CHAPTER 1

Echocardiographic Assessment of Cardiac Output and Ejection Fraction

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Echocardiography has been shown to be an effective, noninvasive method of assessing cardiac function. Furthermore, it has been shown that non-cardiologists can safely and accurately use echocardiography to assess cardiac function.\(^1\)

Cardiac output (CO) is the volume of blood pumped out of the heart per minute. Measured in liters/minute, CO can be calculated using heart rate (HR) and stroke volume (SV).

\[
CO = SV \times HR
\]

Echocardiography can be used to measure SV using a method called pulsed waveform Doppler. This method allows approximation of the stroke volume by taking two measurements:

1) The left ventricular outflow tract (LVOT) area.

2) The range of velocities of blood flow across the LVOT, also known as velocity time integral (VTI).\(^2,3\)

\[
SV = LVOT \text{ area} \times VTI
\]

**Highlights**

- Echo is an effective, noninvasive method for measuring CO.
- Non-cardiologists can measure CO.
- \(CO = SV \times HR\)
- \(SV = LVOT \text{ area} \times VTI\)
**SECTION 2**

Measuring LVOT Area

The LVOT area is approximated by assuming that it is the shape of a circle. Thus, the area is determined by measuring the diameter of the LVOT. Once obtained, a person’s LVOT area does not change. You only need to obtain the LVOT once, and future CO measurements can use old LVOT area measurements.

To determine the LVOT area, first obtain a parasternal long axis view of the heart (Figure 1.1, Image 1.1).

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**HIGHLIGHTS**

LVOT is measured in the parasternal long axis view.

Zoom in on the LVOT to get good visualization of the aortic root.

Measure LVOT in mid-systole, with maximal separation of leaflets.
Zoom in on the LVOT, ensuring good visualization of the aortic root. The diameter should be obtained by measuring from inner edge to inner edge at the level of the aortic annulus (attachment of valve leaflets). Importantly, the diameter of the LVOT should be measured perpendicular to the direction of the LVOT and should be performed in mid-systole, with maximal separation of leaflets (Image 1.2).

**Image 1.2 - LVOT Diameter**

![Image of LVOT Diameter](image)

Measured in mid-systole at base of Ao leaflets.

LVOT area can then be calculated using the following formula:

\[
LVOT\ area\ (cm^2) = \frac{\Pi \times (diameter)^2}{4}
\]
SECTION 3

Measuring VTI

To calculate VTI, pulsed wave Doppler is used to measure the velocity of flow across the LVOT. First, obtain an apical long axis or apical 5 chamber view of the heart (Figure 1.2, Image 1.3, Figure 1.3, Image 1.4).

**Figure 1.2 - Apical 5 Chamber View**

**Image 1.3 - Apical 5 Chamber VTI Measurement**

**Highlights**

- Measure VTI in the apical long axis or 5 chamber view.
- $SV \ (cm^3) = \text{Area LVOT} \ (cm^2) \times VTI \ (cm)$
- The Doppler wave should be in line with the flow of blood through the LVOT.
- In patients with an irregular heart rate, the VTI measurement should be obtained 5-10 times and then averaged.
It is important that the pulsed wave Doppler gate is in line with the LVOT, thus the necessity that an apical window is obtained. Select the pulsed wave Doppler mode and place the Doppler gate parallel to and in the center of the LVOT at the level where the diameter was measured (Images 1.3, 1.4). This will produce a waveform tracing of the velocities of blood flowing out of the LVOT with each heartbeat. The waveform will be negative since the blood is flowing away from the transducer. Next, select “LVOT VTI” through the machine’s measurement package (often found under “CO” or “Ao” in the cardiac measurements section) and trace the waveform of one ejection period (Image 1.5).
Doing this will calculate a value for VTI by taking area under the curve. This measurement is essentially taking the integral of a velocity, so the VTI will be reported in cm. Stroke volume can then be calculated using the following formula:

\[ SV \ (cm^3) = \text{Area LVOT (cm}^2\text{)} \times VTI\ (cm) \]

\[ SV = \left( \frac{\pi(diameter)^2}{4} \right) \times VTI \]

**PITFALLS**

1. When measuring VTI, the Doppler wave should be in line with the flow of blood through the LVOT. Any angle between the Doppler wave and LVOT can result in underestimation of VTI, and therefore SV.

2. When measuring diameter of LVOT, ensure that the measurement is taken in mid-systole. Failure to do this can result in underestimation of LVOT area, and therefore SV.

3. In patients with an irregular heart rate, the VTI measurement should be obtained 5-10 times and then averaged.
Patient care often requires an assessment of a patient’s volume status to direct a course of treatment. Sepsis, hypotension, pancreatitis and many other pathologic states require rapid volume repletion. However, overzealous administration of fluid can be detrimental, particularly in patients with known congestive heart failure. In such cases, echocardiography can be used as a non-invasive method of determining the fluid status of a patient using the passive leg raise (PLR) maneuver. The PLR maneuver enables the physician to deliver a reversible, endogenous fluid bolus of approximately 250cc immediately. To perform PLR, initial measurement of SV should be obtained with the patient in a semi-recumbent position with the head of the bed at 45° (Figure 1.4).
The patient should then be placed recumbent with legs elevated to 45°, and SV should be reassessed after approximately 30-90 seconds\(^4\) (Figure 1.5). A change in SV of \(>10\%\) after PLR has been shown to be a sensitive predictor of volume responsiveness.\(^5,6\) If stroke volume does not increase, the patient is likely not to benefit from fluid administration. To perform PLR consistently and accurately (Movie 1.1), it is helpful to have a foam wedge to ensure appropriate elevation of legs during the second measurement of SV.

