American Journal of Emergency Medicine 35 (2017) 1335–1347

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Review

A review of hemodynamic monitoring techniques, methods and devices for the emergency physician

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ARTICLE INFO

Article history:
Received 18 October 2016
Received in revised form 15 March 2017
Accepted 16 March 2017

Keywords:
Hemodynamic monitoring
Emergency department
Fluid responsiveness
Shock
Point of care ultrasound

ABSTRACT

The emergency department (ED) is frequently the doorway to the intensive care unit (ICU) for a significant number of critically ill patients presenting to the hospital. Hemodynamic monitoring (HDM) which is a key component in the effective management of the critically ill patient presenting to the ED, is primarily concerned with assessing the performance of the cardiovascular system and determining the correct therapeutic intervention to optimise end-organ oxygen delivery. The spectrum of hemodynamic monitoring ranges from simple clinical assessment and routine bedside monitoring to point of care ultrasonography and various invasive monitoring devices. The clinician must be aware of the range of available techniques, methods, interventions and technological advances as well as possess a sound approach to basic hemodynamic monitoring prior to selecting the optimal modality. This article comprises an in depth discussion of an approach to hemodynamic monitoring techniques and principles as well as methods of predicting fluid responsiveness as it applies to the ED clinician. We review the role, applicability and validity of various methods and techniques that include; clinical assessment, passive leg raising, blood pressure, finger based monitoring devices, the mini-fluid challenge, the end-expiratory occlusion test, central venous pressure monitoring, the pulmonary artery catheter, ultrasonography, bioreactance and other modern invasive hemodynamic monitoring devices.

Contents

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http://dx.doi.org/10.1016/j.ajem.2017.03.036
0735-6757/

1. Introduction

Hemodynamic monitoring (HDM) is a key component in the effective management of the critically ill patient. Over the past decade, there have been significant advances in HDM techniques and devices with regards their application in the intensive care unit (ICU) and operating room settings. With the progress and development of emergency medicine globally, an increasing emphasis has been placed on employing accurate diagnostic techniques capable of guiding the early management of the undifferentiated critically ill patient presenting to the emergency department (ED). Coinciding with the turn of the century, there has been increasing emphasis on instituting early management for a number of ED presentations regarded as "time dependent". Idioms such as the golden hour [1], early-goal-directed-therapy (EGDT) [2], time is muscle [3] and time is brain [4] have been promulgated to emphasize the importance of timely management of trauma, sepsis, ST elevation myocardial infarction and stroke patients presenting to the ED.

Despite the fact that a substantial proportion of individuals undergoing stabilization in the ED resuscitation room ultimately require ongoing care in the ICU setting [5], the applicability and role of various HDM techniques and their application in the ED environment have been poorly defined. Whilst the ED is regarded as the gateway to hospital admission [6], the ED resuscitation room may be regarded as the doorway to the ICU. Good overall patient outcomes are dependent on timely appropriate management of the critically ill patient in the ED. As a result, advanced monitoring and interventional tools which were previously regarded the niche of the ICU and operating room environments, have been introduced to the ED. However, bearing in mind challenges and limitations specific to the ED setting the accuracy, reliability, applicability, invasiveness, cost and user friendliness of available HDM devices must be taken into account prior to implementation [7-9].

At its core, HDM is concerned with two fundamental entities within the human circulatory system, oxygen delivery (DO_2) and oxygen consumption (VO_2) where DO_2 (ml O_2/min) = $CO \times Hb \times 1.34 \times SaO_2$ and VO_2 (ml O_2/min) = $CO \times Hb \times 1.34 \times (SaO_2 - SvO_2)$. CO = cardiac output in ml/min, Hb = haemoglobin in g/100 ml, SaO_2 = percentage of arterial oxygen saturation and SvO_2 = percentage of mixed venous oxygen saturation [10,11]. An increase in DO_2 is likely in volume-responders whilst non-responders may actually display a drop in DO_2 as a consequence of the hemodilutory effects of volume infusion [12].

In essence, the concept of HDM is primarily concerned with assessing the performance of the cardiovascular system and its ability to deliver sufficient oxygen to meet the metabolic demands of the body [13]. The value of HDM in the ED is threefold in that it is useful in identifying the presence and nature of shock, secondly it guides appropriate therapeutic interventions and finally it provides an assessment tool for response to therapy [14,15]. For HDM to be truly effective in the ED setting various techniques, methods, interventions and technological advances coupled with a sound clinical approach to basic hemodynamic monitoring must be incorporated prior to selecting the optimal modality [8,15]. An understanding of the manner in which the reading is derived, as well as the accuracy of the device in specific clinical scenarios is paramount [16].

In this article the spectrum of haemodynamic monitoring techniques ranging from basic clinical examination to advanced invasive monitoring as well as their practicality and applicability to the ED environment is reviewed.

2. Shock

Shock can be defined as the inadequate delivery and utilization of oxygen at the cellular level. The various categories of shock determining management principles include cardiogenic, hypovolemic, distributive and obstructive subtypes (Table 1) [17]. The goal of intervention following hemodynamic monitoring is to achieve an increase in cardiac output with a subsequent improvement in tissue oxygenation. A sensible management approach would be to identify and treat abnormalities in order of firstly correcting any cardiac rate and rhythm disturbances, then optimising intravascular volume and systemic vascular resistance and finally attending to myocardial pump function and obstruction related disturbances. HDM aims to assess these elements and determine the appropriate choice of therapy (Fig. 1) [18-20].

3. Understanding the role of fluids in light of the frank starling curve

An initial fluid bolus is a frequent reflex response amongst clinicians faced with a victim in circulatory shock. However, as a consequence of shock related microcirculatory and cellular dysfunction, just half the number of individuals receiving a fluid bolus are expected to respond with a corresponding increase in DO_2 (volume responders) and only

Table 1
Description of the 4 categories of shock.

Cardiogenic	Hypovolemic
Dysrhythmias – extreme bradycardia or tachycardia	Haemorrhagic, trauma – external hemorrhage, intrathoracic, intraabdominal, pelvis and retroperitoneal, long bones
Acute coronary syndrome	Haemorrhagic, non-trauma – gastrointestinal (UGIB, LGIB), ruptured ectopic pregnancy, ruptured AAA
Acute myocarditis	Non-haemorrhagic – diarrhoea, vomiting, heat stroke, excessive sweating
Cardiomyopathies	
Post traumatic myocardial injury	
Valvular heart disease	
Distributive	**Obstructive**
Neurogenic shock (high spinal cord transection)	Tension pneumothorax
Anaphylactic shock[b]	Pericardial tamponade
Septic shock[a]	Pulmonary embolism

[a] Also has an associated 1) hypovolemic component as a result of widespread capillary leak of fluid into the extravascular compartment secondary to cytokine release and loss of the endothelial glycocalyx and 2) cardiogenic component secondary to cytokine mediated myocardial depression.
[b] Also has an associated hypovolemic component as a result of widespread capillary leak of fluid into the extravascular compartment.

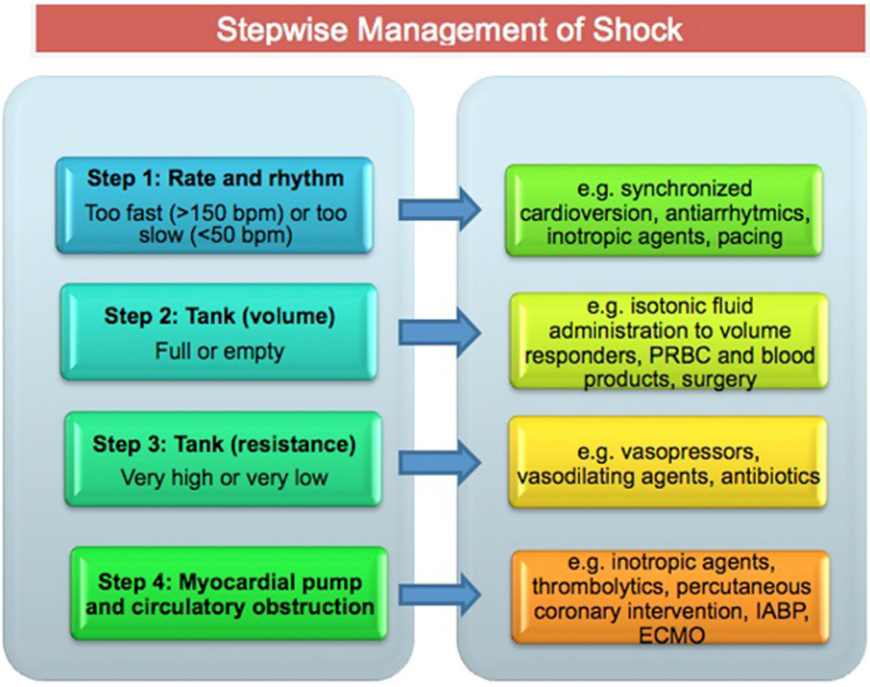

Fig. 1. Stepwise management of the undifferentiated patient presenting with shock to the ED. Each step must be attended to before proceeding to the next step.

half of DO_2 responders will display a corresponding increase in oxygen consumption (VO_2) [12].

It must be understood that fluid depletion does not necessarily equate to fluid responsiveness [19]. From the frank-starling curve (Fig. 2) [21], it can be understood that a fluid depleted individual who is on the steep portion of the curve will respond to a fluid bolus with a corresponding increase in CO and DO_2, whereas a fluid depleted individual who is on the flat portion of the curve will not respond to a fluid bolus with no improvement in CO or DO_2 but rather a worsening of oedema [22] and even mortality

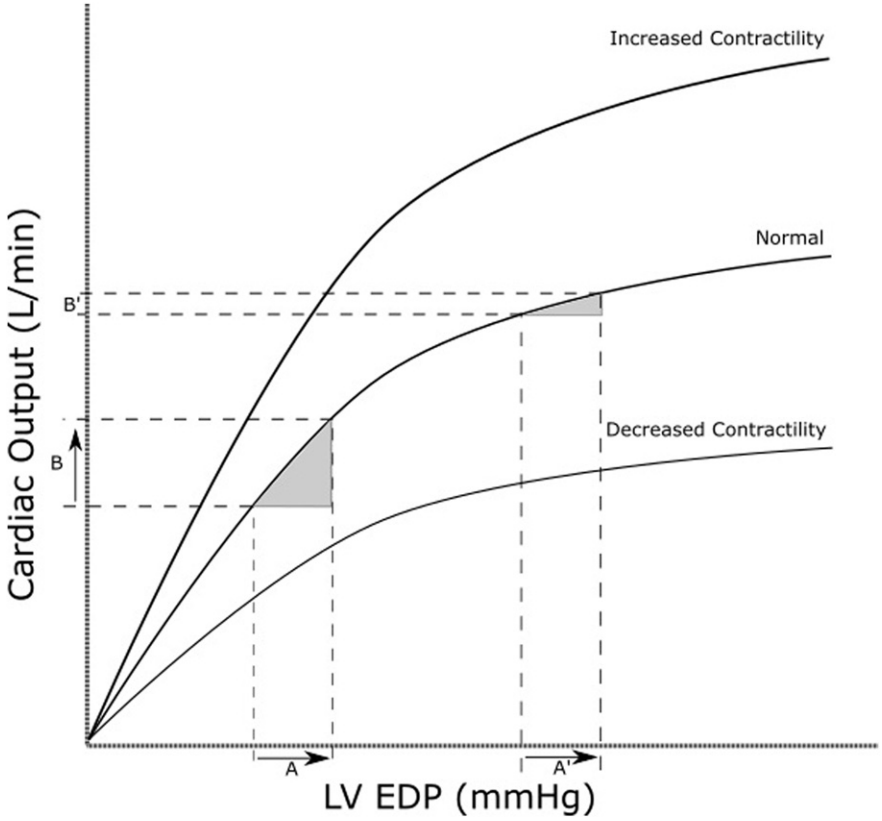

Fig. 2. The Frank-Starling curve. A shocked patient on the STEEP portion of the curve will respond to a fluid bolus with an increase in LVEDP (A) that translates to an increase in cardiac output (B). A shocked patient on the FLAT portion of the curve will NOT respond to a fluid bolus. Despite an increase in LVEDP (A^1), there is a negligible increase in cardiac output (B^1). Hence fluid overload and oedema will result as a consequence of fluid administration in this patient.

