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Basic Hemodynamics:
A Global View of Cardiovascular Dynamics

The introduction of echocardiography has revolutionized medicine by allowing immediate assessment of function and pathology of the heart. Ultrasound is now allowing us to other components of the thorax including the lung and pulmonary vasculature. Use of the pulmonary artery (PA) catheter has decreased over time due to a lack of strong evidence proven benefits as well as complications such as arrhythmias, pulmonary artery rupture, and infection. Additionally use may in fact increase length of stay.

Unfortunately though it seems like the reduced use has been associated with a reduction in understanding of basic hemodynamic function and the impression that echocardiography provides as accurate information in as timely a manner.

Understanding hemodynamic status though is not as simple as visualization with echocardiography or measurement with central lines. A full understanding of hemodynamics should include ALL of the tools available in a logical educational presentation that builds on the most basic tools and culminates with a comprehension from a global perspective. We will look at hemodynamics by incorporating the **electrocardiogram (EKG**), **central venous pressure (CVP), advanced hemodynamic tracings**, **echocardiography,** and the **mathematical formulas** than indicate the condition of the relationships. Once one global cardiovascular physiology then one will be able to understand the use of pharmacologic agents as well as other interventions meant to control function

The Electrocardiogram (ECG)

The ECG is a representation of the electrical activity of the conduction system of the heart. The mechanical events that occur are slightly delayed. We often think of the mechanical events as those that occur within the ventricle without appreciating that critical things are happening in the atrium at the same time. Ventricular activity cannot be maximized unless atrial events are optimized. It is important that activity of the all of the cardiac chambers are appreciated (Figure XXX).

Figure XXX- Mechanical Events Associated with ECG

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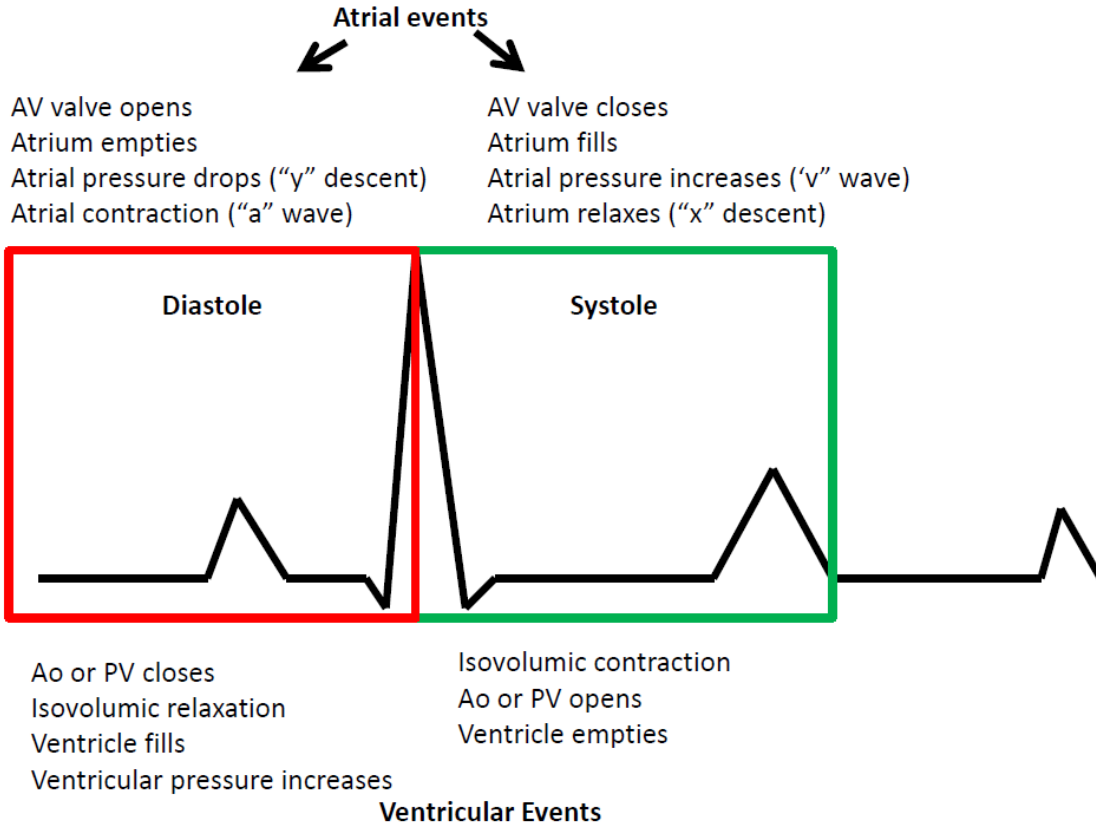


