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### Ultrasound-guided haemodynamic state assessment

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The haemodynamic state refers to the integration of myocardial and vascular systems, and involves both left and right hearts, and systolic and diastolic phases. The assessment of the haemodynamic state can be performed with echocardiography, and provides a higher level of diagnosis than conventional pressure- and flow-based monitoring. Whilst hypotension alerts the practitioner about the existence of haemodynamic abnormality, it does not provide sufficient information to identify the cause or the underlying haemodynamic state. The premise of haemodynamic state monitoring is that better diagnosis will lead to more rational therapy, which in turn may improve the outcome.

The haemodynamic state can be classified into seven broad categories: normal, empty, vasodilation, systolic failure, primary diastolic failure, systolic and diastolic failure and right ventricular failure. These are identified as patterns based upon ventricular size, ventricular function and left atrial (LA) filling pressure. Patients may have an abnormal haemodynamic state (such as a systolic failure), but may not need active treatment if they are haemodynamically stable. However, if treatment is required, it can be directed according to the underlying haemodynamic state. For example, a patient with systolic failure may benefit from inotrope support, whereas an empty state acquires volume infusion and vasodilation requires vasopressor support.

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Haemodynamic assessment is a core skill of all practitioners involved in managing critically ill patients. Conventional haemodynamic monitoring includes vital signs, such as heart rate and blood pressure, or pressure- and flow-based measurements, such as central venous or pulmonary artery

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pressure monitoring, and cardiac output. Whilst these are useful measurements to monitor the change in a patient's condition, they are not particularly effective in identifying what the underlying haemodynamic abnormality is.

The following example is illustrative: an elderly patient undergoing surgery for abdominal aortic aneurysm repair becomes profoundly hypotensive after induction of general anaesthesia. The heart rate is 80 beats per min, pulmonary capillary wedge pressure (PCWP) is 15 mmHg and cardiac index is  $1.8 \text{ l min}^{-1} \text{ m}^{-2}$ . This conventional monitoring allows the clinician to identify the presence of a serious haemodynamic abnormality, but it is neither diagnostic of what the underlying haemodynamic state is, nor of the cause. In this example, the patient could have a dilated and poorly contracting heart, or a small and stiff left ventricle (LV) or even right ventricular failure.

Echocardiography has been used as a diagnostic monitor in critically ill patients who present hypotension, low cardiac output, pulmonary oedema or metabolic derangement such as increased acidosis. It has repeatedly been shown that in critically ill patients, the use of echocardiography leads to a change in diagnosis and change in haemodynamic management in 40–60% of the cases.<sup>1–4</sup> This has traditionally been performed using trans-oesophageal echocardiography (TOE); however, if imaging is adequate, then the same information can be obtained using trans-thoracic echocardiography (TTE). Left ventricular volume and systolic function can be directly assessed using echocardiography, rather than being inferred from pressure and flow assessment.<sup>5</sup> Echocardiography can also be used to identify the cause of haemodynamic assessment, such as valve pathology, or systolic wall motion abnormality that may be consistent with ischaemia or infarction.

The haemodynamic state is the integration of both myocardial and vascular systems, and involves both left and right hearts, and systolic and diastolic phases of the cardiac cycle. Abnormality in any of these parts of the cardiovascular system can lead to haemodynamic abnormality. Equally, the patient can have an abnormal haemodynamic state (e.g., severe systolic failure), but not suffer haemodynamic compromise. The use of cardiac ultrasound (whether trans-thoracic or trans-oesophageal) is quick and accurately categorises the underlying haemodynamic state.

The haemodynamic state can be broadly categorised into seven states (see Table 1):

- a. normal
- b. empty (hypovolaemia)
- c. primary diastolic failure (diastolic heart failure, or heart failure with normal ejection fraction (EF))
- d. primary systolic failure
- e. systolic and diastolic failure
- f. right ventricular failure
- g. vasodilation

### Practice tips

- Hypotension is a sign that there is an underlying haemodynamic problem.
- Hypotension does not define the cause of the problem.
- Rational treatment follows accurate diagnosis of the underlying haemodynamic state.

