Right Ventricular Structure, Function and Assessment in Anaesthesia and Intensive Care

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The right ventricle - structural and functional importance for anaesthesia and

intensive care

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Key Points:

1. **The right ventricle is anatomically and physiologically different from the left ventricle and requires a different approach to assessment and management.**

2. **The right ventricle can be considered to be relatively tolerant of preload but intolerant of increased afterload.**

3. **In the normal RV, perfusion of the RV occurs during both systole and diastole providing myocardial oxygen delivery throughout the cardiac cycle.**

4. **Four key transthoracic echocardiography parameters when assessing the right ventricle are RV size, RV systolic function, RV systolic pressure and septal position.**

5. **RV dysfunction may occur with minimal clinical findings; therefore, a high index of suspicion is required when diagnosing RV dysfunction.**

Learning Objectives

By reading this article you should be able to:

- **Explain the key differences between the left ventricle and right ventricle with particular focus on anatomy and physiology.**

- **Be able to define the role of preload, afterload and ventricular interdependence with regards to right ventricle.**

- **Be aware of the role of CVP monitoring, pulmonary artery catheters and trans-oesophageal echocardiography in diagnosing and managing RV dysfunction.**

- **Describe the key principles of right ventricle assessment using trans-thoracic echocardiography.**

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Introduction

In 1616, Sir William Harvey was the first physician to realise the importance of the

right ventricle (RV) and its interactions with the pulmonary circulation, yet up until the

mid-20th century little emphasis had been placed on the RV. Prior to the 1950s, the

main focus was that of the left ventricle (LV) with the RV being thought of as little

more than passive structure with the sole purpose of providing a conduit between the

systemic and pulmonary circulations. By the 1950s, cardiac surgeons began to

understand the importance of the RV as they attempted to develop techniques for

palliation of right-heart hypoplasia. The crucial role of the RV is now well recognised in

a wide variety of cardiac and non-cardiac conditions.

The RV differs significantly from the LV in terms of its' anatomy, and physiology. As a

result, a different approach is required in terms of assessment and management. In

order to manage patients with RV dysfunction it is important to have a good

understanding of RV anatomy and physiology and their clinical application. The aim of

this article is to provide an understanding of the structure and function of the RV for

the anaesthetist and intensivist.

Anatomy of the RV

The body of the RV receives blood from the right atrium (RA), whilst the outflow tract, transfers the blood to the pulmonary artery (PA). These two areas are separated by a ridge, the crista supraventricularis, which extends in to the ventricular cavity. The RV is the most anterior chamber in the normal heart residing immediately behind the sternum. The RV is crescent-shaped in cross-section and triangular in side-profile. The ventricle is composed of two layers of muscular fibres - superficial circular fibres which are continuous with the subepicardial fibres of the LV and deeper longitudinal fibres. In contrast, the LV has a more complex structure and movement pattern, with three layers of fibres compared to the two of the RV. The thin, free wall of the RV wraps around the more muscular wall of the LV.

Perfusion of the Right Ventricle

The RV blood supply is dependent on the dominance of the coronary system for each individual. Eighty-percent of the population are known to have a RV supplied by the right coronary artery. Unlike the LV, perfusion of the RV occurs during both systole and diastole providing myocardial oxygen delivery throughout the cardiac cycle. This phenomenon only occurs in healthy hearts however, in the diseased state such as in patients with pulmonary hypertension (PH), increased intra-cavity pressure during systole means the distribution of blood to the RV during the cardiac cycle is more like that of the LV, occurring only during diastole.

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Although, the RV ejects the same cardiac output (CO) as the LV, RV stroke work is only

around a quarter that of the LV. The RV is thin-walled, with approximately one-sixth of

the muscle mass of the LV. Under normal conditions, ejection is maintained despite

lesser muscle mass as the RV is coupled with the pulmonary circulation which has a

lower vascular resistance and greater distensibility in comparison with the systemic

circulation. As such both pulmonary artery pressure and vascular resistance are

approximately a fifth of that the systemic circulation. (Table 1). The ability of the lung

to recruit partially collapsed or unused vessels as CO increases, for example during

exercise, serves to maintain coupling and accounts for the minimal changes

demonstrated in pulmonary arterial pressure and reduction in pulmonary vascular

resistance (PVR) seen on exercise.