Pressure Tracings

We have now discussed the ECG and the accompanying events that that occur in the heart during both systole and diastole. The ECG only reflects electrical activity. It is the mechanical events that generate pressures and move blood forward (good) or back (pathology).

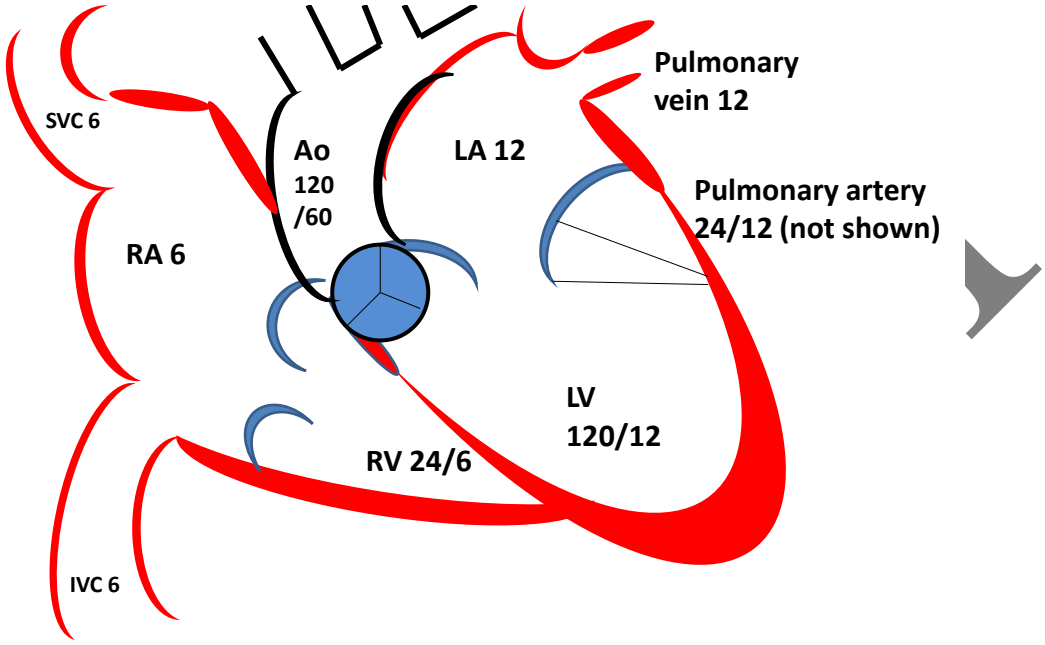
We often think of the right and left ventricles as very separate entities, yes we know that they interact but we often do not realize that functionally they perform must deal with the events.

The simplest way to start is to review the normal pressures. The easiest way I have been taught is to remember the "rule of '6's"'. This is based on the fact that the normal pressure in each chamber of the heart is some simple multiple of 6.

Such pressure tracings are obtained with the pulmonary artery catheter inserted through the right internal jugular vein. Tempe et al studies the distances the line needs to be inserted to reach specific chambers (Table XXX). Distances were more in those undergoing valve surgery compared with those undergoing CABG. Th elength o finsertin to reach the pulmonary artery and pulmonary capillary wedge was directly related to the height of the patient.