### Using echocardiography to determine haemodynamic state

There are four steps to determine the basic haemodynamic assessment:

1. Estimate volume
2. Estimate systolic function
3. Estimate filling pressure
4. Final assessment (putting it all together)

**Table 1**  
Haemodynamic states and approaches to therapy.

Haemodynamic State	Characteristics	Treatment approach
Normal	Normal volume, function and left atrial pressure. Normal vascular resistance	None required
Empty	Reduced ventricular volume but normal function and low atrial pressure	Infuse volume
Primary diastolic failure	Small, stiff left ventricle with preserved systolic function. High left atrial filling pressure	Control heart rate, maintain preload, treat concomitant vasodilation with a vasopressor, consider low-dose inotropes to support right ventricular function, but avoid high-dose inotropes if possible.
Primary systolic failure	Dilated and poorly contracting left ventricle. Normal left atrial filling pressure	These patients are often quite stable under anaesthesia, inotropes are appropriate if global systolic performance is inadequate
Systolic and diastolic failure	Dilated and poorly contracting left ventricle, but with high left atrial filling pressure.	The key principle is to improve systolic function at a reduced preload, producing an adequate cardiac output at a lower left atrial pressure. Therapy includes inodilators, reducing preload (e.g. diuretics), increasing heart rate, and avoiding therapies that may increase pressure around the heart (such as high PEEP)
Vasodilation	(usually) well filled left ventricle with increased ejection fraction and normal left atrial pressure	vasoconstrictors
Right ventricular failure	Dilated and poorly contracting right heart, with raised left and right atrial filling pressure. The dilated right heart compresses the left ventricle, causing left ventricular diastolic dysfunction.	Treatment is similar to that for systolic and diastolic failure; inodilators that also reduce pulmonary vascular resistance such as dobutamine or milrinone may be preferable. Consider nitric oxide in refractory states.

### Step 1: estimate left ventricular end diastolic volume (Preload)

*Categorise as hypovolaemic, normal or dilated*

Prior to the introduction of echocardiography, the estimation of left ventricular volume was 'inferred' from measuring the right atrial or PCWP. As a general rule, the practitioner estimates that as the right atrial or PCWP increases, so does the end diastolic volume. What cannot be determined from invasive pressure monitoring, however, is the compliance of the LV. A PCWP of 18 mmHg could occur in the patient with a dilated LV and, equally, could occur in a patient with an under-filled LV he or she suffers from severe diastolic dysfunction. Echocardiography, however, can be used to directly estimate left ventricular volume and has been well validated against other methods of volume estimation.<sup>6–10</sup> There are several methods to estimate volume:

- a. M-mode echocardiography provides a one-dimensional estimation of preload. Traditionally, the cursor is directed through the base of the LV at the level of the mitral valve chordae. When using TTE, the M-mode is obtained from a parasternal long- or short-axis view, through the base of the LV. Left ventricular end diastolic dimension (LVEDD) is measured at the onset of the QRS on the electrocardiogram (ECG), but is preferably defined as the frame after the mitral valve closure or that frame in the cardiac cycle in which the cardiac dimension is largest. The range of 'normal values' will vary between centres and across populations, but a 'working range' of values for normal is 3.0–5.6 cm; however, this can vary depending on the study population. It is recommended to index the LVEDD to the patient's body surface area (BSA). The normal indexed range is 2.3–3.1 cm m<sup>-2</sup>. If the LVEDD exceeds 5.6 cm, then the ventricle is dilated, and if it is less than 3 cm,

it is hypovolaemic. There are several formulae for converting the single dimension into a volume, such as Teichholz formula, but this translation is not a practical help in determining the preload. Clearly, there will be errors induced in using a one-dimensional measurement when it used to determine a three-dimensional (3-D) structure. It is particularly important that one must beware of using this dimension when there is a significant wall motion abnormality at the base, as this will lead to over-estimation of preload.

- b. Simpson's method: this calculation method is available on most echocardiography machines. It divides a 2-D image of the ventricle into a series of segments and then calculates a volume based on assumptions about the geometry of the ventricle. Whilst there is reasonable agreement between Simpson's method and other methods of volume assessment for TTE, there is much greater potential for error when using TOE because of the risk of foreshortening the LV in the mid-oesophageal views.