Ejection of blood from the RV occurs following a reduction in free wall surface area

and shortening of longitudinal fibres, beginning at the inflow tract and moving in a

'peristaltic' manner towards the RV outflow tract. Compare this to the LV where blood

is ejected due to a concentric contraction of the LV free wall and septum, combined

with a twisting movement of the heart. [1]

Right Ventricular Cardio-dynamics

The performance of the RV is affected by variety of interacting factors including

preload, afterload, and ventricular interdependence. [2] The LV pressure-volume loop

has a familiar rectangular shape, with parallel sides due to well defined isovolumetric

contraction and relaxation phases, and sharp 'corners' where the beginning and end

of both systole and diastole can be easily identified. In comparison RV isovolumetric

contraction time is shorter than that of the LV as during early systole, the pulmonary

valve opens when RV pressure exceeds that of the low-pressure PA. The point of end-

systole is less well defined in the RV leading the RV pressure volume-loop to appear

almost triangular in shape. This occurs as ejection of blood from the RV can continue

despite falling RV pressure due to the momentum of blood in the low-pressure

system[1] ; as a result the isovolumetric ventricular relaxation phase is shortened or

absent in the RV. (Figure 1)

- *Preload*

Preload is defined as the initial stretching of the cardiac fibre prior to contraction and

is influenced by a variety of factors including atrial contractility, ventricular

compliance, venous return, wall tension and heart rate. Conceptually RV end-diastolic

volume (EDV) and end-diastolic pressure (EDP) are considered to be indices of

preload. The RV is described as being 'tolerant' of pre-load; low muscular mass means

the comparatively compliant RV is able to dilate (up to a point) in the face of excessive

volume. With ongoing distention however, decompensation occurs and failure ensues.

Excessive RV dilatation is commonly complicated by dilation of the tricuspid valve

annulus and the development of tricuspid regurgitation (TR). Significant TR leads to

further volume overload and reduces forward flow, reducing CO. Volume overload of

the RV can distort the LV shape and impair LV filling and function (see below –

'ventricular interdependence'.) [2]

- *Afterload*

RV afterload is the load the RV has to overcome during the ejection phase of the

cardiac cycle and in its most complete sense is influenced by pulmonary vascular

resistance, distensibility and pulse wave reflections taking place within the pulmonary

vascular bed. The RV is less well equipped to deal with acute changes in afterload due

to a reduction in cardiac muscle mass compared with the LV; as such the RV is

considered to be intolerant of afterload (Figure 2). In clinical practice, PVR is used as

an index of afterload (Figure 3). In chronically increased afterload, the RV pressure-

volume loop gradually shifts to become rectangular in shape and appear similar to the

normal LV pressure-volume loop.

- *Ventricular Interpendance*

Ventricular interdependence describes the concept whereby the function, volume or

pressure in one ventricle can directly influence that of the other.[3] Both the LV and the

RV are interdependent as they are contained within the relatively in-distensible

pericardial sac with a shared ventricular septum. Whilst these interactions are present

continuously, it is in times of dysfunction that interdependence becomes most

evident.[4] When RV pressure or volume overload occurs, the loaded RV can affect LV

performance and result in a decreased LV preload and contractility. In normal hearts,

LV end-diastolic pressure (EDP) usually exceeds RV-EDP. In times of RV overload, RV-EDP may exceed LV-EDP forcing the ventricular septum towards the LV during diastole. This distorts the normal LV shape (creating a so called "D-shaped" ventricular cavity (Figure 4)), reducing LV diastolic compliance and impairing LV filling.[5] In addition, these alterations in LV geometry have a direct effect on LV systolic function, by reducing the mechanical efficiency of LV contraction.[6] The clinician must be wary of the apparently empty, well contracting LV, which may appear reassuring when seen on echocardiography; in conditions of RV failure, the LV can be under-filled due to compression by, and reduced delivery from the inter-dependant RV.

The haemodynamics of the right heart are affected by respiratory effort.[7] During spontaneous breathing, inspiration leads to a negative intrathoracic pressure 'sucking' blood into the thoracic cavity and improving RV filling whilst simultaneous distension of the pulmonary vasculature leads to a reduction in RV afterload, promoting right-sided CO. Contrast this to mechanical ventilation, where there is a cyclical increase in intrathoracic pressure during inspiration. As intrathoracic pressure increases, filling is impeded and extra-alveolar capillaries are compressed, increasing afterload. These phenomena can lead to the exhibition of marked systolic pressure / stroke volume variation in mechanically ventilated patients with RV dysfunction; caution must be exercised not to interpret these changes as being reflective of hypovolaemia.

Assessment of the RV

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Clinical assessment should focus on evaluating signs and symptoms of RV dysfunction and establishing the precipitating event or underlying aetiology. Not all of the investigations discussed may be readily available, particularly in the emergency setting.

- *Jugular Venous Pressure (JVP) and Central Venous Pressure (CVP)*

Assessing the JVP can be of enormous value in a patient with RV failure. A raised JVP reflects raised atrial pressure and is therefore a specific sign of right-sided failure. Kussmaul's sign, an increase in JVP on inspiration, can be indicative of the cause of RV failure. Increased venous return combined with impaired RV diastolic compliance, occurring in RV infarction and constrictive pericarditis, results in disproportionately increased RAP on inspiration.

