Guidelines for performing a comprehensive haemodynamic assessment with ultrasound. FUSIC® HD.

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Introduction

This document has been written as a guideline for performance of a FUSIC® HD ultrasound examination. It outlines the clinical questions and concepts that underpin the examination, details of how to perform it and also provides a full data set of the views and measurements that should be performed for a complete haemodynamic assessment with ultrasound. It is recommended that all of these views and measurements be obtained where possible. All elements of the haemodynamic system are interconnected, and interdependent, making a full scan desirable. However, it is recognised that the purpose of this exam is to answer specific clinical questions that might make a more focused exam acceptable. By the same token, a repeat scan may be to follow up on one specific question and focus only on this.

Whilst a FUSIC® HD exam is a comprehensive assessment of haemodynamics, it is not a comprehensive echocardiogram. Practitioners should understand the limitations this presents and have a low threshold for referring a patient for a comprehensive echo from a level 2 practitioner if the cardiac views reveal anything outside of the competencies of this assessment tool or if a more detailed assessment is required. On the other hand, a FUSIC® HD study includes views and measurements not done in a fully comprehensive echocardiogram meaning a level 2 echo cannot replace a full HD study. The abdominal views are only for assessing blood flow in specific vessels. They are in no way meant to replace abdominal imaging for other reasons.

Both the British [1] and American Societies of echocardiography [2] have published their own guidelines to performing a comprehensive echo and we acknowledge that much of this document stands on their shoulders. FUSIC® HD practitioners are encouraged to read these documents to understand the differences between haemodynamic assessment and comprehensive echocardiography.

A FUSIC® HD exam asks 10 specific clinical questions. While these questions form the basis of what is included in the FUSIC HD dataset they do not provide a logical order for image acquisition. This guideline outlines these clinical questions and their relevance and then provides a logical framework for obtaining all the views and measurements required. Measurement techniques are described at the end of the document.

Accreditation process

The accreditation pathway is outlined in table 1. Full details can be found on the ICS website.

FUSIC® HD accreditation process	
Prerequesites	Independent competence in history taking, physical examination, interpreting clinical investigations, and understanding of disease processes in critically ill patients , AND…
	Accreditation in FUSIC® heart (formerly FICE) *or* BSE level 1 *or* FEEL, AND…
	Accreditation in FUSIC® lung *or* FAMUS
Program components	Registration with ICS
	E-learning completion
	Course - optional
	Logbook - 50 studies, of which 20 must be directly observed by a registered FUSIC® HD trainer. The full minimum data-set must be attempted in each scan (although it is recognised that not all views are possible in some patients). The 50 cases should clearly demonstrate that the trainee has consistently covered all areas of the syllabus and seen a wide breadth of pathology. There should be a clinically relevant conclusion to each scan.
	Assessment: Competencies must be signed off by a registered trainer before proceeding to examination. Centrally run exam consisting of: Logbook assessment MCQ exam of pathology video clips and theoretical knowledge Practical exam (performing a selected part of the minimum data set on a live model).
	Timeline for completion: A total of 24 months for all components. However the logbook must be collected (first scan to last scan) within 12 months
Trainer requirements	At least one of: ***FUSIC® HD*** accredited for at least 12 months ***OR*** hold a Level 2 Transthoracic Echocardiography (TTE) (i.e. BSE, EACVI, ASE) ***OR*** a European Diploma in Advanced Critical Care Echocardiography (EDEC) ***OR*** be a Cardiologist with regular sessional commitments to TTE.
	The trainer is responsible for directly observing at least 20 of the trainees logbook scans (preferably spread out over the course of training so progression can be demonstrated), reviewing all other logbook scans, signing off competencies and ensuring that the candidate is competent to attempt the practical exam

Table 1. The FUSIC® HD accreditation process

Clinical questions and pathologies

FUSIC® Heart (formerly known as FICE) level scanning, which is a prerequisite to learning a HD scan, addresses 5 key questions. Table 2 lists these and compares them to the additional questions addressed by a FUSIC® HD scan. The pathologies that can be detected with a FUSIC® HD scan are listed in table 3.

	FUSIC® Heart Questions	FUSIC® HD Questions
1	Is the left ventricle dilated and/or significantly impaired?	Is stroke volume and/or ventricular-arterial coupling abnormal?
2	Is the right ventricle dilated and/or significantly impaired?	Is stroke volume responsive to fluids, vasopressors or inotropes?
3	Are there features of low venous return/ stressed venous volume?	Is the aorta abnormal?
4	Is there a pericardial effusion?	Is the aortic valve, mitral valve or tricuspid valve severely abnormal?
5	Is there a pleural effusion?	Is there LVOTO (left ventricular outflow tract obstruction)?
6		Is there a regional wall motion abnormality?
7		Is left atrial pressure raised?
8		Is pulmonary artery pressure raised – and how is the right ventricle responding?
9		Are there ultrasound features of cardiac tamponade?
10		Is there venous congestion?

Table 2. The clinical questions FUSIC® Heart and FUSIC® HD are designed to answer

Pathologies detected by FUSIC® HD	
Left ventricular disease	Left ventricular (LV) hypertrophy (LVH) Dilatation Regional wall motion abnormalities (RWMAs) Impaired systolic function (acute vs chronic) LV-aortic coupling Raised left atrial (LA) pressure Left Ventricular Outflow Tract (LVOT) obstruction
Right ventricular disease	Right ventricular (RV) hypertrophy (RVH) Dilatation Impaired systolic function (acute vs chronic) Raised pulmonary arterial (PA) pressure Volume overload RV to PA coupling
Mitral valve disease	Significant thickening, calcification, restriction Significant prolapse Significant regurgitation Systolic anterior motion
Aortic valve disease	Significant thickening, calcification, restriction Significant regurgitation

Pathologies detected by FUSIC® HD	
Tricuspid valve disease	Significant thickening, calcification, restriction Significant regurgitation
Aortic disease	Root dilatation Thoracic dissection Abdominal aneurysm
Atrial disease	Dilatation Raised LAP
Volume overload	Raised intracardiac pressures Functional tricuspid regurgitation Enlarged IVC Pleural effusions Pericardial effusions Venous congestion
Reduced stressed venous volume/venular pressure)	Hyperdynamic heart Responsive to increased venular tone (vasopressor or volume challenge) Vasopressor responsive VTI/stroke volume
Abnormal flow	Stroke volume Cardiac output
Venous congestion	Enlarged IVC Abnormal venous flows • Portal vein • Hepatic vein • Renal vein and artery
Coupling	LV to aorta (Ejection fraction - visual or measured) RV to pulmonary (RV size and function, Doppler profiles) Organ to systemic circulation - venous flow patterns

Table 3. The pathologies detectable with FUSIC® HD

The clinical questions and their relevance are described in detail below.

Q1. Is stroke volume and/or ventricular-arterial coupling abnormal?

Diagnosing and treating shock is one of the most important roles of an intensive care clinician. Despite this, published evidence demonstrates that it is very difficult to identify a low cardiac output state clinically. Calibrated continuous stroke volume monitors exist, but these are rarely found in locations that patients are referred from.

Ultrasound can quantify stroke volume (SV) using the equation:

$$SV = \text{LVOT VTI} \times \pi (\text{LVOT Diam}/2)^2$$

(LVOT VTI = velocity-time integral in the left ventricular outflow tract; LVOT Diam = diameter of the left ventricular outflow tract)

(As stroke volume = stroke distance x cross sectional area)

And ultrasound has the advantage of being portable. Hence, stroke volume is the subject of our first FUSIC® HD question.

A rapid, qualitative assessment of stroke volume can be performed simply using LVOT volume-time integral (VTI).

- LVOT VTI 18-22cm is normal (but heart rate dependent)
- LVOT VTI <17 or >23 should be considered abnormal

For accuracy, it is important to measure at least 3 traces in sinus rhythm (SR), and 5 in atrial fibrillation (AF), and use the average value.
LVOT VTI alone is a quick and useful surrogate of SV and its trends. However, there are times when a quantitative assessment is required. To do this requires measurement of LVOT diameter. This is notoriously difficult and the SV calculation risks squaring any error involved. So, it is essential to know how to measure LVOT diameter correctly as outlined below.

- LVOT diameter is almost never less than 2cm in an adult

Once you know the LVOT diameter, LVOT VTI and heart rate, you can calculate cardiac output using the equation

$$CO = HR \times LVOT\ VTI \times \pi\ (LVOT\ Diam/2)^2.$$

SV and LVEF are related. But both must be interpreted in context of the following haemodynamic factors:

- LV chamber size
- Preload and afterload
- Degree of inotropy
- Clinical context (gross valvular abnormality, sepsis etc.)

For instance, two hearts could have the same SV while one is dilated and chronically impaired (low LVEF), and the other acutely under-filled and hyperdynamic (high LVEF). In acute, severe MR, a normal ventricle should appear hyperdynamic, so a combination of normal SV, LV size and LVEF indicates LV impairment. The same is true for a patient on inotropic support.

The S^1 wave measured with tissue Doppler of the MV annulus shows good correlation with EF and is simple to measure if EF measurement is not possible.

Left Ventricular Ejection Fraction (LVEF)

Why it's included:
LVEF is now included in the FUSIC® HD competency framework as it is the most practical and clinically useful surrogate of left ventricular–arterial coupling. While previously avoided in favour of fractional shortening (FS), LVEF offers a more widely understood and physiologically integrative measure that captures both contractility and afterload.

Load dependence is a feature, not a flaw:
LVEF is load dependent — and while this makes it unsuitable as a pure measure of contractility, it is *precisely* what makes it a valuable indicator of coupling. It reflects how well the ventricle adapts to the prevailing arterial load. For example, in vasodilated states such as sepsis, afterload may be so low that a failing ventricle maintains a normal-appearing LVEF. However, when vascular tone is restored (e.g. with vasopressors), the increase in afterload can lead to a reduction in LVEF, unmasking previously hidden systolic dysfunction.

Mathematical equivalence to coupling metrics:
In single-beat models (e.g. Chen method), the ratio of arterial to ventricular elastance (Ea/Ees) simplifies to a function of LVEF:

$$Ea / Ees = (1 / EF) - 1$$

This means that in most clinical scenarios, LVEF is effectively a surrogate for the ventricular–arterial coupling ratio, making it a pragmatic and meaningful metric at the bedside.

How to measure it:

- Some echocardiography machines offer automated or AI-derived LVEF calculations (e.g. auto-simpson or machine-learning-based algorithms). These may be used if available and image quality is sufficient.
- Use a high-quality apical 4-chamber and/or biplane view
- Trace the endocardial borders at end-diastole and end-systole
- Apply the Simpson's biplane method if possible:

$$EF = ((EDV - ESV) / EDV) \times 100$$

Where EDV = end-diastolic volume, ESV = end-systolic volume

- If image quality is suboptimal or quantification not feasible, LVEF may be estimated visually (with appropriate experience)

Interpretation:

- Normal: LVEF ≥55%
- Borderline-low: LVEF 50–54%
- Impaired: LVEF 36–49%

- Severely impaired: LVEF ≤35%

Practical points:

- Should be estimated visually or calculated if feasible
- Must be interpreted in context of loading conditions, inotropy, LV size, and clinical state
- Supports better haemodynamic decision-making and prognostication

Q2. Is stroke volume responsive to fluids, vasopressors or inotropes?

Another important question intensive care clinicians need to know is whether or not giving IV fluid, vasopressors or inotropes will improve stroke volume. A FUSIC® heart study may indicate a fluid tolerance profile (i.e. high or low) based on evaluation of the LV and RV size and systolic function, but it cannot answer this.

The commonest cause of haemodynamic instability in a critically ill patient is low venous return from reduced mean systemic pressure and stressed venous volume, caused by either vasoplegia or more rarely hypovolaemia. Unfortunately, echo cannot distinguish between these two causes, prior to treatment, as both share the same appearances (a small, hyperdynamic heart). A hyperdynamic state, with a high stroke volume, is frequently seen after fluid loading and vasopressors have been started. A high SV is a hallmark of resuscitated vasodilation and effectively rules out hypovolaemia.

In ICU patients hypovolaemia is unusual outside the context of trauma or significant GI losses, and should only be suspected when the history and/or physical examination make this a possibility (i.e. bleeding or extracellular fluid loss such as vomiting and diarrhoea). Giving empirical fluid therapy risks being counter-productive through fluid overload, which causes significant harm. [3] Vasoplegia will respond much better to vasopressors than fluids. [4]

If the ventricles are preload-responsive, they will eject a higher SV after stressed venous volume is increased - either via fluid volume-loading (a state referred to as fluid-responsiveness) or via vasoconstriction induced by vasopressors (vasopressor responsiveness). Respiratory variation in intra-thoracic pressure, caused by mechanical ventilation, also induces cyclical changes in venous return and stroke volume. [5]

The presence of preload responsiveness is a state of normality and does not mean that a patient necessarily needs treatment.