The 2-D imaging of the heart in the apical four chamber (Ap4ch) and apical two chamber (Ap2ch) views allows for the calculation of the LVEDV by using the area-length and Simpson's biplane method. The endocardial border is traced at end diastole and the ultrasound system automatically calculates the volume/s.

The normal LVEDV range is:

Men: 67–155 ml (indexed 35–75 ml m<sup>-2</sup>)

Women: 56–104 ml (indexed 35–75 ml m<sup>-2</sup>)

- c. In TOE, the filling and function 'thumbprint' is the left ventricular transgastric mid-view: this view, otherwise known as the 'mid-papillary view', is familiar to most practitioners of TOE and is the view recommended for the serial assessment of preload. To measure the end diastolic area (EDA), the equivalent TTE view is parasternal short axis (Psax) at mid-LV level: this is where the papillary muscles are visualised.

To measure the EDA, trace around the blood-pool area, which means including tracing around the papillary muscles. This convention has grown in popularity following the introduction of automatic border detection systems. Royse et al.<sup>11</sup> described 'normal values' for left ventricular EDA in patients with normal EF, and contrasted this to patients with reduced EF. A normal value will depend on the size of the patient, that is, a small patient is likely to have a smaller EDA than a large patient; however, as a rule of thumb, the normal range is between 8 and 14 cm<sup>2</sup>. An LVEDA <8 cm<sup>2</sup> is consistent with hypovolaemia, and >14 cm<sup>2</sup>, consistent with a dilated LV (Fig. 1).

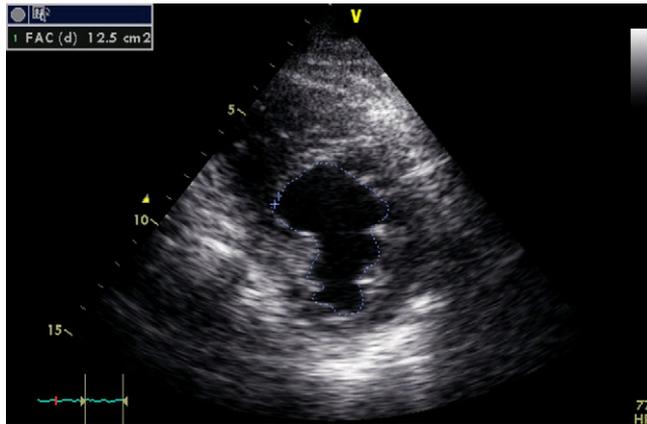
It is noteworthy that preload refers to the volume status of the ventricle and not to a filling pressure. The volume should be categorised based on an echocardiography examination irrespective of what the filling pressure might be.

#### *Estimate the right ventricular volume*

The right ventricle (RV) is crescent shaped and appears to wrap around the LV. The estimates of volume are less quantifiable for the RV than they are for the left as it is not a regular geometric shape. For an assessment of the haemodynamic state, it is easiest to remember the rule of thumb: the RV should appear two-thirds the size of the LV. If the RV is the same size as the left, or larger, then it is dilated. In addition, the RV should appear to end before reaching the left ventricular apex. Another sign of right ventricular dilatation is when apical displacement of the RV is present such that it appears to end at the same point as the left ventricular apex (Figs 2 and 3).

#### **Practice tips**

- Categorise left ventricular volume as 'hypovolaemic', 'normal' or 'dilated'.
- Categorise right ventricular volume as 'normal' or 'dilated'.



**Fig. 1.** Left ventricular short axis view at the mid-papillary level showing a normal left ventricular end diastolic area (12.5 cm<sup>2</sup>) consistent with normal ventricular filling.