The limited ability of CVP to identify patients who will respond positively to a 'fluid challenge' means CVP monitoring has been falling out of favour in the general intensive care patient. The utility of CVP monitoring as a surrogate monitor of RV function however is often under-appreciated. Increasing CVP in a patient with RV dysfunction is an ominous sign. The trend of the CVP in response to fluids and inotropes can provide useful information as well as waveform interpretation. In the presence of severe TR, a dominant v-wave occurs during ventricular systole as a result of retrograde RA filling. This is followed by an associated sharp y descent occurring as a result of high RA volume. Pulmonary hypertension can lead to a dominant a-wave as the RA is contracting against increased resistance.

- *Laboratory*

Investigations should include full blood count, renal and liver function tests, troponin and lactate which may demonstrate evidence of organ hypoperfusion as a result of RV dysfunction. A raised BNP and troponin may be due to a variety of RV failure aetiologies. Studies have demonstrated the role of a raised BNP in diagnosing RV dysfunction and predicting morbidity in patients with pulmonary hypertension[8]. Troponin and BNP have been shown to be a predictor of RV dysfunction in acute pulmonary embolus[9].

- *Electrocardiography*

The ECG can often be normal in patients with RV dysfunction. There may however be evidence of right axis deviation, right bundle branch block or RV hypertrophy. The classic triad of a deep S-wave in lead one, Q-wave and an inverted T-wave in lead three may be present, demonstrating evidence of RV strain and acute cor pulmonale. This pattern is traditionally described in the context of ECG findings in PE but occurs in less than ten-percent of patients.

- *Chest X-ray (CXR)*

The value of CXR when assessing the RV is limited due its' position as an anterior cardiac structure and therefore only contributes to a small portion of the heart

border. The RV is normally best viewed on a lateral CXR where an enlarged RV will fill the retrosternal space and occupy more than 50% of the area between the diaphragm and sternal angle. The LV may also displace backwards which results in cardiomegaly. There may be evidence of the underlying aetiology.

Trans-thoracic echocardiography (TTE)

The mainstay of RV imaging in clinical practice is trans-thoracic echocardiography (TTE). TTE is widely available and can be used to assess both structure and function. Echocardiographic imaging of the RV is challenging however with both the complex shape of the RV and its retrosternal position contributing to these challenges.

Whilst the LV is broadly circular in cross-section therefore volumes can be estimated by summing the volumes of multiple slices of a known thickness ('Simpson's rule'); in contrast, the RV cannot be easily modelled geometrically leading to difficulty in assessing volumes and function on TTE. As a result, numerous surrogate echocardiographic indices of RV function have been described; all have significant limitations and vary in their performance when compared to (gold standard) cardiovascular magnetic resonance techniques.

TTE is nonetheless the most widely used clinical tool for bedside assessment of RV function. Whilst LV function is commonly described quantitatively in terms of ejection fraction, it can be appreciated that due to the difficulties in measuring RV volumes

with TTE, RV TTE reporting tends to be more qualitative in nature. The guidelines for trans-thoracic echocardiography examination of the RV recommend both a qualitative and quantitative assessment.[10] Apical 4-chamber, para-sternal long and short axis and subcostal views provide images for a comprehensive assessment of RV function and RV systolic pressure (RVSP). Discussion of the comprehensive echocardiographic assessment of RV function is out with the scope of this article, however there are a number of parameters, relevant to RV function that are commonly reported in any formal echocardiography report. Table 2 discusses these parameters with the aim of better informing the non-echocardiographer on the interpretation of such reports. Some of the more complex parameters are discussed in more detail below.

- *RV Size*

Qualitative assessment of the RV allows for a comparison of the relative size of the RV and the LV when viewed in an apical-4 chamber view. Caution should be exerted when using this method as it is dependent on the LV being normal in shape and size. Quantitative assessment measures the diameter at the base, mid-level and length.

- *RV Systolic Function*

RV systolic function has been measured using a variety of parameters; tricuspid annular plane systolic excursion (TAPSE) and fractional area change (FAC) are two of the most traditionally used. TAPSE is an easily obtainable measure derived from the

apical four-chamber view using M-mode. The M-mode curser is placed through the tricuspid annulus and measures the amount of longitudinal motion of the annulus at peak systole. Impaired RV systolic function has a TAPSE value of <17mm. [11]

FAC is obtained from a four-chamber view by tracing the RV endocardium in systole and diastole, beginning at the annulus, along the free wall to the apex, and back along the interventricular septum to the annulus. It is expressed as a percentage change in the RV area between end-diastole and end-systole. Normal value is > 35%.[11] It has been shown to correlate well with RV ejection fraction measured by cardiac magnetic imaging.