Absence of preload responsiveness is a pathological state that means the circulation is already likely volume overloaded and that the heart is failing. This is a clear sign to not give fluid. Also, deliberately ‘resuscitating’ to this point causes fluid overload and is potentially dangerous.

A genuinely hypovolaemic patient, who is already maximally vasoconstricted via sympathetic activation, will not increase their stroke volume with vasopressors but will respond significantly to even a small fluid bolus.

A vasoplegic patient, who is maximally vasodilated, will respond much better to vasopressors than fluids, reassuring the clinician that vasopressors are recruiting stressed venous volume. Vasopressors have been shown to increase venous return and cardiac output in septic patients even when LV failure is present. [6]

Preload responsiveness can be predicted with ultrasound using the following methods:

PW Doppler of the LVOT:

- Respiratory variation of >12% in LVOT VTI or peak velocity. This is calculated using the equation

$$\% \text{ Variation} = 100 \times (V_{max} - V_{min}) / ((V_{max} + V_{min}) \times 0.5)$$

where V = LVOT VTI or peak velocity. Note that the max is divided by the mean and not the minimum.

- The following preconditions must apply: Sinus rhythm, mechanically ventilated with no spontaneous breaths, tidal volumes of at least 8ml/kg, normal intra-abdominal pressure, and an intact thorax
- A failing right or left ventricle can result in a false positive

IVC:

- Respiratory variation (distensibility index) of >12% in IVC diameter. This is calculated using the equation

$$DI\ IVC = 100 \times (D_{max} - D_{min}) / ((D_{max} + D_{min}) \times 0.5)$$

where DI is distensibility index and D is diameter. Note the mean is used again here.

- The same caveats apply as for LVOT variation measurements.

These caveats make these methods unusable in many patients. In which case, LVOT VTI can be measured before and after a fluid challenge, a passive leg-raise or starting a vasopressor infusion to see if it increases.

Measuring LVOT VTI after starting an inotrope will also demonstrate whether this is effective in increasing stroke volume.

Combining this preload responsiveness assessment with the venous congestion parameters described below should allow the clinician to make the right decision on whether volume loading, volume removal, vasopressors or inotropes are the correct treatment.

Q3. Is the aorta abnormal?

Aortic root dissection carries a high mortality and is frequently overlooked. As such, ultrasound can be pivotal to making this diagnosis and enabling life-saving treatment.

An abnormally dilated aortic root can be detected by measuring the end of the aortic root at the sinotubular junction and the ascending aorta 2cm distally. If >4cm in diameter, this should prompt referral for an expert scan and/or cross-sectional imaging. The presence of an intimal dissection flap, seen within the aortic root on ultrasound, confirms the diagnosis of thoracic dissection.

A FUSIC® HD level practitioner should regard aortic root dissection as a rule-in, not rule-out, diagnosis. If this is detected, a pericardial effusion and aortic regurgitation (AR) should be looked for. And vice versa.

With small modifications from standard echo windows, 2D ultrasound can be used to image the entire aorta, from its root to bifurcation, making it an important target in a patient with undifferentiated shock.

The aortic root is seen in the PLAX view. The aortic arch and all its major vessels can be visualised in the suprasternal view. The descending thoracic aorta can be visualised in long-axis from a modified PSAX view at the MV level and in the A2C view. The abdominal aorta can be visualised in long and short-axis from the epigastrium to the pelvis.

Flow in the aorta should be pulsatile using colour and spectral Doppler. Continuous flow suggests a shunt. Turbulent flow suggests dissection.

Any aortic abnormality seen in a FUSIC® HD study should be followed immediately by further imaging and/or expert review.

For further guidance on aortic assessment, see the ‘Evaluating Aorta’ section (below).

Q4. Is the aortic valve, mitral valve or tricuspid valve severely abnormal?

Some acute valvular pathology, such as papillary muscle rupture secondary to myocardial infarction (MI) and acute AR secondary to aortic dissection, requires immediate surgical intervention. Chronic valvular pathology, such as stenosis and regurgitation, can have dramatic

effects on the way the heart handles different loading conditions. Therefore, the clinician needs to know if haemodynamically significant valve disease is present.

Quantifying valve disease in a critically unwell patient is a complex task that requires extensive knowledge and experience in echocardiography, and is considerably beyond the scope of FUSIC® Heart. Instead, FUSIC® HD utilises mostly 2D and some colour Doppler to identify valvular obstruction (severe leaflet thickening, calcification and immobility) or regurgitation (large, highly turbulent colour signals) and visually differentiate severe from non-severe disease. FUSIC® HD practitioners will also be able to identify acute or severe valvular pathology and refer to an expert in a timely fashion. They should also be able to distinguish functional (no intrinsic valve disease e.g. valve leaflets stretched apart by chamber dilatation) from non-functional (intrinsic disease of the valve apparatus) regurgitation. This will require the learner to see a range of valvular pathology with expert mentorship.

Dimensionless Index (DI) is a useful application of CW Doppler for detecting severe aortic stenosis (AS) when comprehensive echo is not immediately available:

DI utilises the fact that AS causes elevated velocities through the AV. When CW Doppler is placed through the AV in either A5C or A3C, it may demonstrate two traces superimposed on each other: a shorter, denser one from the LVOT and a taller, less dense one from the AV. The ratio of these peak velocities (LVOT / aortic) gives the DI. To ensure accuracy, it is better to calculate the DI by measuring the VTI across the AV with CW Doppler and measuring the LVOT VTI with PW Doppler. The ratio of peak velocities are acceptable but VTI is preferred.

$$DI = VTI_{LVOT} / VTI_{AV}$$

- DI = 1 is normal
- DI < 0.25 demonstrates severe AS

DI has the advantage of being simple to perform, not requiring any other measurements, and is not affected by cardiac output or body surface area.

Knowing the direction of a TR jet also helps to obtain an optimal measurement of TR Vmax (described below), which is a key indicator of raised left and right heart pressures.

Infective endocarditis is another important diagnosis. A FUSIC® HD practitioner would not be expected to rule this out, but should have the appropriate knowledge to recognise obvious vegetations and refer to a Level 2 expert for a more detailed assessment.

Q5. Is there LVOTO (left ventricular outflow tract obstruction)?

Left ventricular outflow tract obstruction (LVOTO) is an important and potentially reversible cause of haemodynamic instability. It may mimic left ventricular failure and lead to harmful mismanagement — particularly if treated with diuretics, vasodilators, or inotropes.

In critical illness, LVOTO most commonly occurs when basal septal hypertrophy (especially in older patients) combines with a hyperdynamic state (low preload and/or afterload). This narrows the LVOT and increases blood velocity. As velocity rises, pressure drops — drawing the mitral valve leaflets (MVLs) into the LVOT during mid to late systole. This causes systolic anterior motion (SAM), which in turn generates dynamic LVOTO and acute mitral regurgitation (MR).

Recognising LVOTO is crucial because it is treatable, and the required treatment is often opposite to that of left heart failure:

- **Fluid resuscitation** if hypovolaemic
- **Cessation of inotropes** and chronotropes
- **Beta blockade** (e.g. esmolol)
- **Pure vasopressors**, which increase venous return and restore preload and afterload, helping to maintain end-systolic volume and reduce SAM

How to recognise LVOTO on echocardiography:

- 2D imaging (PLAX, A5C, A3C):
  Look for systolic anterior motion (SAM) of the MVLs towards the septum in mid to late systole. This movement is often subtle and fast. Freeze the image and scroll frame-by-frame to improve detection.
- M-mode (PLAX):
  May demonstrate anterior MV leaflet movement and/or fluttering of the aortic valve due to early partial closure in late systole.
- Colour Doppler:
  - Posteriorly directed MR jet
  - Turbulent flow distal to the LVOTO (often in late systole)
- PW Doppler:
  - Normal velocities in the proximal LVOT
  - Sudden increase in velocity with aliasing just distal to the obstruction

Key teaching point:
Systolic anterior motion (SAM) is the most common cause of LVOTO in critical care, but not the only one. LVOTO refers to a haemodynamically significant systolic flow limitation in the LVOT. Assessment should include both anatomical findings and clinical context, and management should be guided by the underlying mechanism. Flow obstruction can also occur at the mid-ventricular level, particularly in hyperdynamic, hypovolaemic states, and should be considered

when SAM is absent but flow acceleration is seen.

Q6. Is there a regional wall motion abnormality?

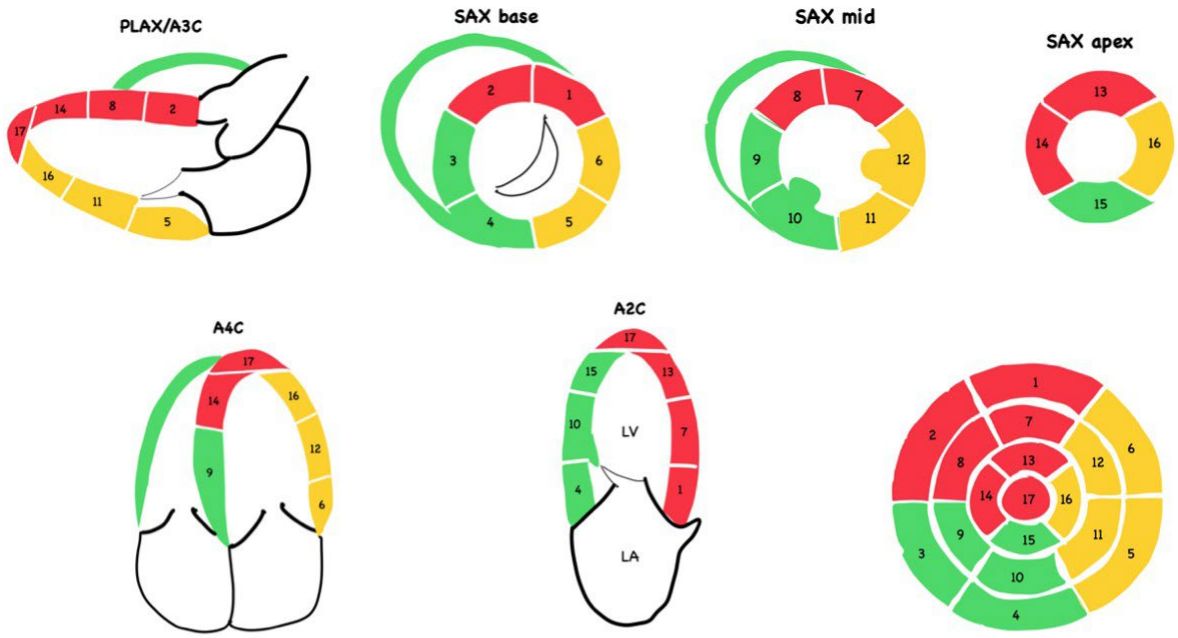


Figure 1. LV segments and coronary artery territories

LV segments				Coronary arteries
Basal	**Mid**	**Apical**	**Apex**	
1. Anterior	7. Anterior	13. Anterior	17. Apical	LAD
2. Anteroseptal	8. Anteroseptal	14. Septal		
3. Inferoseptal	9. Inferoseptal	15. Inferior		RCA/PDA
4. Inferior	10. Inferior	16. Lateral		
5. Inferolateral	11. Inferolateral			LCx
6. Anterolateral	12. Anterolateral			

Table 4. 17 segment coronary artery territories

Evaluating global systolic function is a core FUSIC® Heart skill. Assessing regional wall motion requires further experience. But it is an important means of identifying patents with ischaemic heart disease who might benefit from coronary intervention.

In systole, normal endocardial excursion is >5mm and normal wall thickening is >30%. [7] Wall thickening is a better gauge of regional function than endocardial movement as an akinetic segment may be moved because it is attached to another moving part. Wall motion is described

as normokinetic, hypokinetic (reduced thickening and movement), akinetic (no thickening or movement), dyskinetic (no thickening and moves away from the centre of the left ventricle in systole) or aneurysmal (bulges eccentrically in systole and diastole). When hypo/akinetic segments are limited to a certain distribution, usually relating to coronary artery territories, they are known as RWMAs.

RWMAs can be seen early in ischaemia, sometimes even before ECG changes are present. So being able to detect them can help the intensive care clinician make a timely diagnosis. The LV walls have been divided up into 17 segments, with different segments being seen in various standard echo views. [8] The coronary artery territories in each view are illustrated in figure.