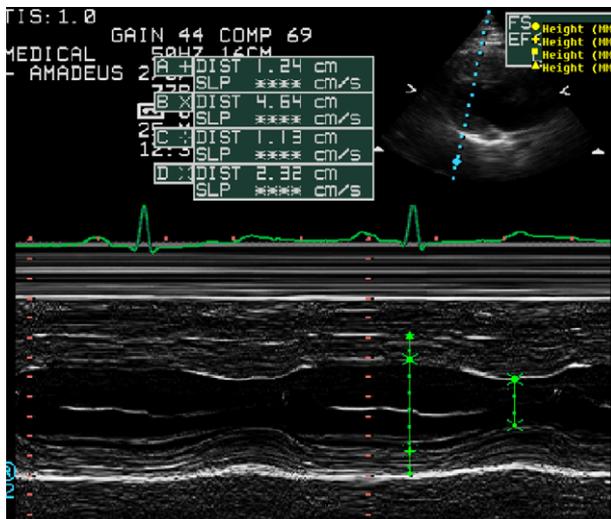
### Step 2: estimate ventricular function: categorise as ‘increased’, ‘normal’ or ‘decreased’

Systolic function is measured by estimating EF. This can be performed by the visual estimation, or using quantification methods as described above. For M-mode measurements, fractional shortening (FS) is determined from the LVEDD and the left ventricular end systolic dimension (LVESD).

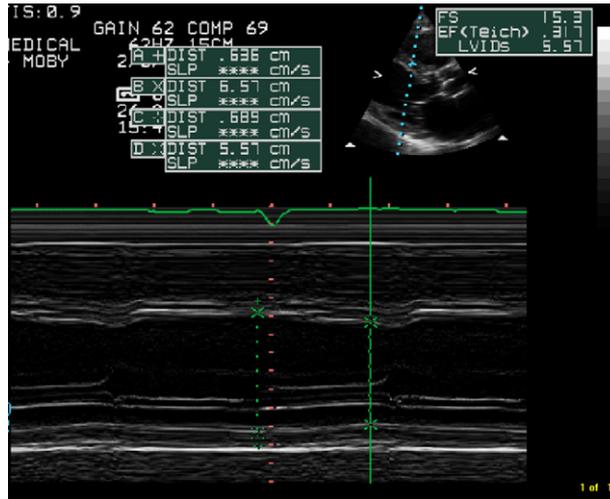
The equation for calculating the percentage of FS is:

$$FS(\%) = (LVEDD - LVESD) / LVEDD \times 100$$

When using 2-D measurements (fractional area change, FAC) or 3-D estimates (3-D echocardiography, or Simpson’s rule), the value for FAC or EF is similar.



**Fig. 2.** M-Mode of the left ventricle showing normal end-diastolic dimension (4.64 cm) and increased fractional shortening (50%) consistent with increased ventricular function.



**Fig. 3.** M-Mode of the left ventricle showing a dilated left ventricle (6.57 cm) and reduced fractional shortening (15%) consistent with reduced systolic function.

$$\text{FAC}(\%) = (\text{LVEDA} - \text{LVESA}) / \text{LVEDA}$$

$$\text{EF}(\%) = (\text{LVEDV} - \text{LVESV}) / \text{LVEDV}$$

### Practice tips

- Increased systolic function: FS >44% or FAC/EF >65%
- Normal systolic function: FS 28–44% or FAC/EF 50–65%
- Decreased systolic function: FS <28% or FAC/EF <50%

### Right ventricular function

The RV is crescent shaped, wraps around the LV and consequently cannot be entirely visualised in any single 2-D echo view. Although multiple methods for quantitative echocardiographic RV assessment have been described, in clinical practice, the assessment of RV structure and function remains mostly qualitative. The RV is sensitive to changes in afterload, and alterations in RV size and function are indicators of increased pulmonary vascular resistance and load transmitted from the left-sided chambers. RV size and wall thickness are essential assessments when evaluating RV function.

### Practice tip

- Categorise right ventricular function as 'normal' or 'reduced'.

