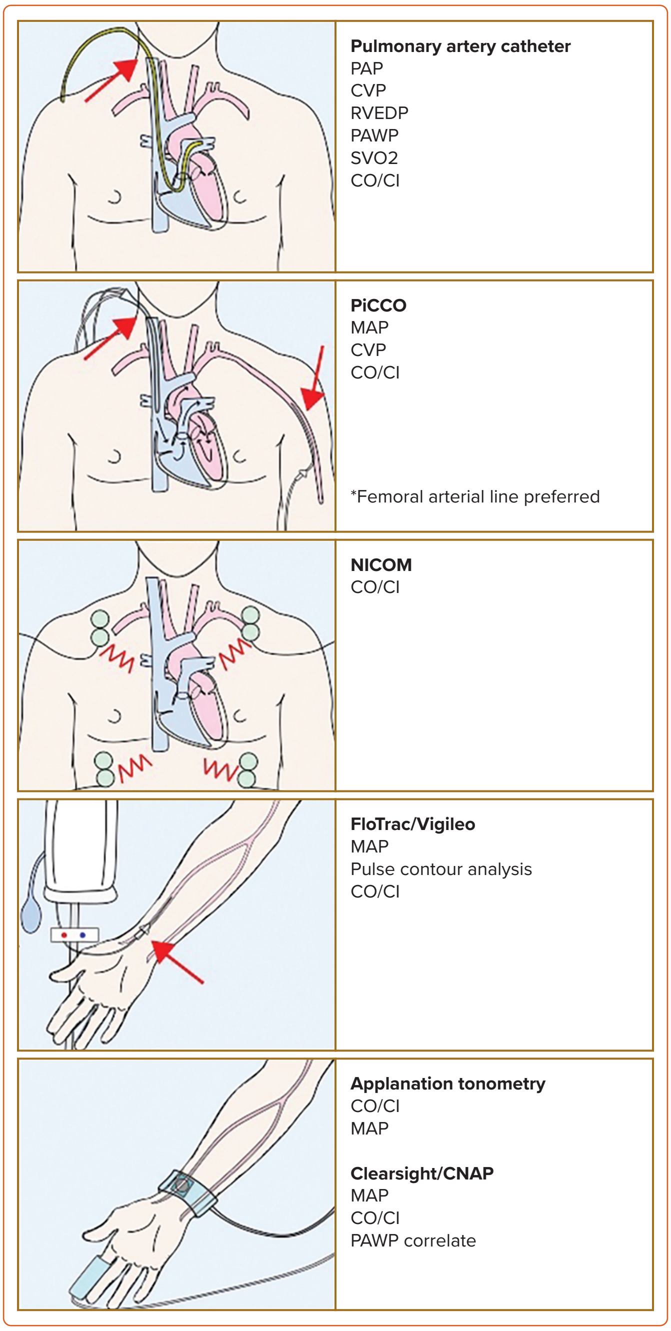
A myriad of invasive, minimally invasive, and noninvasive hemodynamic assessment modalities exists that supplement physical examination (*Figure 1 and Table 1*). These techniques rely on various physiological principles and assumptions to measure hemodynamic parameters. The aims of this review are to summarize the available literature on the mechanisms and clinical validity of various hemodynamic monitoring modalities as well as providing a contemporary update on pulmonary artery catheter usage. We have characterized each modality based on its level of invasiveness.

Physical Examination in Shock

The bedside physical examination is the oldest method of patient evaluation and can detect the presence of shock. Skin mottling, cool extremities, and delayed capillary refill have been correlated with mortality in patients with septic shock.[7,8] Similarly, physical examination findings help grade severity of CS. Created in 1967, the Killip Classification grades heart failure (HF) severity post-MI based on progression from the presence of an S3 gallop or isolated rales, to pulmonary edema, to overt CS.[9] The 2019 Society for Cardiovascular Angiography and Interventions (SCAI) Clinical Expert Consensus Statement on Classification of Cardiogenic Shock was developed based on physical examination in conjunction with biochemical and hemodynamic profiles. The SCAI physical exam can detect the worsening of CS on a continuum from isolated tachycardia and elevated jugular venous pressure; to cool extremities, pulmonary rales, oliguria, altered mentation, and narrow pulse pressure; to peri-arrest and arrest.[6,8]

While physical examination certainly has a part to play in the initial diagnosis of shock and the degree of shock severity, it may not be reliable

Figure 1: Hemodynamic Monitoring Devices and Associated Measured Indices



**Noninvasive venous waveform analysis is not shown. CO/CI = cardiac output/cardiac index; CVP = central venous pressure; PAP = pulmonary artery pressure; PAWP = pulmonary artery wedge pressure; MAP = mean arterial pressure; NICOM = noninvasive cardiac output monitor; RVEDP = right ventricular end diastolic pressure; SvO_2 = mixed venous oxygen saturation.*

in differentiating shock etiology. As early as 1984, Eisenberg et al. noted the pitfalls of physical diagnosis when they reported that physicians could only accurately estimate a pulmonary artery wedge pressure (PAWP) by physical diagnosis 30% of the time compared with pulmonary artery catheter (PAC) monitoring.[10] Similarly, a 1994 study by Mimoz et al. demonstrated 56% accuracy in predicting patient hemodynamic profiles by physical exam alone.[11] More recent observational studies in adults and children have had similar diagnostic inaccuracy.[12,13] Evidence to date suggests that while it is an important screening index for the presence and severity of shock, physical examination alone is not adequate to determine the cause of shock nor risk stratification.[14]

An Update on Pulmonary Artery Catheters

PAC has been used for the direct measurement of hemodynamic profiles for several decades. It provides direct measurement of intracardiac pressures as well as estimates of cardiac output (CO) and cardiac index. PAC allows for the estimation of CO by two techniques – indirect Fick and bolus thermodilution (Td). Each has its pitfalls but Td is preferred over indirect Fick even in low output and severe tricuspid regurgitation.[15–18] While measuring Td, it is critical that injections should be made in triplicate and all values within 10% of each other to account for beat-to-beat and manual injection variabilities. Furthermore, injection should occur at the same point of the respiratory cycle for consistent measurements.[19]

The original 1976 Forrester hemodynamic classification categorizes shock and CS treatment based on PAWP ('wet' versus 'dry') and cardiac index ('warm' versus 'cold') alone, thereby installing the PAC as a cornerstone of early CS management.[20] Once enshrined as a permanent fixture in the management of intensive care patients, the PAC then became a focus of intense debate in the early 2000s after studies noted an increased rate of complication without a clear reduction in mortality.[21–23]

The 2005 ESCAPE trial then sought to determine the safety and efficacy of routine consecutive day use of PACs in patients hospitalized for chronic decompensated HF, and ultimately found no mortality benefit.[24] It is worth noting that although the ESCAPE trial was a negative study, it was not focused on patients with general decompensated HF rather than the management of a CS population. The average systolic blood pressure in the study cohort was 106 mmHg and a very small percentage (<5%) would have met the clinical definition of CS. Although it did not meet its primary endpoint, ESCAPE's secondary functional endpoints consistently favored PAC-directed therapy, especially in exercise capability and quality of life.[24] However, subsequent meta-analyses of the use of PAC concluded a lack of mortality benefit with PAC placement and even a trend towards harm.[25,26] In addition to unclear mortality benefit, PACs have been criticized for invasiveness and increased use of resources when there are potential alternatives, such as less invasive hemodynamic diagnostic devices.[27]