**Figure 1.5 - PLR Legs Elevated Position**

**Movie 1.1 - One Minute Ultrasound Passive Leg Raise Demo**
Section 5
Assessing the Ejection Fraction

Although most echocardiographers use the “eyeball” technique to estimate the left ventricular ejection fraction (LVEF), there are additional methods for estimation. These include:

- Fractional shortening (FS)
- Simpson’s Method (Method of Discs)
- E-point septal separation (EPSS)

Fractional Shortening

Fractional shortening uses an anterior-posterior measurement of the left ventricular dimension to estimate ejection fraction. This measurement does not take into account the 3D shape of the ventricle and is fraught with error in patients with prior CAD history and wall motion abnormalities. The measurement is made at end systole (LVESD - end systolic diameter) and end diastole (LVEDD - end diastolic diameter). This measurement should be made 1cm apically from the septal-aortic attachment and perpendicular to the long axis of the heart in the parasternal long axis or parasternal short axis window and should bisect the chordae tendineae (Image 1.6, 1.7). A normal fractional shortening is between 25-45%.

Pearl: The FS is typically \( \frac{1}{2} \) the total ejection fraction

\[
FS = \frac{LVEDD - LVESD}{LVEDD} \times 100
\]
**Simpson’s Method**

The Simpson’s Method of Discs uses a computer package and a bit of upper level algebra to estimate the end diastolic and end systolic volumes of the LV. It requires a cardiac package on your ultrasound machine and adequate views of the apical 4 chamber and apical 2 chamber with traced endocardial borders in both systole and diastole for each. This method is the most time consuming, but it gives the most accurate estimation of LVEF. Its specific description is beyond the scope of this book, but more information can be found here.

**EPSS**

E-Point septal separation or EPSS is a commonly taught and easy method for assessment of the left ventricular ejection. This method has been described and used since the late 1970s. EPSS is a quick and dirty estimation of the LVEF and depends on free movement of the mitral valve. Thus, it can be inaccurate in disease processes that affect the MV such as mitral stenosis, calcification, significant aortic insufficiency and dilation of the mitral annulus from dilated cardiomyopathy. Nevertheless, in most instances the EPSS offers the novice sonographer a quick and easy method to estimate the LVEF.

To measure the EPSS, a parasternal long axis window is obtained and the image is maximized so that the mitral valve is well visualized. If the MV is touching the septum on visual inspection, the patient’s EF can be described as normal, or greater than 55%.
If the MV is not touching the septum, the distance from the MV during the E-point and the septum should be measured (Image 1.8). A distance <0.7cm is consistent with a normal EF. A distance >1.0cm is consistent with a reduced ejection fraction.

**STEPS TO EVALUATE EPSS**

**STEP 1:** Obtain parasternal long axis window (Figure 1.6).

**STEP 2:** Obtain adequate view of MV (Movie 1.2).

**STEP 3:** Place M-mode ice pick through tip of MV (Image 1.9).
STEP 4: Initiate M-mode tracing (Image 1.10).

**IMAGE 1.10 - M-Mode Button**

STEP 5: Identify the E and A waves of the MV (Images 1.11, 1.12).

**IMAGE 1.11 - M-Mode Through Mitral Valve**

STEP 6: Measure distance from tip of MV to septum (Image 1.13).

**IMAGE 1.13 - E and A Waves of Mitral Valve**

**IMAGE 1.12 - Normal EPSS and Thus LVEF**
Example of normal EPSS in patient with normal LVEF (Image 1.14):

**IMAGE 1.14 - Location of EPSS Measurement**

Examples of elevated EPSS in patient with severely reduced LVEF (Image 1.15, Movie 1.3):

**IMAGE 1.15 - Increased EPSS and Thus Reduced LVEF**

**MOVIE 1.3 - Increased EPSS and Thus Reduced LVEF**
A recent MRI study created an equation that can be used to estimate a LVEF based on the EPSS distance:

\[ \text{LVEF} = 75.5 - 2.5 \times \text{EPSS} \]

Although EPSS is usually an accurate measure of LVEF, there are a few occasions when the EPSS may falsely underestimate the LVEF:

1) Aortic insufficiency (AI): The AI regurgitant jet can push down the MV anterior leaflet in diastole causing the EPSS to be elevated despite a normal EF (Image 1.16).

2) Mitral Stenosis (MS): Although rare, moderate to severe MS can cause decreased excursion of the MV anterior leaflet, and thus elevate the EPSS.

**SUMMARY**

In conclusion, there are multiple ways to estimate a patient’s LVEF. Although these methods may be useful, none have been proven to be more accurate than a trained eye using visual estimation. Practice is the best way to learn to estimate LVEF. In the meantime while you are learning, EPSS and FS may be acceptable methods to estimate the LVEF.


Acute right ventricular (RV) dysfunction may occur in patients with critical illness and is often challenging to diagnose and treat. Echocardiography can assist at the bedside to diagnose the etiology (e.g., pulmonary embolus) and monitor the response to therapy. Echocardiography can also identify patients with chronic RV failure if they present with other illnesses; knowledge of pre-existing RV dysfunction may be important during their acute management (e.g., avoiding massive fluid resuscitation in patients with hypotension and severe RV dysfunction).

Hemodynamic assessment has classically focused on the left ventricle (LV), with little focus on the right. As this chapter will discuss, however, the right ventricle is sensitive to changes in pulmonary and systemic pressure and volume, which may affect a patient’s hemodynamic status and acute management.

**Highlights**

Echocardiography of the RV can be helpful in:
- PE
- Fluid resuscitation
- Chronic RV failure
- Hemodynamic status
The right ventricle is the most anterior chamber of the heart, lying directly behind the sternum. The RV has a complex geometric shape and is often described as a “U” shaped structure, as it wraps around the left ventricle. The RV can be visualized from the standard cardiac views; the inferior vena cava (IVC) should also be included in the assessment of the RV because it helps determine right atrial pressures. The RV can be visualized from the following four views:

1. Parasternal long-axis (PLA) view (Movie 2.1).

**Movie 2.1 - Parasternal Long-Axis View**

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**Highlights**

The RV has 3 distinct portions:
- Inflow Portion
- Apex
- Outflow Portion

The RCA perfuses the RV free wall.

The moderator band is perfused by the LAD.
2. Parasternal short-axis view (Movie 2.2).