- *Assessment of pulmonary artery pressure*

Systolic pulmonary artery pressure (SPAP) is considered the same as RV systolic pressure (RVSP) in the absence of stenotic pulmonary valves disease (very rare out-with the congenital cardiac population). RVSP is derived by the addition of the pressure gradient between the RV and the RA, to the pressure in the RA. In order to measure the RV-RA pressure gradient the maximum velocity of the TR jet must be calculated. The maximum velocity measured as the peak regurgitation is converted to pressure using Bernoulli's law (Pressure (p) = 4 x volume $(v)^2$). The maximum velocity of the TR jet should be measured in either an apical four-chamber or a short-axis view.

RA pressure is estimated from the IVC diameter and the presence of inspiratory collapse in a subcostal view. The measurement should be made at end-expiration and just proximal to the hepatic veins that lie proximal to the ostium of the RA. [10] An IVC < 17mm with a collapsibility index of 50% has an RA pressure of 5mm. An IVC >17mm with collapsibility > 50% has an RA pressure of 10 whereas the same IVC diameter but with an index < 50% has a RA pressure of 15.

In positive-pressure ventilated patients, the degree of collapsibility of the IVC cannot be used as a reliable marker of RA pressure. In these patients' however RA pressure is commonly measured by central venous cannulation.

- *Septal Morphology*

Chronic RV dilatation, for example due to isolated volume overload as occurs in tricuspid regurgitation (TR), results in the RV apex progressively replacing the LV as the true apex of the heart. If viewed in a para-sternal short axis window, the LV will appear D-shaped due to flattening of the ventricular septum.[10] The geometry of the LV will also alter during RV pressure overload due to shifting of the septum to the left away from the centre of the RV and towards the centre of the LV. The LV cavity will appear D-shaped in the short-axis view predominantly during systole. [10]

- *Trans-oesophageal echocardiogram (TOE)*

Intra-operative TOE is now being widely used for high-risk surgical procedures and is commonplace in cardiac anaesthesia. It is also useful in haemodynamically unstable patients both in the theatre and in intensive care to guide management strategies. TOE has the advantage over TTE of better quality imaging, particularly in patients who are ventilated with positive-pressure ventilation, chest wall injuries or during cardiothoracic surgery.[12] Consideration should be given to high-risk patients for an on-table, real-time TOE RV assessment.

- *Pulmonary artery catheter (PAC)*

PAC are the diagnostic gold standard for the diagnosis of PH. They have fallen out of favour in the general ICU population; however; they may aid the diagnose of RV failure and allow appropriate management of these patients. The PAC allows right-sided measurements to be performed and thermo-dilution allows RV (cardiac) output to be measured. The PAC allows for the accurate measurement of CO which in turn allows the clinician to calculate PVR, as a surrogate marker for afterload. Acute RV dysfunction is suggested by an CVP greater than pulmonary capillary wedge pressure (PCWP), a low cardiac index (CI) and stroke volume index (SVI) and mixed venous oxygen saturations less than 55%. [13]

Conclusion

A sound understanding of RV anatomy and physiology is essential for the anaesthetist

and intensivist. By considering the basic principles of preload, afterload, contractility,

perfusion and ventricular interdependence allows for a structured approach to clinical

aspects of RV failure and its management.

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MCQs

1. With regards to anatomy of the right ventricle (RV)

a) The sinus and the outflow tract of the RV are separated by a ridge known as the crista ventricularis

b) Is crescent shaped in side section

c) Is perfused during systole and diastole

d) The RV wall thickness is one quarter that of the LV

e) Ejection of blood from the RV occurs following lengthening of the longitudinal fibres

2. Transthoracic echocardiography of the right ventricle (RV)

a) Uses surrogate indices of RV function

b) Is gold standard for assessment of the RV function

c) Commonly uses TAPSE as a measurement of RV systolic pressure

d) Calculates RV systolic pressure using the Venturi principle

e) Showing flattening of the ventricular septum in diastole suggests volume overload

3. In relation to physiology of the right ventricle (RV):

a) Systemic vascular resistance can be used as a surrogate marker of afterload.

b) An increase in RV loading leads to an increase in both RV and LV end-diastolic volumes.

c) Normal movement of the inter-ventricular septum is inward towards the LV during systole.

d) The RV pressure-volume loop appears triangular as the point of end-diastole is not as well defined.