### Step 3: estimate LA filling pressure: categorise as 'low', 'normal' or 'high'

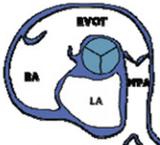
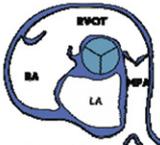
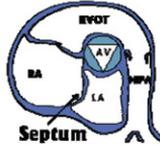
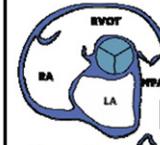
When evaluating the basic haemodynamic state, it is important to categorise LAP into 'high' or 'normal'. It is of some additional use to determine when the LAP might be 'low', as this can help identify an empty

state. There are many ways to estimate the LAP with echocardiography, but none of them are particularly accurate in determining the ‘raw’ pressure and will always be less accurate than invasive pressure monitoring. The definition of the ‘high’ LAP state is somewhat arbitrary and based on the years of experience with invasive pressure monitoring, such as with the use of a pulmonary artery catheter. It is probably reasonable, however, to define a ‘high’ LAP state as exceeding 15 mmHg. The following methods are examples of estimating LAP state, and are arranged in the order of this author’s personal preference.

- a. **The shape and movement of the interatrial septum.** The normal direction of the interatrial septum moves from left to right for most of the cardiac cycle. During mid-systole, however, there is transient reversal so that it bows from right to left. As the LAP rises, this directional change is reduced, and in the elevated LAP state, the interatrial septum remains bowed from left to right throughout the cardiac cycle. These changes are accentuated by ventilation such that there is increased movement of the interatrial septum with ventilation. The transition between normal and high pressure is quite easily seen when the septum does not move during inhalation, but is seen to move right to left with exhalation (if the patient is mechanically ventilated).<sup>12</sup> The interatrial septum remaining fixed left to right with ventilation indicates raised LAP. When the atrium is empty, the movement of the interatrial septum is increased such that there is marked movement in both directions through the cardiac cycle. The interatrial septum may appear concertinaed or buckled upon itself, and this shape reflects a low LAP state.<sup>13</sup> The best analogy to help one conceptualise the movement is to think of the left atrium as a water-filled balloon. When the balloon is full of water and the pressure is high, the balloon is circular in shape and if one is to cut a slice across the balloon, it would appear as a semi-circle going outwards. This is analogous to the ‘fixed curvature’ (FC) seen in the high LAP state. If a little water is let out, a tap on the edge with the hand would move the wall inwards briefly before it springs out, which is analogous to the ‘systolic reversal’ (SR) seen with normal LAP. Finally, if the balloon is relatively empty, the walls of the balloon would be concertinaed or would shrink down and appear to overlap and a small tap of the hand will produce excessive motion. This is analogous to the ‘systolic buckling’ (SB) pattern of the interatrial septum and is illustrated in Table 2 below.

Royle et al.<sup>13</sup> categorised these three states and investigated how they change with increasing or decreasing the LAP. In a study performed in patients undergoing cardiopulmonary bypass, the shape and movement of the interatrial septum was observed whilst draining blood out and then re-infusing it

**Table 2**  
Estimation of left atrial pressure.

Left Atrial Filling Pressure (Interatrial septum motion)					
Low Pressure		Normal Pressure		High Pressure	
					
Diastole	Systole	Diastole	Systole	Diastole	Systole
Systolic buckling		Systolic reversal		Fixed curvature	

Septum is atrial septum; AV is aortic valve; LA is left atrium; RA is right atrium; LV is left ventricle; RV is right ventricle; MPA is main pulmonary artery.

through the aortic cannula. They identified that the direction of change is proportional to the LAP state; that is, as volume is decreased, the direction changes towards the lower LAP state, and as volume is infused, the LAP state changes to a higher state. The actual PCWP is less well defined by categorical measurement such as interatrial septal movement. As one would expect, there is a range of PCWPs for each category, but, in general terms, the trend in PCWP is in the same direction as the change in the interatrial septal pattern.

#### LA size

Increased LA size is associated with adverse cardiovascular outcomes. The increase is commonly related to increased wall tension and secondary to increased filling pressure. The size of the LA can be estimated visually using 2-D echo and is seen in all the standard imaging planes. The M-mode linear dimensions can be obtained from the parasternal long axis view. Although a large LA can occur with normal LAP, it is more commonly associated with high LAP.