MOVIE 2.2 - Parasternal Short-Axis View

3. Apical four-chamber (A4C) view (Movie 2.3).

MOVIE 2.3 - Apical Four-Chamber View

4. Subcostal long-axis view (Movie 2.4).

MOVIE 2.4 - Subcostal Long-Axis View

The RV is sometimes described as three distinct portions due to its complex geometric shape:

1. Inflow (or body) portion
   a. Begins at the tricuspid valve and extends to the apex.
   b. This portion is smooth-walled with minimal trabeculations.
2. Apex
   a. Begins after the inflow portion.
   b. Is heavily trabeculated and contains the moderator band.
   c. When the apex is hypertrophied, thick trabeculations may be mistaken for intra-cardiac tumors or thrombus.\(^4\)
3. Outflow portion
   a. Begins distal to the apex, ends just proximal to the pulmonary valve.
   b. Is the portion visualized in the PLA view (Movie 2.1).
BORDERS OF THE RV

The lateral border of the RV is the free wall; it is normally thin and should measure 5mm or less. Diseases or conditions causing increased pulmonary vascular resistance (e.g., pulmonary hypertension) and increased afterload (e.g., left heart failure) may cause hypertrophy and increase the thickness of the free wall. Diseases such as hypertrophic cardiomyopathies and infiltrative cardiomyopathies may also result in increased free wall thickness without an increase in afterload. The free wall should be measured from the subcostal view during diastole; this is the view where the free wall is most perpendicular to the plane of the ultrasound beam and hence, the most accurate measurement (Movie 2.5).

MOVIE 2.5 - Measuring RV Free Wall

The interventricular septum is the medial border of the RV and it is shared with the left ventricle. The interventricular septum normally remains concave with respect to the LV throughout both systole and diastole; changes in this relationship, however, represent RV pathology and will be described later in this chapter (Movie 2.6).

MOVIE 2.6 - Interventricular Septum

The tri-leaflet tricuspid valve (TV) comprises the inferior border of the RV, and consists of the anterior, posterior, and septal leaflets. The superior border is poorly defined due to the complex geometric shape of the RV, but the apex of the apical 4-chamber view is typically chosen. Of note, the tricuspid valve inflow view is usually the only view in which the posterior leaflet of the TV can be viewed (Movie 2.1).
**Pattern of Contractility**

The RV and the LV differ in their pattern of contraction. The LV contracts in multiple planes; however, the majority of contraction occurs concentrically around the midpoint of the LV. The RV, on the other hand, contracts longitudinally; contracting from the tricuspid valve towards the apex and outflow tract. This longitudinal pattern of contraction is often described as “peristaltic” or like an “accordion” (Movie 2.7).

**RV Coronary Blood Flow**

The majority of RV coronary blood flow is from the right coronary artery (RCA). The RCA perfuses the RV free wall and the inferior wall of the LV. The posterior descending artery, which is a branch of the RCA, perfuses the inferior portion of the interventricular septum. The moderator band is perfused by the left anterior descending artery (LAD); branches of the LAD may also perfuse the RV apex.

The RV has approximately 25% of the muscle mass of the left ventricle, despite the fact that the RV must deliver the same amount of cardiac output as the LV. The RV is able to match the LV in cardiac output because the pulmonary vascular system is a high-flow and low-resistance circuit. The contractile force of the RV is mostly supplied by the free wall of the RV, but up to 33% of the force is contributed by the interventricular septum with contribution from the LV.
**Assessing RV Size**

The thin-walled right ventricle is sensitive to acute changes in pressure and/or volume. The RV may acutely dilate in response, increasing its overall dimensions and volume in response to these changes. It is therefore important to determine the RV size during initial assessment with echocardiography, as it may help lead to the cause of the presenting disease.

**Qualitative Assessment: The “Eyeball” Method**

The simplest method of assessing RV size is an “eyeball” method using the apical 4-chamber view. The RV normally appears triangular-shaped and is two-thirds the size of the LV from this view, i.e., RV:LV is 0.6:1, as measured in diastole (Movie 2.8).

**Highlights**

The simplest method of assessing the RV size is the “eyeball” method using the apical 4-chamber view.

The normal RV/LV ratio is 0.6:1 in diastole.

Pressure and volume overload can be identified by a “D” shaped RV.

**Movie 2.8 - “Eyeball” Method**
As the RV becomes mild to moderately dilated, the RV:LV ratio increases to 0.7-1:1. When the RV is severely dilated, the RV:LV ratio is >1:1. Although this method is qualitative and an "eyeball" assessment, it has been found to be relatively accurate with good inter-observer reliability.

### Qualitative Assessment of RV Size

<table>
<thead>
<tr>
<th>Qualitative Assessment of RV Size</th>
<th>RV:LV Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0.6:1</td>
</tr>
<tr>
<td>Mild-Moderately Dilated</td>
<td>0.7-1:1</td>
</tr>
<tr>
<td>Severely Dilated</td>
<td>&gt;1:1</td>
</tr>
</tbody>
</table>

### Quantitative Assessment

A quantitative assessment of RV size can also be made from the apical four-chamber view. Three measurements are taken during diastole; two transverse diameters and one longitudinal (Movie 2.9).

#### MOVIE 2.9 - Quantitative Assessment

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**The RV is Enlarged When:**

1. The base of the RV (or annulus of TV) measures > 35mm.
2. The mid-cavity of the RV is > 42mm.
3. The longitudinal diameter of the RV (base to apex) is > 86mm.

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### Pearls for Identifying the RV

Severe RV dilation causes distortion of the ventricular anatomy to the extent that the novice sonographer may mistake the RV for the LV in the apical four-chamber view. Remember the following pearls to distinguish the RV from the LV (Movie 2.10):

1. The tricuspid valve is more apically displaced within the RV, as compared to the mitral valve within the LV.
2. The RV always contains the moderator band, while the LV does not.
3. Tilting the probe anteriorly will visualize the “fifth” chamber of the apical view, also known as the aortic outflow track; this ventricle must therefore be the LV.