Chamber	Average distance	95% CI
Right ventricle	24.6 cm	24.2-24.9 cm
Pulmonary artery	36 cm	35.6-36.5 cm
Wedge	42.8 cm	42.2-43.5 cm

Figure XXX- The Pressure "Rule of 6's"



Hemodynamic "Rule of 6's"

Volumes

Pressures do not necessarily correlate with the individual chamber volume. There are published normal values for chamber volume as well as size.

Table XXX- Normal Chamber volumes

Chamber	Male	Female
Left ventricle EDV	106 ± 22 cc	76±15 cc
LV ESV	41 ± 10 cc	28 ±7 cc
LV EDD	50.2±4.1 mm	45.0±3.6 mm
LV ESD	32.4±3.7 cc	28.2±3.3 mm
LV EF (%)	52-72%	54-74%
RV EF (%)	58 ±6.5%	58±6.5%

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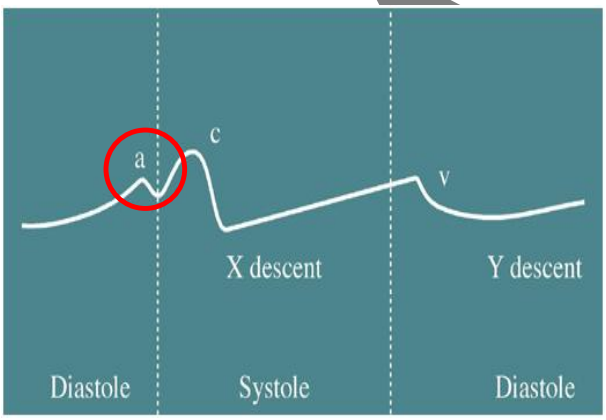
The Central Venous Pressure Tracing (CVP)

The central venous pressure (CVP) may be measured either in the superior or inferior vena cava or within the right atrium itself. When a pulmonary artery catheter is in place measurement generally occurs at the superior cavoatrial junction. There are three "waves" that are noticeable but often difficult to closely distinguish due to the nature of the monitoring systems which unfortunately cannot be stopped in real time to allow assessment. These "waves" reflect events rather than distinguish individual points in time. Waves may reflect active events such as atrial contraction of more passive phases such as filling. It is important to understand that the pressures in the left atrium have the same morphology as those on the right.

Most ventricular filling from the atrium (80%) is passive but the final 20-% is active and relies upon contraction of the atrium. This muscular contraction results additional filling but also an increase in atrial pressure (Figure XXX). This pressure increase is the last microsecond in which the pressure in the atrium exceeds that in the right ventricle (*diastole*).

Figure XXX- "a" wave

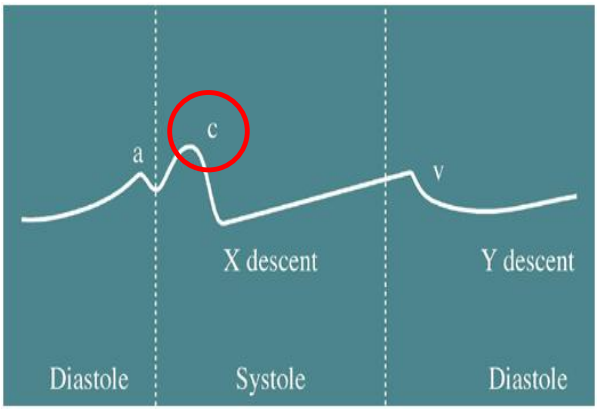


"a" wave is due to atrial contraction at the end of diastole. Pressure is increased as the atrium contracts. Conditions such as contraction against a closed or stenotic valve will increase the size of the wave

Pathology:
The "a" wave will *not* be seen in atrial fibrillation. The "a" wave may be more prominent in conditions such as tricuspid stenosis or where ventricular filling relies more on atrial filling that normal, ie cor pulmonale. A "cannon 'a' wave" occurs when the atrium contracts against a *closed* tricuspid valve.