Non-ischaemic RWMAs also exist - notably Takotsubo cardiomyopathy, which causes symmetrical RWMAs that extend beyond coronary territories. Takotsubo cardiomyopathy is an important diagnosis to the intensivist because its immediate treatment requires urgent discontinuation of any beta-agonists, which may seem counter-intuitive when presented with clinical signs of poor ventricular function.

Q7. Is left atrial pressure raised?

Being able to identify patients with raised left atrial (LA) pressure is of key importance in critically ill patients. Ultrasound can identify raised LA pressure and help clinicians to distinguish between cardiac and non-cardiac pulmonary oedema, avoid injudicious intravenous fluid administration when LA pressure is high, and monitor the response to fluid administration or removal.

FUSIC® HD aims to establish whether LA pressure is probably high, probably normal, or indeterminate. It does not attempt to quantify a specific pressure. This approach enables determination of the likely aetiology of pulmonary oedema and supports fluid and haemodynamic decision-making in the acute setting. It does not aim to provide formal grading of left ventricular diastolic dysfunction, which is complex and beyond the scope of FUSIC® HD.

Step 1: Lung ultrasound and interatrial septum

The combination of:
- B-lines on lung ultrasound, and
- Bowing of the interatrial septum into the right atrium throughout the cardiac cycle

makes raised LA pressure very likely. When both are present, no further Doppler assessment is required. B-lines alone are non-specific in critical illness and must be interpreted in context.

Step 2: Echocardiographic assessment of LA pressure

If Step 1 is negative or equivocal, further assessment is performed using Doppler and chamber size.

Left atrial size

LA size increases when LA pressure has been elevated over a period of days. Although LA volume indexed to body surface area is the reference standard, this is time-consuming and technically demanding. In FUSIC® HD, LA size is assessed pragmatically to determine whether it appears clearly enlarged or clearly normal.

Acceptable approaches include:

- Visual assessment (“eyeballing”)
- LA diameter measured in PLAX at end-systole
- Single-plane LA area in the apical four-chamber view at end-systole

As a guide LA enlargement is suggested by:

- LA diameter >45 mm in women or >50 mm in men
- LA area >25 cm²

Mitral inflow and tissue Doppler (E/e¹)

Pulsed-wave Doppler is used to measure the early mitral inflow velocity (E wave). Tissue Doppler imaging at the mitral annulus measures early diastolic myocardial velocity (e¹), reflecting LV relaxation.

Conceptually, E reflects the pressure gradient driving transmitral filling, whereas e¹ reflects the resulting myocardial lengthening associated with LV filling. The ratio E/e′ therefore behaves as a practical surrogate of diastolic operating stiffness (pressure relative to filling).

e¹is measured at the septal and lateral annulus and averaged.

The ratio E/e¹ is interpreted as follows:

- ≥14 suggests raised LA pressure
- <8 supports normal LA pressure
- 8–13 is indeterminate and must be interpreted alongside other findings

e¹ is used only as part of the E/e¹ calculation and is not interpreted independently as neither E nor e′ alone reliably reflects LA pressure in critically ill patients.

Tricuspid regurgitation velocity (TR Vmax)

If tricuspid regurgitation is present, the peak velocity of the TR jet (TR Vmax) may support the presence of raised LA pressure.

- TR Vmax >2.8 m/s increases the likelihood of raised LA pressure

However, TR Vmax should only be used for LA pressure estimation when:

- Right ventricular systolic function is preserved, and
- There is no evidence of increased pulmonary vascular resistance

In the presence of significant lung disease (e.g. pneumonia, ARDS), high PEEP, pulmonary embolism, pulmonary hypertension, or RV failure, TR Vmax reflects pulmonary vascular load rather than LA pressure and should not be used for LA pressure inference.

Integrating findings

LA pressure is classified as:

Probably high

- Positive Step 1 (B-lines + IAS bowing), or
- ≥2 markers suggesting raised LA pressure on Step 2

Probably normal

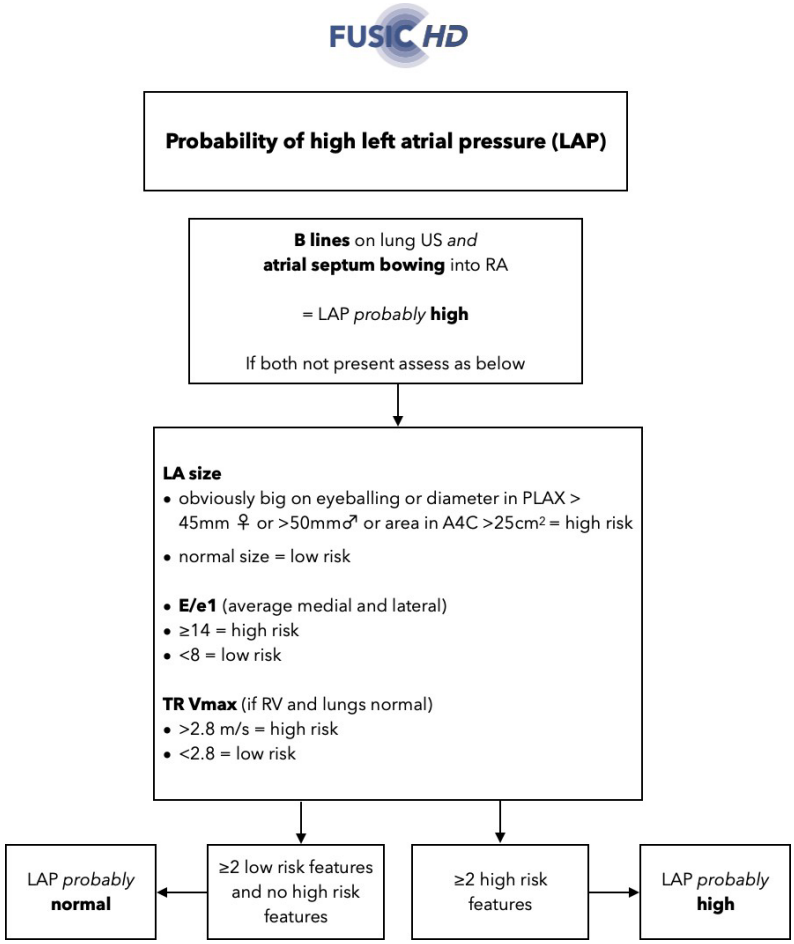
- ≥2 markers supporting normal LA pressure and no markers suggesting high LA pressure

Indeterminate

- Findings do not meet criteria for either category

Figure 2 shows an algorithm for estimation of LA pressure adapted from ASE guidance and modified for use in critically ill patients.

Figure 2. Algorithm for estimation of left atrial pressure.



Q8. Is pulmonary artery pressure raised – and how is the right ventricle responding?

Understanding pulmonary artery (PA) pressure and right ventricular (RV) function is critical to predicting haemodynamic responses to interventions like mechanical ventilation, fluid loading, or vasopressors. These assessments help identify patients with RV–pulmonary arterial (RV–PA) uncoupling, pulmonary hypertension (PH), or acute RV failure — all common and under-recognised problems in critical care.

As such, it is a key question for FUSIC HD.

8.1 Estimating Pulmonary Artery Pressure

The cornerstone of PA pressure assessment is identifying TR using colour Doppler, then measuring TR Vmax. [10] Most ventilated patients have some degree of TR.

PA pressure can be estimated according to the equation:

$$\text{PA pressure} = 4 \times \text{TR Vmax}^2 + \text{RAP}$$

However, rather than attempting to quantify PA pressure in this way, emphasis is now on measuring TR Vmax to detect the probability of raised PA pressure, as follows: [11]

- TR Vmax <2.8 indicates low risk of raised PA pressure
- TR Vmax >3.4 indicates high risk of raised PA pressure
- TR Vmax 2.8 - 3.4 indicates probable raised PA pressure

Important caveats:

- Use multiple views (e.g. PSAX AV, RV inflow, A4C) and record the highest velocity
- Underestimation occurs with poor alignment, poor RV function, or severe TR
- Overestimation may occur with isolated long R–R intervals
- This method is invalid in the presence of pulmonary stenosis

Acute vs Chronic Pulmonary Hypertension

Increased PA pressure can be acute or chronic, and differentiating between them on echo can help with diagnosis and prognosis. Echo clues include:

- In acute cor pulmonale (e.g. pulmonary embolism), the right ventricle is dilated but typically unable to generate very high pressures. TR Vmax is often <3.5 m/s, and the RV wall is typically not hypertrophied.
- In chronic pressure overload, the RV free wall becomes hypertrophied as well as dilated, enabling it to generate higher pressures and velocities.

Measure RV wall thickness in a zoomed subcostal view; an RV free wall >5 mm is considered abnormal.

Right Ventricular Size and Dimensions

RV size offers essential insight into pressure or volume overload and should be assessed alongside TR Vmax and functional indices. Use the apical 4-chamber view, measuring inner-edge to inner-edge at end-diastole:

Measurement	Normal Cut-off	Notes
Basal diameter	<45 mm	Simplified cut-off; BSE: <43 mm (women), <47 mm (men)
Mid diameter	<40 mm	BSE: <35 mm (women), <42 mm (men)
Longitudinal length	<85 mm	BSE: <80 mm (women), <87 mm (men)

Interpret in context — borderline values may still be significant depending on function, pressures, and clinical setting.

Right Ventricular Function – Fractional Area Change (FAC)

RV FAC is now the preferred metric for RV systolic function in FUSIC® HD. It provides a more comprehensive view than longitudinal measures such as TAPSE or RV S′ because it integrates changes in both axes and better reflects RV–PA coupling.
FAC = ((RV diastolic area – RV systolic area) / RV diastolic area) × 100

FAC (%)	Interpretation
≥30%	Normal
<30%	Impaired

- Acquire a clear apical 4-chamber view
- Trace the RV endocardial borders at end-diastole and end-systole
- Avoid foreshortening or inclusion of the RVOT
- FAC is less angle-dependent and correlates with outcome in PH and critical illness

Note: BSE reference ranges vary by sex; FUSIC® HD adopts a simplified 30% threshold to maintain usability while preserving sensitivity.

RV Pressure vs Volume Overload

Septal configuration provides additional insight:

- D-shaped septum in systole → RV pressure overload (e.g. acute PE)
- D-shaped septum in diastole → RV volume overload (e.g. severe TR)
- Throughout cycle → Combined pressure–volume overload

Septal flattening is easier to identify in parasternal short axis at papillary muscle level.

Additional Doppler Features Suggesting Pulmonary Hypertension

Doppler indicators can enhance diagnostic confidence, especially in borderline or ambiguous cases.

RVOT Doppler and Pulmonary Valve Assessment:

Feature	Threshold	Implication
RVOT acceleration time	<105 ms	Suggests raised PA pressure
Systolic notching	Present	Often in proximal obstruction (e.g. PE)
Early diastolic PR velocity	>2.2 m/s	Suggests elevated PA pressures
PA diameter	>25 mm	Enlarged pulmonary artery

How to measure:

- RVOT acceleration time: PW Doppler in PSAX or PLAX outflow view, sample just proximal to pulmonary valve; measure time from onset to peak flow (sweep speed 100 mm/s)
- Early diastolic PR velocity: CW Doppler through pulmonary valve, usually in PSAX
- PA diameter: PSAX, mid diastole, half way between PV and PA bifurcation

Differentiating Pre- vs Post-Capillary Pulmonary Hypertension

Pre-Capillary PH	Post-Capillary PH
Lung disease	LV systolic or diastolic dysfunction
Severe RV and RA enlargement	LA enlargement, significant left heart valve disease
RVOT notching (early)	Features of raised LAP
No signs of raised LAP	Often coexists with LV pathology

Always interpret in the clinical context — echo indicators support, but do not definitively diagnose, the type or cause of PH.

Q9. Are there ultrasound features of cardiac tamponade?

FUSIC® Heart practitioners can identify pericardial and pleural effusions, and distinguish between them. FUSIC® HD goes further to evaluate whether or not the pericardial effusion is consistent with cardiac tamponade. While this is a clinical diagnosis, certain echo features are characteristic.

In the presence of a haemodynamically significant pericardial collection, cardiac chambers are affected during their relaxation, when they are at their lowest pressures. And those with the lowest pressure are usually affected first. Hence, the right atrium is first to collapse in ventricular systole (free wall inversion for >1/3 of systole is highly predictive), followed by the right ventricle in diastole. A pericardial effusion with any right sided chamber collapse should prompt a high suspicion of tamponade. Left-sided chamber collapse is a late phenomenon. Raised RA pressure leads to a dilated, non-collapsing IVC and other signs of venous congestion.