#### MOVIE 2.10 - RV Identity Pearls
Assessing RV Systolic Function

Several diseases may lead to RV systolic dysfunction (e.g., RV acute myocardial infarction, sub-massive pulmonary embolism, etc.). Early detection may assist in the diagnosis, as well as monitoring response to therapy. Assessing systolic function, however, may be difficult due to the complex geometry of the RV and the longitudinal pattern of contraction. Two methods commonly used to assess RV systolic function are the:

1) Tricuspid annular plane of systolic excursion (or TAPSE) method
2) RV fractional area change (or RVFAC) method

Both methods assess RV systolic function from the apical four-chamber view.

TAPSE Method

The RV contracts longitudinally from the base to the apex (RV7). Therefore, the more the annulus of the TV is displaced during systole, the better the systolic function. Conversely, the less the TV annulus moves, the poorer the systolic function. TAPSE is a simple method in which the annular displacement of the TV is measured over a cardiac cycle and is performed from the apical four-chamber view. A normal TAPSE is 16-20mm, and a TAPSE less than 16mm is abnormal and indicates reduced RV systolic function.5

Adding an M-mode to this assessment may improve accuracy. To use M-mode, place the cursor to the lateral border of the tricuspid annulus and observe contractility over a few cardiac cycles and then freeze the image; calipers are then used to measure the annular displacement (Movie 2.11).

MOVIE 2.11 - Measuring TAPSE

RVFAC Method

RV systolic function can also be assessed by the RV fractional area change method. This method assesses the change in RV area from diastole to systole. To perform the method of RVFAC, the RV is imaged from the apical four-chamber view and attempts to minimize RV foreshortening should be made. If available on your machine, the appropriate cardiac package should then be opened. The image should first be frozen and cine back to end-diastole, then the RV en-
docardium is traced completely around the chamber including trabeculations, papillary muscles, and the moderator band. Once this area is completely traced, an area is calculated and will appear on the screen; this is the end-diastolic area (or EDA). Next, cine to the end of systole during the same cardiac cycle and trace the RV endocardium to obtain the end-systolic area (or ESA).

The RVFAC will automatically be derived or can be calculated by using the formula:

\[
RV \text{ Fractional Area Change} \\
FAC = \frac{EDA - ESA}{EDA} \times 100
\]

An RVFAC less than 35% is considered to be abnormal systolic function, while greater than 40% is considered normal. Although this quantitative method of RV systolic assessment is validated and widely accepted, it is not uncommon for experienced cardiologists to officially use the “eyeball” method assessment.

**VENTRICULAR OVERLOAD**

The thin-walled RV pumps blood to the left heart with a similar cardiac output as the muscular LV. The RV achieves this under normal filling and loading conditions because the pulmonary system is a low-pressure system. If the RV becomes overwhelmed by either pressure or volume overload, however, ventricular function may become abnormal. There are two categories of RV dysfunction:

1) Pressure overload leading to systolic dysfunction.
2) Volume overload leading to diastolic dysfunction.

**SYSTOLIC (PRESSURE) OVERLOAD**

Acute or chronic increases in RV afterload (e.g., pulmonary embolism, pulmonary hypertension, etc.) lead to systolic (or pressure) overload. The RV accommodates increases in pressure by “ballooning” out the RV free wall. As a result, the interventricular septum becomes flattened or “D” shaped when observed from the parasternal short-axis view (Movie 2.12).

**MOVIE 2.12 - Systolic Overload**
Septal flattening (also known as paradoxical septal motion or septal dyskinesia) occurs during both systole and diastole, in contrast to the normal shape of the interventricular septum, which is concave with respect to the LV in both systole and diastole. In extreme cases, LV filling and cardiac output may be compromised when the septum flattens and encroaches on the LV cavity.

The RV may accommodate chronic increases in afterload through hypertrophy of the free wall. Recall that measurement of the free wall should be made during diastole from the subcostal view, as the ultrasound beam is most perpendicular to the free wall; RV free wall measurements of greater than 5mm indicate hypertrophy from chronic overload (Movie 2.5). Other echocardiographic signs of chronic RV pressure overload include hypertrophy of apical trabeculations, papillary muscles, and the moderator band.

A clinically challenging situation arises when attempting to determine whether a patient with chronic RV afterload develops acute-on-chronic pressure overload (i.e., patient with chronic pulmonary hypertension develops an acute pulmonary embolism). In these cases, prior echocardiograms should be reviewed and compared to the current echocardiogram for any changes.

**Diastolic (Volume) Overload**

Acute volume (or diastolic) overload may occur with massive volume resuscitation (e.g., massive transfusion in hemorrhagic shock) and may lead to RV diastolic dysfunction. If volume overload occurs, the RV can acutely adapt by dilating or “ballooning” out the free wall in a manner similar to acute pressure overload; septal flattening (a “D” shaped septum) visualized in the parasternal short-axis view. Although this “D” shaped septum looks similar to pressure overload, the difference is that septal flattening from volume overload occurs at the end of diastole, while the septum is normal in contour during systole (Movie 2.13). As was the case in pressure (or systolic) overload, volume overload may compromise LV filling and cardiac output when the septum flattens and encroaches on the LV cavity.

**Movie 2.13 - Diastolic Overload**
NON-INVASIVE MEASUREMENT OF PASP

Invasive right heart catheters are typically used to measure pulmonary artery systolic pressures (PASP) for diagnosing or monitoring certain conditions (e.g., pulmonary hypertension). B-mode ultrasound and Doppler, however, can be used to non-invasively estimate right heart pressures. Estimation of PASP requires deriving two measurements:

1) The peak RV systolic velocity (measured from tricuspid valve).
2) The right atrial pressure (measured from IVC).