PAC is a diagnostic tool, not a treatment modality. As any other diagnostic tool, it cannot improve mortality by its mere placement. However, appropriate interpretation of real-time PAC hemodynamic profiles can easily capture hemodynamic changes while delineating the relative contributions and severity of right ventricular (RV) versus left ventricular (LV) failure in CS. Nuanced interpretation can then guide appropriate treatment strategy including initiation of inotropes as well as escalation to MCS therapy. Garan et al.'s analysis from the Cardiogenic Shock Working Group cohort found that complete PAC hemodynamic profiling in CS was associated with lower in-hospital mortality across all SCAI classifications even when adjusted for CS etiology, presence of MCS, and local PAC usage trends (adjusted OR: 1.57; 95% CI [1.06–2.33]). This study also found that an incomplete PAC hemodynamic profile portended similar in-hospital mortality risk to having no PAC profiling at all, which they theorized may have been due to an underestimation of RV contribution to CS.[28] Accordingly, the most recent European Society of Cardiology guidelines classify clinical presentations of acute HF not only by Forrester-based CO and PAWP, but also by the presence of elevated RV end diastolic pressure.[29]

The ESCAPE trial showed potential benefits of PAC in high volume centers, perhaps a reflection of its usefulness among those who are more experienced with hemodynamic evaluation.[11] By allowing medical providers to make better informed decisions, PAC could ultimately

Table 1: Comparison of Minimally Invasive and Noninvasive Hemodynamic Monitor to Pulmonary Artery Catheter-measured Cardiac Output

Authors	Patient Population	Comparison Groups	n	Measured Outcome	Clinical Primary Outcome/Endpoint	Results	Clinical Significance
Squara 2007[60]	Post-cardiac surgery	NICOM versus PAC (Td)	110	CO (l/min)	None	• Mean PAC versus NICOM (R = 0.64, slope = 0.71 (95% CI [0.70–0.72]). Bias 0.06 ± 0.71 • Fluid challenge PAC lag time (7.1 ± 3.1 min for negative challenge, 6.8 ± 3.2 min for positive challenge) versus NICOM (3.4 ± 1.3 min and 4.0 ± 2.2 min respectively; p=0.01, 0.003)	NICOM estimated CO with acceptable accuracy and precision
Marque et al. 2013[54]	Septic shock	FloTrac (VCI) versus PAC (Td)	18	Cardiac index (l/min/m^2)	None	CC = 0.47 (p<0.01, r^2 = 0.22)	Poor correlation in septic shock
Rich et al.2013[61]	Pulmonary hypertension	NICOM versus CO (Fick and Td)	50	CO (l/min)	None	• Fick mean CO = 4.84 ± 1.39 versus Td 5.69 ± 1.74 (r=50.60, p,0.001) and NICOM 4.73 ± 1.15 (r=50.83, p=0.001) • NICOM versus PAC-Fick coefficient of variance = 3.5 ± 0.3% versus 9.6 ± 6.1%, p=0.001	NICOM is precise and reliable in measuring CO at rest and with vasodilator challenge
Wagner et al. 2015[72]	Post-cardiac surgery	AT versus PAC (Td)	50	CO (l/min)	None	• Td vs AT-CO = 4.7 ± 1.2 versus 4.9 ± 1.1; % error = 34% • AT-CO trend concordance 95%	Noninvasive assessment of changes in CO
Ganter et al. 2016[52]	Septic shock	FloTrac versus PAC (Td)	47	CO (l/min)	None	Bland and Altman analysis mean bias ± 2 SD of 0 ± 2.14 (% error = 34.5%)	Poor correlation in septic shock
Asamoto et al. 2017[53]	Off-pump CABG and liver transplant recipients	FloTrac versus LiDCO versus PAC (Td)	21	Cardiac index (l/min/m^2)	None	• FloTrac versus PAC: Bias = -0.44; % error = 74.4; R^2 = 0.48 • LiDCO versus PAC, Bias = -0.38; % error = 53.5; R^2 = 0.75	LiDCO better than FloTrac, but neither were within acceptable limits of error
Lamia et al. 2018[38]	Post-cardiac surgery Pertinent exclusions: LVEF <45%, arrhythmias, valvular dysfunction, and MCS	PAC (Td) versus FloTrac/NICOM/ LiDCOplus/PiCCOplus	21	CO (l/min)	None	• PAC = 5.7 ±1.5 versus LiDCO = 6.0 ± 1.9 (bias −0.10, r=0.83, p=0.0028) • FloTrac = 5.9 ± 1.0 (bias −0.40, r=0.73, p=0.0258) • PiCCO = 5.7 ± 1.8 (bias 0.18, r=0.85, p=0.0019) • NICOM = 5.3 ± 1.0 (bias −0.71, r=0.87, p=0.0011)	Dynamic changes in CO trended congruently across devices but wide range of inter-device bias
Lin et al. 2018[55]	Cardiac surgery with CPB	FloTrac versus PAC (CoTd)	32	CO (l/min)	None	• Pre-CPB FloTrac versus PAC: % error 61.82; bias = 0.16 (−2.15, 2.47), concordance 64.10%. • Post-CPB FloTrac versus PAC: % error 51.80; bias = 0.48, concordance 62.16%	Poor correlation between FloTrac and PAC
Wagner et al. 2018[78]	Post-cardiac surgery	CNAP versus PAC	51	CO (l/min)	None	• Calibrated-CNCO – PAC-CO, -0.3 (SD ± 0.5; -1.2 to +0.7; 19% error) • Uncalibrated-CNCO – PAC-CO +0.5 (SD +/- 1.3; 49% error)	CNAP requires frequent calibrations with PAC
Rali et al. 2020[62]	Cardiogenic shock	NICOM versus PAC (Fick and Td)	50	CO (l/min)	None	• NICOM-Fick r=0.132, CC 0.101 (0.008–0.191) p=0.033, bias 0.763 • NICOM-Td r=0.275, CC 0.133 (0.073–0.192), p<0.001, bias 0.484	NICOM correlates poorly to Fick or Td derived via PAC in CS
Alvis et al. 2021[88]	Ambulatory heart failure	NIVA versus PAC	84	PAWP (mmHg)	30-day hospital admission for heart failure exacerbation	NIVA score positively correlated with PAWP (r=0.92, n=106, p<0.0001) Discharge NIVA score predicted 30-day admission with an AUC of 0.84, a NIVA score >18 predicted admission with a sensitivity of 91% and specificity of 56%	NIVA can risk-stratify HF patients

AT = applanation tonometry; AUC = area under curve; CABG = coronary artery bypass graft; CC = correlation coefficient; CNAP = continuous noninvasive arterial pressure waveform; CNCO = continuous noninvasive cardiac output; CO = cardiac output; CoTd = continuous thermodilution; CPB = cardiopulmonary bypass; LVEF = left ventricular ejection fraction; MCS = mechanical circulatory support; NICOM = noninvasive cardiac output monitor; PAC = pulmonary artery catheter; NIVA = noninvasive venous waveform analysis; PAWP = pulmonary artery wedge pressure; Td = bolus thermodilution; VCI = vena cava inferior.

improve mortality in patients with CS. In an observational study by Ranka et al. analyzing the National Readmissions Database for patients admitted with acute CS (n=236,156), PAC-guided therapy was associated with a significant (31%) reduction in mortality during index hospitalization, a 17% reduction in 30-day HF readmissions rate and sixfold increase in usage of an LV assist device and orthotopic heart transplants during readmission.[30] Hernandez et al. also found that despite the recent decline in the use of PAC in patients with CS, treatment guided by PAC assessment resulted in lower mortality during index hospitalization (n=915,416; 35.1% versus 39.2%, OR 0.91, 95% CI [0.88–0.95]; p<0.001).[31] These, among other recent studies, have certainly revived discussion and debate about the need for appropriately interpreted PAC profiles as a powerful tool in CS management.[32,33] While these studies have advocated for the role of PAC hemodynamic assessment in CS, randomized controlled trials will help solidify it.