The "c" wave is an event in early *systole*. During early systole the ventricle is most filled. The "c" wave occurs at the immediate onset of ventricular contraction prior to the opening of the pulmonary valve (isovolumic contraction). The tricuspid valve bulges into the right atrium causing an increase in atrial pressure (Figure XXX).

Figure XXX- "c" wave

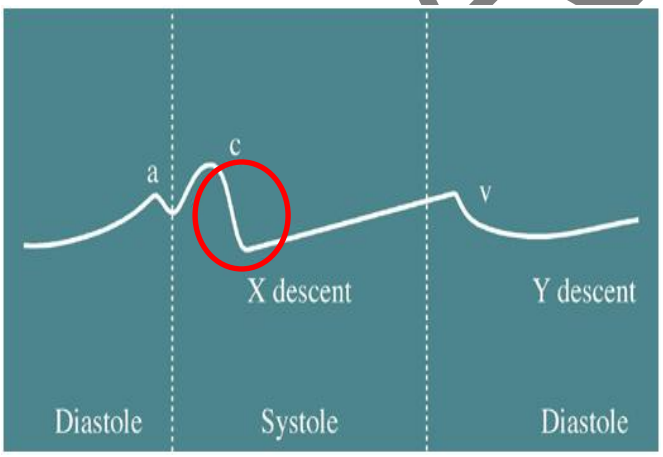


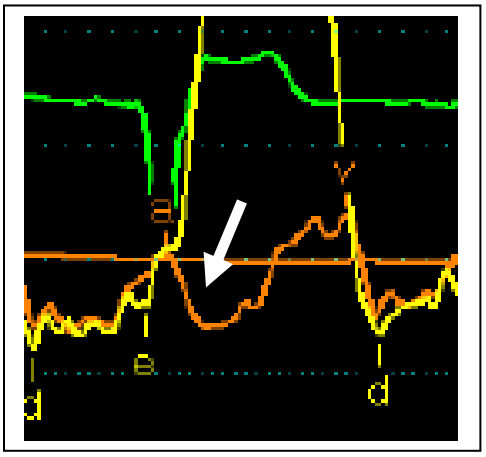
Pathology:
The "c" wave may be prominent in tricuspid regurgitation.

The "c" wave is due to bulging of the tricuspid valve into the atrium at the start of ventricular systole. Any condition which results in more bulging or volume entry into the atrium during early systole will increase the "c" wave

The "X" descent follows the "c" wave. As the annulus of the tricuspid valve descends towards the apex of the heart during ventricular systole, the atrium relaxes and there is an associated pressure drop or "descent". This is correlated with the Tricuspid Annular Plane Systolic Excursion (TAPSE) during echocardiography.

Figure XXX- "X" descent





The "x" descent occurs as the atrium relaxes and the tricuspid valve is pulled downwards. This is correlated with the tricuspid annular plane systolic excursion (TAPSE)

The "v" wave occurs during ventricular systole. The ventricle is contracting, thus pressures are higher than in the atrium. The tricuspid valve is closed. During this time blood is filling the atrium from the superior and inferior venacava. This filling increases pressure and the "v" wave is seen.