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4. Regarding assessment of the right ventricle (RV)

a) Cardiac magnetic resonance imaging should be used first line in all patients to assess the right ventricle

b) $S_1Q_3T_3$ is seen in over 50% of patients with RV dysfunction

c) In the presence of tricuspid regurgitation, a dominant v wave may be seen when examining the CVP trace.

d) Pulmonary artery catheters will allow cardiac output calculations to be made and therefore allow calculation of pulmonary vascular resistance and afterload.

e) In positive-pressure ventilated patients, the degree of collapsibility of the IVC is a reliable marker of RA pressure

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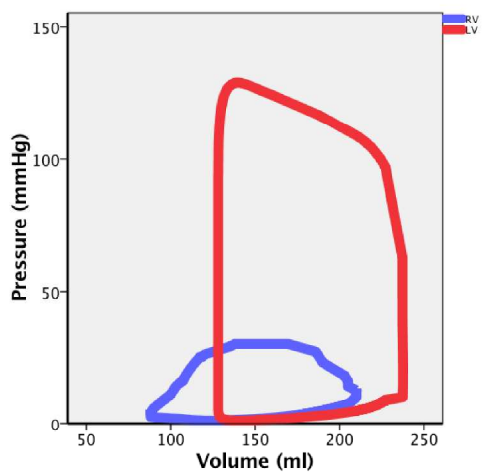43
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Figure 1. Pressure-volume curves of the left and right ventricle. Note the lower pressures and more triangular appearance of the PV loop for the right ventricle (blue) compared with the more rectangular left ventricle (red).

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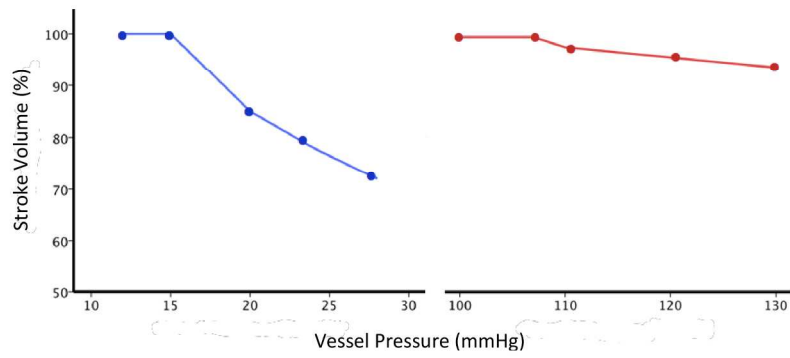42
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Figure 2. Comparison of RV (blue) and LV (red) adaptation to afterload. Note how the RV stoke volume falls rapidly in response to increased afterload compared to the LV.

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$$PVR = \frac{(MPAP - PCWP) \times 80}{CO}$$
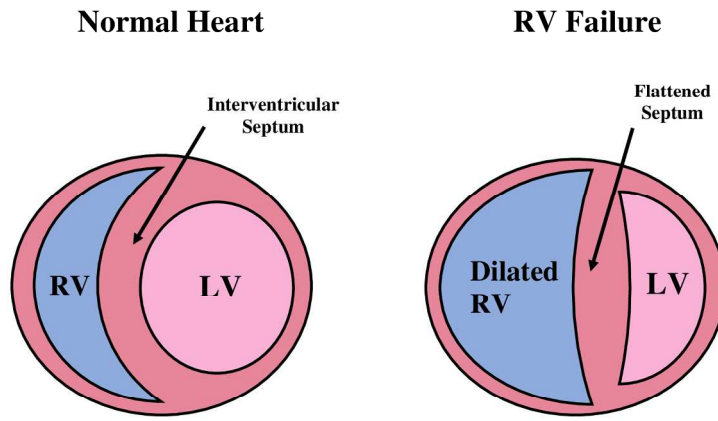
Normal Heart   **RV Failure**

Figure 4. Graphical representation of ventricular interdependence.

297x209mm (200 x 200 DPI)