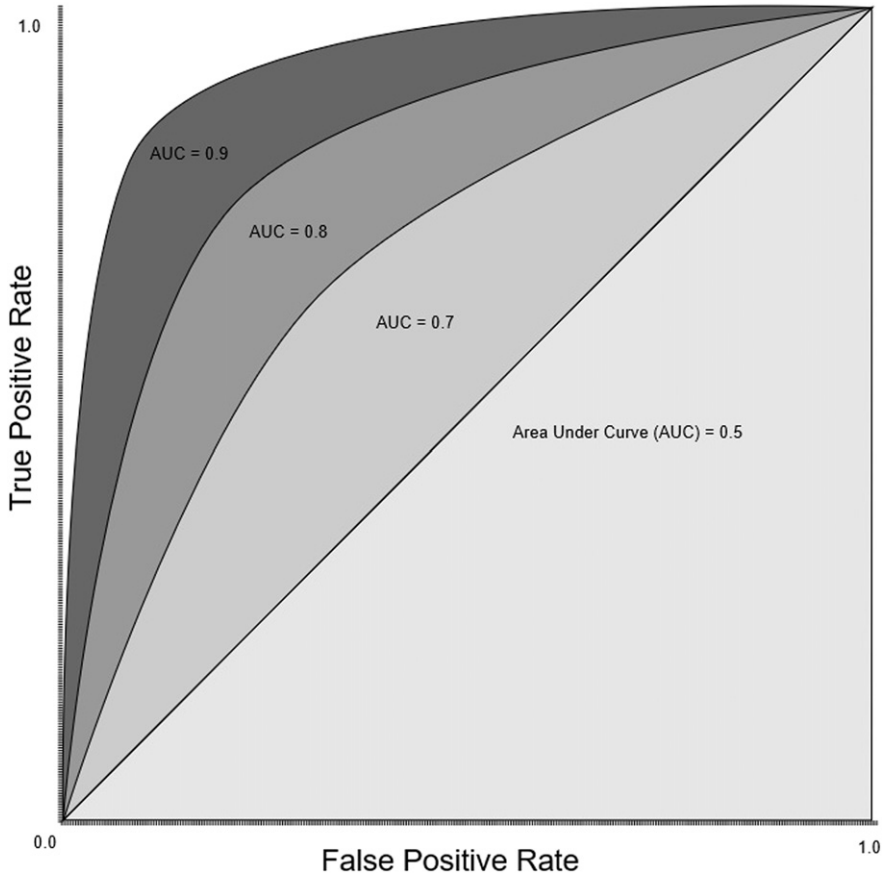

Fig. 3. The receiver operating curve, a statistical tool that compares the true positive rate (sensitivity) versus the false positive rate (1-specificity) of various measured and derived variables of interest. For an area under the curve (AUC) of 0.5, the true positive rate is equal to the false positive rate which renders the test worthlessness and is synonymous to flipping a coin. An AUC of <0.8 is inadequate, whereas an AUC of >0.9 is associated with very good accuracy.

[23,24]. This places a high level of importance on hemodynamic monitoring as a tool to accurately determine a patients fluid status and ongoing requirements whilst at the same time preventing fluid overload [13,25].

The receiver operating curve (ROC) is a statistical tool that is useful to determine the reliability of various measured and derived variables in predicting fluid responsiveness. The true positive rate, also referred to as the probability of detection or sensitivity, is plotted against the false positive rate (1 – specificity), which is also known as the probability of a false alarm. An area under the ROC (AUROC) of 0.5 implies that the true positive rate is equal to the false positive rate which renders the test worthless and is synonymous to flipping a coin. AUROC values > 0.8 or 0.9 imply adequate or very good accuracy of the monitoring technique (Fig. 3) [26].

4. Predicting fluid responsiveness

Response to a fluid bolus can be predicted by 1) determining the degree of change in various measured or derived variables in response to variations in the respiratory cycle (inspiration and expiration). These variables are generally determined by means of invasive monitoring devices in fully ventilated patients. 2) Measuring the percentage increase in other measured or derived variables that are generally determined with the use of less invasive monitoring devices in response to a passive leg raise (PLR) maneuver. Table 2 summarizes the reliability of various variables that may be obtained with invasive and non-invasive monitoring devices and their ability to predict fluid responsiveness.

4.1. Respiratory variation

With an increase in intrathoracic pressure during inspiration in the mechanically ventilated patient, venous return and stroke volume are both diminished when compared to the expiratory phase of respiration. The difference is usually <10%, however under certain conditions in the volume depleted patient, larger differences in various measured variables that include pulse pressure variation (PPV), systolic pressure variation (SPV) and stroke volume variation (SVV) are able to predict fluid responsiveness with varying degrees of accuracy [27]. Conditions that limit the routine use of devices that are capable of determining these variables include the fact that the patient must be paralyzed, fully ventilated with a tidal volume > 8 ml/kg and have a set RR < 17 breaths per minute. Validity of these variables are further compromised in patients with dysrhythmias, severe peripheral vascular disease, aortic valvular regurgitation, pronounced vasoconstriction or vasodilation, right ventricular failure, pericardial tamponade or constriction and raised intra-abdominal pressure [28,29].

Based on the above limitations and the need for an invasive monitoring device that generally requires both arterial and central venous access to determine these respiratory variation based indices (PPV, SPV, SVV), this method may be less feasible and applicable to most ED settings. The use and validity of these devices and techniques are described below.

4.2. Passive leg raising

By placing the patient in a supine position and then passively raising both legs to a 30–45° angle above the horizontal plane for a 30–90 s

period induces a reversible auto-transfusion of approximately 300 ml of blood from the lower extremities to the heart, causing an increase in left ventricular preload and thereby challenging the Frank-Starling curve. This method is contra-indicated in patients with underlying abdominal hypertension and lower extremity trauma [30,31].

An increase in cardiac output (CO), velocity time integral (VTI), aortic blood flow velocity, carotid artery blood flow time or end-tidal CO_2 ($ETCO_2$) at the end of a PLR maneuver has been shown to predict response to a fluid bolus in patients with spontaneous breathing activity. In a recent meta-analysis that included 991 adult patients from twenty-one studies, the pooled AUROC was 0.95 ± 0.01 (sensitivity 85%, specificity 91%) for a passive leg raise (PLR) induced change in cardiac output ≥ 10 ± 2%, whereas the pooled AUROC for a PLR induced change in pulse pressure (PP) was only 0.77 ± 0.05 (sensitivity 56%, specificity 83%). The authors concluded that PLR-induced changes in CO are very reliable in predicting a response to a fluid bolus and whilst the specificity of a PLR-induced change in PP remains acceptable, the sensitivity is poor [32]. Table 2 mentions the details of 3 studies that have demonstrated the reliability of VTI in predicting fluid responsiveness [33-35].

A study comprising 79 subjects including patients with spontaneous breathing activity and cardiac rhythm disturbances showed that a PLR-induced increase in aortic blood flow velocity ≥ 10% predicted response to a fluid bolus with a sensitivity of 97% and specificity of 94% [31]. Changes in carotid artery blood flow time (FTc) in response to a PLR have also shown promise in predicting volume responsiveness [36-39]. One of these studies conducted in 70 healthy blood donors with a mean blood loss of 452 ml showed a mean percentage increase in corrected carotid artery flow time of 8.3% after a PLR-maneuver was predictive of fluid responsiveness [36]. A PLR-induced increase in $ETCO_2$ > 5% was associated with a sensitivity of 71% and specificity of 100% for predicting fluid responsiveness in 65 spontaneously breathing individuals [40]. Another smaller study in patients undergoing fixed mechanical ventilation, showed that a PLR-induced increase in $ETCO_2$ > 5% was associated with an AUROC of 0.97 [41].

The PLR is an easy to perform maneuver that requires minimal training. However the PLR must be interpreted in conjunction with changes in CO, VTI, aortic blood flow velocity, carotid artery flow time or $ETCO_2$. The accurate determination of CO generally requires the use of invasive monitoring devices that require both arterial and central venous access and are therefore less applicable to the ED. Other methods of determining CO that are more applicable to the ED setting and require minimal training, although accuracy and validity are questionable include finger based monitoring devices and bioreactance. VTI, aortic blood flow velocity and carotid artery flow time are all determined by Doppler ultrasonography, however operator dependency and the associated steep learning curve limit its widespread use [42]. $ETCO_2$ can be easily determined with continuous waveform-capnography devices that are generally available in the ED. The use and validity of these devices and techniques are described below.

5. Currently available hemodynamic monitoring devices

Currently available methods for assessing hemodynamic status and predicting fluid responsiveness in the shocked patient range from simple to complex and include clinical assessment, serial biomarker interpretation, blood pressure (BP) monitoring, ultrasonography, bioreactance (NICOM™) monitoring (Cheetah Medical, Portland, OR, USA) and other non-invasive methods such as pulse oximeter plethysmography waveform analysis (e.g. Masimo Corporation, Irvine, CA, USA), pulse oximeter based continuous cardiac output monitoring (esCCO™, Nihon Kohden, Japan), finger cuff based hemodynamic monitoring (ClearSight™, Edwards Lifesciences, Irvine CA, USA), minimally invasive methods such as central venous pressure (CVP) assessment, the FloTrac/Vigileo™ System (Edwards Lifesciences, Irvine CA, USA), the COstatus™ (transonic systems, Ithaca, NY, USA) and more invasive methods such as the Pulmonary Artery Catheter (PAC), the Pulse index Continuous Cardiac Output (PiCCO™) system (Pulsion Medical Systems SE, Munich, Germany), the Lithium Dilution Cardiac Output (LiDCO™) system (LiDCO Ltd., Cambridge, UK), and the Volume View/EV 1000™ system (Edwards Lifesciences, Irvine CA, USA) [43-54].