Minimally Invasive Hemodynamic Monitoring

Pulse Index Continuous Cardiac Output Monitoring

Transpulmonary thermodilution or lithium dilution devices, such as the pulse contour CO (PiCCO) monitoring system (Pulsion Medical Systems/ Getinge) and the LiDCO system (LiD-COplus, LiDCO), estimate CO by transthoracic thermodilution and lithium indicator dilution, respectively.[34,35] They are less invasive than PAC in that they do not transverse the heart, but they still require central access. PiCCO is performed by injecting a cold fluid bolus via a central venous catheter and measuring the resultant thermodilution via a thermistor-tipped femoral artery catheter.[27] The thermodilution curve (blood temperature versus time) translates to estimated CO by the Stewart–Hamilton equation. CO measured by PiCCO has been shown to be within acceptable agreement (r=0.97, p<0.0001) with PAC-based intermittent bolus thermodilution estimation of CO in critically ill patients.[36] Once calibrated with thermodilution, PiCCO algorithmically incorporates pulse contour analysis for continuous CO and stroke volume variation measurement, quantitative estimation of extravascular lung water (EVLW), and other calculated hemodynamic parameters. However, frequent recalibration is required.[37]

In a study of 20 patients admitted to the intesive care unit (ICU) after cardiac surgery with arterial line and PAC monitoring, cross-comparison of PAC derived CO was performed with PiCCO and LiDCO estimations; mean CO measurements were similar, though accuracy suffered during dynamic changes in CO.[34] A newer cross-comparison between PAC, PiCCO, and LiDCO devices demonstrated tight inter-device measurement of dynamic CO trends in post-cardiac surgery patients without significant cardiac dysfunction, arrhythmia, or valvular abnormalities (PAC-PiCCO r=0.85, p=0.0019; PAC-LiDCO r=0.83, p=0.0028), suggesting that prior inaccuracies may have been algorithmically corrected.[38] Among patients with CS, studies comparing PAC to PiCCO found adequate concordance with the cardiac index, including in patients with valvular abnormalities or arrhythmias.[39,40] PiCCO has also been shown to demonstrate concordance with transthoracic echocardiography in estimating cardiac output.[27,35]

Location of central venous catheter as well as presence of MCS devices can affect the accuracy of PiCCO measurements. Herner et al. described significantly lower estimations in cardiac functional index when catheters were placed in a femoral location instead of gold standard jugular or subclavian venous access, though later iterations of PiCCO monitoring algorithms have some provisions to correct for venous catheter location.[41] Thermodilutional-derived global ejection fraction has shown more accuracy to date than thermodilutional-derived cardiac functional index regardless of venous catheter location.[41,42] PiCCO accuracy can also be affected by MCS, such as intra-aortic balloon pump (IABP) counter-pulsation.[39] The device detects every augmentation during IABP support as a new systole, resulting in inaccurate estimation of heart rate. Literature regarding PiCCO monitoring with other forms of MCS such as ventricular assist devices or veno-arterial extracorporeal membrane oxygenation is sparse.

More recently, PiCCO monitoring has also been used with adjunct carotid tonometry in the measurement of effective arterial elastance (Ea), which is defined as the ratio between central end-systolic pressure and stroke volume. Ea has been proposed as an alternative to systemic vascular resistance when measuring LV afterload and measuring the ventricular-arterial decoupling that occurs in shock states.[6,43]

The PiCCO system can provide a qualitative estimate of EVLW and has been proposed as a tool in management and prognostication of acute lung injury, acute respiratory distress syndrome (ARDS), and cardiogenic pulmonary edema.[27,44] As the cold saline bolus is injected, the downslope of the thermodilution curve is used to estimate total pulmonary and thermal volumes, and EVLW is then estimated as the difference of intrathoracic blood volume and intrathoracic thermal volume.[27,45] Targeting EVLW in sepsis and ARDS management has not revealed benefit. A multicenter randomized controlled trial of 350 patients demonstrated no mortality benefit of EVLW versus central venous pressure (CVP)-guided fluid balance in septic shock or ARDS.[46] Indeed, little correlation is reported between EVLW estimates and shock subtype or ICU mortality.[47,48] Given the evolution of fluid balance assessment in recent years, larger-scale prospective studies will be critical in determining the utility of EVLW estimations with PiCCO monitoring in critically ill patients.

Similar to PiCCO, the LiDCO system provides CO measurements by lithium indicator dilution generating a curve of concentration over time. A lithium chloride indicator is injected in either a central or peripheral venous line, then arterial concentrations of the lithium are measured by serial blood draws through an arterial line sensor.[49] With three sequential dilution measurements, the coefficient of error in measurement of CO is as low as 5% in hemodynamically stable, ventilated intensive care patients.[50] Initial inaccuracies reported during dynamic CO shifts seem to have improved in later algorithms, though notably patients with severe cardiac dysfunction (LV ejection fraction [LVEF] <45%), MCS, valvular dysfunction, and arrhythmias were excluded.[34,38] It remains unclear to what extent these hemodynamic assessments affect clinical outcomes. Furthermore, an important caveat is that these systems only assess CO and do not provide the complete hemodynamic picture (including pulmonary artery pressure, PAWP, etc.) which is more valuable than any one parameter alone.[28]

Uncalibrated Pulse Contour Analysis

The FloTrac/Vigileo system (Edwards Lifesciences) uses pulse contour analysis derived from the arterial line to estimate stroke volume. When combined the patient's demographic data via the Vigileo monitor, it can also provide estimations of CO, cardiac index, and stroke volume variation with suboptimal accuracy.[35] This technique does not require calibration with PAC-measured CO but also does not provide estimates of intra-cardiac pressures such as CVP, PAP, or pulmonary capillary wedge pressure.[35,51]

FloTrac has been criticized for poor correlation with PAC measured CO, with a widely variable percentage error (up to 68%) across all generations of monitors and across several studies and settings (ICU, postoperative, septic shock patients).[51–56] While its utility in accurate estimation of cardiac index is limited, it may be useful in trending change in cardiac index as a mark of volume responsiveness.[57–59]

Noninvasive Hemodynamic Monitoring

Noninvasive Cardiac Output Monitoring

Noninvasive Cardiac Output Monitor (NICOM, Cheetah Medical) measures intrathoracic bioimpedance by alternating AC currents through thoracic pulsatile blood flow. It then indirectly calculates the stroke volume as the derivative of the change in the NICOM signal amplitude between systole and diastole. This measurement is dependent upon the diffusion of oscillating electric currents through the thoracic cavity.