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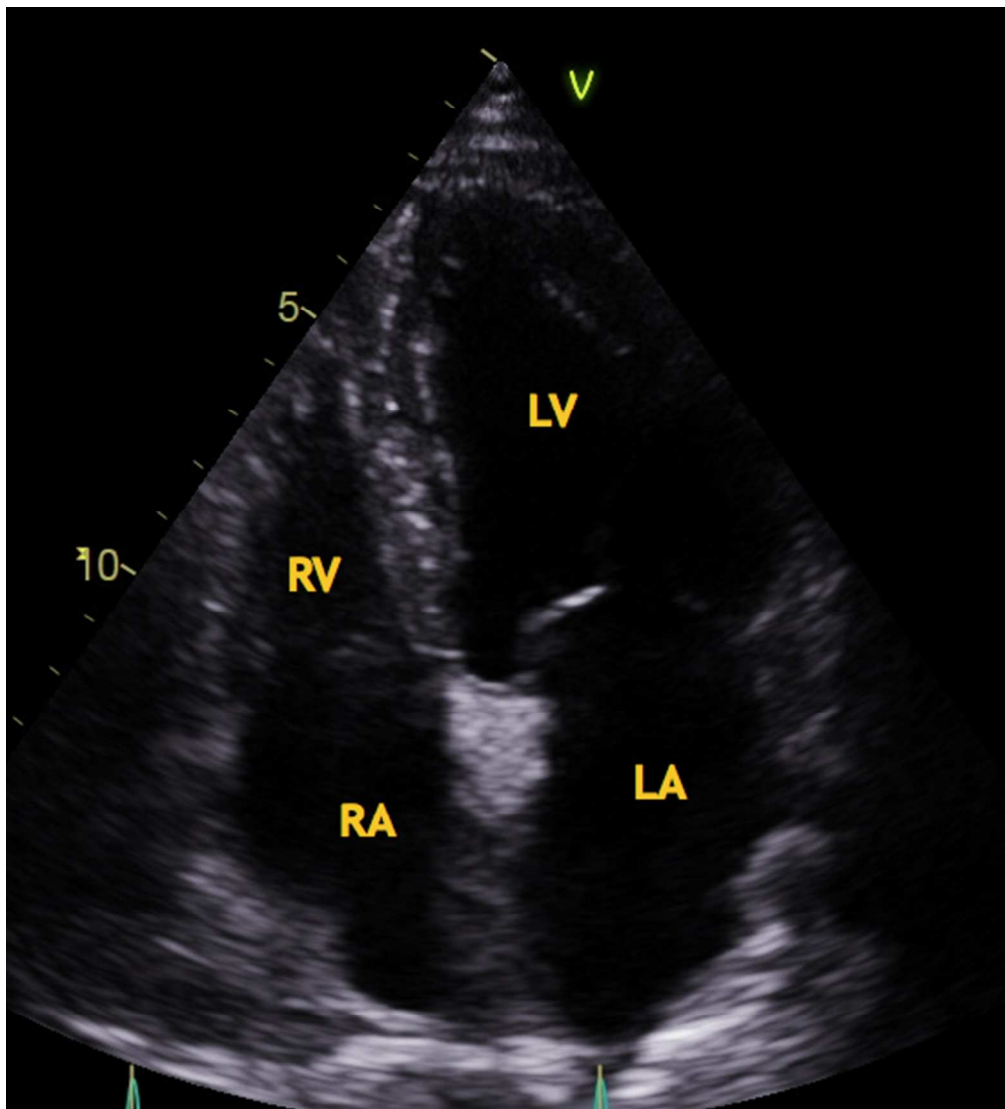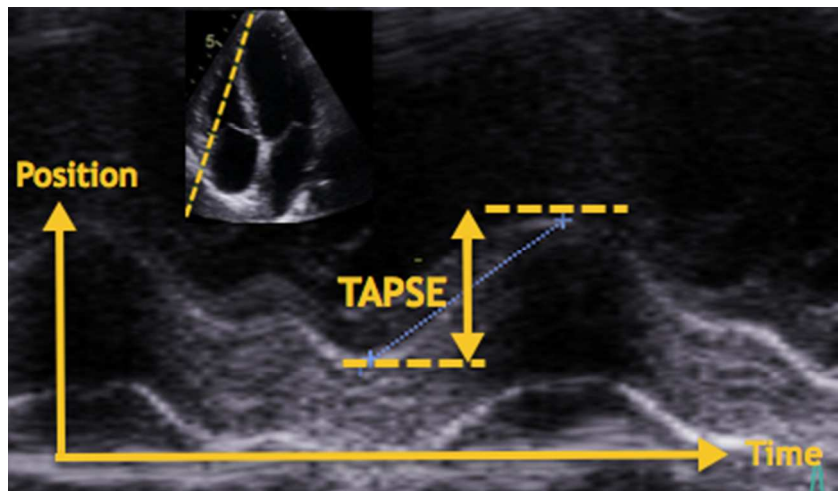31
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Figure 5: Transthoracic echocardiography images. Images to be used in conjunction with Table2. 5.a: Normal Apical 4 chamber TTE view.
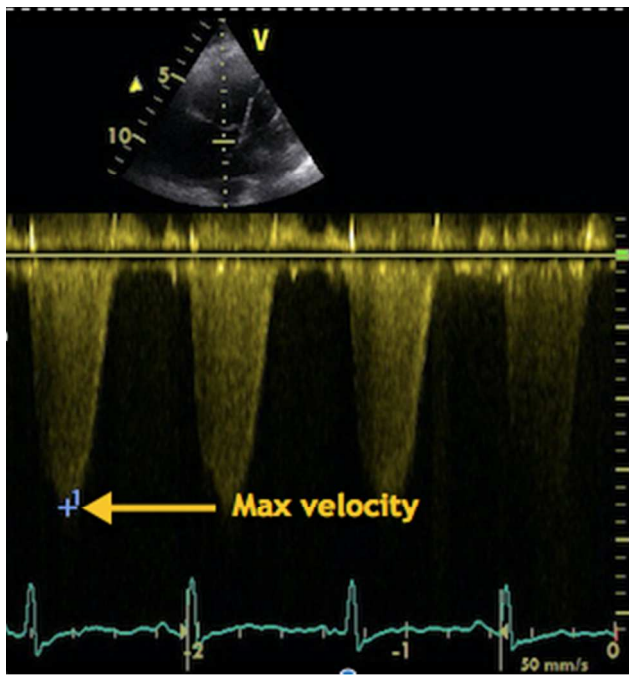
194x214mm (72 x 72 DPI)

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5b: Calculation of right ventricle systolic function using TAPSE.