Normal spontaneous ventilation causes peak flow velocity variation through the tricuspid and mitral valves. Pulsus paradoxus associated with tamponade is an exaggeration of this, and can be assessed with PW Doppler at the tips of the TV and MV leaflets. Because tamponade lies somewhere on a continuous spectrum, there is no absolute value for percentage variation that confirms it, which is reflected by various texts using different thresholds. Commonly quoted values are peak E velocity variation of >40% through the TV and >25% through the MV. Velocity variation is attenuated by mechanical ventilation; as such, it should not be relied on to assess mechanically ventilated patients.

The exception to all of this is post-cardiac surgery, when regional tamponade is most common and most often missed, even by experienced operators. Tamponade cannot be excluded post-cardiac surgery by an apparently normal HD study. Expert input is always required if this is suspected.

Q10. Is there venous congestion?

High venous pressures reduce organ blood flow and can cause organ dysfunction and injury. The method described here has a high specificity for predicting acute kidney injury and outperforms CVP or isolated IVC measurements. [12] Venous congestion has also been linked with post-operative delirium. [13] Heart disease, lung disease and volume overload are all causes of venous congestion, as high pressure anywhere downstream of the veins will result in high venous pressures. The most common cause is left ventricular failure (systolic and diastolic).Other causes include MR, pulmonary disease, RV failure and TR - all of which can be detected with US.

Iatrogenic fluid overload, if enough to cause high right atrial pressure (heart failure), is also a common cause in critically ill patients, due to poorly judged fluid resuscitation.

A CVP transducer will provide solid evidence (CVP has a quasi linear relationship with AKI [14] and CVPs of >8mmhg have been shown to be particularly harmful). [15] However, patients do not always have CVP monitoring, and the absolute CVP value does not necessarily predict whether venous flow is affected. If CVP is unavailable, or if the effect the CVP is having on the venous system is in doubt, then ultrasound should be used.

High venous pressures first manifest as IVC dilatation, then as flow abnormalities in the great veins, which can be assessed with PW Doppler [16]

IVC

The IVC is the first vein to interrogate in a subcostal view. Imaging in its short axis can help ensure accuracy.

- An IVC diameter of <2cm makes congestion unlikely.
- An IVC diameter >2cm raises suspicion of venous congestion prompting continuation of the venous scan.

Hepatic vein

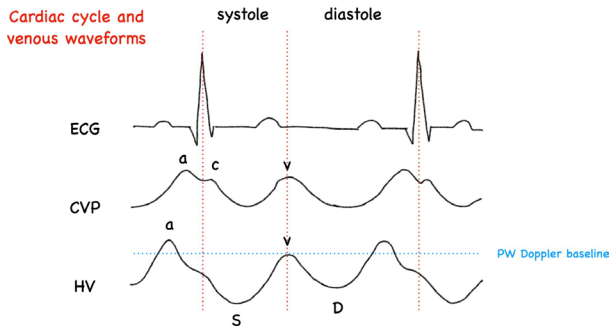


Figure 3. The CVP and HV waveforms related to the cardiac cycle

PW Doppler is then applied to the hepatic vein. Imaging is obtained from a subcostal view or from the anterior to mid axillary line in the 5-7th intercostal space with the marker pointing cephalad and the beam rotated between ribs and directed towards patient's left flank-hip area. Normally an S wave (caused by ventricular systole) and a D (caused by passive RV filling) wave is generated below the baseline, with the S wave being bigger than the D wave. If available, an ECG trace should be used to distinguish these, but they are usually paired so easy enough to identify.

- If the S wave is smaller than the D wave (S/D <1), this is a sign of a mild abnormality.

- If the S wave is inverted above the baseline (meaning TR is present), then this is a sign of a severe abnormality.

Portal vein
With a small amount of angulation from the hepatic vein view, the portal vein can be easily imaged (and vice versa). Normal flow is mostly continuous with a small amount of variation. As congestion develops, flow becomes more pulsatile. This can be visualised with colour Doppler but is quantified with PW Doppler.

The Pulsatility Index is:

PI = (Vmax - Vmin) / Vmax

- PI 0.3-0.5 is mildly abnormal
- PI >0.5 severely abnormal

Renal vessels
Finally, the renal interlobar veins and arteries are interrogated with PW Doppler. Each kidney should be imaged in their long axis, as superficially as possible, and a colour flow box placed over the whole kidney to identify the location of the interlobar vessels between the medullary pyramids. This will allow appropriate placement of the PW Doppler sample gate.

Renal artery:
The arterial Renal Resistive Index is:

RRI = (Vmax - Vmin) / Vmax

- RRI >0.7 is abnormal

RRI is increased in venous congestion but other factors affect arterial systolic and diastolic flow velocities and therefore RRI. These are classified into:

1. pulse pressure
2. a combination of interstitial and venous pressure
3. vascular compliance

Therefore RRI is not specific for venous congestion. It is affected by other systemic haemodynamic factors (cardiac function, arterial compliance and heart rate) and other renal factors (reno-vascular disease, obstruction and compression).

RRI >0.7 precedes AKI in sepsis by up to 48h, and a low CVP and RRI have been shown to have the best renal outcomes in sepsis. Some experts are therefore now using RRI to guide vasopressor management in sepsis.

Renal Vein:
As in the other veins, flow should be relatively continuous. With increasing venous pressures, flow becomes increasingly pulsatile, and then interrupted - first biphasic (S and D waves) and then monophasic - diastolic flow (D wave) only. Venous flows are a much more specific indicator of venous congestion than arterial flows and RRI.

It is really only necessary to observe whether flow is continuous or interrupted. Expressed mathematically, the maximum and minimum velocities can be measured allowing calculation of the Venous Impedance Index (VII). Interrupted flow (biphasic or monophasic), where flow returns to baseline, means that VII = 1.

The Venous Impedance Index is:

VII = (Vmax - Vmin) / Vmax

While a VII of >0.7 is abnormal, it becomes particularly significant when it is 1.

- VII = 1 with systolic and diastolic flow (discontinuous with S and D waves present) - mildly abnormal
- VII = 1 with only diastolic flow (discontinuous with only D wave) - severely abnormal

Scoring
A venous excess ultrasound (VExUS) score has been validated to quantify venous congestion into grades of increasing severity. [12] This is outlined in table 5.

VExUS score	
Grading score	
Grade 0	**IVC <20mm**
Grade 1	**IVC >20mm *plus*** no or mild abnormalities in any pattern
Grade 2	**IVC >20mm *plus*** severe abnormality in 1 pattern
Grade 3	**IVC >20mm *plus*** severe abnormalities in >1 pattern
Abnormality patterns	
Hepatic vein	Mild - S < D Severe - S above baseline
Portal vein	Mild - PI 0.3-0.5 Severe - PI >0.5
Interlobar renal vein	Mild - Interrupted S and D phase Severe - Interrupted only D phase
Other causes for abnormal venous flow patterns	

VExUS score
Primary organ disease (hepatic, renal) Vessel thrombosis or stenosis Raised intra abdominal pressure Portal Vein PI >0.5 in young age, athletes, low BMI, hyperdynamic states

Table 5. VExUS scoring

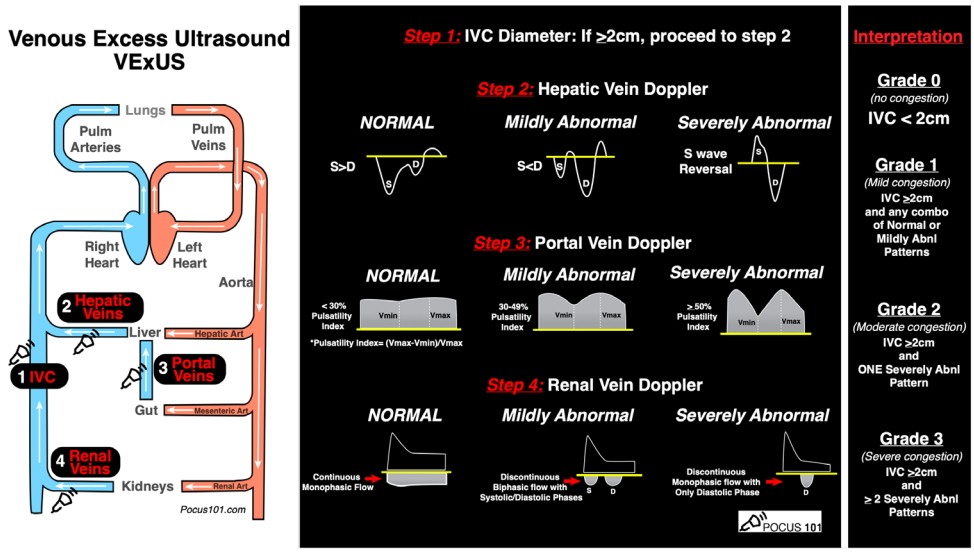


Figure 4. Normal and abnormal venous waveforms in VExUS scoring (with permission from POCUS101).

Clinical uses

Indications for performing a venous congestion scan are listed in table 6. Lung and heart ultrasound should also be performed to look for possible causes (pulmonary disease, cardiac disease and fluid overload). Manifestations of fluid overload that can be seen with ultrasound include a dilated right heart, functional TR, pulmonary oedema, pleural effusions, pericardial effusions and intra-abdominal fluid (ascites, gall bladder wall thickening).

Indications for a venous congestion scan
Assessment of fluid balance Acute kidney injury (including acute-on-chronic) Newly deranged LFTs (especially if associated with AKI) Delirium To guide fluid removal (diuretics or RRT) LV or RV failure CVP >8

Table 6. Indications for performing a venous congestion scan

When thinking about fluid balance, a congestive pattern strongly suggests that fluid removal will be beneficial (tamponade is an obvious exception to this). A non-congested pattern suggests fluid administration will be tolerated without too much harm (but does not suggest that fluid is needed). Any findings should be considered in conjunction with other signs of fluid balance - history, cumulative fluid balance, presence of peripheral oedema, AKI, CVP etc.

Seeing how venous flows respond to interventions is particularly important. For example, VII reduction would be a target of fluid removal.

Performing the scan

Clinicians undertaking FUSIC® HD are expected to already be accredited in FUSIC® Heart and Lung which provide the fundamentals on which a full haemodynamic scan is built. FUSIC® Heart teaches 4 basic cardiac views while HD introduces additional views. These can be seen in table 7.

FUSIC® Heart views	Additional views for FUSIC® HD
Parasternal long axis (PLAX) Parasternal short axis (PSAX) Apical 4 chamber (A4C) Subcostal long axis (SC)	Right ventricular inflow (RVI) Right ventricular outflow (RVO) Apical two-chamber (A2C) Apical three-chamber (A3C) Subcostal short-axis (SSAX) Aortic views - Suprasternal (SS), modified PLAX, modified SAX, modified A2C, abdominal aorta (AA) Hepatic Renal

Table 7. Additional views obtained in FUSIC® HD compared to FUSIC® Heart

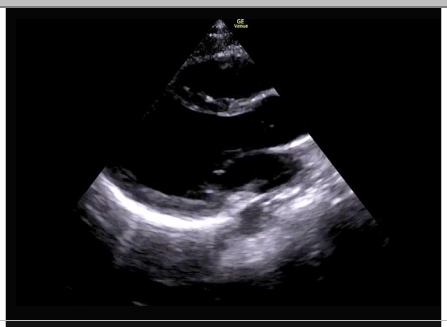
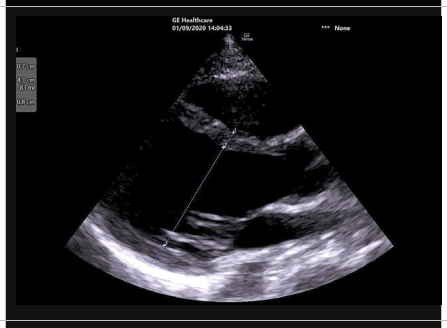
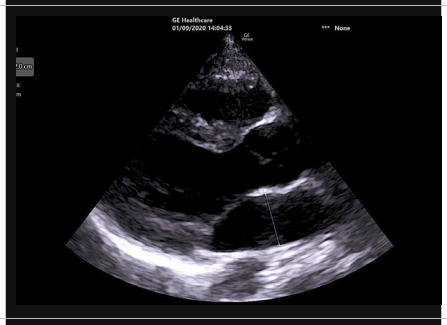
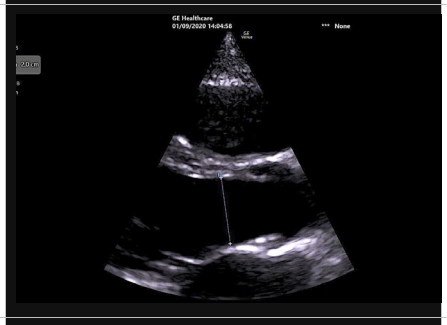
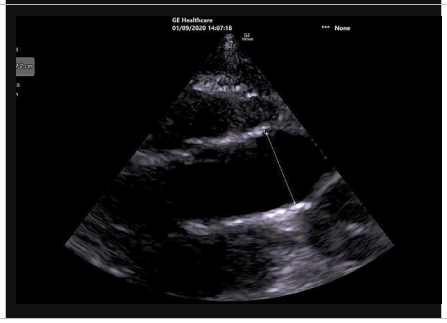
The views, and measurements and calculations in each view, required for a full FUSIC® HD scan are listed in table 6. Table 7 provides a step by step sequence of the suggested order of image acquisition with details of probe placement, example images, technical settings, measurements and calculations to be performed, and what to look for in each view. Several of the aortic views are modified standard echo windows. The operator can obtain these views whilst on the relevant echo view or perform them separately. If an aortic dissection is suspected it would be advisable to perform these separately. Readers are encouraged to look at the FUSIC® section of the Intensive Care Society website for e-learning resources such as videos of how to perform various parts of a full HD scan. Reference ranges of normal, and significantly abnormal, values is displayed in table 8.