Studies evaluating the validity of the use of NICOM when compared with PAC have yielded mixed results. Squara et al. assessed CO in post-cardiac surgery patients by both NICOM and thermodilution by PAC and demonstrated that NICOM was a reliable method of measuring CO in this cohort.[60] Rich et al. then demonstrated that NICOM was comparable to PAC in precision when assessing hemodynamics in patients with pulmonary artery hypertension.[61] However, NICOM has not been reliable in assessing hemodynamics in patients with CS and acute decompensated HF. In a cross-sectional prospective clinical trial, Rali et al. found that NICOM correlated poorly with indirect Fick and thermodilution measurements of CO in patients with CS.[62] It is plausible that these errors in measurement may be a result of interstitial and pulmonary edema and increased preload states in patients with chronic HF and low flow state in CS. The correlation did not improve with normalization of the cardiac index >2.2 l/min/m^2 or with the achievement of euvolemic status (CVP <5 mmHg or pulmonary artery systolic pressure <25 mmHg).[62]

Arterial Applanation Tonometry

Arterial applanation tonometry noninvasively estimates the aortic pressure waveform as a correlate of cardiac hemodynamics. It is performed by securing a pressure sensor (tonometer) over the wrist to partially flatten the radial artery and capture the arterial pulse. The resultant pulse waveform then undergoes a Fourier transformation algorithm to estimate a central aortic pressure waveform.[63] Since the arterial pressure waveform contour is primarily determined by the force and duration of ventricular ejection, aortic impedance, and peripheral vasculature resistance it can be calibrated to estimate hemodynamics including CO. The T-line® system (Tensys® Medical) is a well-known applanation tonometer that estimates hemodynamics by formulaically auto-calibrating a pulse contour analysis of radial tonometry based on demographic and biometric patient data.[35,64,65]

Arterial tonometry has demonstrated accuracy in measuring beat-to-beat blood pressure variation and mean arterial pressure (MAP) in anesthetized surgical patients and critically ill non-cardiac patients.[66–69] However, in critically ill patients with severe HF, arrhythmias, or valvular disorders, MAP estimations with arterial tonometry are less accurate than traditional arterial line monitoring, with a near 40% error reported.[70] Small proof-of-concept studies comparing CO estimations of arterial tonometry to PAC in critically ill patients found that appropriately positioned and calibrated arterial tonometers were able to estimate CO with a 23–34% margin of error.[71,72] However, a follow-up study comparing arterial tonometry to PAC measurements in patients undergoing major abdominal surgery had an error rate of 43%.[73] Applanation tonometry is strongly affected by vasoactive medications, obesity, and arrhythmias, and loses precision in large hemodynamic shifts or changes in vascular tone.[69,70]

Arterial tonometry is gaining traction for hemodynamic estimations in an ambulatory setting, with promising application in screening for hypertension, obstructive sleep apnea, coronary artery disease, and LV hypertrophy, among other pathologies.[63] It also has potential as an alternative to Doppler ultrasound to measure blood pressure in patients with an LV assist device and may be useful as a continuous wearable device.[74,75] However, further improvements are needed for it to have consistently accurate arterial blood pressure and CO estimates in critically ill patients.

Volume Clamp Method-derived Pulse Contour Analysis

ClearSight (Edwards Lifesciences) and Continuous Noninvasive Arterial Pressure Waveform (CNAP, CNSystems) noninvasive hemodynamic measuring systems estimate CO via photoplethysmography of the finger pressure arterial waveform. In these volume clamp method devices, an occlusive band around the finger regulates the external pressure needed to keep a continuous arterial blood volume in the finger throughout systole and diastole.[27] The resultant pulse contour analysis is then used to estimate CO and stroke volume variation.

In the surgical setting and in hemodynamically stable ICU patients, ClearSight and CNAP have demonstrated an approximate 25% margin of error when calibrated with thermodilution, and 25–45% error when auto-calibrated.[73,76–78] However, ClearSight and CNAP are not usually thermodilutionally calibrated in clinical practice, and larger ICU studies found much higher margins of error and standard deviations of measurement in auto-calibrated ClearSight measurements of undifferentiated shock patients.[79,80] ClearSight and CNAP are particularly affected by hemodynamic shifts that require recalibration, vasopressor use, arrhythmias, and peripheral arterial disorders, which may limit their broad application in accurate hemodynamic assessment of critically ill patients.[77,81]

More recent data demonstrate that ClearSight and CNAP may be useful to track fluid responsiveness. Boisson et al. found that thermodilutionally calibrated ClearSight versus PiCCO in the operating room accurately trended increase in CO after 250 ml fluid boluses.[77] In hemodynamically unstable patients, auto-calibrated ClearSight was able to trend increase in MAP and cardiac index over time with fluid resuscitation of patients in the emergency room or rapid response.[82,83] Similarly finger photoplethysmography has been used to measure pulse amplitude ratio, defined as the ratio of pulse pressure at the end of a Valsalva maneuver to before the onset of Valsalva, which can estimate PAWP in HF patients as well as help identify hospitalized HF patients at increased risk of 30-day HF events.[84,85]

Noninvasive Venous Waveform Analysis

The high capacitance, low compliance venous system has not been widely studied in noninvasive hemodynamic monitors to date due to limitations in collecting and measuring low-frequency venous signals. Noninvasive venous waveform analysis (NIVA) has recently been used to measure venous distension, and thereby estimate volume status and PAWP.

NIVA technology uses piezoelectric sensing over the superficial wrist veins to detect and amplify venous signaling, then applies a Fourier transformation and algorithm to the signal to estimate PAWP.[86] An initial study comparing NIVA estimates of PAWP to right heart catheterization measurements demonstrated a sensitivity of 80% and specificity of 53% in detecting a PAWP of >18 mmHg.[87] With further refinement, NIVA technology may be used as an adjunct or alternative to implantable PAP monitoring systems such as CardioMEMS, which have in turn shown to be useful in ameliorating HF exacerbations and hospitalizations.[87–89] NIVA has also been proposed as a method to direct volume removal during hemodialysis.[86] However, inpatient application of NIVA is yet to be evaluated.

Transthoracic Echocardiography

Critical care echocardiography (CCE) has gained significant popularity with increased availability of mobile echocardiography machines and training opportunities.[90] The noninvasive nature of CCE is especially appealing and echocardiography has long been validated as a reliable measure of hemodynamics.[91,92]

While the full scope of CCE application exceeds the limits of this review, it is worth noting that CCE can estimate all advanced hemodynamics with relative accuracy and tracking aortic velocity time index (VTI) is a reliable means to track change in CO over time or in response to fluid administration.[93,94] CCE also adds vital information about cardiac structural details such as regional wall motion abnormality; valvular pathology; and diastolic dysfunction.[93] Echocardiographic findings help with appropriate interpretation of hemodynamic data, such as tricuspid regurgitation affecting interpretation of CVP. Jentzer et al. recently discovered that LVEF at admission measured by formal transthoracic echocardiography in acute HF correlated to SCAI shock stages ($p<0.001$ across all stages) and independently predicted mortality based on LVEF and E/e' ratio.[95] This study prompted renewed discussion about the need for invasive hemodynamic monitoring if echocardiographic-derived hemodynamic measurements not only provide the above-mentioned benefits, but also demonstrate strong correlation to shock stage.[96] However, echocardiography only provides a single snapshot into the hemodynamic profile of a patient which, while extremely valuable, may change rapidly in the intensive care setting. In complex cardiac patients, CCE and continuous invasive hemodynamic monitoring such as PAC may then serve the most value when used to both detect and diagnose shock evolution.