The accuracy, reliability, validity and applicability vary with each of the above devices and methods. Choice of monitoring technique must be individualized and is dependent on the underlying patient pathology (e.g. dysrhythmias, right ventricular failure), whether the patient is fully ventilated or spontaneously triggering ventilator breaths or not-ventilated, the invasiveness of the monitoring technique, clinician experience and whether dynamic (continuous) or static monitoring is required [16,19,55].

6. Assessing hemodynamic status non-invasively

6.1. Clinical assessment and serial biomarker measurements

A clinician's auditory, visual and tactile assessment of a patient constitutes hemodynamic monitoring at its most basic, non-invasive level. In the majority of cases a goal directed history, clinical examination and basic bedside monitoring are adequate in determining appropriate management of the shocked patient (Fig. 2). Whilst these elements are subjective and have variable accuracy and precision as definitive diagnostic tools, they pave the way in choosing the appropriate HDM technique and device.

Prior to the administration of fluids, the clinician must assess for the presence of oedema. Stevenson and Perloff in 1989 showed that a combination of peripheral oedema, pulmonary oedema or a raised jugular venous pressure was associated with a specificity of 100% and sensitivity of 58% for predicting fluid overload [56]. Most other clinical findings including an inadequate urine output and a derangement in the level of consciousness are inaccurate indicators of tissue perfusion as a result of the common presence of various confounding pathologies including acute kidney injury, the presence of drugs and other medications and other co-morbid pathologies in the critically ill patient population [57,58].

Serial measurements of circulatory system biomarkers such as the mixed venous oxygen saturation (S_VO_2) and lactate are useful indicators of the adequacy of tissue oxygenation. A low S_VO_2 may suggest the presence of a low CO state, anaemia, hypoxaemia or an increase in oxygen consumption, whereas supranormal S_VO_2 levels in septic patients may actually reflect maldistribution of blood flow or inadequate mitochondrial oxygen utilization and may in fact be associated with poorer outcomes [59,60]. Lactate has been shown to be more useful than S_VO_2 in identifying patients that may benefit from the administration of fluid [61,62].

6.2. Blood pressure monitoring

A “normal” BP is commonly regarded as a systolic/diastolic pressure of 120/80 mm Hg, which equates to a mean arterial pressure (MAP) of 94 mm Hg. International sepsis guidelines recommend a target MAP of >65 mm Hg in patients with severe sepsis and septic shock [63] and the MAP's to achieve adequate abdominal, cerebral and renal perfusion pressures have been recommended as above 60 mm Hg, 70 mm Hg and 85 mm Hg respectively [64-66], whereas geriatrics generally have higher blood pressure readings with an upward shift in the autoregulatory range [67]. These inconsistencies and other limitations [68,69] render blood pressure monitoring a poor HDM tool and rather inaccurate in predicting end organ perfusion and fluid responsiveness.

Furthermore the clinician must exercise caution in the following two scenarios where the true perfusion pressure is underestimated by the observed MAP. Firstly when there is an increase in extravascular pressure such as in individuals with a raised intracranial pressure, abdominal hypertension or a limb compartment syndrome and secondly in individuals with a significant increase in right atrial pressure which

Table 2
Summary of the reliability of various variables obtainable with invasive and non-invasive monitoring devices and their usefulness in predicting fluid responsiveness.

Variable	Reliability for determining fluid responsiveness	Study population	Advantages / limitations of technique or device
Variables obtained through non-invasive monitoring methods			
Blood pressure monitoring			
ΔMAP, ΔPP	Poor correlation (r = 0.52 and r = 0.56 respectively) for predicting fluid responsiveness [69]		Cuff size, site of measurement and correct application of cuff impact measurements obtained. Oscillometric based devices are less accurate at higher and lower extremes [68] For intra-arterial monitoring; calibration, leveling, zeroing and the absence of kinking, air bubbles or other obstructions must be ensured [69]
Ultrasonography			
Respiratory variation induced ultrasonographic changes			
Δ aortic blood flow (L/min) >18%	Sensitivity 90%, Specificity 94%, AUROC 0.93	38 fully ventilated patients without dysrhythmias [86].	Static. Ultrasonography is operator dependent and has a steep learning curve. Patients must be fully ventilated. Unreliable in the presence of arrhythmias.
Δ aortic blood flow time corrected for HR (FTc) (ms)	Sensitivity 55%, Specificity 94%, AUROC 0.76	38 fully ventilated patients without dysrhythmias [86].	As above
Δ peak aortic blood flow velocity (m/sec) >12%	Sensitivity 100%, Specificity 89%	19 sedated and ventilated patients [87].	As above
Δ left ventricular end diastolic area (m^2)	Poor correlation ($r^2 = 0.11$) for predicting fluid responsiveness [87].		As above
Δ inferior vena caval (IVC) diameter	1) >12% distensibility on expiration: PPV 93%, NPV 92% [88] 2) >18% distensibility on expiration: Sensitivity 90%, Specificity 80% [89]	1) 39 fully ventilated patients [88]. 2) 23 fully ventilated patients [89].	Static. Ultrasonography is operator dependent and has a steep learning curve. In the spontaneously breathing patient, changes in IVC diameter correlates with CVP readings but not fluid responsiveness [90,91]
Δ superior vena caval (SVC) collapsibility > 36% on inspiration	Sensitivity 90%, Specificity 100%	66 fully ventilated patients [92].	As above
Passive leg raise (PLR) induced ultrasonographic changes			
Δ subaortic velocity time integral (VTI) >12%	1) Sensitivity 77%, Specificity 100%, AUROC 0.96 [33] 2) Sensitivity 69%, Specificity 89%, AUROC 0.90 [34]	1) 24 spontaneously breathing patients [33]. 2) 34 spontaneously breathing patients [34].	Static. Can be used in spontaneously breathing patient but not in the presence of arrhytmias.
Δ carotid artery VTI >20%	Sensitivity 94%, Specificity 86%	34 spontaneously breathing patients [35].	As above
Δ carotid artery blood flow time (FTc) (ms) >5%	Sensitivity 66%, Specificity 77%	70 spontaneously breathing blood donors [36].	As above
Bioreactance (NICOM™ device)			
PLR induced increase in CO/SV	1) Sensitivity 88%, Specificity 100% [93]. 2) Sensitivity 94%, Specificity 100% [35]. 3) AUROC ± 0.5 [44].	1) 75 post cardiac surgery patients [93]. 2) 34 spontaneously breathing patients [35]. 3) 48 spontaneously breathing patients [44].	Dynamic. Can be used in the spontaneously breathing patient and also in the presence of arrhythmias. Use is limited by inconsistent evidence.
Finger based monitoring devices			
Plethysmographic waveform analysis (e.g. Masimo™)			
Variation in plethysmographic waveform amplitude (Δ POP)	Sensitivity 85%, Specificity 85%, AUROC 0.89	Meta-analysis of160 fully ventilated patients in sinus rhythm [94].	Dynamic. Except for *esCCO™*, the patient must be fully ventilated with a tidal volume of at least 8 ml/kg. Pulse oximeter based waveform analysis is dependent on peripheral perfusion which is decreased in hypothermia, shock,

			edema and vasoconstrictor therapy. These imitations are frequent findings in patients most require hemodynamic monitoring.
Increase in plethysmographic variation index (PVI)	Sensitivity 83%, Specificity 89%, AUROC 0.95	Meta-analysis of 173 fully ventilated patients in sinus rhythm [94].	As above
Increase in PVI >19%	Sensitivity 94%, Specificity 87%, AUROC 0.97	31 ventilated and sedated patients in the ED [95].	As Above
estimated Continuous Cardiac Output (esCCO™) monitor			
Continuous cardiac output monitoring	Inconsistent results mostly showing poor reliability, precision and correlation when compared to established methods [46,101-105]		As Above
ClearSight™			
ΔPPV, ΔSVV	1) AUROC 0.57 and 0.50 [106] 2) Good correlation, r = 0.88 and r = 0.87 [107]	1) 45 ventilated patients following cardiac surgery [106]. 2) 19 post-operative fully ventilated patients [107].	As above
Mini-fluid challenge			
ΔCO, ΔSVV, ΔPPV after 100ml colloid bolus over 1 minute	CO, AUROC 0.78 SVV, AUROC 0.91 PPV, AUROC 0.92	49 ventilated (<8ml/kg) and deeply sedated patients without dysrhythmias [110].	Static. Can't be performed in the presence of arrhythmias. Patients must be deeply sedated and mechanically ventilated. The measurement of VTI is operator dependent.
ΔVTI, ΔPPV after 100ml colloid bolus over 1 minute	VTI, AUROC 0.90 PPV, AUROC 0.55	39 ventilated and sedated patients [109].	As above
End-expiratory occlusion test			
ΔC0 >5% after 15s end expiratory occlusion	AUROC 0.97	34 ventilated patients with dysrhythmias and some spontaneous breathing activity [[111].	Static. Can be performed in pts with arrhythmias
Variables obtained through invasive monitoring methods			
CVP	AUROC 0.55	Meta-analysis of 685 patients from 29 studies [27].	Dynamic. Poor marker of fluid status and poor marker of fluid responsiveness.
GEDVI	AUROC 0.56	Meta-analysis of 685 patients from 29 studies [27].	Static. Good marker of fluid status, but poor marker of fluid responsiveness. Thermodilution is inaccurate in the presence of intrathoracic hemorrhage and intra-cardiac shunts.
LVEDAI	AUROC 0.64	Meta-analysis of 685 patients from 29 studies [27].	As above
ΔSVV >±11-13%	AUROC 0.84	Meta-analysis of 685 patients from 29 studies [27].	Dynamic. Patient must be fully ventilated with a tidal volume of at least 8 ml/kg and a RR < 17bpm. Validity is compromised in patients with 1) arrhythmias 2) severe peripheral vascular disease 3) AV regurgitation 4) pronounced vasoconstriction / vasodilation 5) RV failure 6) pericardial tamponade / constriction 7) raised intra-abdominal pressure
ΔSPV >±11-13%	AUROC 0.86	Meta-analysis of 685 patients from 29 studies [27].	As above
ΔPPV >±11-13%	AUROC 0.94	Meta-analysis of 685 patients from 29 studies [27].	As above

can no longer be ignored in the MAP equation (MAP = (CO × SVR) − RAP) [68].

As a result of unpredictable changes in arterial compliance and pulse wave amplification, especially in critically ill patients, variables such as MAP and pulse pressure (PP) (PP = SBP − DBP) are unable to reliably predict fluid responsiveness [69,70]. Therefore, although better than nothing, monitoring of SBP, DBP, MAP and PP are only crude indicators of end organ perfusion and must be augmented with clinical findings and other methods of hemodynamic monitoring in the sick patient.