147x85mm (72 x 72 DPI)
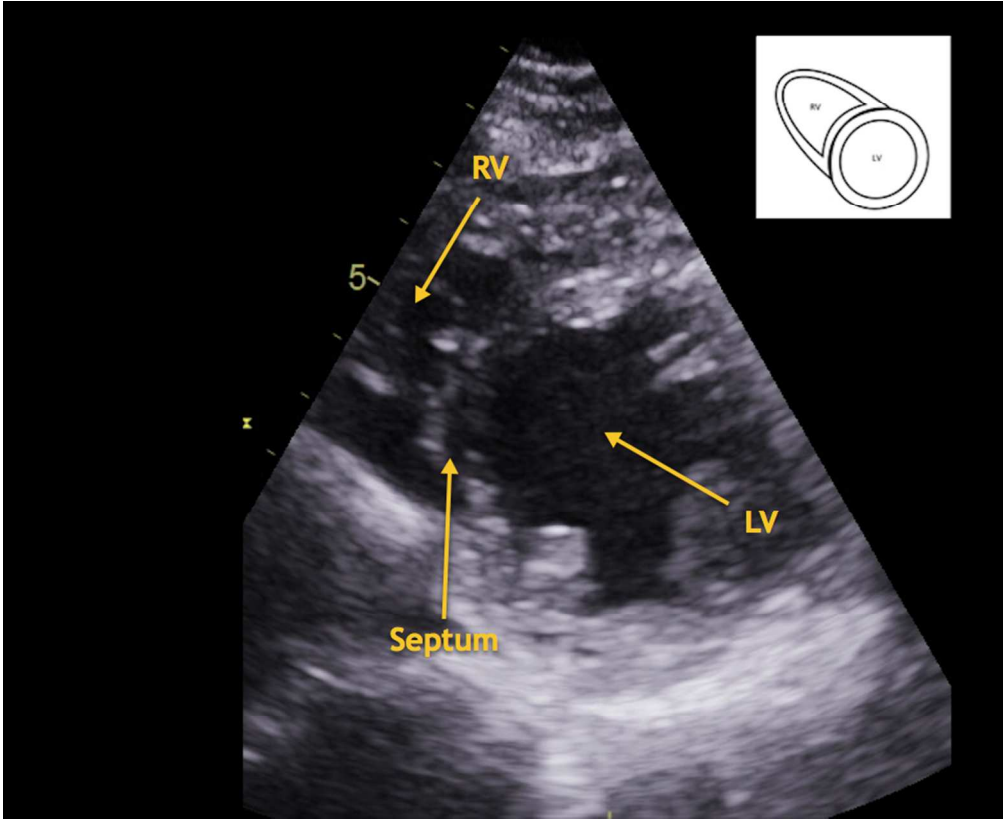
5c: Calculation of right ventricle systolic pressure using the maximum velocity of the TR jet.

110x117mm (72 x 72 DPI)

5d: Normal Short axis TTE view.

338x275mm (72 x 72 DPI)

Table 1: Comparison left ventricle versus the right ventricle

	Right Ventricle	Left Ventricle
Shape	Crescent	Ellipsoidal
Structure	2 layers of fibres	3 layers of fibres
Free Wall Thickness	1-5mm	8-10mm
Circulation	Low-pressure, low-resistance	High-pressure, high-resistance
Stroke Volume (SV)	70-90ml	70-90ml
Ejection Fraction (EF)	65%	70-80%
Ventricular Pressure (diastole)	0-8mmHg	4-12mmHg
Ventricular Pressure (systole)	15-30mmHg	90-140mmHg
Afterload	PVR <250 dynes-sec/cm^{-5}	SVR 800-1200 dynes-sec/cm^{-5}
Adaptation to disease	Tolerant of preload	Tolerant of afterload