The first step is to obtain the peak RV systolic velocity, which is done by imaging the tricuspid valve (TV) in either the apical four-chamber view, RV inflow view (from the parasternal long-axis view), or parasternal short-axis view at the level of the aortic valve. Next, once the TV is visualized, a color Doppler box is placed over the valve to look for tricuspid regurgitation (TR). If TR is observed, the cursor for continuous wave (CW) Doppler is placed through the middle of the jet and the peak velocity is obtained. If the ultrasound machine has the appropriate calculation package, the peak RV systolic velocity of the jet is measured and the calculation package will automatically convert this value into a pressure (Movie 2.14).

The next step is to estimate the right atrial pressure (RAP). The RAP is obtained by visualizing the IVC from the subxiphoid view. The absolute size of the IVC and the change in diameter with respirations will estimate the RAP. IVC measurements should be made just distal to the junction of the IVC to the right atrium, just inferior to the first hepatic vein. For how to measure the IVC, see the Fluid Responsiveness chapter in Introduction to Bedside Ultrasound Volume 1.

The final step is to add the peak RV systolic pressure and RAP to derive the PASP. A PASP greater than 36mmHg is considered elevated. Limitations of this method include lack of an acoustic window to visualize the TV and the inability to define a TR jet (even with the coexistence of pulmonary hypertension).
Section 4
RV Pathology

Highlights

RV pressure overload and systolic dysfunction may suggest acute pulmonary embolism.

McConnell’s sign has a very poor specificity, possibly as low as 30%.

Signs of pericardial tamponade include: RA systolic collapse, RV diastolic collapse, reciprocal respiratory changes in the RV and LV filling, and plethora of the IVC.

Acute Pulmonary Embolism

Acute pulmonary embolism causes significant morbidity and mortality worldwide, and bedside echocardiography can be used as a tool to help detect the presence of this disease when other suggestive clinical signs and symptoms are also present.

Bedside echocardiography does not have adequate sensitivity to rule out pulmonary embolism alone, but may be suggested if found in conjunction with other findings. For example, RV pressure overload and systolic dysfunction may suggest acute pulmonary embolism if a significant pulmonary clot burden is present. Acute pressure overload and RV dilation may also lead to dilation of the tricuspid valve annulus and tricuspid regurgitation, which can be detected with echocardiography (Movie 2.15).

Movie 2.15 - Pulmonary Embolism
The presence of acute RV dysfunction with the acute onset of hypotension may also help with the decision to treat a pulmonary embolism with thrombolytic therapy in the proper clinical context.\textsuperscript{17}

McConnell’s sign was a commonly cited echocardiographic finding associated with acute pulmonary embolism. McConnell’s sign is defined by the presence of RV mid-wall hypokinesis with normal contraction of the apex (Movie 2.16). First described in 1996, McConnell’s sign was once believed to be a very specific echocardiographic finding for an acute pulmonary embolism.\textsuperscript{18} Since its initial description, however, McConnell’s sign has been found in several other clinical conditions (e.g., pulmonary hypertension, acute respiratory distress syndrome, RV myocardial infarction, etc.). Casazza et al. demonstrated that McConnell’s sign has a very poor specificity (30\%) for acute PE, and found that a significant number of patients with McConnell’s sign actually had an acute RV myocardial infarction (AMI).\textsuperscript{19}

\textbf{Acute RV Myocardial Infarction}

Bedside ultrasound can assist in the diagnosis of an acute myocardial infarction of the RV.\textsuperscript{4,5} Depending on the size and duration of the occlusion, echocardiography may detect segmental wall motion abnormality (i.e., hypokinesis, akinesis, or dyskinesis) and/or a reduction in myocardial thickening.\textsuperscript{3} The presence of McConnell’s sign (hypokinesis of the mid RV free wall with normal apical contraction) may also suggest AMI, and other modalities should be used to distinguish the clinical picture from acute pulmonary embolism and other diseases.\textsuperscript{19}

When an AMI of the RV is suspected, the RV should be inspected in multiple views to visualize the geometrically complex ventricle\textsuperscript{5}:

1) Parasternal long-axis view examines the RV outflow tract.
2) Parasternal short-axis view (papillary muscle level) visualizes anterior, lateral and portions of the inferior wall.
3) Apical four-chamber view visualizes the lateral RV free wall and apex.
4) Subcostal long-axis view visualizes the inferior RV free wall.
5) Subcostal short-axis view visualizes the RV outflow tract and a portion of the RV inferior wall.

Particular attention should also be given when imaging the inferior wall of the LV and the inferior LV septum because the RCA also provides perfusion to these territories.
PERICARDIAL TAMponADE AND THE RV

Pericardial effusions may occur from a variety of disorders (e.g., HIV, uremia, trauma) and ultrasound can be used to diagnose the effusion, as well as assist with ultrasound-guided pericardiocentesis. Pericardial tamponade is the physiologic state in which increasing pressure within the pericardial space may exceed the pressure within the heart (or the intra-cardiac pressure). Over time, this may result in reduced cardiac filling, cardiac output, and hemodynamic collapse. Tamponade is a clinical diagnosis, but ultrasound can demonstrate early signs of impending tamponade, even before hypotension develops. See the Basic Cardiac chapter in Introduction to Bedside Ultrasound Volume 1 for additional information.

TAMponADE: A REVIEW OF PHYSIOLOGY

Accumulation of fluid in the pericardial space progressively reduces filling of the low-pressure right-sided heart chambers, regardless of the initial insult or underlying pathology. If the pericardial fluid accumulates chronically, the pericardium can adapt by increasing in size to accommodate the rising intra-pericardial pressures. On the other hand, rapidly accumulating effusions are not well tolerated, and small but rapid effusions can acutely result in cardiovascular collapse. Whether an effusion develops acutely or chronically, there is a critical point where the rising intra-pericardial pressures exceed the pressures within the right side of the heart; this results in a reduction in cardiac filling and subsequently cardiac output.