6.3. Ultrasonography

Point of care ultrasound imaging is now widely regarded as a part of the furniture and armamentarium of the emergency department (ED) [71,72]. Despite the drawback of it being operator dependent and its associated steep learning curve [42], ultrasound imaging is still an appealing tool for HDM due to the fact that it is non-invasive, safe, free of ionizing radiation and can be performed at the bedside [73]. In the past decade there have been significant strides in the application of ultrasonography in the field of hemodynamic monitoring.

Various protocols describing the ultrasongraphic assessment of the circulatory system have been described [74-81]. The findings of regional wall motion abnormalities (RWMA), a poorly contractile myocardium, low ejection fraction, dilated cardiac chambers, valvular stenosis, regurgitations and the presence of comet tail artifacts or "B-lines" suggest a cardiogenic aetiology, whereas an absence of lung sliding and comet tail artifacts with the presence of a lung-point sign on lung ultrasonography may suggest a missed tension pneumothorax [82].

Ultrasonographic features suggesting the need for urgent drainage of a pericardial tamponade include a dilated inferior vena cava (IVC), systolic collapse of the right atrium for more than a third of the cardiac cycle and diastolic collapse of the right ventricle [83]. Diagnostic criteria suggesting pulmonary embolism include dilatation of right sided cardiac chambers, elevation of pulmonary artery pressures and mural thrombi on transthoracic views [84] as well as features of deep vein thrombosis (DVT) on limited compression ultrasonography (LCUS) [85] whereas in trauma patients the presence of fluid in the pleural and abdominal cavities suggests the presence of hemorrhagic shock [76].

With regards to the application of ultrasonography in predicting fluid responsiveness, various methods using 2-dimensional, motion (M) or Doppler modes of imaging have been described that predominantly assess the variation in blood flow and blood flow velocity with the respiratory cycle. Variables described include the degree of variation in aortic blood flow (l/min), aortic blood flow velocity (m/s), peak aortic blood flow velocity (m/s), carotid artery blood flow time (ms), and vena caval diameter (mm). Changes in the velocity time integral (VTI) which is a surrogate measure of stroke volume after a passive leg raise maneuver or fluid bolus have also been shown to predict fluid responsiveness. In the spontaneously breathing patient changes in the IVC diameter correlate with CVP measurements but not fluid responsiveness. Reliability of the above methods are presented in Table 2 [33-36,86-92]. Some of these variables are more accurately assessed with trans-esophageal ultrasonography which is an invasive technique requiring specialized training [86].

6.4. Bioreactance

Bioreactance continuously measures the time delay (phase shift) between the electrical current that is applied to the thorax and voltage that is returned. These phase shifts correlate with aortic blood volume and are used to determine stroke volume. The NICOM™ monitor provides continuous, non-invasive hemodynamic monitoring via 4 sensor pads applied over the thorax. Current evidence to support its routine use is inconsistent (Table 2). Whilst some studies have proven the NICOM™ monitor reliable in determining CO and fluid responsiveness when coupled with a PLR maneuver [35,93], Kupersztych-Hagege and colleagues demonstrated an AUROC of just over 0.5 and concluded that the NICOM™ device was inaccurate in estimating CO or predicting fluid responsiveness when coupled with a PLR test [44]. Large scale ED based studies are required prior to defining its role in this setting.

6.5. Finger based monitoring devices

With regard to pulse oximeter derived plethysmographic waveform analysis; both the respiratory variation in the plethysmographic waveform amplitude (ΔPOP) and the plethsmographic variability index (PVI) have shown promise in predicting fluid responsiveness. Sandroni and colleagues in their meta-analysis that included 10 studies reported a pooled AUROC of 0.89 and 0.95 for the ability of ΔPOP and PVI in predicting fluid responsiveness [94]. More recently, Feissel and colleagues reported an AUROC of 0.90 for PVI in an ED based study that looked at patients in the early phase of septic shock [95]. The need for patients to be fully ventilated with no spontaneous respirations and a tidal volume of at least 8 ml/kg limits its usefulness in the ED environment [96]. Other limitations relate to the fact that pulse oximeter based waveform analysis is dependent on peripheral perfusion which is commonly affected by hypothermia, shock, vasoconstrictor agents and the site of measurement [97-99]. These limitations are generally more common in patients actually requiring HDM. In another study, probes placed on the ear lobe or forehead where changes in vascular tone are less variable, were shown to be more accurate in predicting fluid responsiveness then finger based probes [100].

The estimated continuous cardiac output (esCCO™) monitoring device continuously estimates cardiac output by determining the pulse wave transit time (PWTT) (time taken for blood from the heart to reach the finger-tip) which is measured as the time between the peak of the electrocardiogram (ECG) R-wave and the oximeter pulse wave rise point seen at the finger-tip. Since measurements of PWTT depend on peripheral perfusion, the limitations of plethysmographic waveform analysis mentioned above are also applicable here. Various studies in recent years have reported inconsistent results with most reporting poor reliability, precision and correlation when compared to established methods [46,101-105].

The ClearSight™ device continuously measures BP, CO, SVV and PPV via an inflatable finger cuff. As with pulse oximeter plethysmography and esCCO™, there is inconsistent evidence supporting its reliability, especially in patients with poor peripheral perfusion, hypothermia and peripheral oedema [47,106,107].

6.6. The mini-fluid challenge

An increase in the ultrasonographic VTI after administering a small volume of fluid (100 ml) can predict fluid responsiveness without the detrimental effects associated with large fluid boluses [108]. Muller and colleagues demonstrated that an increase in VTI of >10% after a 100 ml colloid bolus over 1 min proved reliable in its ability to predict fluid responsiveness (AUROC 0.92) [109]. Another study that included 29 patients that were challenged with a 100 ml colloid bolus demonstrated an AUROC of 0.90 for predicting an increase in VTI. In the same study, fluid induced changes in pulse pressure variation (PPV) did not prove reliable (AUROC 0.55) [110]. However the mini-fluid challenge test is restricted to deeply sedated and mechanically ventilated patients without cardiac dysrhythmias.

6.7. The end-expiratory occlusion test

Interruption of the respiratory cycle at the end of expiration will avert the expected cyclical changes in venous return and cardiac output. A study that included 34 mechanically ventilated patients with dysrhythmias and some spontaneous breathing activity showed that a 5% increase in CO after a 15 s end-expiratory hold maneuver was predictive

of fluid responsiveness with an AUROC of 0.97 [111]. The end-expiratory occlusion test together with ultrasonography and the mini-fluid challenge are static hemodynamic monitoring methods in contrast to bioreactance monitoring systems and finger based monitoring devices which allow for continuous monitoring.

7. Invasive hemodynamic monitoring devices

7.1. Central venous pressure monitoring

CVP is a useful marker of right ventricular function but has no value in predicting fluid responsiveness in the critically ill patient. This is because CVP is influenced by frequent and unpredictable changes in vascular tone, intra-thoracic pressure, ventricular compliance and myocardial geometry in the critically ill patient [112-116]. In a meta-analysis that included 803 patients from 24 studies, a meager 16% of patients responded to a fluid bolus with the pooled AUROC of just 0.56 for the ability of CVP to predict fluid responsiveness. The authors concluded that neither a high, normal or low CVP, nor the response of CVP to fluid loading should be utilized to guide the fluid management strategy of any patient [48]. Despite the evidence, sepsis management consensus guidelines continue to advocate the use of CVP measurements in guiding fluid administration [63,117].

7.2. Pulmonary artery catheter

Since its introduction in the 1970's, the pulmonary artery catheter has been widely used as a diagnostic tool in critically ill patients. Based on the principle of thermodilution, CO and other variables are estimated by measuring the temperature in the pulmonary artery after injecting a bolus of a cold saline solution into the right atrium. Insertion of a PAC has a steep learning curve and can be rather challenging with complications that include pulmonary infarction and hemorrhage, rupture of the balloon tip and cardiac arrhythmias [118]. A meta-analysis of 13 pooled studies that included 5686 patients concluded that the use of the PAC was not associated with mortality, ICU length of stay (LOS), hospital LOS, or cost benefits [51]. Hence the PAC has largely been replaced by modern less-invasive HDM devices that are discussed below.

7.3. Modern invasive HDM devices

These devices require a CVP catheter as well as an intra-arterial line to allow for continuous hemodynamic monitoring. Peripheral intra-arterial lines are sufficient when using the FloTrac/Vigileo™, COstatus™ and LiDCO™ devices, whereas the PiCCO™ and Volume View/EV 1000™ are more invasive and require the placement of a femoral intra-arterial line [29].

All of these devices have the ability to continuously estimate CO with varying degrees of accuracy. With the FloTrac/Vigileo™ device, CO is calculated using an algorithm based on the pressure recording analytical method (PRAM) which assumes the patients vascular compliance and elastance based on weight, age, sex and height. Hence CO values are less accurate in situations where there are major changes in vascular compliance, in patients with underlying aortic regurgitation and where there is an over or under dampening of the arterial waveform [119]. The COstatus™ estimates CO by making use of ultrasound technology to determine changes in blood flow and velocity after injecting warm saline [120].

The LiDCO™ device which is based on the lithium dilution method, determines the changes in lithium concentration after injecting small amounts of lithium chloride to intermittently estimate CO [121], whereas the PiCCO™ and the Volume View/EV 1000™ utilize the principles of transpulmonary thermodilution by determining downstream temperature changes after injecting ice-cold saline in the femoral vein. Thermodilution derived variables have their limitations and are inaccurate in the presence of an intracardiac shunt or intrathoracic hemorrhage [119,122].

The LiDCO™, PiCCO™ and Volume View/EV 1000™ devices are also capable of continuously estimating cardiac output based on arterial pressure waveform analysis. The LiDCO™ and PiCCO™ employ the pulse power analysis and pulse contour analysis methods respectively, whereas the Volume View/EV 1000™ can be linked to the FloTrac/Vigileo™ or ClearSight™ devices to continuously measure CO. After changes in vascular compliance, these devices require calibration via the measurement of CO with the various dilution methods described above [28,29].

In addition to estimating CO, other useful parameters that may be determined by the above devices include stroke volume (SV), extravascular lung water index (EVLWI), global end diastolic volume index (GEDVI), stroke volume variation (SVV), systolic pressure variation (SPV), pulse pressure variation (PPV), systemic vascular resistance index (SVRI) and $S_{CV}O_2$ (Table 3). Of these variables, SVV, SPV and PPV have been proven useful in predicting fluid responsiveness (Table 2) [27]. EVLWI is a measure of pulmonary interstitial and alveolar space fluid accumulation (pulmonary oedema) and has prognostic implications [123,124].

8. Does HDM improve patient outcomes

Despite recent advances, there is still considerable debate with regard to overall outcomes and benefit with the implementation of HDM devices [15,125,126]. In the critically ill patient, various other factors such as irreversible cellular injury and the inability to utilize oxygen at the tissue level impact outcomes [12]. In fact the Pulmonary Artery Catheter which for a long time was regarded the standard in HDM,

Table 3
Summary of available invasive modalities.