#### Practice tips

The normal American Society of Echocardiography (ASE) values for LA size measured by M-mode are:

3.0–4.0 cm (men) 2.7–3.8 cm (women)  
or indexed for BSA as:  
1.5–2.3 cm m<sup>-2</sup> (men) 1.5–2.3 cm m<sup>-2</sup> (women)

#### Doppler methods of LAP estimation

Several methods using the various Doppler measurements have been validated for estimating LAP. The problem with all these measurements is a relatively high frequency of false negatives. If the Doppler estimate is indicative of a true high LAP, then it has a relatively low false positive risk; however, many patients will have normal Doppler profiles with raised LAP.

- Pulmonary Vein Doppler<sup>14</sup>: the normal profile is for systolic predominance of flow in the pulmonary veins. Diastolic predominance is indicative of raised LAP, as is a pulmonary vein 'a' wave velocity >35 cm s<sup>-1</sup>.
- Difference in the duration of mitral and pulmonary vein 'a' wave<sup>15</sup>: normally, the pulmonary vein 'a' wave is shorter than the transmitral 'a' wave. If the pulmonary vein wave is larger than the mitral wave, then this is indicative of raised LAP.
- Peak mitral E velocity/annular tissue Doppler E velocity (E/E' ratio).<sup>16</sup>

An E/E' ratio of >15 in awake patients is consistent with raised LAP. This has not yet been validated in anaesthetised or ventilated patients.

- Estimation of right atrial pressure (RAP) as a surrogate for LAP.

As a general principle, if the RAP is elevated, so also will the LAP. A raised RAP can be used in the haemodynamic state assessment concept discussed in this article. TTE has been used for the estimation of RAP for a long time as it is well validated. It may be easier to estimate RAP than LAP using TTE. A 2-D echocardiography is used in estimating the RAP by observing the size and contraction of the inferior vena cava (IVC) during inspiration. This response is a reflection of right-sided haemodynamics. The IVC is imaged from the subcostal acoustic window. An increase in venous return to the right heart occurs during inspiration, resulting in a decreased pressure in the IVC. This results in a decrease in blood

volume and intraluminal pressure and the collapse (or reduction) in IVC diameter. The reverse occurs during expiration. With increasing filling pressure in the right heart, this flow is impeded, resulting in diminished or absent collapse of the IVC. Increased vessel and hepatic vein size may result; therefore, both the IVC diameter and the degree of inspiratory collapse are noted and used to estimate RAP. Brief sudden inspiration such as sniffing aids in accentuating the inspiration affect. A summary is shown in Table 3.

#### Step 4: final assessment

The key difference between echocardiography and invasive pressure monitoring, when used to diagnose haemodynamic state, is that echocardiography allows direct assessment of volume, systolic function, and also filling pressure. This combination of knowledge allows us to estimate preload, ventricular function and, importantly, to estimate ventricular compliance. Only when we can estimate compliance and volume together can we differentiate diastolic heart failure from other haemodynamic states.

Table 4 is a summary guide to interpretation, but the process is quite simple. For example, if preload, function and filling pressure, all are normal, then we have defined the first haemodynamic state, that is, 'normal'.

- a. **Normal haemodynamic state** – characterised by normal LVEDA, normal EF estimate and normal LAP.
- b. **Empty (hypovolaemic)** – characterised by reduced LVEDA, normal or increased EF estimate and low LAP.
- c. **Primary diastolic failure** – the ventricle will appear hypovolaemic (reduced LVEDA), have normal EF estimate but high LAP. This haemodynamic state is very difficult to appreciate because it looks normal. It is 'a conceptual leap' to believe that the normal-looking ventricle constitutes heart failure. The key to identifying the state is to see a normal-looking ventricle operating at a high filling pressure state.
- d. **Primary systolic failure** – characterised by increased LVEDA (dilated ventricle), reduced EF estimate and normal LAP. Essentially, the compliance of this ventricle is normal or increased. It is important to differentiate between dilated cardiomyopathy that is associated with normal versus increased filling pressure because the haemodynamic performance may be quite different.
- e. **Systolic and diastolic failure** – in this haemodynamic state, the LVEDA is increased, the EF reduced and the LAP increased. These patients may represent the worst end of the heart failure spectrum and may be associated with right ventricular failure as well. The diastolic failure is evident because of the raised filling pressure.
- f. **Right ventricular failure** – characterised by a dilated RV, with reduced inward excursion and an elevated LAP state. Although isolated right ventricular failure can occur, it is frequently associated with LV failure as well. The RV will compress the LV, causing left ventricular diastolic dysfunction (and the raised LAP state).