Several signs of impending tamponade can be detected with bedside ultrasound. These include:

- Right atrial systolic collapse
- Right ventricular diastolic collapse
- Reciprocal respiratory changes in the RV and LV filling
- Plethora of the inferior vena cava

RA SYstolic COLLapSE

The right atrial (RA) free wall is thin and sensitive to increases in intra-pericardial pressures. As a result, critical intra-pericardial pressures result in systolic collapse of the RA free wall (best viewed from apical four-chamber view) and is a sign of tamponade physiology. Although brief RA systolic collapse may be normally seen, collapse persisting for greater than 1/3 of atrial systole is 94% sensitive and 100% specific for pericardial tamponade (Movie 2.17).

MOVIE 2.17 - Pericardial Tamponade
RV Diastolic Collapse

RV free wall collapse during diastole may also be seen with pericardial tamponade. RV collapse is best visualized in either the parasternal long-axis or subcostal long-axis view (Movie 2.18).

Once the appropriate window is obtained, an indentation of the RV can be visualized during diastole. An M-mode cursor may be placed through the RV free wall to better visualize the abnormal free wall motion\(^1\) (Movie 2.19).

Diastolic collapse of the RV free wall is less sensitive than RA systolic collapse (60% vs. 94%) but is a very specific sign of tamponade (100%\(^4\)). The sensitivity of RV diastolic collapse may be reduced, however, with conditions such as RV free wall hypertrophy and infiltrative cardiomyopathies (e.g., amyloidosis); diastolic collapse may be masked despite relatively elevated intra-pericardial pressures\(^2\).

Respirophasic Doppler Inflow Velocity

Inspiration causes negative intra-thoracic pressures, which pulls open (i.e., away from the septum) the free wall of the right ventricle under normal conditions. This creates a negative pressure within the right side of the heart, leading to increased venous return and right-sided cardiac filling. Increases in right heart filling result in an increased RV inflow velocity through the tricuspid valve. When expiration begins, the left side of the heart fills, resulting in an increased LV inflow velocity through the mitral valve; the right-heart simultaneously normalizes the RV inflow velocity. Over a normal respiratory cycle there should be no more than 25% and 15% variation of inflow velocity for the tricuspid and mitral valves, respectively\(^1\).

An exaggeration in the variation of inflow velocities is observed when tamponade physiology is present. This occurs because the RV free wall cannot pull open against the dense pericardial effusion. Therefore, as the right heart fills during inspiration, the septum is pushed over into the LV to accommodate RV filling; the result is that
the LV fills less effectively and the MV inflow velocity is reduced. With expiration, the LV inflow velocities increase and also become exaggerated as the septum shifts back towards the RV. Pericardial tamponade should be suspected when variations of more than 40% or 25% are observed for tricuspid and mitral inflow velocities, respectively. Of note, the previous description is the physiologic explanation of pulsus paradoxus (i.e., reduction in systolic BP by 10-12mmHg during inspiration) observed in pericardial tamponade.

The apical four-chamber view is the best view to assess variation of inflow velocities. To perform this exam, the sample volume for pulsed wave Doppler is placed at the tips of the leaflets of each valve. The velocities recorded above the baseline (i.e., toward the probe) are then measured for the maximum and minimum velocities and the percentage of variation recorded. This measurement is sometimes made with a respirometer to determine the phase of respiration, although not mandatory. The Doppler sweep speed can be reduced to 12.5 meters per second to visualize more cardiac cycles, especially if the patient has tachycardia (Movie 2.20).

**Inferior Vena Cava (IVC) Plethora**

Pericardial tamponade increases right heart pressures. This is reflected by a dilated or plethoric IVC with less than 50% respiratory variation (recall that IVC size and respiratory variation reflect RA pressure). Although IVC plethora is not a specific sign of pericardial tamponade (40%), it has good sensitivity (97%). A plethoric IVC can be used to confirm pericardial tamponade when other echocardiographic signs are also present and may help rule it out if a pericardial effusion is present (effusion but no tamponade) (Movie 2.21).

**Conclusion**

In summary, the geometrically complex RV is thin walled and has only 25% of the musculature of the LV, yet it produces the same amount of cardiac output. Several diseases can affect the RV, directly or indirectly, leading to hemodynamic compromise. Familiarity with the various echocardiographic changes in RV morphology may lead to earlier identification of disease and potentially better patient outcomes by minimizing hemodynamic compromise.
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Diastole is the period of the cardiac cycle between the closure of the aortic valve and closure of the mitral valve. The primary function of diastole is to fill the left ventricle in preparation for systole.

For the purpose of this chapter diastology is the echocardiographic evaluation of the relaxation and compliance of the left ventricle (LV) and one of the most complicated and difficult aspects of echocardiography. This evaluation is considered an advanced skill, but it can be a valuable part of the echo evaluation in patients with dyspnea or signs of heart failure in the emergency department. According to the European Society of Cardiology, about 50% of patients with heart failure have “diastolic-only” failure, and thus a normal ejection fraction (EF). With 5 million Americans suffering from heart failure and an annual related health care cost of 29.6 billion dollars, this disease represents a significant burden on the economy and greatly contributes to the health care crisis in the U.S.

A thorough diastolic evaluation should be performed on patients whenever a complete echocardiogram is performed. The diastolic evaluation itself takes only a few minutes and can help identify elevations in cardiac filling pressures and offer prognostic information. Diastolic dysfunction has been shown to more appropriately correlate with cardiac filling pressures than LV EF. For this reason, a more focused diastolic exam can be performed when interested in the relation of diastolic dysfunction to symptomatic heart failure in patients with dyspnea, weight gain, or other concerning findings for volume overload.
Diastolic function in the evaluation of critically ill patients is an area of significant interest in critical care echocardiography. We know that diastolic and systolic dysfunction can help elicit those patients that are at risk of being fluid intolerant. Some providers have even used changes in diastolic function to guide fluid therapy, although this has not been proven in the literature. While beyond the scope of this text, diastolic function can be used for multiple applications in critically ill patients, but it requires significant expertise to do so. An important application of diastolic dysfunction in sepsis is its use as a predictor of mortality. In one study, isolated diastolic dysfunction was found to have the highest hazard score of any predictor, even low urine output.
The physiology of diastolic function is possibly the most complicated aspect of the topic, but it is paramount in understanding how to apply echocardiography for diastolic evaluation. The diastolic filling period of the cardiac cycle can be broken into two phases:

1) E Wave: The early filling phase, which is dominated by myocardial relaxation and the left atrial (LA) filling pressure.