Modality	Variables	Method of determining variables such as CO, SVV, PPV, EVLWI, GEDVI	Invasiveness
Central venous pressure (CVP) catheter	CVP, $S_{CV}O_2$		CVP catheter
Pulmonary artery catheter (PAC)	CVP, S_VO_2, PAP, RVEDP, LVEDP (PCWP), SV, CO	Thermodilution	Pulmonary artery catheter
FloTrac™	SV, CO, SVV, SVRI	Pulse contour analysis	Requires peripheral arterial line and CVP catheter
COstatus™	SV, CO, SVRI	Ultrasound technology and blood flow	Requires peripheral arterial line and CVP catheter
Lithium dilution cardiac output (LiDCO™)	SV, CO, PPV, SPV, SVV, SVRI	Lithium dilution and pulse power analysis	Requires peripheral arterial line and CVP catheter
Pulse index continuous cardiac output (PiCCO™)	SV, CO, EVLW, GEDVI, ITTV, ITBV, PBV, SVV, SVRI.	Thermodilution and pulse contour analysis	Requires femoral arterial line and CVP catheter
Volume View/EV 1000™	SV, CO, EVLW, GEDVI, ITTV, ITBV, PBV, SVV, SVRI.	Thermodilution and pulse contour analysis	Requires femoral arterial line and CVP catheter

CVP – central venous pressure; $S_{CV}O_2$ – central venous oxygen saturation S_VO_2 – mean venous oxygen saturation; PAP – pulmonary artery pressure; RVEDP – right ventricular end-diastolic pressure; LVEDP – left ventricular end-diastolic pressure; PCWP – pulmonary capillary wedge pressure; SV – stroke volume; CO – cardiac output; SVV – stroke volume variation; SVRI – stroke volume variation index; PPV – pulse pressure variation; SPV – systolic pressure variation index; EVLW – extra-vascular lung water index; GEDVI – global end-diastolic volume index; ITTV – intra-thoracic thermal volume; ITBV – intra-thoracic blood volume; PBV – pulmonary blood volume.

has now fallen out of favor due to its failure to show clinical outcome benefit [51]. A drawback of current HDM devices is their inability to monitor the microcirculation [127]. Recent advances in sublingual hand held microcirculatory monitoring devices have shown promise in this regard [128], however its value with regards outcome benefit is not yet known. Large scale randomized trials are required to measure the true impact of current and newer HDM systems [129].

9. Implementation, practical application and future direction of HDM in the emergency department

HDM devices and techniques have predominantly been designed and described for use in the ICU environment. Invasive devices such as the FloTrac/Vigileo™, COstatus™, LiDCO™, PiCCO™ and Volume View/EV 1000™ are frequently available in the ICU setting. Unlike the ICU, most ED's currently do not have the capacity and capability with respect to experience, manpower and equipment to implement these invasive HDM devices. However, with appropriate training, simple techniques and devices such as the PLR maneuver coupled with ultrasonography or $ETCO_2$ monitoring is well within the realms of the ED clinician. Considering the complexity of most of these devices, one might argue that their use be restricted to academic centers. However, there is no reason that these modalities should not be implemented in non-academic centers that frequently manage complex clinical cases provided they have personnel with the appropriate expertise and training.

The emergency department is a busy, pressured environment and often an extension of the intensive care environment. An ideal hemodynamic monitoring device would be one that is reliable, validated, easy to use, safe, readily available, non-invasive, has a rapid response-time,

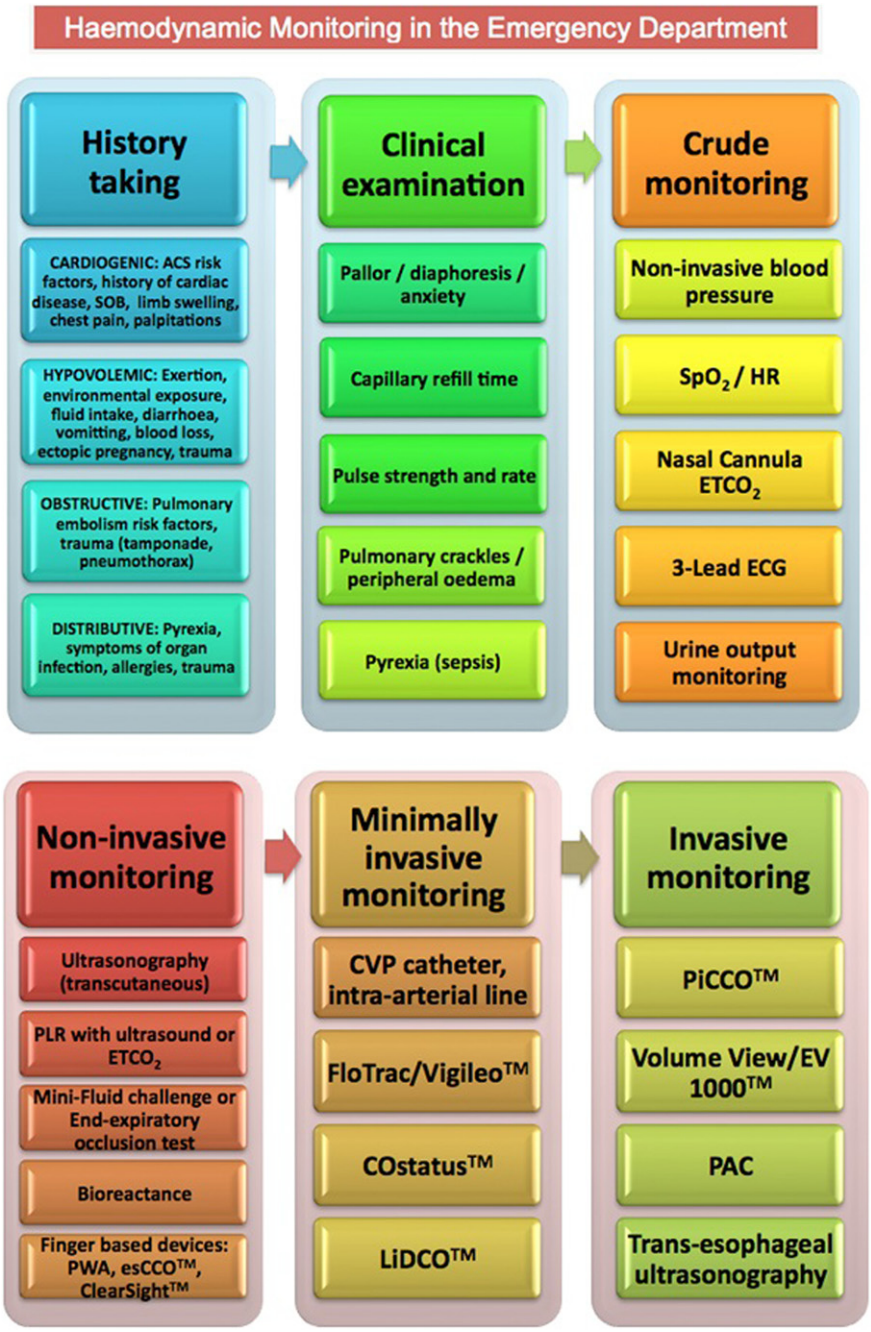

Fig. 4. An approach to hemodynamic monitoring from simple clinical assessment to invasive techniques.

applicable to the spontaneously breathing patient, not labor or time intensive, not operator dependent, cost effective and continuously provides measurements of relevant, accurate and reproducible variables. A device with all of these properties does not currently exist.

HDM methods and techniques requiring invasive monitoring devices and the need to fully ventilate and sedate/paralyze patients are generally impractical and not desired in the ED setting. However as a result of hospital overcrowding, many ED's are required to take care of critically ill patients for prolonged periods, hence necessitating the need for clinical expertise and advanced HDM devices in relevant centers [130,131]. Clinical assessment coupled with ultrasonography should always be the first step in assessing the hemodynamic state and is useful in guiding initial therapy in most clinical scenarios [132]. Despite ultrasound techniques being limited by its operator dependency, its steep learning curve and its inability to provide continuous monitoring, it is still the most practical method of hemodynamic monitoring and determining fluid responsiveness when coupled with PLR-maneuver induced changes in VTI or carotid artery blood flow time [33-36] and various ultrasound shock assessment protocols [74-81]. In addition to ultrasonography being non-invasive, it is now readily available in most ED's. With the growth of emergency medicine as a specialty internationally, emergency physicians and trainees are generally familiar with the basic principles of ultrasonography. With practice [71] and after completing specifically designed ultrasound based training courses, the non-radiologist clinician can easily acquire the necessary competence to reliably evaluate the anatomy of interest with ultrasound guidance [133,134]. Dinh and colleagues demonstrated that VTI measurements obtained by trained ED physicians correlated with that measured by certified cardiac sonographers (r 0.82) [135].

When ultrasonography is not available, the PLR-maneuver coupled with $ETCO_2$ monitoring has also shown promise in predicting fluid responsiveness [40], whilst the mini-fluid challenge and end-expiratory occlusion test may also be considered in the ventilated patient [110, 111]. Although totally non-invasive and user friendly, bioreactance based devices are marred by inconsistent evidence and require further validation [44], whereas finger based monitoring devices are dependent on good peripheral circulation making them unreliable in critically ill patients most requiring HDM [97-99]. CVP monitoring alone has no value and should not be used to determine fluid status or predict response to a fluid bolus [48]. Fig. 4 illustrates a step-wise approach to HDM ranging from simple clinical assessment to advanced monitoring methods.

In summary, for the critically ill hemodynamically unstable patient presenting to the ED, management based on clinical assessment and simple bedside monitoring is sufficient in the majority of cases. This may be supplemented with non-invasive monitoring modalities such as bedside ultrasound assessment and determination of hemodynamic variables (e.g. VTI), the mini-fluid challenge or $ETCO_2$ monitoring coupled with the PLR maneuver. Where experiences and resources exist, more advanced HDM devices and techniques must be considered, especially when a delay to ICU admission is expected. For the latter objectives to become a reality in the ED, dedicated training programs, seminars and courses in HDM aimed at the emergency clinician must be designed. Considering the paucity of hemodynamic monitoring in the Emergency Medicine literature, opportunity exist for replicating many of these published studies in the ED setting.

10. Conclusion

Hemodynamic monitoring to guide appropriate therapy is an important intervention that is commonly neglected in critically ill patients presenting to the ED. An initial approach that includes clinical assessment, basic bedside monitoring and point of care ultrasonography coupled with a PLR-maneuver is sufficient to guide appropriate therapy in most instances. However the ED clinician must be knowledgeable with regards to advanced methods/techniques of HDM which may be necessary in complex presentations.

Conflicts of interest

The authors' hereby certify that this submission is not under publication consideration elsewhere, and is free of conflict of interest.