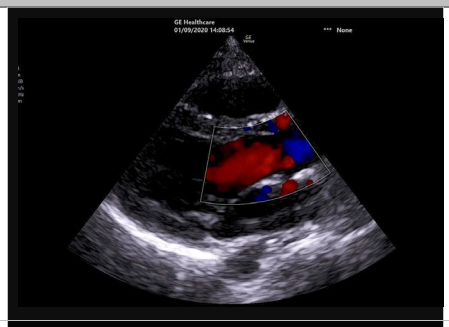
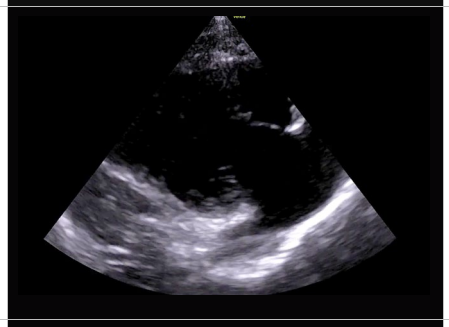
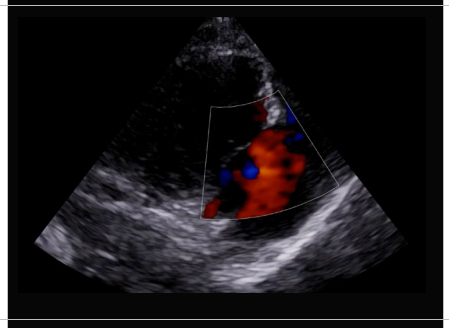
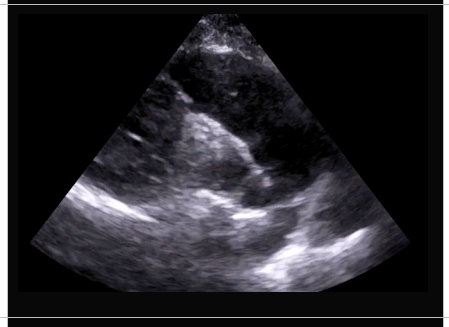
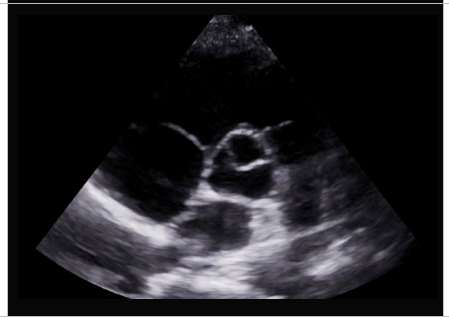
FUSIC® HD views, measurements and calculations	
PLAX	Inspect LV size and systolic function Inspect RVOT size and systolic function Measure LV basal diameter at end-diastole Measure LV basal wall thickness at end-diastole Calculate wall thickening % Measure LA diameter at end-systole Measure diameter of the ST junction and ascending aorta 2cm distally Measure LVOT diameter at mid-systole Inspect AV and MV for general appearance (thickness, calcification, mobility) Inspect MV for SAM/LVOT obstruction Inspect AV and MV for regurgitation using colour Doppler Inspect for pleural and pericardial effusions
RV inflow	Inspect RV size and systolic function Inspect TV for general appearance (thickness, calcification, mobility) Inspect TV for TR using colour Doppler Measure TR Vmax (if TR present) using CW Doppler
RV outflow	Inspect RVOT size Measure PA diameter Measure RVOT acceleration time Measure early diastolic PR velocity Inspect for systolic notching of RVOT Doppler
PSAX AV level	Inspect position and movement of inter-atrial septum Inspect AV and TV for general appearance (thickness, calcification, mobility) Inspect TV for TR using colour Doppler Measure TR Vmax (if TR present) using colour Doppler Measure PA diameter Measure RVOT acceleration time Measure early diastolic PR velocity Inspect for systolic notching of RVOT Doppler
PSAX MV level	Inspect MV leaflets for general appearance (thickness, calcification, mobility) Inspect for RWMAs Inspect for pericardial effusion
PSAX papillary level	Inspect LV for size and systolic function Inspect for pericardial effusion Inspect for RWMAs Inspect RV for size and function Inspect inter-ventricular septum for position and movement
PSAX apex	Inspect for twisting motion Inspect for RWMAs Inspect for pericardial effusion

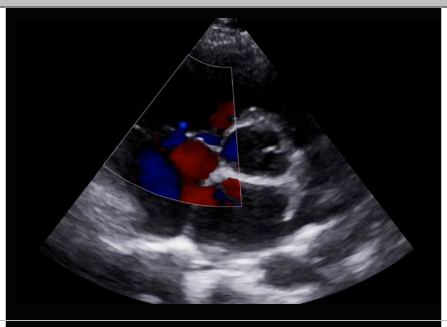
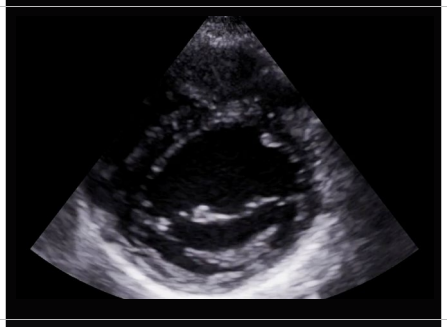
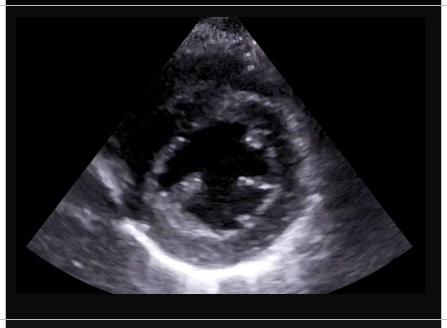
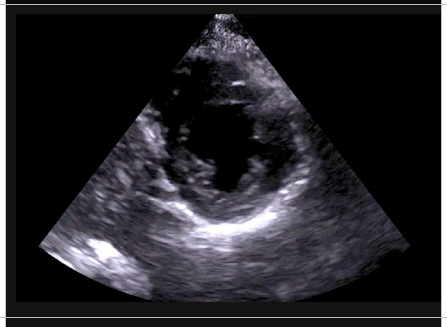
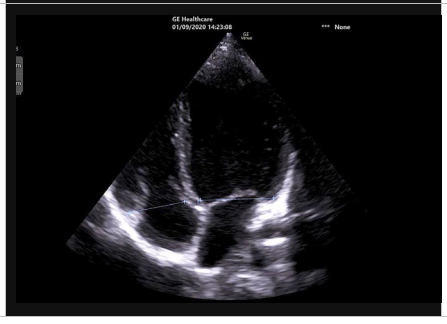
FUSIC® HD views, measurements and calculations	
A4C	Inspect all four chambers for size Inspect for pericardial effusion Inspect LV for systolic function Inspect for RWMAs Measure LVEDA and LVESA Calculate LVEF Inspect RV for systolic function Measure basal, mid and longitudinal RV dimensions Measure RV/LV basal ratio Measure RV area in diastole and systole Calculate RV fractional area change Measure LA area end systole Measure TAPSE and MAPSE using M-mode Inspect MV and TV for general appearance (thickness, calcification, mobility) Inspect MV and TV for regurgitation using colour Doppler Measure TR Vmax (if present) using CW Doppler Measure E and A velocities using PW Doppler Measure e^1 using TDI at both the septal and lateral MV annuli (then average results) Calculate E/A and E/e^1 Measure LV S^1 using TDI at the lateral MV annulus Measure RV S^1 using TDI at the lateral TV annulus Measure LV area at end diastole and end systole Calculate ejection fraction (average with A2C for biplane EF)
A5C	Inspect AV for general appearance (thickness, calcification, mobility) Inspect AV for regurgitation using colour Doppler Measure AV VTI with CW Doppler Measure LVOT VTI using PW Doppler (including looking for LVOT obstruction) Calculate LVOT VTI or Vmax respiratory variation using an appropriate sweep speed Measure percentage change in LVOT VTI with passive leg raise or fluid challenge Inspect MV for SAM Measure dimensionless index using CW Doppler across LVOT/AV (if both traces are visible simultaneously)
A2C	Inspect LV for systolic function Inspect for RWMAs Measure LVEDA and LVESA Calculate biplane LVEF Inspect MV for general appearance (thickness, calcification, mobility) Inspect MV for regurgitation using colour Doppler Inspect for pericardial effusion Measure LV area at end diastole and end systole Calculate ejection fraction (average with A4C for biplane EF)
A3C	Inspect LV for systolic function Inspect for RWMAs Inspect AV and MV for general appearance (thickness, calcification, mobility) Inspect AV and MV for regurgitation using colour Doppler Inspect MV for SAM Measure LVOT VTI using PW Doppler (including looking for LVOT obstruction) Calculate LVOT VTI or Vmax respiratory variation using an appropriate sweep speed Measure percentage change in LVOT VTI variation with passive leg raise or fluid challeng Measure dimensionless index using CW Doppler across LVOT/AV (if both traces are visible simultaneously) Inspect for pericardial effusion
FUSIC® HD views, measurements and calculations	

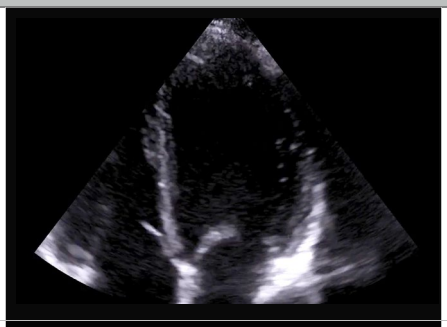
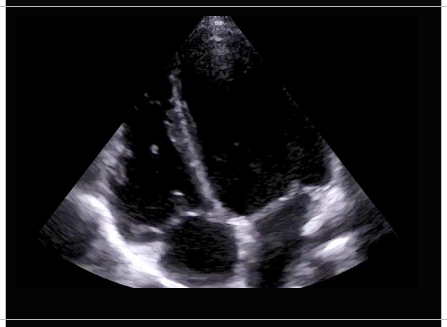
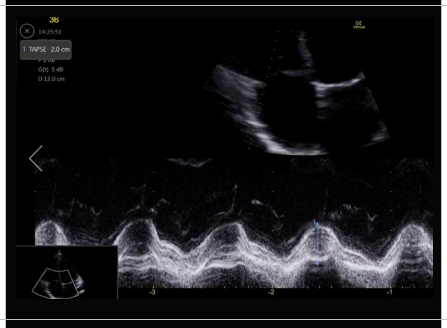
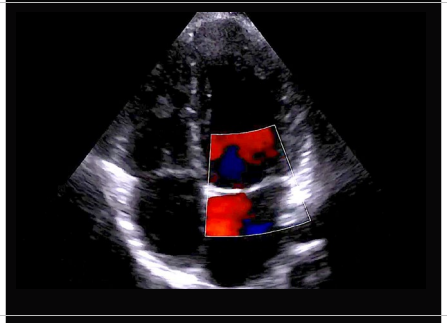
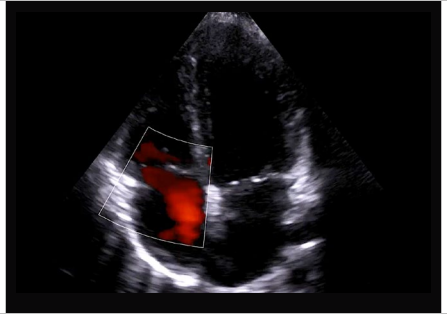
Subcostal	Inspect all four chambers for size and systolic function Inspect for pericardial effusion Inspect for ascities Measure RV free wall thickness at end-diastole in zoomed view
IVC	Measure IVC diameter 1-4cm proximal to RA in long and short axis Inspect respiratory variation
Hepatic vein	Inspect flow pattern using colour Doppler Inspect waveform appearance using PW Doppler Measure S/D ratio
Portal vein	(Can be assessed subcostally, RUQ or mid-axillary) Inspect size and general appearance Inspect flow pattern (red, red/blue, blue) using colour Doppler Inspect flow pattern using PW Doppler Measure Pulsatility Index using PW Doppler
Renal	Inspect size and general appearance (e.g. hydronephrosis) Inspect interlobar vessels between medullary pyramids using colour Doppler Inspect arterial and venous flows and waveform appearance at segmental artery/vein using PW Doppler Measure Renal artery Resistive Index Measure Venous Impedance Index
Aorta modified PLAX	Modified PLAX Inspect aortic root Measure ascending aorta diameter (3-4cm from aortic valve)
Aorta suprasternal	Inspect aortic arch and great vessels Inspect flow pattern with colour Doppler
Aorta modified PSAX	Inspect descending aorta in long-axis (running across the far field) Inspect flow pattern with colour Doppler
A4C	Inspect upper descending aorta in short-axis
Aorta modified A2/3C	Inspect descending thoracic aorta (distal to subclavian artery) in long-axis (running across the far field) Inspect flow pattern with colour Doppler
Subcostal	Inspect abdominal aorta in long and short-axis Measure aortic diameter
Epigastric	Inspect abdominal aorta in long and short-axis Measure aortic diameter