#### Summary: how can diagnosis of the haemodynamic state influence management?

The evaluation that has been outlined above allows the practitioner to determine the broad categories of haemodynamic abnormality. The treatment of these conditions can be very different, even though the signs and symptoms presenting to the practitioner appear the same. Although the management of systolic failure appears straightforward presently, the management of diastolic failure

**Table 3**

Estimating RAP from IVC size and effect with inspiration.

IVC size (cm)	Inspiration affect	Estimated mean RAP (mmHg)
Small < 1.5	collapse	0–5
Normal 1.5–2.5	↓ ≥ 50%	5–10
Normal 1.5–2.5	↓ ≤ 50%	10–15
Dilated > 2.5	↓ ≤ 50%	15–20
Dilated IVC and Hepatic veins	no collapse	>20

**Table 4**

Summary of haemodynamic state assessment.

Haemodynamic state							
	Normal	Empty	Vasodilation	Systolic failure	Primary diastolic failure	Systolic & Diastolic failure	RV failure
Volume	N	▼	N	▲	N/▼	▲	RV ▲
Systolic function	N	N/▲	▲	▼	N	▼	RV ▼
Filling pressure	N	▼	N	N	▲	▲	▲

N is normal; arrows indicate increased or decreased.

is very different. For example, the use of an inodilator such as dobutamine or milrinone is reasonably standard therapy for patients with dilated cardiomyopathy. It appears logical that increasing systolic function facilitating ejection simultaneously will improve global myocardial performance. The primary limitation in diastolic heart failure, however, is that stroke volume is reduced because of reduced preload rather than because of poor systolic function. The use of an inodilator may reduce preload even further because of tachycardia (reduced filling time) and increased EF. The aim of this article is not to outline detailed therapeutic options, but rather to highlight that accurate diagnosis will lead to logical choice of therapy. The exact choice of what type of volume to infuse, or what inotrope combination is best for each condition, is largely a matter of experience and familiarity. Broad categories of the therapy for each of the haemodynamic states are described in Table 1.

### Research agenda

There is little doubt that haemodynamic state assessment can be identified using echocardiography, either with TTE or TOE. The main areas of research required are identification of whether haemodynamic state assessment alters patient management and, ultimately, whether this improves the outcome. There is also the question of training novices to perform limited echocardiography and haemodynamic state assessment. Training is covered in greater detail elsewhere in literature; however, Royse et al<sup>17</sup>, compared the accuracy of novice assessment of the haemodynamic state with that of an expert echocardiographer. The learning period was very brief, such that after 10 supervised studies, and a further 10 unsupervised studies, the information required for haemodynamic state assessment could be attained reliably by the novice, whereas a further 20 studies were required for good agreement on more complex measurements such as cardiac output.

### Conclusion

Haemodynamic state assessment is a higher order of diagnosis than conventional measurement of blood pressure, heart rate and chamber filling pressures. The haemodynamic state represents the combination of ventricular and vascular systems, involves left and right hearts and systolic and diastolic phases of the cardiac cycle. Although clinical signs such as hypotension alert the practitioner about the existence of haemodynamic abnormality, they does not provide sufficient diagnostic information to determine what the haemodynamic state is, or the cause of it. We treat the haemodynamic state rather than simply treating the vital sign. The premise of haemodynamic state monitoring is that better treatment will lead to more rational therapy, and that might in turn improve outcome.

There are four simple steps to perform:

1. assess ventricular size – categorise as hypovolaemic, normal or dilated;
2. assess ventricular function – categorise as increased, normal or decreased;
3. estimate LA filling pressure – categorise as low, normal or high and
4. integrate these findings to determine the haemodynamic state.

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