References

[1] Lerner EB, Moscati RM. The golden hour: scientific fact or medical "urban legend"? Acad Emerg Med 2001;8:758–60.
[2] Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med 2008;36:296–327. http://dx.doi.org/10.1097/01.CCM.0000298158.12101.41.
[3] Dubinsky I. Time is muscle. Can Fam Physician Médecin Fam Can 1996;42:1269–70 [1278–9].
[4] Saver JL. Time is brain–quantified. Stroke 2006;37:263–6. http://dx.doi.org/10.1161/01.STR.0000196957.55928.ab.
[5] Simpson HK, Clancy M, Goldfrad C, Rowan K. Admissions to intensive care units from emergency departments: a descriptive study. Emerg Med J 2005;22:423–8. http://dx.doi.org/10.1136/emj.2003.005124.
[6] Hogan AJ, Bouknight RR. The emergency department as a gateway for hospitalization: evidence from the National Medical Expenditure Survey. J Health Care Poor Underserved 2002;13:288–97.
[7] Nguyen HB, Huang DT, Pinsky MR. Hemodynamic monitoring. In: Tintinalli JE, Stapczynski JS, Ma OJ, Yealy DM, Meckler GD, Cline DM, editors. Tintinalli's emergency medicine A comprehensive study guide. McGraw-Hill Education: New York, NY; 2016.
[8] Maurer C, Wagner JY, Schmid RM, Saugel B. Assessment of volume status and fluid responsiveness in the emergency department: a systematic approach. Medizinische Klin - Intensivmed Und Notfallmedizin 2015. http://dx.doi.org/10.1007/s00063-015-0124-x.
[9] García X, Pinsky MR. Clinical applicability of functional hemodynamic monitoring. Ann Intensive Care 2011;1:35. http://dx.doi.org/10.1186/2110-5820-1-35.
[10] Joseph Varon REJF. Handbook of practical critical care medicine. Illustrated. Springer Science & Business Media; 2012.
[11] Hamzaoui O, Monnet X, Teboul JL. Evolving concepts of hemodynamic monitoring for critically ill patients. Indian J Crit Care Med 2015;19:220–6. http://dx.doi.org/10.4103/0972-5229.154556.
[12] Monnet X, Julien F, Ait-Hamou N, Lequoy M, Gosset C, Jozwiak M, et al. Lactate and venoarterial carbon dioxide difference/arterial-venous oxygen difference ratio, but not central venous oxygen saturation, predict increase in oxygen consumption in fluid responders. Crit Care Med 2013;41:1412–20. http://dx.doi.org/10.1097/CCM.0b013e318275cece.
[13] Wernerman J, Sigurdsson GH. Intravenous fluid management in critically ill patients: for everybody or for a dedicated team? Acta Anaesthesiol Scand 2016;60:831. http://dx.doi.org/10.1111/aas.12720.
[14] Malbrain ML, Reuter DA. Hemodynamic treatment algorithms should follow physiology or they fail to improve outcome. Crit Care Med 2012;40:2923–4. http://dx.doi.org/10.1097/CCM.0b013e31825f6cd0.
[15] Pinsky MR. Functional haemodynamic monitoring. Curr Opin Crit Care 2014;20:288–93. http://dx.doi.org/10.1097/MCC.0000000000000090.
[16] Vos JJ, Scheeren TWL. How to "validate" newly developed cardiac output monitoring devices. J Clin Monit Comput 2016;30:147–8. http://dx.doi.org/10.1007/s10877-015-9793-5.
[17] Cecconi M, de Backer D, Antonelli M, Beale R, Bakker J, Hofer C, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med 2014;40:1795–815. http://dx.doi.org/10.1007/s00134-014-3525-z.
[18] Michard F, Teboul J-L. Predicting fluid responsiveness in ICU patients. Chest 2002;121:2000–8. http://dx.doi.org/10.1378/chest.121.6.2000.
[19] Marik PE, Monnet X, Teboul J-L. Hemodynamic parameters to guide fluid therapy. Ann Intensive Care 2011;1:1. http://dx.doi.org/10.1186/2110-5820-1-1.
[20] Benes J, Kirov M, Kuzkov V, Lainscak M, Molnar Z, Voga G, et al. Fluid therapy: double-edged sword during critical care? Biomed Res Int 2015;2015. http://dx.doi.org/10.1155/2015/729075.
[21] Woodcock TE, Woodcock TM. Revised Starling equation and the glycocalyx model of transvascular fluid exchange: an improved paradigm for prescribing intravenous fluid therapy. Br J Anaesth 2012;aer515. http://dx.doi.org/10.1093/bja/aer515.
[22] Braunwald E, Sonnenblick E, Ross J. Mechanism of cardiac contraction and relaxation. Hear Dis, Philadelphia: WB Saunders 1988:383–425.
[23] Vincent J-L, Weil MH. Fluid challenge revisited. Crit Care Med 2006;34:1333–7. http://dx.doi.org/10.1097/01.CCM.0000214677.76535.A5.
[24] Boyd JH, Forbes J, Nakada T, Walley KR, Russell JA. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. Crit Care Med 2011;39:259–65. http://dx.doi.org/10.1097/CCM.0b013e3181feeb15.
[25] Sadaka F, Juarez M, Naydenov S, O'Brien J. Fluid resuscitation in septic shock. J Intensive Care Med 2013;885066613478899. http://dx.doi.org/10.1177/0885066613478899.
[26] Detector performance analysis using ROC curves n.d. http://www.mathworks.com/help/phased/examples/detector-performance-analysis-using-roc-curves.html?requestedDomain=www.mathworks.com (accessed September 19, 2016).
[27] Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a