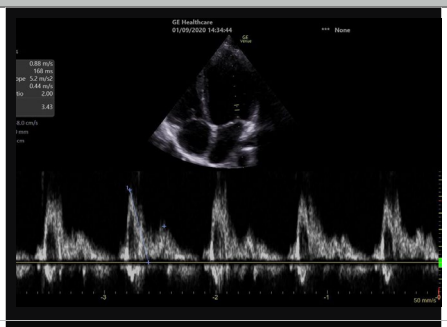
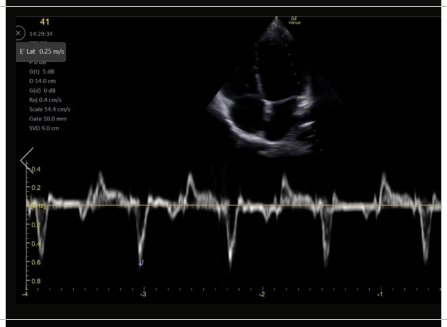
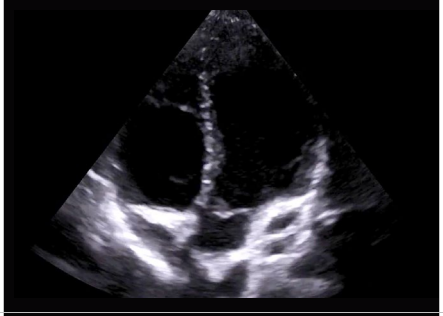
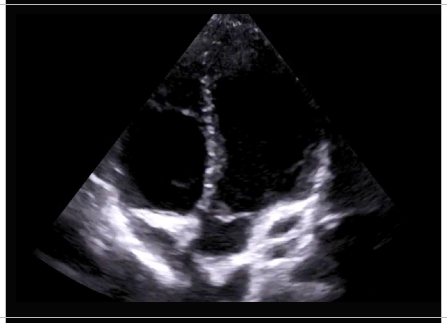
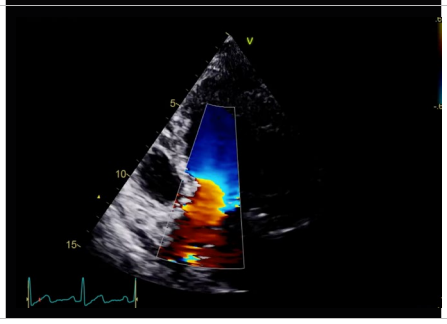
Table 8. Standard views and measurements

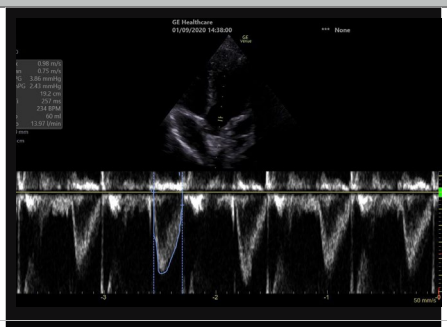
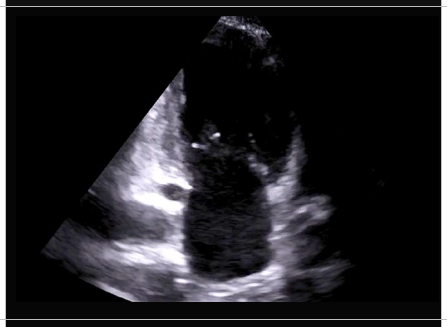
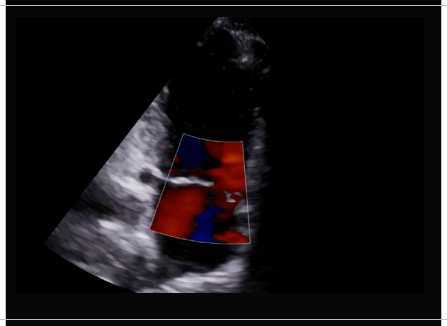
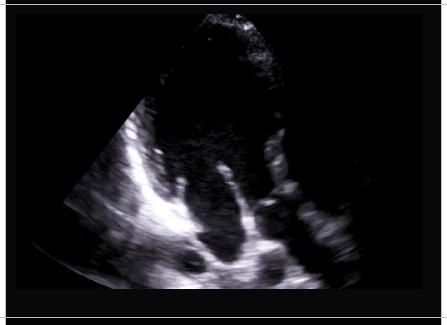
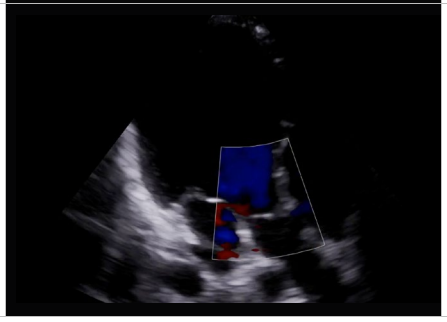
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
PLAX (2D) Deep view Left sternal border Marker to right shoulder Highest rib space possible			• Pleural space • Pericardial space
PLAX (2D)		• LVIDd • IVSd • LVPWd • LA diameter end systole Do not include any RV wall in septal measurement	• Chamber size and function • Valves (thickening, calcification, mobility, apposition, SAM MV) • Pericardium • Ventricular walls (thickness and motion)
PLAX (2D)		• LA size end systole Measure perpendicularly to atrial walls	
PLAX (2D) focused on LVOT Reduced depth and width to focus on LVOT *or* Zoom		• LVOT diameter Mid systole Measure at the hinge points of the AV leaflets	
PLAX (2D) focused on ascending aorta Higher rib space may improve view		Measure the root at the ST junction and ascending aorta 2cm distally (inner edge to inner edge)	Size, dissection flap

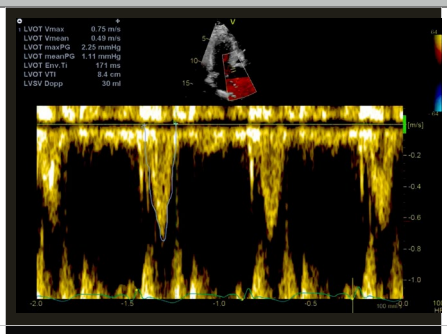
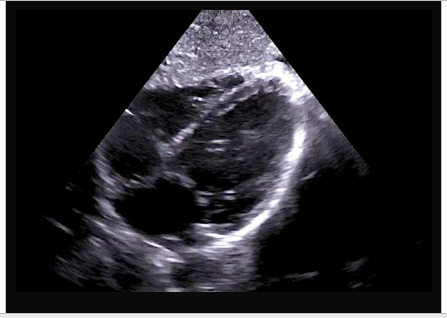
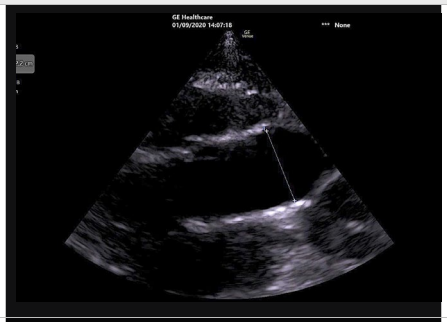
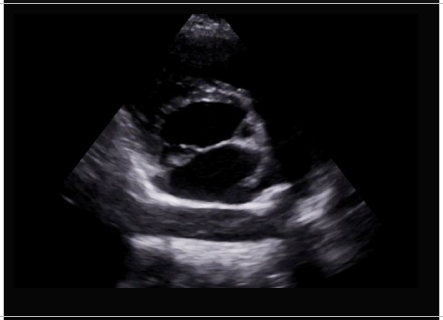
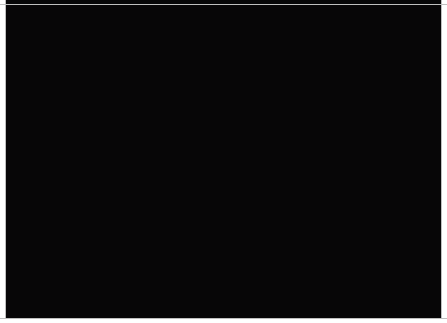
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
PLAX (CFM)			• MV and AV • Look for abnormal flow (regurgitation, turbulence)
RV inflow (2D) and CFM Tilt beam towards right hip			• RV size • RV Systolic function • TV appearance
RV inflow (CFM)		TR Vmax with CW Doppler aligned with jet	Size and direction of TR jet
RV outflow Tilt towards left shoulder Slight clockwise rotation		PW Doppler RVOT RVOT Acceleration time CW Doppler PV Early diastolic PR velocity	RVOT size Doppler envelope morphology
PSAX AV level (2D) Rotate 90^0 clockwise from PLAX view Superior tilt		RVOT diameter PW Doppler RVOT RVOT Acceleration time CW Doppler PV Early diastolic PR velocity	• Atrial septal movement • AV/TV appearance Doppler envelope morphology

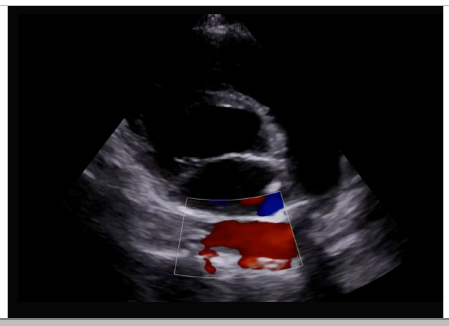
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
PSAX AV level (CFM)		• TR Vmax	Size and direction of TR jet
PSAX MV level Inferior tilt from AV level			• LV size and function • Septal position and movement • LV RWMAs • RV size and function • Anterior and posterior MV leaflets
PSAX papillary level Inferior tilt from MV level			• LV size and function • Septal position and movement • LV RWMAs • RV size and function
PSAX apex Inferior tilt from papillary level			• LV RWMAs • LV function (look for twisting motion)
A4C (2D) Probe directed apex to base of heart Marker to left side of patient		• RV/LV basal ratio • LA area end systole	• Chamber size • LV and RV systolic function • MV and TV appearance