Appropriate training and competency among non-ultrasonographers remain the most significant limitation in widespread CCE usage. While there are CCE training programs provided by several professional organizations, there is no current formal consensus on number of training hours or exams needed to ensure competency.[97] In response to this, the American Society of Echocardiography has recently developed a Critical Care Echocardiography board certification to attempt standardization of CCE skills.[98]

Conclusion

Several minimally invasive and noninvasive modalities exist to assess hemodynamic parameters. Most of these modalities still require optimization and validation for widespread usage. In the interim, comprehensive invasive hemodynamic profiling of patients in shock with echocardiography, and in select cases, PAC – which overall does not appear to improve clinical outcomes – remains pivotal in ensuring timely diagnosis and optimal treatment, especially in the increasingly complex patient population of the modern day CICU. ❑

1. Katz JN, Shah BR, Volz EM, et al. Evolution of the coronary care unit: clinical characteristics and temporal trends in healthcare delivery and outcomes. *Crit Care Med* 2010;38:375–81. https://doi.org/10.1097/CCM.0b013e3181cb0a63; PMID: 20029344.
2. Sinha SS, Sjoding MW, Sukul D, et al. Changes in primary noncardiac diagnoses over time among elderly cardiac intensive care unit patients in the United States. *Circ Cardiovasc Qual Outcomes* 2017;10:e003616. https://doi.org/10.1161/CIRCOUTCOMES.117.003616; PMID: 28794121.
3. Miller PE, Thomas A, Breen TJ, et al. Prevalence of noncardiac multimorbidity in patients admitted to two cardiac intensive care units and their association with mortality. *Am J Med* 2021;134:653–61.e5. https://doi.org/10.1016/j.amjmed.2020.09.035; PMID: 33129785.
4. Bohula EA, Katz JN, van Diepen S, et al. Demographics, care patterns, and outcomes of patients admitted to cardiac intensive care units: the Critical Care Cardiology Trials Network prospective North American multicenter registry of cardiac critical illness. *JAMA Cardiol* 2019;4:928–35. https://doi.org/10.1001/jamacardio.2019.2467; PMID: 31339509.
5. Berg DD, Bohula EA, van Diepen S, et al. Epidemiology of shock in contemporary cardiac intensive care units. *Circ Cardiovasc Qual Outcomes* 2019;12:e005618. https://doi.org/10.1161/CIRCOUTCOMES.119.005618; PMID: 30879324.
6. Hsu S, Fang JC, Borlaug BA. Hemodynamics for the heart failure clinician: a state-of-the-art review. *J Card Fail* 2022;28:133–48. https://doi.org/10.1016/j.cardfail.2021.07.012; PMID: 34389460.
7. Cecconi M, Hernandez G, Dunser M, et al. Fluid administration for acute circulatory dysfunction using basic monitoring: narrative review and expert panel recommendations from an ESICM task force. *Intensive Care Med* 2019;45:21–32. https://doi.org/10.1007/s00134-018-5415-2; PMID: 30456467.
8. De Backer D, Bakker J, Cecconi M, et al. Alternatives to the Swan-Ganz catheter. *Intensive Care Med* 2018;44:730–41. https://doi.org/10.1007/s00134-018-5187-8; PMID: 29725695.
9. Mello BH, Oliveira GB, Ramos RF, et al. Validation of the Killip-Kimball classification and late mortality after acute myocardial infarction. *Arq Bras Cardiol* 2014;103:107–17. https://doi.org/10.5935/abc.20140091; PMID: 25014060.
10. Eisenberg PR, Jaffe AS, Schuster DP. Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients. *Crit Care Med* 1984;12:549–53. https://doi.org/10.1097/00003246-198407000-00001; PMID: 6734221.
11. Mimoz O, Rauss A, Rekik N, et al. Pulmonary artery catheterization in critically ill patients: a prospective analysis of outcome changes associated with catheter-prompted changes in therapy. *Crit Care Med* 1994;22:573–9. https://doi.org/10.1097/00003246-199404000-00011; PMID: 8143466.
12. Nowak RM, Sen A, Garcia AJ, et al. The inability of emergency physicians to adequately clinically estimate the underlying hemodynamic profiles of acutely ill patients. *Am J Emerg Med* 2012;30:954–60. https://doi.org/10.1016/j.ajem.2011.05.021; PMID: 21802880.
13. Razavi A, Newth CJL, Khemani RG, et al. Cardiac output and systemic vascular resistance: clinical assessment compared with a noninvasive objective measurement in children with shock. *J Crit Care* 2017;39:6–10. https://doi.org/10.1016/j.jcrc.2016.12.018; PMID: 28088009.
14. Drazner MH, Hellkamp AS, Leier CV, et al. Value of clinician assessment of hemodynamics in advanced heart failure: the ESCAPE trial. *Circ Heart Fail* 2008;1:170–7. https://doi.org/10.1161/CIRCHEARTFAILURE.108.769778; PMID: 19675681.
15. Narang N, Thibodeau JT, Levine BD, et al. Inaccuracy of estimated resting oxygen uptake in the clinical setting. *Circulation* 2014;129:203–10. https://doi.org/10.1161/CIRCULATIONAHA.113.003334; PMID: 24077170.
16. Hoeper NM, Maier R, Tongers J, et al. Determination of cardiac output by the Fick method, thermodilution, and acetylene rebreathing in pulmonary hypertension. *Am J Respir Crit Care Med* 1998;160:535–41. https://doi.org/10.1164/ajrccm.160.2.9811062; PMID: 10430725.
17. Opotowsky AR, Hess E, Maron BA, et al. Thermodilution vs estimated Fick cardiac output measurement in clinical practice: an analysis of mortality from the Veterans Affairs Clinical Assessment, Reporting, and Tracking (VA CART) program and Vanderbilt University. *JAMA Cardiol* 2017;2:1090–9. https://doi.org/10.1001/jamacardio.2017.2945; PMID: 28877293.
18. Hoeper MM, Bogaard HJ, Condliffe R, et al. Definitions and diagnosis of pulmonary hypertension. *J Am Coll Cardiol* 2013;62(25 Suppl):D42–50. https://doi.org/10.1016/j.jacc.2013.10.032; PMID: 24355641.
19. Stevens JH, Raffin TA, Mihm FG, et al. Thermodilution cardiac output measurement effect of the respiratory cycle on its reproducibility. *JAMA* 1985;253:2240–2. https://doi.org/10.1001/jama.253.15.2240; PMID: 3974116.
20. Forrester JS, Diamond G, Chatterjee K, Swan HJ. Medical therapy of acute myocardial infarction by application of hemodynamic subsets. *N Engl J Med* 1976;295:1404–13. https://doi.org/10.1056/NEJM197612162952505; PMID: 790194.
21. 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 controlled trial. *JAMA* 2003;290:2713–20. https://doi.org/10.1001/jama.290.20.2713; PMID: 14645314.
22. 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;348:5–14. https://doi.org/10.1056/NEJMoa021108; PMID: 12510037.
23. Reade MC, Angus DC. Pac-Man: game over for the pulmonary artery catheter? *Crit Care* 2006;10:303. https://doi.org/10.1186/cc3977; PMID: 16420664.
24. Binanay C, Califf RM, Hasselblad V, et al. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial. *JAMA* 2005;294:1625–33. https://doi.org/10.1001/jama.294.13.1625; PMID: 16204662.
25. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. *JAMA* 2005;294:1664–70. https://doi.org/10.1001/jama.294.13.1664; PMID: 16204666.
26. Rajaram SS, Desai NK, Kalra A, et al. Pulmonary artery catheters for adult patients in intensive care. *Cochrane Database Syst Rev* 2013;2:CD003408. https://doi.org/10.1002/14651858.CD003408.pub3; PMID: 23450539.
27. Teboul JL, Saugel B, Cecconi M, et al. Less invasive hemodynamic monitoring in critically ill patients. *Intensive Care Med* 2016;42:1350–9. https://doi.org/10.1007/s00134-016-4375-7; PMID: 27155605.
28. Garan AR, Kanwar M, Thayer KL, et al. Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower in-hospital mortality. *JACC Heart Fail* 2020;8:903–13. https://doi.org/10.1016/j.jchf.2020.08.012; PMID: 33121702.
29. McDonagh TA, Metra M, Adamo M, et. al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J* 2021;42:3599–726. https://doi.org/10.1093/eurheartj/ehab368; PMID: 34447992.
30. Ranka S, Mastoris I, Kapur NK, et al. Right heart catheterization in cardiogenic shock is associated with improved outcomes: insights from the nationwide readmissions database. *J Am Heart Assoc* 2021;10:e019843. https://doi.org/10.1161/JAHA.120.019843; PMID: 34423652.
31. Hernandez GA, Lemor A, Blumer V, et al. Trends in utilization and outcomes of pulmonary artery catheterization in heart