systematic review of the literature. Crit Care Med 2009;37:2642–7. http://dx.doi.org/10.1097/CCM.0b013e3181a590da.
[28] Montenij LJ, de Waal EEC, Buhre WF. Arterial waveform analysis in anesthesia and critical care. Curr Opin Anaesthesiol 2011;24:651–6. http://dx.doi.org/10.1097/ACO.0b013e32834cd2d9.
[29] Benington S, Ferris P, Nirmalan M. Emerging trends in minimally invasive haemodynamic monitoring and optimization of fluid therapy. Eur J Anaesthesiol 2009;26:893–905. http://dx.doi.org/10.1097/EJA.0b013e3283308e50.
[30] Mahjoub Y, Touzeau J, Airapetian N, Lorne E, Hijazi M, Zogheib E, et al. The passive leg-raising maneuver cannot accurately predict fluid responsiveness in patients with intra-abdominal hypertension. Crit Care Med 2010;38:1824–9. http://dx.doi.org/10.1097/CCM.0b013e3181eb3c21.
[31] Monnet X, Rienzo M, Osman D, Anguel N, Richard C, Pinsky MR, et al. Passive leg raising predicts fluid responsiveness in the critically ill. Crit Care Med 2006;34:1402–7. http://dx.doi.org/10.1097/01.CCM.0000215453.11735.06.
[32] Monnet X, Marik P, Teboul J-L. Passive leg raising for predicting fluid responsiveness: a systematic review and meta-analysis. Intensive Care Med 2016. http://dx.doi.org/10.1007/s00134-015-4134-1.
[33] Lamia B, Ochagavia A, Monnet X, Chemla D, Richard C, Teboul J-L. Echocardiographic prediction of volume responsiveness in critically ill patients with spontaneously breathing activity. Intensive Care Med 2007;33:1125–32. http://dx.doi.org/10.1007/s00134-007-0646-7.
[34] Maizel J, Airapetian N, Lorne E, Tribouilloy C, Massy Z, Slama M. Diagnosis of central hypovolemia by using passive leg raising. Intensive Care Med 2007;33:1133–8. http://dx.doi.org/10.1007/s00134-007-0642-y.
[35] Marik PE, Levitov A, Young A, Andrews L. The use of bioreactance and carotid Doppler to determine volume responsiveness and blood flow redistribution following passive leg raising in hemodynamically unstable patients. Chest 2013;143:364–70. http://dx.doi.org/10.1378/chest.12-1274.
[36] Mackenzie DC, Khan NA, Blehar D, Glazier S, Chang Y, Stowell CP, et al. Carotid flow time changes with volume status in acute blood loss. Ann Emerg Med 2015;66:277–282.e1. http://dx.doi.org/10.1016/j.annemergmed.2015.04.014.
[37] Mackenzie D, Khan N, Blehar D, Stowell C, Noble V, Liteplo A. Ultrasound measurement of carotid flow time changes with volume status. Crit Care 2014;18:P131. http://dx.doi.org/10.1186/cc13321.
[38] Hossein-Nejad H, Mohammadinejad P, Lessan-Pezeshki M, Davarani SS, Banaie M. Carotid artery corrected flow time measurement via bedside ultrasonography in monitoring volume status. J Crit Care 2015;30:1199–203. http://dx.doi.org/10.1016/j.jcrc.2015.08.014.
[39] Blehar DJ, Glazier S, Gaspari RJ. Correlation of corrected flow time in the carotid artery with changes in intravascular volume status. J Crit Care 2014;29:486–8. http://dx.doi.org/10.1016/j.jcrc.2014.03.025.
[40] Monnet X, Bataille A, Magalhaes E, Barrois J, le Corre M, Gosset C, et al. End-tidal carbon dioxide is better than arterial pressure for predicting volume responsiveness by the passive leg raising test. Intensive Care Med 2013;39:93–100. http://dx.doi.org/10.1007/s00134-012-2693-y.
[41] Monge García MI, Gil Cano A, Gracia Romero M, Monterroso Pintado R, Pérez Madueño V, Díaz Monrové JC. Non-invasive assessment of fluid responsiveness by changes in partial end-tidal CO2 pressure during a passive leg-raising maneuver. Ann Intensive Care 2012;2:9. http://dx.doi.org/10.1186/2110-5820-2-9.
[42] Pinto A, Pinto F, Faggian A, Rubini G, Caranci F, Macarini L, et al. Sources of error in emergency ultrasonography. Crit Ultrasound J 2013;5(Suppl. 1):S1. http://dx.doi.org/10.1186/2036-7902-5-S1-S1.
[43] Dentinger AM, Hoctor RT. Non-invasive hemodynamic state monitoring using ultrasound. Conf Proc IEEE Eng Med Biol Soc 2010;2010:5306–9. http://dx.doi.org/10.1109/IEMBS.2010.5626322.
[44] Kupersztych-Hagege E, Teboul J-L, Artigas A, Talbot A, Sabatier C, Richard C, et al. Bioreactance is not reliable for estimating cardiac output and the effects of passive leg raising in critically ill patients. Br J Anaesth 2013;111:961–6. http://dx.doi.org/10.1093/bja/aet282.
[45] Westphal GA, Silva E, Gonçalves AR, Caldeira Filho M, Poli-de-Figueiredo LF. Pulse oximetry wave variation as a noninvasive tool to assess volume status in cardiac surgery. Clin (São Paulo, Brazil) 2009;64:337–43. http://dx.doi.org/10.1590/s1807-59322009000400012.
[46] Permpikul C, Leelayuthachai T. Non-invasive estimated continuous cardiac output (escCO) during severe sepsis and septic shock resuscitation. J Med Assoc Thai 2014;97(Suppl. 3):S184–8.
[47] Ameloot K, Palmers P-J, Malbrain MLNG. The accuracy of noninvasive cardiac output and pressure measurements with finger cuff: a concise review. Curr Opin Crit Care 2015;21:232–9. http://dx.doi.org/10.1097/MCC.0000000000000198.
[48] Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 2008;134:172–8. http://dx.doi.org/10.1378/chest.07-2331.
[49] Argueta E, Berdine G, Pena C, Nugent KM. FloTrac® monitoring system: what are its uses in critically ill medical patients? Am J Med Sci 2015;349:352–6. http://dx.doi.org/10.1097/MAJ.0000000000000393.
[50] Crittendon I, Dreyer WJ, Decker JA, Kim JJ. Ultrasound dilution: an accurate means of determining cardiac output in children. Pediatr Crit Care Med 2012;13:42–6. http://dx.doi.org/10.1097/PCC.0b013e3182196804.
[51] Rajaram SS, Desai NK, Kalra A, Gajera M, Cavanaugh SK, Brampton W, et al. Pulmonary artery catheters for adult patients in intensive care. Cochrane Database Syst Rev 2013:CD003408. http://dx.doi.org/10.1002/14651858.CD003408.pub3.
[52] Litton E, Morgan M. The PiCCO monitor: a review. Anaesth Intensive Care 2012;40:393–409.
[53] 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. http://dx.doi.org/10.1186/cc2852.
[54] Kiefer N, Hofer CK, Marx G, Geisen M, Giraud R, Siegenthaler N, et al. Clinical validation of a new thermodilution system for the assessment of cardiac output and volumetric parameters. Crit Care 2012;16:R98. http://dx.doi.org/10.1186/cc11366.
[55] Guerin L, Monnet X, Teboul J-L, Marik PE, Monnet X, Teboul JL, et al. Monitoring volume and fluid responsiveness: from static to dynamic indicators. Best Pract Res Clin Anaesthesiol 2013;27:177–85. http://dx.doi.org/10.1016/j.bpa.2013.06.002.
[56] Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. JAMA 1989;261:884–8.
[57] Hoste EAJ, Bagshaw SM, Bellomo R, Cely CM, Colman R, Cruz DN, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. Intensive Care Med 2015;41:1411–23. http://dx.doi.org/10.1007/s00134-015-3934-7.
[58] Esper AM, Martin GS. The impact of comorbid [corrected] conditions on critical illness. Crit Care Med 2011;39:2728–35. http://dx.doi.org/10.1097/CCM.0b013e318236f27e.
[59] Gattinoni L, Brazzi L, Pelosi P, Latini R, Tognoni G, Pesenti A, et al. A trial of goal-oriented hemodynamic therapy in critically ill patients. SvO2 Collaborative Group. N Engl J Med 1995;333:1025–32. http://dx.doi.org/10.1056/NEJM199510193331601.
[60] Vincent J-L, Rhodes A, Perel A, Martin GS, Della Rocca G, Vallet B, et al. Clinical review: update on hemodynamic monitoring–a consensus of 16. Crit Care 2011;15:229. http://dx.doi.org/10.1186/cc10291.
[61] Velissaris D, Pierrakos C, Scolletta S, de Backer D, Vincent JL. High mixed venous oxygen saturation levels do not exclude fluid responsiveness in critically ill septic patients. Crit Care 2011;15:R177. http://dx.doi.org/10.1186/cc10326.
[62] Teboul J-L, Hamzaoui O, Monnet X. SvO2 to monitor resuscitation of septic patients: let's just understand the basic physiology. Crit Care 2011;15:1005. http://dx.doi.org/10.1186/cc10491.
[63] Dellinger RP, Levy MM, Rhodes A, Annane D, Gerlach H, Opal SM, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2012. Crit Care Med 2013;41:580–637. http://dx.doi.org/10.1097/CCM.0b013e31827e83af.
[64] Kirkpatrick AW, Roberts DJ, de Waele J, Jaeschke R, Malbrain MLNG, de Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med 2013;39:1190–206. http://dx.doi.org/10.1007/s00134-013-2906-z.
[65] Carney NA, Ghajar J. Guidelines for the management of severe traumatic brain injury. Introduction. J Neurotrauma 2007;24(Suppl. 1):S1–2. http://dx.doi.org/10.1089/neu.2007.9997.
[66] James M, Bouchard J, Ho J, Klarenbach S, LaFrance J-P, Rigatto C, et al. Canadian Society of Nephrology commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. Am J Kidney Dis 2013;61:673–85. http://dx.doi.org/10.1053/j.ajkd.2013.02.350.
[67] Lionakis N, Mendrinos D, Sanidas E, Favatas G, Georgopoulou M. Hypertension in the elderly. World J Cardiol 2012;4:135–47. http://dx.doi.org/10.4330/wjc.v4.i5.135.
[68] Augusto J-F, Teboul J-L, Radermacher P, Asfar P. Interpretation of blood pressure signal: physiological bases, clinical relevance, and objectives during shock states. Intensive Care Med 2011;37:411–9. http://dx.doi.org/10.1007/s00134-010-2092-1.
[69] Pierrakos C, Velissaris D, Scolletta S, Heenen S, De Backer D, Vincent J-L. Can changes in arterial pressure be used to detect changes in cardiac index during fluid challenge in patients with septic shock? Intensive Care Med 2012;38:422–8. http://dx.doi.org/10.1007/s00134-011-2457-0.
[70] Qi H, Gu Q, Liu N, Zhang B. Mean arterial pressure as an indicator of fluid responsiveness in patients with septic shock. Zhonghua Wei Zhong Bing Ji Jiu Yi Xue 2013;25:32–5. http://dx.doi.org/10.3760/cma.j.issn.2095-4352.2013.01.009.
[71] Laher AE, Wells M. ED neuro-ultrasonography and the mental foramen: practice makes perfect. Am J Emerg Med 2016;34:2240–1.
[72] Coşkun F, Akıncı E, Ceyhan MA, Sahin Kavaklı H. Our new stethoscope in the emergency department: handheld ultrasound. Ulus Travma Acil Cerrahi Derg 2011;17:488–92.
[73] Claudon M, Guillaume L. Diagnostic imaging and radiation hazards. Rev Prat 2015;65:79–81.
[74] Perera P, Mailhot T, Riley D, Mandavia D. The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically lll. Emerg Med Clin North Am 2010;28:29–56 vii 10.1016/j.emc.2009.09.010.
[75] Breitkreutz R, Price S, Steiger HV, Seeger FH, Ilper H, Ackermann H, et al. Focused echocardiographic evaluation in life support and peri-resuscitation of emergency patients: a prospective trial. Resuscitation 2010;81:1527–33. http://dx.doi.org/10.1016/j.resuscitation.2010.07.013.
[76] Kirkpatrick AW, Sirois M, Laupland KB, Liu D, Rowan K, Ball CG, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the Extended Focused Assessment with Sonography for Trauma (EFAST). J Trauma 2004;57:288–95.
[77] Rapid assessment by cardiac echo + (RACEplus) n.d. https://www.ttsh.com.sg/RACEplus-course/ (accessed September 20, 2016).
[78] Advanced focused cardiothoracic ultrasound n.d. http://usabcd.org/node/76 (accessed September 20, 2016).
[79] Romero-Bermejo FJ, Ruiz-Bailen M, Guerrero-De-Mier M, Lopez-Alvaro J. Echocardiographic hemodynamic monitoring in the critically ill patient. Curr Cardiol Rev 2011;7:146–56. http://dx.doi.org/10.2174/157340311798220485.
[80] Chew MS. Haemodynamic monitoring using echocardiography in the critically ill: a review. Cardiol Res Pract 2012;2012:139537. http://dx.doi.org/10.1155/2012/139537.
[81] McLean AS. Echocardiography in shock management. Crit Care 2016;20:275. http://dx.doi.org/10.1186/s13054-016-1401-7.
[82] Husain LF, Hagopian L, Wayman D, Baker WE, Carmody KA. Sonographic diagnosis of pneumothorax. J Emerg Trauma Shock 2012;5:76–81. http://dx.doi.org/10.4103/0974-2700.93116.
[83] Ristić AD, Imazio M, Adler Y, Anastasakis A, Badano LP, Brucato A, et al. Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J 2014;35:2279–84. http://dx.doi.org/10.1093/eurheartj/ehu217.