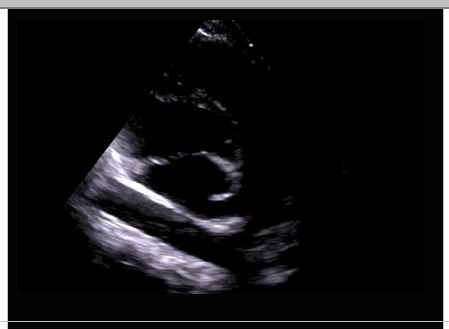
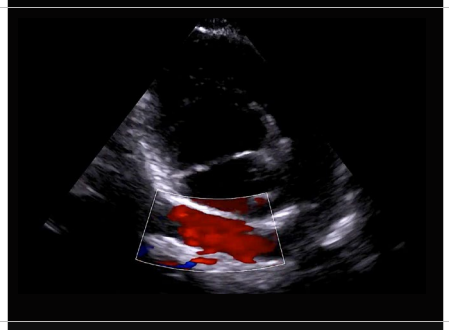
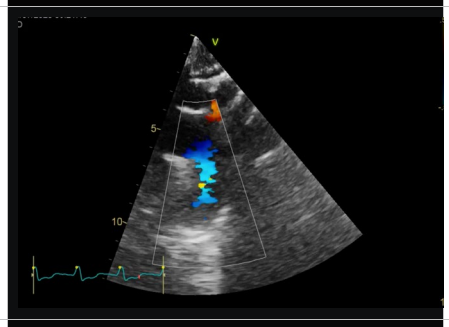
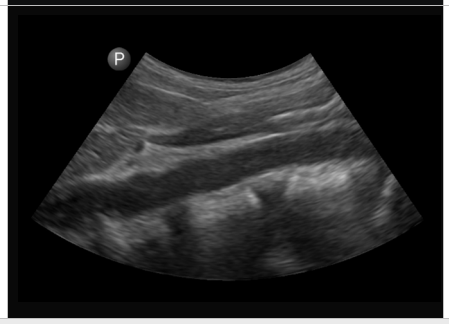
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
A4C (2D) LV view Reduce depth +/- narrow width to focus on LV		• LVEDA • LVESA • calculate LVEF	• Chamber size • LV systolic function • MV appearance
A4C RV focused Tilt to patient's right Rotate probe to maximise RV dimensions		• RVEDA • RVESA • calculate RV FAC • TAPSE • S1 (TDI) • RV basal diameter • RV mid diameter • RV longitudinal diameter	RV size and function
A4C (MM) focused and zoomed on MV and TV annulus			
A4C (CFM) Colour box appropriate size			Size and direction of MR and TR jets
A4C (CFM) Colour box appropriate size		• TR Vmax	

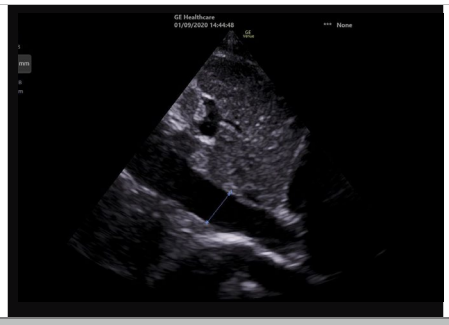
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
A4C (PW) tips of MV leaflets Use colour Doppler to help identify LV inflow to position PW gate		• E velocity • A velocity • calculate E/A ratio	Use CFM to help position PW sample box
A4C (TDI)		• e¹ lateral MV annulus • e¹ medial MV annulus • Calculate E/e¹ • LV S¹ lateral MV annulus* • RV S¹ lateral TV annulus* *S¹ not necessary if LVEF measured	
A5C (2D) Tilt anteriorly from A4C view			AV appearance
A5C (CW)		• VTI AV	
A5C (CFM)			• AV for regurgitation • Turbulence in LVOT

FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
A5C (PW)		• LVOT VTI • calculate LVOT respiratory variation using appropriate sweep speed • % change LVOT VTI with PLR or fluid challenge Dimensionless Index = VTI_{LVOT} / VTI_{AV}	
A2C (2D) Rotate 60⁰ anti-clockwise from A4C view		• LVEDA • LVESA • LVEF biplane	• LV systolic function • RWMAs • MV appearance
A2C (CFM) Colour box appropriate size			MV regurgitation - size and direction of jet
A3C (2D) Rotate 60⁰ anti-clockwise from A2C view			• LV systolic function • RWMAs • MV for SAM
A3C (CFM) Colour box appropriate size			AV and MV regurgitation - size and direction of jet

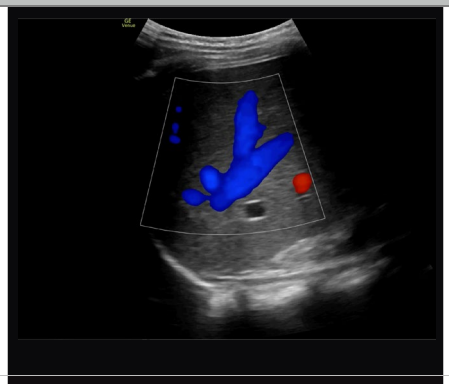
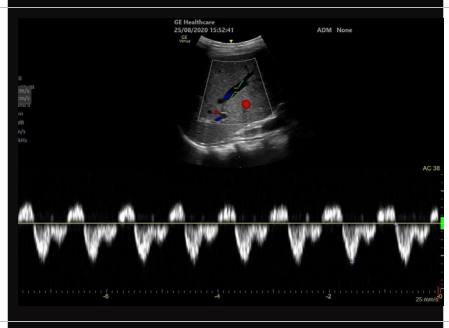
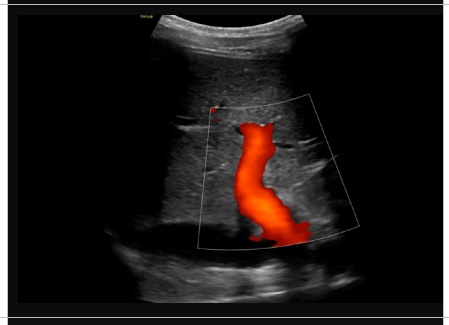
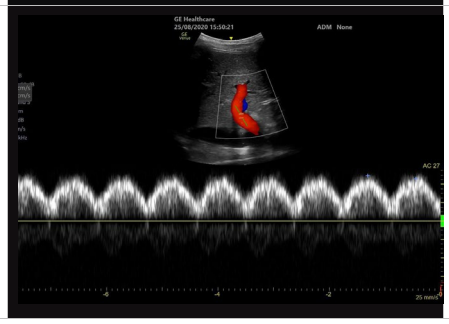
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
A3C (PW)		• LVOT VTI	
Subcostal Subxiphoid, probe flat, marker to left shoulder		• RV free wall thickness end diastole (focused and zoomed)	• Chamber size • LV and RV function • Pericardial effusion • Ascites
Aortic views			
PLAX (2D) focused on ascending aorta Higher rib space may improve view		Measure ascending aorta 3-4 cm from AV (inner edge to inner edge)	Size, dissection flap
PSAX (2D) MV level modified for descending aorta (tilt anteriorly and anticlockwise) 2D			Dissection flap
			

PSAX (CFM) MV level modified for descending aorta (tilt anteriorly and anticlockwise) CFM			Colour flow pattern

FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
A2/3C (2D) modified for descending aorta (90 rotation from A4C)			Dissection flap
A2/3C (CFM) modified for descending aorta			Colour flow pattern
Suprasternal Marker at 1 o'clock Angle down to cut through right nipple and tip of left scapula			Dissection flap Colour flow pattern
Epigastric Abdominal aorta long and short axis Xiphisternum to umbilicus		measure diameter	Dissection flap Colour flow pattern
Venous views			

IVC (2D) Subcostal phased array (PA) or curvilinear (CL) Marker to 12 o'clock Slide probe to patients R to align plane with IVC		measure diameter 1-4cm proximal to RA in long and short axis In long axis ensure US beam slicing through widest diameter	Respiratory variation in diameter

FUSIC® HD full data set

View	Image	Measurements and settings	Inspect
Hepatic vein (colour) PA Subcostal or CL Anterior to mid axillary line 5-7th intercostal space, marker cephalad, rotated between ribs, direction towards patient's left flank-hip		Colour scale 20-40cm/s	Colour flow pattern
Hepatic vein (PW)		Scale +20 to -30 cm/s Baseline mid to high Sweep speed 25 mm/s Measure: S wave D wave S/D ratio	Waveform appearance
Portal vein (colour) Anterior to mid axillary line 5-7th intercostal space, marker cephalad, rotated between ribs, direction towards patient's left flank-hip		Colour scale 20-40 cm/s	Size and appearance colour flow pattern
Portal vein (PW)		Sweep speed 25 mm/s Baseline mid to low Measure : Max and min velocity Calculate: Pulsatility index	PW Doppler waveform pattern

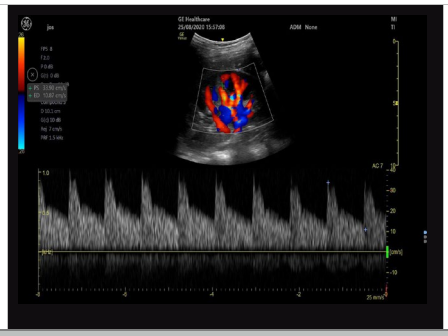
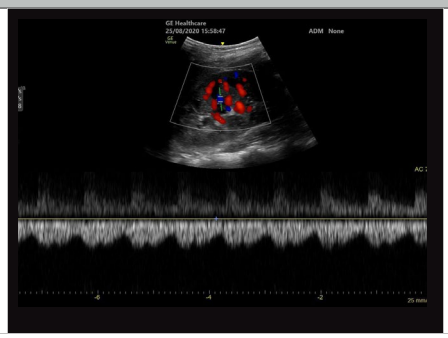
Renal (colour) and PW Position probe in long axis of each kidney		Colour scale 20-40 cm/s Sweep speed 25 mm/s Baseline mid to low Measure: Peak systolic and end diastolic arterial flow Calulate: Renal resistive index (RRI)	• Identify interlobar arteries and veins • PW Doppler waveform pattern
FUSIC® HD full data set			
View	Image	Measurements and settings	Inspect
Renal (colour) and (PW)		Baseline - mid Optional - Measure max and min velocities and calculate Venous Impedance Index (VII) *Arterial and venous traces often obtained from same sample volume*	• Identify interlobar arteries and veins • PW Doppler waveform pattern - normal or interrupted (biphasic or monophasic)

Table 9. suggested sequence of image acquisition and full data set

Measurement techniques

Measuring dimensions

LVIDd, IVSd/PWIDd wall thickness, LVOT diameter, and LA diameter are all measured in a PLAX view - ideally the mid PLAX view (with the LV cavity and LVOT next to each other in the field). The lower PLAX view (with the LV cavity lying higher than the LVOT in the field) may visualise the secondary MV cords within the LV cavity and compromise accuracy of its basal dimension measurement. The higher PLAX view mainly visualizes the aortic root.

Freeze the image at end-diastole and measure the septum, the LV cavity (inside edge to inside edge) and infero-lateral wall along a line cutting perpendicularly through the long-axis of the LV at the tips of the open MV leaflets. Ensure that no RV wall is included in the septal measurement (RV and LV wall can be distinguished in the septum) and that no trabeculations or papillary muscles are included. Repeat these measurements at end-systole.

Focus on the LVOT by reducing depth and width or by using the zoom function.

Measure the LVOT diameter in mid-systole at the hinge points of the AV leaflets. Errors are squared in stroke volume calculations, so care should be taken to measure this accurately. LVOT diameter is almost never <2cm.

LA diameter is measured at end-systole, from the sinus of valsalva to the posterior LA wall, in the

same plane as the closed AV leaflets.

LA area is measured in the A4C view by tracing around the inside border of the LA at end systole.

The basal RV/LV ratio is measured in the A4C view. The diameter of the base of the RV and LV are measured at end diastole at their widest point (not the annulus). Measurements should be taken from the inner edge to inner edge of the ventricular walls and not include any valvular apparatus or trabeculations. These measurements are for a ratio calculation and also a measure of RV size (in conjunction with other RV measurements).
RV wall thickness is measured most easily in the subcostal view at end diastole at mid ventricular level. Correct placement of the focus point and zooming the image will increase accuracy. Trabeculations should not be included in the measurement.

Using M-mode

TAPSE and MAPSE are measured using M-mode in the A4C view through the respective lateral annulus. Accurate measurement requires focussing the image on point being measured. The longitudinal movement of the annulus should be as parallel to the M-mode line as possible which may require repositioning of the probe. Using zoom improves accuracy. It is essential to follow the white line of the leading edge of the annulus so the same part of the annulus is measured at its nearest and furthest distances. It is vertical displacement of the trace that is measured, not its slope.

M-mode is also used through the LVOT in the PLAX view to assess for SAM. Set sweep speed to around 50 mm/s.

Using pulsed-wave Doppler

With PW Doppler, a sampling volume is placed at a specific point. Unlike CW Doppler, it does not transmit and receive continuously, but waits a specified time for the pulse to reach the set depth and return to the probe, thus ignoring any signals outside the sampling volume. In laminar flow conditions, PW Doppler produces a trace with a characteristic inner and outer envelope.

If the pulse repetition frequency (PRF) is low (as happens with increased depth), or the velocity of what is being measured (usually blood flow) is high, then the machine is not sampling fast enough to know in what direction flow is occurring. This is known as aliasing.

Aliasing is represented in PW Doppler by the tip of the PW trace being cut-off and translated to the opposite side of the baseline. The velocity this happens at is called the Nyquist limit. Aliasing is reduced by increasing the velocity scale, adjusting the baseline to increase the Nyquist limit in a particular direction, and increasing the frequency (for example reducing the depth of the sample volume).