failure with and without cardiogenic shock. *J Card Fail* 2019;25:364–71. https://doi.org/10.1016/j.cardfail.2019.03.004; PMID: 30858119.
32. Sionis A, Rivas-Lasarte M, Mebazaa A, et al. Current use and impact on 30-day mortality of pulmonary artery catheter in cardiogenic shock patients: results from the CardShock study. *J Intensive Care Med* 2020;35:1426–33. https://doi.org/10.1177/0885066619828959; PMID: 30732522.
33. Osman M, Syed M, Patel B, et al. Invasive hemodynamic monitoring in cardiogenic shock is associated with lower in-hospital mortality. *J Am Heart Assoc* 2021;10:e021808. https://doi.org/10.1161/JAHA.121.021808; PMID: 34514850.
34. Hadian M, Kim HK, Severyn DA, Pinsky MR. Cross-comparison of cardiac output trending accuracy of LiDCO, PiCCO, FloTrac and pulmonary artery catheters. *Crit Care* 2010;14:R212. https://doi.org/10.1186/cc9335; PMID: 21092290.
35. Pour-Ghaz I, Manolukas T, Foray N, et al. Accuracy of non-invasive and minimally invasive hemodynamic monitoring: where do we stand? *Ann Transl Med* 2019;7:421. https://doi.org/10.21037/atm.2019.07.06; PMID: 31660320.
36. Sakka SG, Reinhart K, Meier-Hellmann A. Comparison of pulmonary artery and arterial thermodilution cardiac output in critically ill patients. *Intensive Care Med* 1999;25:843–6. https://doi.org/10.1007/s001340050962; PMID: 10447543.
37. Hamzaoui O, Monnet X, Richard C, et al. Effects of changes in vascular tone on the agreement between pulse contour and transpulmonary thermodilution cardiac output measurements within an up to 6-hour calibration-free period. *Crit Care Med* 2008;36:434–40. https://doi.org/10.1097/01.CCM.OB013E318161FEC4; PMID: 18091547.
38. Lamia B, Kim HK, Severyn DA, Pinsky MR. Cross-comparisons of trending accuracies of continuous cardiac-output measurements: pulse contour analysis, bioreactance, and pulmonary-artery catheter. *J Clin Monit Comput* 2018;32:33–43. https://doi.org/10.1007/s10877-017-9983-4; PMID: 28188408.
39. Schmid B, Fink K, Olschewski M, et al. Accuracy and precision of transcardiopulmonary thermodilution in patients with cardiogenic shock. *J Clin Monit Comput* 2016;30:849–56. https://doi.org/10.1007/s10877-015-9782-8; PMID: 26429134.
40. Hilty MP, Franzen DP, Wyss C, et al. Validation of transpulmonary thermodilution variables in hemodynamically stable patients with heart diseases. *Ann Intensive Care* 2017;7:86. https://doi.org/10.1186/s13613-017-0307-0; PMID: 28831765.
41. Herner A, Heilmaier M, Mayr U, et al. Comparison of cardiac function index derived from femoral and jugular indicator injection for transpulmonary thermodilution with the PiCCO-device: a prospective observational study. *PLOS ONE* 2018;13:e0200740. https://doi.org/10.1371/journal.pone.0200740; PMID: 30063736.
42. Huber W, Gruber A, Eckmann M, et al. Comparison of pulmonary vascular permeability index PVPI and global ejection fraction GEF derived from jugular and femoral indicator injection using the PiCCO-2 device: a prospective observational study. *PLOS ONE* 2017;12:e0178372. https://doi.org/10.1371/journal.pone.0178372; PMID: 29040264.
43. Jozwiak M, Millasseau S, Richard C, et al. Validation and critical evaluation of the effective arterial elastance in critically ill patients. *Crit Care Med* 2019;47:e317–24. https://doi.org/10.1097/CCM.0000000000003645; PMID: 30664009.
44. Wang H, Cui N, Su L, et al. Prognostic value of extravascular lung water and its potential role in guiding fluid therapy in septic shock after initial resuscitation. *J Crit Care* 2016;33:106–13. https://doi.org/10.1016/j.jcrc.2016.02.011; PMID: 27021852.
45. Jozwiak M, Teboul JL, Monnet X. Extravascular lung water in critical care: recent advances and clinical applications. *Ann Intensive Care* 2015;5:38. https://doi.org/10.1186/s13613-015-0081-9; PMID: 26546321.
46. Zhang Z, Ni H, Qian Z. Effectiveness of treatment based on PiCCO parameters in critically ill patients with septic shock and/or acute respiratory distress syndrome: a randomized controlled trial. *Intensive Care Med* 2015;41:444–51. https://doi.org/10.1007/s00134-014-3638-4; PMID: 25605469.
47. Tagami T, Ong MEH. Extravascular lung water measurements in acute respiratory distress syndrome: why, how, and when? *Curr Opin Crit Care* 2018;24:209–15. https://doi.org/10.1097/MCC.0000000000000503; PMID: 29608455.
48. Werner M, Wernly B, Lichtenauer M, et al. Real-world extravascular lung water index measurements in critically ill patients: pulse index continuous cardiac output measurements: time course analysis and association with clinical characteristics. *Wien Klin Wochenschr* 2019;131:321–8. https://doi.org/10.1007/s00508-019-1501-x; PMID: 31069475.
49. Pearse RM, Ikram K, Barry J. Equipment review: an appraisal of the LiDCO plus method of measuring cardiac output. *Crit Care* 2004;8:190–5. https://doi.org/10.1186/cc2852; PMID: 15153237.
50. Cecconi M, Dawson D, Grounds RM, Rhodes A. Lithium dilution cardiac output measurement in the critically ill patient: determination of precision of the technique. *Intensive Care Med* 2009;35:498–504. https://doi.org/10.1007/s00134-008-1292-4; PMID: 18802681.
51. Mayer J, Boldt J, Poland R, et al. Continuous arterial pressure waveform-based cardiac output using the FloTrac/Vigileo: a review and meta-analysis. *J Cardiothorac Vasc Anesth* 2009;23:401–6. https://doi.org/10.1053/j.jvca.2009.03.003; PMID: 19464625.
52. Ganter MT, Alhashemi JA, Al-Shabasy AM, et al. Continuous cardiac output measurement by un-calibrated pulse wave analysis and pulmonary artery catheter in patients with septic shock. *J Clin Monit Comput* 2016;30:13–22. https://doi.org/10.1007/s10877-015-9672-0; PMID: 25721853.
53. Asamoto M, Orii R, Otsuji M, et al. Reliability of cardiac output measurements using LiDCOrapid and FloTrac/Vigileo across broad ranges of cardiac output values. *J Clin Monit Comput* 2017;31:709–16. https://doi.org/10.1007/s10877-016-9896-7; PMID: 27300325.
54. Marque S, Gros A, Chimot L, et al. Cardiac output monitoring in septic shock: evaluation of the third-generation FloTrac-Vigileo. *J Clin Monit Comput* 2013;27:273–9. https://doi.org/10.1007/s10877-013-9431-z; PMID: 23361128.
55. Lin SY, Chou AH, Tsai YF, et al. Evaluation of the use of the fourth version FloTrac system in cardiac output measurement before and after cardiopulmonary bypass. *J Clin Monit Comput* 2018;32:807–15. https://doi.org/10.1007/s10877-017-0071-6; PMID: 29039063.
56. Kaufmann T, Clement RP, Hiemstra B, et al. Disagreement in cardiac output measurements between fourth-generation FloTrac and critical care ultrasonography in patients with circulatory shock: a prospective observational study. *J Intensive Care* 2019;7:21. https://doi.org/10.1186/s40560-019-0373-5; PMID: 31011425.
57. Krige A, Bland M, Fanshawe T. Fluid responsiveness prediction using Vigileo FloTrac measured cardiac output changes during passive leg raise test. *J Intensive Care* 2016;4:63. https://doi.org/10.1186/s40560-016-0188-6; PMID: 27721980.