2) A Wave: The late filling phase, which is dominated by the LA contraction and left ventricular compliance.

This relationship is often described as a “push-pull” filling of the left ventricle (Figure 3.1).

The early filling phase represents the LV pulling blood from the LA by creating a relative negative intraventricular pressure by the post systolic relaxation of the LV chamber. This effect is secondary to the tor-
sional recoil of the left ventricle. In a normal heart, almost all of the filling of the LV occurs during the “pull.” As diastolic function worsens, the LV loses the ability to “pull” blood into the LV and the body has to retain fluid to increase the LA loading pressure. This represents a change from “pull” to “push”, where LA pressure has to elevate to overcome the poor relaxation and compliance of the left ventricle (Movie 3.1).

Most diastolic dysfunction is caused by longstanding hypertension. This disease leads to thickened LV walls from the constant increased afterload, which become stiff from hypertrophy. This stiffening causes worsening relaxation and compliance and impairs LV filling, which leads to decreased cardiac output, activation of the renin-angiotensin II system, and eventually increased preload and filling pressures. This is the body’s method of continuing to fill a thickened, stiff LV: by increasing filling pressures through an elevation in preload. This process causes the symptoms we all know as heart failure (Figure 3.2).
Section 3

Evaluating Diastolic Function

Highlights

Qualitative: Large LA, poor descent of the base.

Quantitative: PW Doppler of MV inflow, TDI of septal MV annulus.

Key Values:
- Elevated PCWP > 12mmHg
- Elevated LVEDP > 16mmHg
- Normal E velocity < 100cm/s
  - Normal e’ velocity > 8cm/s
  - Normal E/e’ < 15

Qualitative Evaluation

Diastolic dysfunction can be inferred from two key observational findings on bedside ultrasound. First, a patient with elevated filling pressures, and thus diastolic dysfunction, typically has a dilated, large left atrium. In fact, LA volume has been found to be very strongly associated with diastolic dysfunction and expresses the severity of diastolic dysfunction. The left atrial area is best measured in the apical 4 chamber view (Image 3.1).

Image 3.1 - Measurement of LA Area

This window can be difficult to obtain, but a normal atrial size in an otherwise healthy patient is often the extent to which diastolic dysfunction need be evaluated. A typical LA volume is measured at end
systole and is less than 58ml, although a visual estimation is typically adequate for assessing the need for further diastolic assessment. An additional initial analysis of diastolic dysfunction is the visual assessment of descent of the base - the apical displacement of the septal mitral valve annulus during diastole (Image 3.2, Movie 3.2).

Adequate movement of the base is typically acceptable to rule out severe diastolic dysfunction, as greater movement suggests rapid relaxation during early diastole, and thus normal diastolic filling. Slow or minimal descent of the base is consistent with diastolic dysfunction.

**IMAGE 3.2 - Location of Mitral Annulus**

**MOVIE 3.2 - Qualitative Evaluation of the LA and Descent of the Base**
Quantitative Evaluation

Quantitative evaluation of diastolic function is obtained by performing 2 specific Doppler measurements: Mitral valve inflow and tissue Doppler imaging.

Mitral Valve Inflow

Mitral valve (MV) inflow patterns are used to define the degree of diastolic dysfunction. This measurement tells us the velocity at which the blood is traveling as it passes through the MV during diastole. The ability of the LV to relax and comply to the changes in the pressure in the LA affects these velocity profiles. These measurements of the velocity profile can describe the severity of disease.

This Doppler measurement is obtained by placing the pulsed wave (PW) Doppler gate at the very tips of the mitral valve leaflets in the apical 4 chamber window (Illustration 3.1).

The waveform obtained includes the E and A waves, where “E” refers to early filling and “A” refers to the atrial kick (Image 3.3).

Illustration 3.1 - Location of Doppler Gate for MV Inflow

The variations of mitral valve inflow patterns are broken into 4 different types (Figure 3.3).

- Normal: E>A
- Impaired Relaxation: E<A
- Pseudonormal: E>A
- Restrictive: E>>>A
The severity of diastolic dysfunction worsens from normal to restrictive (Figure 3.4).

**Normal:** Normal filling pressures, **LVEDP** < 15mmHg

In normal MV filling, the majority of diastolic filling occurs during the early phase of the cardiac cycle, thus the E is greater than the A wave.

**Impaired Relaxation:** Normal filling pressures, **LVEDP** < 15mmHg

As the LV stiffens, the next chronologic phase in the progression of disease is Impaired Relaxation. This inflow pattern displays the myocardium’s inability to relax, thus less blood flow occurs during the early filling phase (low E velocity) and more occurs during the atrial kick (E<A). Although the myocardium is stiff at this point, the filling pressures are not elevated as the atrium makes up for the impaired relaxation without an increase in preload and filling pressure.

**Pseudonormal:** Increased filling pressure, **LVEDP** > 15mmHg

By this point in the disease process, the body has retained fluid in order to increase preload, and thus filling pressures, so that the LV can be appropriately filled during diastole. Appropriately, the increase in filling pressure and volume of the LA causes a significant pressure difference between the LV and the LA during early diastole, with the pressure in the LA being much higher than the LV. This leads to a higher E velocity and therefore E>A again. This pattern looks very similar to Normal, and thus requires tissue Doppler imaging (TDI) to differentiate. This patient is volume overloaded.
**Restrictive: increased filling pressure, LVEDP > 20mmHg**

Restrictive is the most severe form of the disease and likely represents significant volume overload. On MV inflow, there is little to no A wave and the E wave is typically very large with E velocities approaching 150-200cm/s. The pressure within the LV cavity during end diastole is now so high that the atrial kick is virtually ineffective at filling the LV, and thus there is little to no A wave on the MV inflow pattern.