Measuring E and A velocities
The sweep speed should be set to around 50mm/s. This usually produces several traces on the screen that can be viewed simultaneously. The baseline should be lowered and velocity scale set to maximise the size of the trace. The gain is set appropriately so the trace not too dark or too bright. An ECG must be used to ensure correct delineation of E and A waves and the peak velocity of each should be measured using calipers. For accuracy, at least 3 traces should be evaluated in sinus rhythm, and at least 5 in atrial fibrillation.

Measuring LVOT VTI
In either the A5C or A3C views, the sample box is placed in the LVOT within 1cm of AV. The baseline is raised and velocity set to maximise the size of the trace. A sweep speed 50-100 mm/s is appropriate to trace the VTI. A higher sweep speed makes the PW trace larger and easier to trace accurately. For accuracy, at least 3 traces should be evaluated in sinus rhythm, and at least 5 in atrial fibrillation.

Measuring LVOT VTI or Vmax respiratory variation
A sweep a speed of 25 mm/s will make the trace smaller and show multiple heart beats over the full respiratory cycle, so that the lowest and highest peak velocities can be compared to assess Vmax variation. A sweep speed of 50 mm/s will allow enough cardiac cycles to be seen as well as provide a large enough trace to measure accurately.

Measuring venous flows
The sampling volume should be as parallel as possible to blood flow. Appropriate angulation of the probe and using the angle correct function on the ultrasound machine will improve accuracy. As venous flows are slower than arterial flows, a velocity scale of 20-40 cm/s is usually appropriate. The baseline should be set depending on the waveform you are examining is towards or away from the probe. A sweep speed of 25 mm/s is appropriate. The renal interlobar vessels run together which means that arterial and venous waveforms are often displayed simultaneously. If arterial and venous flows are seen separately then individual traces can be demonstrated.

RVOT and PV Doppler
RVOT acceleration time: PW Doppler in PSAX AV level or PLAX outflow view, sample just proximal to pulmonary valve; measure time from onset to peak flow (sweep speed 100 mm/s).

Using colour Doppler
Colour Doppler is a form of pulsed wave Doppler where flow is assessed at multiple points in a region of interest. It is traditionally colour coded as blue representing flow away from the probe and red towards (BART). Darker shades represent lower velocities; brighter shades higher velocities.

Laminar flow is uniform in colour; turbulent and high-velocity flow is represented by a mosaic of red and blue. Variance is an optional setting, which codes turbulent flow as green.

Frame rate, and therefore image quality, is maximised by making the colour box as narrow and shallow as possible while still including the area of interest.

Gain should be set by gradually increasing it until speckling appears outside the area of interest, then reducing it to the point that this disappears.

Velocity range is also known as scale. The set velocity range represents the Nyquist limit in each direction (above which aliasing will occur). A higher scale will alias less but will not show slower flow. Increasing the scale will make a regurgitant jet look smaller; lowering the scale will make it look bigger. The recommended range for most valve interrogation is 50-70 cm/s.

Lower velocity flows like the portal vein will need a scale of around 20-40 cm/s. Very low velocities may not show up even with a low scale. In such cases, power Doppler, whose colour is directionless, can detect lower velocities (e.g. when looking for venous thrombosis).

Due to the Doppler equation ($\cos 90^0 = 0$) flow will be detected more easily, and measured more accurately, the more parallel it is to the US beam. The measured angle must be between $0\text{-}60^0$ (the closer to 0 the better). Angling the probe to achieve this should always be done. Most machines will also have an angle correct function to correct any deviance from parallel.

Using continuous wave Doppler

CW Doppler measures all blood velocities along the chosen line. Because of this, it creates a trace that is characteristically filled in, with no inner envelope. CW Doppler has the advantage of measuring high blood velocities. However, it does not demonstrate where along this line the peak velocity has been generated.

FUSIC® HD uses CW Doppler through the AV to calculate dimensionless and through the TV to measure TR Vmax (which is critical for risk stratifying patients for pulmonary hypertension and raised LA pressure). Early diastolic PR velocity is measured by placing CW Doppler through pulmonary valve, usually in PSAX aortic level view.

Using tissue Doppler

Tissue Doppler imaging (TDI) is another form of pulsed wave Doppler. Myocardium moves more slowly than blood cells however, and its amplitude is higher, so the signals are subjected to different filter and gain settings by TDI to reveal a characteristic velocity-time trace.

In FUSIC® HD, TDI is used to measure e^1, LV S^1 and RV S^1 the method of which is shown in table 7. It is important to use an ECG trace to time the cardiac cycle and its relation to the observed waveforms so they are identified correctly. LV S^1 is not essential if LVEF has been measured but is a useful marker if LVEF cannot be measured.

Measuring e^1

Place a PW TDI sample box over the lateral and then the medial MV annulus. Set the sweep speed to around 100 mm/s. Raise the baseline (as it is looking at movement away from the probe) and set the velocity to maximise the size of the trace. Adjust the gain appropriately. An ECG must be used to correctly identify e and a waves. Measure the peak velocity of the first diastolic wave (the e1 wave).

Measuring LV S1 and RV S1

PW TDI is placed over MV lateral and then TV lateral annulus. Set the sweep speed 100 mm/s and lower the baseline (as it is looking at movement towards the probe) and set the velocity to maximise the size of the trace. Adjust the gain appropriately, and measure the peak velocity of the systolic wave (the S1 wave).

Evaluating the aorta

Features associated with aortic dissection are dilatation, turbulent, non-pulsatile flow, a dissection flap, aortic regurgitation and a pericardial effusion. These should be looked for, where relevant, in the aortic views described below.

Aortic root and ascending aorta:

This is visualised from the PLAX view with the depth reduced and probe tilted to focus on the aortic root. Tilting superiorly, or moving up a rib space (sometimes combined with angling down), may improve the view. Examine the aortic valve and look for a dissection flap in the aortic root. Measure the diameter of the sinotubular junction (end of root) and the ascending aorta 2cm distally. The aortic root can also be visualised from the A5 and A3C views.

Aortic arch:

Place the probe in the supra-sternal notch with the marker directed to 1 o’clock. Angle down to cut through a line between the right nipple and tip of left scapula.

Thoracic aorta:

From the PSAX view at MV level, tilting anteriorly and anticlockwise will modify the view for the descending aorta. It can also be visualised from a hybrid A2C/A3C view. Centre the aorta in the A4C view and rotate anticlockwise until probe cephalad/caudad in line with the aorta.

Abdominal aorta:

Examine the aorta between the xiphisternum and umbilicus in long and short axis. For further information on aortic assessment see Q3.

Normal values

Comprehensive lists of normal and abnormal values for echo measurements have been published by both the BSE 17 and ASE 18 and these have been used where relevant to provide the information in table 8. Some measurements have been simplified for FUSIC® HD and readers are encouraged to consult the BSE and ASE documents for greater precision.

Measurement	Normal	Significant	Notes
End systole			Frame where AV closes ***or*** Frame prior to MV opening
End diastole			Frame before the MV closes ***or*** largest LV cavity size ***or*** start of QRS complex on ECG ***or*** R wave on ECG
LVIDd (mm)	<50 ♀ <55 ♂	>60 ♀ >65 ♂	
IVSd (mm)	<12	>12	
LVPWd (mm)	<12	>12	
LV wall thickening	>30%	Hypokinetic 0-30% Akinetic - no thickening	
LA diameter PLAX end systole	<4.5cm ♀ <5cm ♂	>4.5cm ♀ >5cm ♂	best sign of high LA pressure is septum bowed into RA throughout cardiac cycle
LA area	10-22 cm^2	>22 cm^2	end ventricular systole
RVOT proximal PLAX/PSAX	<4.5cm	>4.5cm	
RVOT acceleration time	>105ms	<105ms	
RV fractional area change	>30%	<30%	
Early diastolic PR velocity	<2.2m/s	>2.2m/s	
PA diameter	<2.5cm	>2.5cm	mid way between PV and bifurcation
RV basal diameter	<4.5cm	>4.5cm	
RV mid diameter	<4cm	>4cm	
RV longitudinal diameter	<8.5cm	>8.5	
Dimensionless index	1	<0.25 = severe AS	
TAPSE	>1.7cm	<1.7cm	
LVEF	Normal: ≥ 55%	Borderline-low: 50–54% Impaired: 36–49% Severely impaired: ≤ 35%	Biplane measurment where possible
RV S wave cm/s	>9	<9	
LV S (optional)	>7		
E/A ratio	<1	>2	1-2 grey area >2 in young age and hyperdynamic states
Measurement	**Normal**	**Significant**	**Notes**

E/e[1]	<8	>14	average of medial and lateral measurements
RV wall	≤5mm	>5mm	RV hypertrophies quickly when afterload high
E wave velocity variation (tamponade assessment)		TV >40% MV >25%	only in spontaneous respiration.
TR velocity	<2.8	>2.8	4 x V^2 = pressure in mmHg.
IVC	<2cm	>2cm	
Hepatic vein	S wave > D wave	Mild - S < D Severe - S above baseline	Invalid if primary TR or constrictive pericarditis (valid if function TR)
Portal vein pulsatility index (PI)	<0.3	Mild 0.3-0.5 Severe >0.5	Can be >0.5 in young age, athletes, low BMI, hyperdyanamic states
Renal vein venous impedence index (VII)	<0.7	Mild - =1 S and D phase Severe - =1 D phase only	
Renal artery resistive index (RRI)	<0.7	>0.7	Affected by pulse pressure, interstitial and venous pressure, vascular compliance

Table 10. Normal and significantly abnormal values

References

1. Wharton G, Steeds R, Allen J, et al. A minimum dataset for a standard adult transthoracic echocardiogram: a guideline protocol from the British Society of Echocardiography . 2015; 2(1):G9-G24.
2. Mitchell C, Rahko PS, Blauwet LA, et al. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography Journal of the American Society of Echocardiography. 2019; 32(1):1-64.
3. Malbrain ML, Marik PE, Witters I, et al. Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice Anaesthesiol Intensive Ther. 2014; 46(5):361-380.
4. Khan AI, Fischer M, Pedoto AC, et al. The impact of fluid optimisation before induction of anaesthesia on hypotension after induction Anaesthesia. 2020; 75(5):634-641.
5. Miller A, Mandeville J. Predicting and measuring fluid responsiveness with echocardiography . 2016; 3(2):G1-G12.
6. Hamzaoui O, Georger J, Monnet X, et al. Early administration of norepinephrine increases cardiac preload and cardiac output in septic patients with life-threatening hypotension Crit

Care. 2010; 14(4):R142-.
7. Nowosielski M, Schocke M, Mayr A, et al. Comparison of wall thickening and ejection fraction by cardiovascular magnetic resonance and echocardiography in acute myocardial infarction J Cardiovasc Magn Reson. 2009; 11(1):22-.
8. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized Myocardial Segmentation and Nomenclature for Tomographic Imaging of the Heart Circulation. 2002; 105(4):539-542.
9. Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging Journal of the American Society of Echocardiography. 2016; 29(4):277-314.
10. Mercado P, Maizel J, Beyls C, et al. Reassessment of the Accuracy of Cardiac Doppler Pulmonary Artery Pressure Measurements in Ventilated ICU Patients Critical Care Medicine. 2019; 47(1):41-48.
11. Augustine DX, Coates-Bradshaw LD, Willis J, et al. Echocardiographic assessment of pulmonary hypertension: a guideline protocol from the British Society of Echocardiography . 2018; 5(3):G11-G24.
12. Beaubien-Souligny W, Rola P, Haycock K, et al. Quantifying systemic congestion with Point-Of-Care ultrasound: development of the venous excess ultrasound grading system Ultrasound J. 2020; 12(1).
13. Benkreira A, Beaubien-Souligny W, Mailhot T, et al. Portal Hypertension Is Associated With Congestive Encephalopathy and Delirium After Cardiac Surgery Canadian Journal of Cardiology. 2019; 35(9):1134-1141.
14. Legrand M, Dupuis C, Simon C, et al. Association between systemic hemodynamics and septic acute kidney injury in critically ill patients: a retrospective observational study Crit Care. 2013; 17(6):R278-.
15. Mullens W, Abrahams Z, Francis GS, et al. Importance of Venous Congestion for Worsening of Renal Function in Advanced Decompensated Heart Failure Journal of the American College of Cardiology. 2009; 53(7):589-596.
16. Spiegel R, Teeter W, Sullivan S, et al. The use of venous Doppler to predict adverse kidney events in a general ICU cohort Crit Care. 2020; 24(1).
17. Harkness A, Ring L, Augustine DX, et al. Normal reference intervals for cardiac dimensions and function for use in echocardiographic practice: a guideline from the British Society of Echocardiography . 2020; 7(1):G1-G18.
18. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging Journal of the American Society of Echocardiography. 2015; 28(1):1-39.e14.