58. Khwannimit B, Bhurayanontachai R. Prediction of fluid responsiveness in septic shock patients: comparing stroke volume variation by FloTrac/Vigileo and automated pulse pressure variation. *Eur J Anaesthesiol* 2012;29:64–9. https://doi.org/10.1097/EJA.0b013e32834b7d82; PMID: 21946822.
59. Khwannimit B, Jomsuriya R. Comparison the accuracy and trending ability of cardiac index measured by the fourth-generation of FloTrac with the PiCCO device in septic shock patients. *Turk J Med Sci* 2020;50:860–9. https://doi.org/10.3906/sag-1909-58; PMID: 32336075.
60. Squara P, Denjean D, Estagnasie P, et al. Noninvasive cardiac output monitoring (NICOM): a clinical validation. *Intensive Care Med* 2007;33:1191–4. https://doi.org/10.1007/s00134-007-0640-0; PMID: 17458538.
61. Rich JD, Archer SL, Rich S. Noninvasive cardiac output measurements in patients with pulmonary hypertension. *Eur Respir J* 2013;42:125–33. https://doi.org/10.1183/09031936.00102212; PMID: 23100501.
62. Rali AS, Buechler T, Van Gotten B, et al. Non-invasive cardiac output monitoring in cardiogenic shock: the NICOM study. *J Card Fail* 2020;26:160–5. https://doi.org/10.1016/j.cardfail.2019.11.015; PMID: 31751786.
63. Nelson MR, Stepanek J, Cevette M, et al. Noninvasive measurement of central vascular pressures with arterial tonometry: clinical revival of the pulse pressure waveform? *Mayo Clin Proc* 2010;85:460–72. https://doi.org/10.4065/mcp.2009.0336; PMID: 20435839.
64. Wagner JY, Langemann M, Schön G, et al. Autocalibrating pulse contour analysis based on radial artery applanation tonometry for continuous non-invasive cardiac output monitoring in intensive care unit patients after major gastrointestinal surgery – a prospective method comparison study. *Anaesth Intensive Care* 2016;44:340–5. https://doi.org/10.1177/0310057X1604400307; PMID: 27246932.
65. Saugel B, Flick M, Bendjelid K, et al. Journal of clinical monitoring and computing end of year summary 2018: hemodynamic monitoring and management. *J Clin Monit Comput* 2019;33:211–22. https://doi.org/10.1007/s10877-019-00297-w; PMID: 30847738.
66. Dueck R, Goedje O, Clopton P. Noninvasive continuous beat-to-beat radial artery pressure via TL-200 applanation tonometry. *J Clin Monit Comput* 2012;26:75–83. https://doi.org/10.1007/s10877-012-9336-2; PMID: 22258303.
67. Meidert AS, Huber W, Hapfelmeier A, et al. Evaluation of the radial artery applanation tonometry technology for continuous noninvasive blood pressure monitoring compared with central aortic blood pressure measurements in patients with multiple organ dysfunction syndrome. *J Crit Care* 2013;28:908–12. https://doi.org/10.1016/j.jcrc.2013.06.012; PMID: 23910893.
68. Meidert AS, Huber W, Müller JN, et al. Radial artery applanation tonometry for continuous non-invasive arterial pressure monitoring in intensive care unit patients: comparison with invasively assessed radial arterial pressure. *Br J Anaesth* 2014;112:521–8. https://doi.org/10.1093/bja/aet400; PMID: 24355832.
69. Langwieser N, Prechtl L, Meidert AS, et al. Radial artery applanation tonometry for continuous noninvasive arterial blood pressure monitoring in the cardiac intensive care unit. *Clin Res Cardiol* 2015;104:518–24. https://doi.org/10.1007/s00392-015-0816-5; PMID: 25618259.
70. Greiwe G, Hoffmann S, Herich L, et al. Comparison of blood pressure monitoring by applanation tonometry and invasively assessed blood pressure in cardiological patients. *J Clin Monit Comput* 2018;32:817–23. https://doi.org/10.1007/s10877-017-0089-9; PMID: 29204771.
71. Saugel B, Meidert AS, Langwieser N, et al. An autocalibrating algorithm for non-invasive cardiac output determination based on the analysis of an arterial pressure waveform recorded with radial artery applanation tonometry: a proof of concept pilot analysis. *J Clin Monit Comput* 2014;28:357–62. https://doi.org/10.1007/s10877-013-9540-8; PMID: 24322474.
72. Wagner JY, Sarwari H, Schon G, et al. Radial artery applanation tonometry for continuous noninvasive cardiac output measurement: a comparison with intermittent pulmonary artery thermodilution in patients after cardiothoracic surgery. *Crit Care Med* 2015;43:1423–8. https://doi.org/10.1097/CCM.0000000000000979; PMID: 25844700.
73. Wagner JY, Grond J, Fortin J, et al. Continuous noninvasive cardiac output determination using the CNAP system: evaluation of a cardiac output algorithm for the analysis of volume clamp method-derived pulse contour. *J Clin Monit Comput* 2016;30:487–93. https://doi.org/10.1007/s10877-015-9744-1; PMID: 26227161.
74. Zayat R, Drosos V, Schnoering H, et al. Radial artery tonometry to monitor blood pressure and hemodynamics in ambulatory left ventricular assist device patients in comparison with Doppler ultrasound and transthoracic echocardiography: a pilot study. *Artif Organs* 2019;43:242–53. https://doi.org/10.1111/aor.13335; PMID: 30040134.
75. Digiglio P, Li R, Wang W, Pan T. Microflotronic arterial tonometry for continuous wearable non-invasive hemodynamic monitoring. *Ann Biomed Eng* 2014;42:2278–88. https://doi.org/10.1007/s10439-014-1037-1; PMID: 24889715.
76. Frank P, Logemann F, Gras C, Palmaers T. Noninvasive continuous arterial pressure monitoring during anesthesia induction in patients undergoing cardiac surgery. *Ann Card Anaesth* 2021;24:281–7. https://doi.org/10.4103/aca.ACA_120_20; PMID: 34269255.
77. Boisson M, Poignard ME, Pontier B, et al. Cardiac output monitoring with thermodilution pulse-contour analysis vs. non-invasive pulse-contour analysis. *Anaesthesia* 2019;74:735–40. https://doi.org/10.1111/anae.14638; PMID: 30888055.
78. Wagner JY, Korner A, Schulte-Uentrop L, et al. A comparison of volume clamp method-based continuous noninvasive cardiac output (CNCO) measurement versus intermittent pulmonary artery thermodilution in postoperative cardiothoracic surgery patients. *J Clin Monit Comput* 2018;32:235–44. https://doi.org/10.1007/s10877-017-0027-x; PMID: 28540614.
79. Kim SH, Lilot M, Sidhu KS, et al. Accuracy and precision of continuous noninvasive arterial pressure monitoring compared with invasive arterial pressure: a systematic review and meta-analysis. *Anesthesiology* 2014;120:1080–97. https://doi.org/10.1097/ALN.0000000000000226; PMID: 24637618.
80. Eyeington CT, Lloyd-Donald P, Chan MJ, et al. Rapid response team review of hemodynamically unstable ward patients: the accuracy of cardiac index assessment. *J Crit Care* 2019;49:187–92. https://doi.org/10.1016/j.jcrc.2018.09.002; PMID: 30482613.
81. Meidert AS, Saugel B. Techniques for non-invasive monitoring of arterial blood pressure. *Front Med (Lausanne)* 2017;4:231. https://doi.org/10.3389/fmed.2017.00231; PMID: 29359130.
82. Eyeington CT, Lloyd-Donald P, Chan MJ, et al. Non-invasive continuous haemodynamic monitoring and response to intervention in haemodynamically unstable patients during rapid response team review. *Resuscitation* 2019;143:124–33. https://doi.org/10.1016/j.resuscitation.2019.08.025; PMID: 31446156.
83. Koopmans NK, Stolmeijer R, Sijtsma BC, et al. Non-invasive assessment of fluid responsiveness to guide fluid therapy in patients with sepsis in the emergency department: a