TTE Measurement	How to Measure	Values
RV Size (Image 5a)	Two ways to determine: • Qualitative comparison of the relative sizes of the RV and LV. (The normal RV should be 2/3 the size of the LV, only if the LV size is NORMAL) • Diameter at the base, mid-level and length. If values higher than those given, RV dilatation present	Qualitative: • Mild dilation – RV > 2/3 LV • Moderate dilation – RV = LV • Severe dilation – RV larger than LV Diameter: • Base > 42mm • Mid-level > 35mm • Length > 83mm
RV Systolic Function (Image 5b)	Variety of parameters may be used. TAPSE is the commonest: • 'M-mode' cursor through the tricuspid annulus provides a graphical representation of annular position against time	Displacement of the annulus towards the apex during systole is indicative of systolic function • <17mm = RV systolic dysfunction.
RV Systolic Pressure (Image 5c)	$RVSP = 4\,v^2 + CVP$ where v = velocity Max velocity of the TR jet (highlighted in Image 5c) is converted to pressure with Bernoulli's equation ($P=4v^2$)	Normal RVSP = <35mmHg
Septal Position (Image 5d)	Visual assessment of ventricular septal position in parasternal short axis view. In image 5d a parasternal short axis is shown. The LV, RV and interventricular septum are highlighted.	As the RV dilates, the ventricular septum will flatten and the LV will lose its characteristic circular shape and become D-shaped in cross section (see figure 3). • Flattening in diastole only suggests volume overload • Flattening in systole and diastole suggests volume and pressure overload

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Table 2. Quick reference guide for important TTE parameters of the RV. Detailed explanation of each parameter given in the main body of the article. Please see Figure 5 and online for TTE images.

Question 1

In relation to physiology of the right ventricle (RV):

   a) Systemic vascular resistance can be used as a surrogate marker of afterload.

   b) An increase in RV loading leads to an increase in both RV and LV end-diastolic volumes.

   c) Normal movement of the interventricular septum is inward towards the LV during systole.

   d) The RV pressure-volume loop appears triangular as the point of end-diastole is not as well defined.

Answers

   a) False. Pulmonary vascular resistance is a surrogate of afterload. The equation for the calculation of PVR requires the mean pulmonary artery pressure, pulmonary capillary wedge pressure and cardiac output.

   b) False. An increase in RV loading will result in an increase in RV end-diastolic volume and a decrease in LV end-diastolic volume.

   c) True. The septum does move towards the LV during systole.

   d) False. The RV pressure-volume loop is triangular as the point of end-systole is not well defined

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Question 2:

With regards to anatomy of the right ventricle (RV)

   a) The sinus and the outflow tract of the RV are separated by a ridge known as the

       crista ventricularis

   b) Is crescent shaped in side section

   c) Is perfused during systole and diastole

   d) The RV wall thickness is one quarter that of the LV

   e) Ejection of blood from the RV occurs following lengthening of the longitudinal fibres

Answers

   a) False. The ridge is known as crista supraventricularis

   b) False. The RV is crescent-shaped in cross-section and triangular in side section.

   c) True.

   d) False. RV stroke work is only around one quarter that of the LV. The RV is thin

       walled, with approximately one six of the muscle mass of the LV.

   e) False. Ejection occurs due to shortening of the fibres.

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Question 3:

Transthoracic echocardiography of the right ventricle (RV)

a) Uses surrogate indices of RV function

b) Is gold standard for assessment of the RV function

c) Commonly uses TAPSE as a measurement of RV systolic pressure

d) Calculates RV systolic pressure using the Venturi principle

e) Showing flattening of the ventricular septum in diastole suggests volume overload

Answers

a) True. A number of surrogate echocardiographic indices of RV function are used

b) False. Cardiac magnetic resonance imaging is now gold standard for assessment of

the RV due to the complex shape and retrosternal position of the RV

c) False. TAPSE is one way to measure RV systolic function. There a variety of other

measurements available to measure systolic function.

d) False. RV systolic pressure is calculated using the Bernoulli principle. The maximal

velocity of the TR jet is converted to pressure using the Bernoulli principle.

e) True. Flattening in diastole only suggests volume overload. Flattening in systole and

diastole suggests volume and pressure overload.

Question 4:

Regarding assessment of the right ventricle (RV)

a) Cardiac magnetic resonance imaging should be used first line in all patients to assess the right ventricle

b) $S_1Q_3T_3$ is seen in over 50% of patients with RV dysfunction

c) In the presence of tricuspid regurgitation, a dominant v wave may be seen when examining the CVP trace.

d) Pulmonary artery catheters will allow cardiac output calculations to be made and therefore allow calculation of pulmonary vascular resistance and afterload.

e) In positive-pressure ventilated patients, the degree of collapsibility of the IVC is a reliable marker of RA pressure

Answers

a) False. CMR is now the gold-standard for assessment of the RV; however, it is not the first-line assessment. Simple measures including echocardiography are considered first line assessment.

b) False. $S_1Q_3T_3$ occurs as a result of RV strain or cor pulmonale and is seen in less than 10% of patients with PE.

c) True. A dominant v-wave is a result of abnormal RA filling.

d) True. PA catheter thermo-dilution techniques allow for the calculation of cardiac output.

e) False. In this group of patients, RA pressure should be calculated using the CVP.