MV inflow patterns are a spectrum and change with the patient’s volume status. For example, a patient admitted in acute heart failure with an initial echo showing restrictive MV inflow pattern may have a repeat echo on hospital day 3 showing impaired relaxation due to significant diuresis. Once a patient’s MV inflow pattern is impaired, they typically never go back to normal since the damage to the myocardium has already been done. However, patients can move between impaired, pseudonormal, and restrictive as their preload and filling pressures change.

**TISSUE DOPPLER IMAGING**

TDI measures the speed of the myocardium as it moves during diastole. As the LV fills, the ventricle elongates and the base of the heart (the portion including the MV, tricuspid valve, and atria) descends away from the probe. The speed at which the tissue moves while the ventricle is expanding changes as relaxation occurs, and thus diastolic function worsens. As the ventricle becomes stiffer, it moves slower and the descent of the base is diminished.

To obtain this measurement, the tissue Doppler imaging setting on the machine is used. TDI is simply a pulsed wave Doppler setting on some echo machines that automatically decreases the velocity scale to 0-20cm/s below the zero velocity baseline, decreases the wall filter, and decreases the gain.\(^\text{13}\) (Note: You do not have to have a TDI setting on your machine to measure the velocity of the myocardium for this evaluation, although TDI is quicker.) The apical 4 chamber window is again used and the TDI gate is placed on the septum, just next to the MV septal leaflet\(^\text{11}\) (Illustration 3.2).

[ILLUSTRATION 3.2 - TDI Gate Location for TDI Waveform Analysis]
The waveform produced displays an e’ wave and a’ wave, which signify the same aspect of diastole as in the MV inflow pattern: e’ represents early filling, and a’ represents the atrial kick. The e’ and a’ will be much slower than the E and A since they represent tissue movement rather than blood velocities\textsuperscript{13} (Figures 3.4, 3.5, Image 3.4).

A normal TDI displays an e’>a’ where the e’ velocity is > 8cm/s

As the diastolic dysfunction worsens, the e’ wave gets smaller and smaller as the myocardial velocity approaches zero. Once the e’ velocity is < 8cm/s, the patient has diastolic dysfunction. Thus, when differentiating between normal and pseudonormal MV inflow patterns, the TDI measurement can be extremely helpful.

Estimating LVEDP: The LVEDP can be estimated by dividing the E velocity by the e’ velocity (Figure 3.6). If this value is > 15mmHg, then the patient is believed to have an elevated LVEDP\textsuperscript{15,16}

Find out more about the topic of Tissue Doppler Imaging [here](#).
**MOVIE 3.3** - Diastology How-To

**MOVIE 3.4** - One Minute Ultrasound Diastolic Demo
**Acute Dyspnea**

Patients can present with symptoms of an acute heart failure exacerbation without systolic dysfunction. While the majority of these patients also have a reduced ejection fraction, diastolic dysfunction can be the underlying cause rather than systolic dysfunction. Pseudonormal or restrictive patterns identify those patients with elevated LVEDP and may help identify those patients that need diuresis.

**Volume Overload**

In patients with acute heart failure exacerbations, diastolic function can be used to estimate the LVEDP, and thus the response to therapy. As mentioned previously, as LVEDP changes, so does the inflow pattern. Therefore, it is not unusual for patients with heart failure to transition from restrictive to impaired relaxation over the course of their diuresis.

**Massive Volume Resuscitation**

Diastology can be used for directing fluid resuscitation or simply indicating those patients at higher risk for pulmonary edema with massive volume resuscitation. If used to direct fluid resuscitation, one would do so as if guiding volume resuscitation using the PCWP. We know that \( E/e' > 15 \) indicates an elevated LVEDP and thus an elevated PCWP. Thus, one option would be to fluid bolus the patient until the \( E/e' \) reached 15, although this has not been adequately studied. It is important to note that other methods of detecting fluid re-
sponsiveness have been more rigorously studied, such as passive lag raise, Ao velocity variation, or even the IVC changes with respiration. (See the Fluid Responsiveness chapter in Introduction to Bedside Ultrasound Volume 1 for more information). If just being used to identify patients at risk, one would have concerns of fluid tolerance if the patient has impaired relaxation, pseudonormal or restrictive MV inflow patterns at the onset of volume resuscitation.

Also see: Ultrasound Podcast Diastology Part 1 and Part 2.
LIMITATIONS

There are several limitations of diastolic evaluation of the heart using Doppler echocardiography. The first and most important is operator error. Doppler measurements change with the angle of insonation. Additionally, patients with tachycardia often have fused E and A waves, making mitral waveform patterns difficult to address. Arrhythmias, such as atrial fibrillation, destroy the A waves and cause irregular heartbeats, which inevitably change the dP/dT from beat to beat. This makes diastolic evaluation virtually impossible.

There are also other causes of heart failure symptoms in patients with normal ejection fractions. These include constrictive pericarditis and mitral regurgitation.

CONSTRUCTIVE PERICARDITIS

This also causes elevated filling pressures in the setting of a normal LV EF and can present as acute heart failure. However, patients have respiratory variation of MV inflow velocities >25% with expiration, diastolic flow reversal in the hepatic veins, and typically have a septal e’ velocity > the lateral annulus e’ velocity (Image 3.5).
The latter of these findings is unique to constrictive pericarditis and is due to the retained vertical excursion, thus the septal e’ velocity being greater than the lateral e’ velocity.  

**Mitral Regurgitation (MR)**

Moderate to severe MR can lead to significant elevations in peak E velocities of mitral valve inflow. While this increase in E wave velocity does not necessarily mean diastolic dysfunction, the increased E/e’ ratio is predictive of the degree of MR disease and the need for hospitalization and mortality.  

**Conclusion**

In the end, diastology is a more advanced technique than identifying pericardial effusion and estimating global function. Nevertheless, it can be a very valuable tool that is applicable to a very large population of patients. If one takes the time to learn it, patients will reap the benefits.


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