prospective cohort study. *Emerg Med J* 2021;38:416–22. https://doi.org/10.1136/emermed-2020-209771; PMID: 33888514.
84. Gilotra NA, Tedford RJ, Wittstein IS, et al. Usefulness of pulse amplitude changes during the Valsalva maneuver measured using finger photoplethysmography to identify elevated pulmonary capillary wedge pressure in patients with heart failure. *Am J Cardiol* 2017;120:966–72. https://doi.org/10.1016/j.amjcard.2017.06.029; PMID: 28754567.
85. Gilotra NA, Wanamaker BL, Rahim H, et al. Usefulness of noninvasively measured pulse amplitude changes during the Valsalva maneuver to identify hospitalized heart failure patients at risk of 30-day heart failure events (from the PRESSURE-HF Study). *Am J Cardiol* 2020;125:916–23. https://doi.org/10.1016/j.amjcard.2019.12.027; PMID: 31928720.
86. Alvis BD, Polcz M, Miles M, et al. Non-invasive venous waveform analysis (NIVA) for volume assessment in patients undergoing hemodialysis: an observational study. *BMC Nephrol* 2020;21:194. https://doi.org/10.1186/s12882-020-01845-2; PMID: 32448178.
87. Alvis BD, Polcz M, Huston JH, et al. Observational study of noninvasive venous waveform analysis to assess intracardiac filling pressures during right heart catheterization. *J Card Fail* 2020;26:136–41. https://doi.org/10.1016/j.cardfail.2019.09.009; PMID: 31574315.
88. Alvis B, Huston J, Schmeckpeper J, et al. Non-invasive venous waveform analysis (NIVA) correlates with pulmonary capillary wedge pressure (PCWP) and predicts 30-day admission in heart failure patients undergoing right heart catheterization: NIVA scores correlate with PCWP and predicts 30-day admission. *J Card Fail* 2021. https://doi.org/10.1016/j.cardfail.2021.09.009; PMID: 34555524; epub ahead of press.
89. Adamson PB, Abraham WT, Aaron M, et al. CHAMPION trial rationale and design: the long-term safety and clinical efficacy of a wireless pulmonary artery pressure monitoring system. *J Card Fail* 2011;17:3–10. https://doi.org/10.1016/j.cardfail.2010.08.002; PMID: 21187258.
90. Vincent JL, Joosten A, Saugel B. Hemodynamic monitoring and support. *Crit Care Med* 2021;49:1638–50. https://doi.org/10.1097/CCM.0000000000005213; PMID: 34269718.
91. Papolos A, Narula J, Bavishi C, et al. US Hospital use of echocardiography: insights from the Nationwide Inpatient Sample. *J Am Coll Cardiol* 2016;67:502–11. https://doi.org/10.1016/j.jacc.2015.10.090; PMID: 26846948.
92. Mercado P, Maizel J, Beyls C, et al. Reassessment of the accuracy of cardiac Doppler pulmonary artery pressure measurements in ventilated ICU patients: a simultaneous Doppler-catheterization study. *Crit Care Med* 2019;47:41–8. https://doi.org/10.1097/CCM.0000000000003422; PMID: 30379666.
93. Vieillard-Baron A, Millington SJ, Sanfilippo F, et al. A decade of progress in critical care echocardiography: a narrative review. *Intensive Care Med* 2019;45:770–88. https://doi.org/10.1007/s00134-019-05604-2; PMID: 30911808.
94. Zhang Y, Wang Y, Shi J, et al. Cardiac output measurements via echocardiography versus thermodilution: a systematic review and meta-analysis. *PLOS ONE* 2019;14:e0222105. https://doi.org/10.1371/journal.pone.0222105; PMID: 31581196.
95. Jentzer JC, Wiley BM, Anavekar NS, et al. Noninvasive hemodynamic assessment of shock severity and mortality risk prediction in the cardiac intensive care unit. *JACC Cardiovasc Imaging* 2021;14:321–32. https://doi.org/10.1016/j.jcmg.2020.05.038; PMID: 32828777.
96. Fortuni F, Tavazzi G, De Ferrari GM. Pulmonary artery catheter in cardiogenic shock: will the benefits finally outweigh the costs and complications? *JACC Heart Fail* 2021;9:322–3. https://doi.org/10.1016/j.jchf.2020.12.007; PMID: 33795123.
97. Kovell LC, Ali MT, Hays AG, et al. Defining the role of point-of-care ultrasound in cardiovascular disease. *Am J Cardiol* 2018;122:1443–50. https://doi.org/10.1016/j.amjcard.2018.06.054; PMID: 30115421.
98. Kirkpatrick JN, Grimm R, Johri AM, et al. Recommendations for echocardiography laboratories participating in cardiac point of care cardiac ultrasound (POCUS) and critical care echocardiography training: report from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2020;33:409–22.e4. https://doi.org/10.1016/j.echo.2020.01.008; PMID: 32122742.