[84] Jardin F, Dubourg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. Chest 1997;111:209–17.
[85] Frazee BW, Snoey ER, Levitt A. Emergency Department compression ultrasound to diagnose proximal deep vein thrombosis. J Emerg Med 2001;20:107–12.
[86] Monnet X, Rienzo M, Osman D, Anguel N, Richard C, Pinsky MR, et al. Esophageal Doppler monitoring predicts fluid responsiveness in critically ill ventilated patients. Intensive Care Med 2005;31:1195–201. http://dx.doi.org/10.1007/s00134-005-2731-0.
[87] Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. Chest 2001;119:867–73.
[88] Feissel M, Michard F, Faller J-P, Teboul J-L. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. Intensive Care Med 2004;30:1834–7. http://dx.doi.org/10.1007/s00134-004-2233-5.
[89] Barbier C, Loubières Y, Schmit C, Hayon J, Ricôme J-L, Jardin F, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. Intensive Care Med 2004;30:1740–6. http://dx.doi.org/10.1007/s00134-004-2259-8.
[90] Rudski L, Lai W, Afilalo J, Hua L, Handschumacher M, Chandrasekaran K, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and t. J Am Soc Echocardiogr 2010;23:685–713. http://dx.doi.org/10.1016/j.echo.2010.05.010.
[91] Nagdev AD, Merchant RC, Tirado-Gonzalez A, Sisson CA, Murphy MC. Emergency department bedside ultrasonographic measurement of the caval index for noninvasive determination of low central venous pressure. Ann Emerg Med 2010;55:290–5. http://dx.doi.org/10.1016/j.annemergmed.2009.04.021.
[92] Vieillard-Baron A, Chergui K, Rabiller A, Peyrouset O, Page B, Beauchet A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. Intensive Care Med 2004;30:1734–9. http://dx.doi.org/10.1007/s00134-004-2361-y.
[93] Benomar B, Ouattara A, Estagnasie P, Brusset A, Squara P. Fluid responsiveness predicted by noninvasive bioreactance-based passive leg raise test. Intensive Care Med 2010;36:1875–81. http://dx.doi.org/10.1007/s00134-010-1990-6.
[94] Sandroni C, Cavallaro F, Marano C, Falcone C, de Santis P, Antonelli M. Accuracy of plethysmographic indices as predictors of fluid responsiveness in mechanically ventilated adults: a systematic review and meta-analysis. Intensive Care Med 2012;38:1429–37. http://dx.doi.org/10.1007/s00134-012-2621-1.
[95] Feissel M, Kalakhy R, Banwarth P, Badie J, Pavon A, Faller J-P, et al. Plethysmographic variation index predicts fluid responsiveness in ventilated patients in the early phase of septic shock in the emergency department: a pilot study. J Crit Care 2013;28:634–9. http://dx.doi.org/10.1016/j.jcrc.2013.03.011.
[96] Keller G, Cassar E, Desebbe O, Lehot J-J, Cannesson M. Ability of pleth variability index to detect hemodynamic changes induced by passive leg raising in spontaneously breathing volunteers. Crit Care 2008;12:R37. http://dx.doi.org/10.1186/cc6822.
[97] Broch O, Bein B, Gruenewald M, Hocker J, Schottler J, Meybohm P, et al. Accuracy of the pleth variability index to predict fluid responsiveness depends on the perfusion index. Acta Anaesthesiol Scand 2011;55:686–93. http://dx.doi.org/10.1111/j.1399-6576.2011.02435.x.
[98] Antonelli M, Levy M, Andrews PJD, Chastre J, Hudson LD, Manthous C, et al. Hemodynamic monitoring in shock and implications for management. International consensus conference, Paris, France, 27-28 April 2006. Intensive Care Med 2007;33:575–90. http://dx.doi.org/10.1007/s00134-007-0531-4.
[99] Biais M, Cottenceau V, Petit L, Masson F, Cochard J-F, Sztark F. Impact of norepinephrine on the relationship between pleth variability index and pulse pressure variations in ICU adult patients. Crit Care 2011;15:R168. http://dx.doi.org/10.1186/cc10310.
[100] Desgranges F-P, Desebbe O, Ghazouani A, Gilbert K, Keller G, Chiari P, et al. Influence of the site of measurement on the ability of plethysmographic variability index to predict fluid responsiveness. Br J Anaesth 2011;107:329–35. http://dx.doi.org/10.1093/bja/aer165.
[101] Smetkin AA, Hussain A, Fot EV, Zakharov VI, Izotova NN, Yudina AS, et al. Estimated continuous cardiac output based on pulse wave transit time in off-pump coronary artery bypass grafting: a comparison with transpulmonary thermodilution. J Clin Monit Comput 2016. http://dx.doi.org/10.1007/s10877-016-9853-5.
[102] Terada T, Oiwa A, Maemura Y, Robert S, Kessoku S, Ochiai R. Comparison of the ability of two continuous cardiac output monitors to measure trends in cardiac output: estimated continuous cardiac output measured by modified pulse wave transit time and an arterial pulse contour-based cardiac output device. J Clin Monit Comput 2016;30:621–7. http://dx.doi.org/10.1007/s10877-015-9772-x.
[103] Biais M, Berthezène R, Petit L, Cottenceau V, Sztark F. Ability of esCCO to track changes in cardiac output. Br J Anaesth 2015;115:403–10. http://dx.doi.org/10.1093/bja/aev219.
[104] Feissel M, Aho LS, Georgiev S, Tapponnier R, Badie J, Bruyère R, et al. Pulse wave transit time measurements of cardiac output in septic shock patients: a comparison of the estimated continuous cardiac output system with transthoracic echocardiography. PLoS One 2015;10:e0130489. http://dx.doi.org/10.1371/journal.pone.0130489.
[105] Bataille B, Bertuit M, Mora M, Mazerolles M, Cocquet P, Masson B, et al. Comparison of esCCO and transthoracic echocardiography for non-invasive measurement of cardiac output intensive care. Br J Anaesth 2012;109:879–86. http://dx.doi.org/10.1093/bja/aes298.
[106] Fischer M-O, Coucoravas J, Truong J, Zhu L, Gérard J-L, Hanouz J-L, et al. Assessment of changes in cardiac index and fluid responsiveness: a comparison of Nexfin and transpulmonary thermodilution. Acta Anaesthesiol Scand 2013;57:704–12. http://dx.doi.org/10.1111/aas.12108.
[107] Lansdorp B, Ouweneel D, de Keijzer A, van der Hoeven JG, Lemson J, Pickkers P. Non-invasive measurement of pulse pressure variation and systolic pressure variation using a finger cuff corresponds with intra-arterial measurement. Br J Anaesth 2011;107:540–5. http://dx.doi.org/10.1093/bja/aer187.
[108] Vincent J-L. "Let's give some fluid and see what happens" versus the "mini-fluid challenge". Anesthesiology 2011;115:455–6. http://dx.doi.org/10.1097/ALN.0b013e318229a521.
[109] Muller L, Toumi M, Bousquet P-J, Riu-Poulenc B, Louart G, Candela D, et al. An increase in aortic blood flow after an infusion of 100 ml colloid over 1 minute can predict fluid responsiveness: the mini-fluid challenge study. Anesthesiology 2011;115:541–7. http://dx.doi.org/10.1097/ALN.0b013e318229a500.
[110] Mallat J, Meddour M, Durville E, Lemyze M, Pepy F, Temime J, et al. Decrease in pulse pressure and stroke volume variations after mini-fluid challenge accurately predicts fluid responsiveness†. Br J Anaesth 2015;115:449–56. http://dx.doi.org/10.1093/bja/aev222.
[111] Monnet X, Osman D, Ridel C, Lamia B, Richard C, Teboul J-L. Predicting volume responsiveness by using the end-expiratory occlusion in mechanically ventilated intensive care unit patients. Crit Care Med 2009;37:951–6. http://dx.doi.org/10.1097/CCM.0b013e3181968fe1.
[112] Hoeft A, Schorn B, Weyland A, Scholz M, Buhre W, Stepanek E, et al. Bedside assessment of intravascular volume status in patients undergoing coronary bypass surgery. Anesthesiology 1994;81:76–86.
[113] Wagner JG, Leatherman JW. Right ventricular end-diastolic volume as a predictor of the hemodynamic response to a fluid challenge. Chest 1998;113:1048–54.
[114] Oohashi S, Endoh H. Does central venous pressure or pulmonary capillary wedge pressure reflect the status of circulating blood volume in patients after extended transthoracic esophagectomy? J Anesth 2005;19:21–5. http://dx.doi.org/10.1007/s00540-004-0282-0.
[115] Kumar A, Anel R, Bunnell E, Habet K, Zanotti S, Marshall S, et al. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. Crit Care Med 2004;32:691–9.
[116] Gödje O, Peyerl M, Seebauer T, Lamm P, Mair H, Reichart B. Central venous pressure, pulmonary capillary wedge pressure and intrathoracic blood volumes as preload indicators in cardiac surgery patients. Eur J Cardiothorac Surg 1998;13:539–40.
[117] Jones AE, Puskarich MA. The Surviving Sepsis Campaign guidelines 2012: update for emergency physicians. Ann Emerg Med 2014;63:35–47. http://dx.doi.org/10.1016/j.annemergmed.2013.08.004.
[118] Evans DC, Doraiswamy VA, Prosciak MP, Silviera M, Seamon MJ, Rodriguez Funes V, et al. Complications associated with pulmonary artery catheters: a comprehensive clinical review. Scand J Surg 2009;98:199–208.
[119] Mayer J, Boldt J, Poland R, Peterson A, Manecke GR. Continuous arterial pressure waveform-based cardiac output using the FloTrac/Vigileo: a review and meta-analysis. J Cardiothorac Vasc Anesth 2009;23:401–6. http://dx.doi.org/10.1053/j.jvca.2009.03.003.
[120] Galstyan G, Bychinin M, Alexanyan M, Gorodetsky V. Comparison of cardiac output and blood volumes in intrathoracic compartments measured by ultrasound dilution and transpulmonary thermodilution methods. Intensive Care Med 2010;36:2140–4. http://dx.doi.org/10.1007/s00134-010-2003-5.
[121] Linton RA, Band DM, Haire KM. A new method of measuring cardiac output in man using lithium dilution. Br J Anaesth 1993;71:262–6.
[122] Oren-Grinberg A. The PiCCO Monitor. Int Anesthesiol Clin 2010;48:57–85. http://dx.doi.org/10.1097/AIA.0b013e3181c3dc11.
[123] Sakka SG, Klein M, Reinhart K, Meier-Hellmann A. Prognostic value of extravascular lung water in critically ill patients. Chest 2002;122:2080–6.
[124] Jozwiak M, Teboul J-L, Monnet X. Extravascular lung water in critical care: recent advances and clinical applications. Ann Intensive Care 2015;5:38. http://dx.doi.org/10.1186/s13613-015-0081-9.
[125] Chan Y-K, Khan ZH. Hemodynamic monitoring and outcome—a physiological appraisal. Taiwanica]–>Acta Anaesthesiol Taiwan 2011;49:154–8. http://dx.doi.org/10.1016/j.aat.2011.11.002.
[126] Downs EA, Isbell JM. Impact of hemodynamic monitoring on clinical outcomes. Best Pract Res Clin Anaesthesiol 2014;28:463–76. http://dx.doi.org/10.1016/j.bpa.2014.09.009.
[127] Ince C. Hemodynamic coherence and the rationale for monitoring the microcirculation. Crit Care 2015;19(Suppl. 3):S8. http://dx.doi.org/10.1186/cc14726.
[128] Kara A, Akin S, Ince C. Monitoring microcirculation in critical illness. Curr Opin Crit Care 2016;22:444–52. http://dx.doi.org/10.1097/MCC.0000000000000335.
[129] Ramsingh D, Alexander B, Cannesson M. Clinical review: does it matter which hemodynamic monitoring system is used? Crit Care 2013;17:208. http://dx.doi.org/10.1186/cc11814.
[130] Jayaprakash N, O'Sullivan R, Bey T, Ahmed SS, Lotfipour S. Crowding and delivery of healthcare in emergency departments: the European perspective. West J Emerg Med 2009;10:233–9.
[131] Langhan TS. Do elective surgical and medical admissions impact emergency department length of stay measurements? Clin Investig Med Médecine Clin Exp 2007;30:E177–82.
[132] Mackenzie DC, Noble VE. Assessing volume status and fluid responsiveness in the emergency department. Clin Exp Emerg Med 2014;1:67–77. http://dx.doi.org/10.15441/ceem.14.040.
[133] Abbasi S, Farsi D, Hafezimoghadam P, Fathi M, Zare MA. Accuracy of emergency physician-performed ultrasound in detecting traumatic pneumothorax after a 2-h training course. Eur J Emerg Med 2013;20:173–7. http://dx.doi.org/10.1097/MEJ.0b013e328356f754.
[134] Walker A, Brenchley J, Sloan JP, Lalanda M, Venables H. Ultrasound by emergency physicians to detect abdominal aortic aneurysms: a UK case series. Emerg Med J 2004;21:257–9.
[135] Dinh VA, Ko HS, Rao R, Bansal RC, Smith DD, Kim TE, et al. Measuring cardiac index with a focused cardiac ultrasound examination in the ED. Am J Emerg Med 2012;30:1845–51. http://dx.doi.org/10.1016/j.ajem.2012.03.025.