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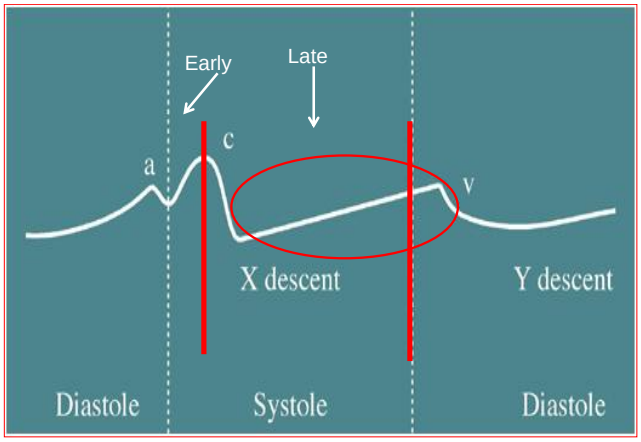
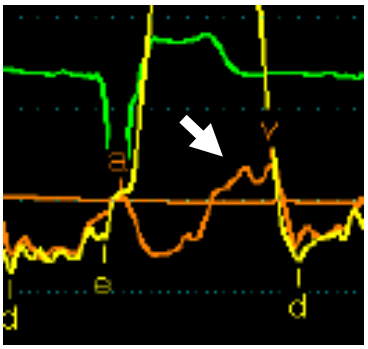
Figure XXX – "v" wave



During systole, the tricuspid valve should be closed. During this time, the right atrium is filling with blood which is increasing the pressure within the right atrium. The wave of increased pressure is called the "v" wave.

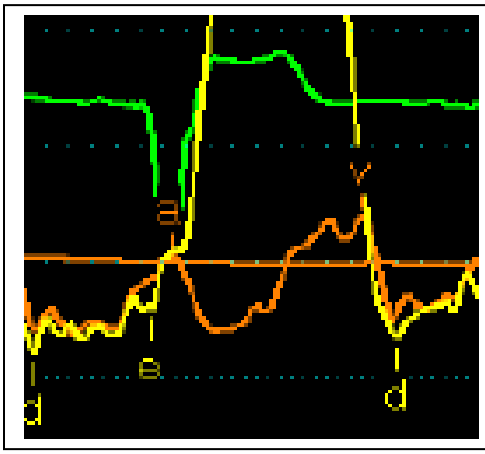
Pathology:
The "v" wave will be prominent in conditions resulting in more rapid filling of the atrium such as tricuspid regurgitation

Close up of atrial filling during systole (LA in this case)



The "y" descent is the last event on the central venous tracing. The "y" descent occurs when the tricuspid valve opens and blood rushes into the right ventricle. This is an early diastolic event.

Figure XXX- "y" descent



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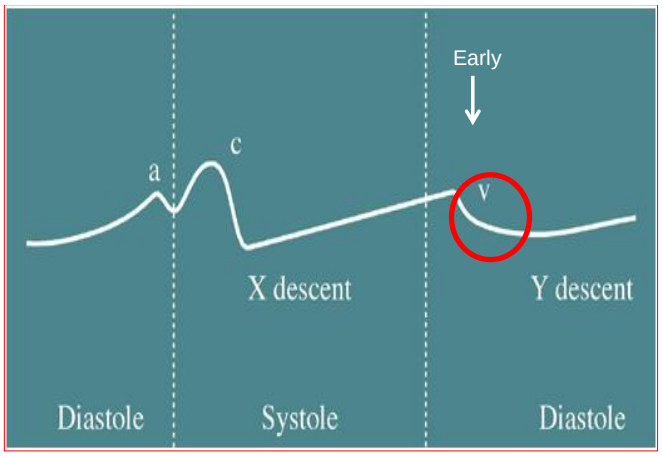

The "y" descent occurs when the tricuspid valve opens and blood flows into the right ventricle

The CVP reflects what is happening in the right atrium. We make the assumption that if the pressure in the atrium at such a time is "a" mmHg then the pressure in the left ventricle must be "d" and this must reflect the filling and function of the left ventricle. This is a bit of a stretch for many reasons which we will discuss.

It is important to begin with the fact that the correlate of the left sided correlate of the central venous pressure is the pulmonary venous pressure (PVP) and of course the left atrial events (LA). We do not monitor these continually because that would require penetration into the LA. Regardless the events are the same.

When the pulmonary artery occlusion pressure (PAOP) or "wedge" pressure is obtained, these waves are quite distinct (Figure XXX).

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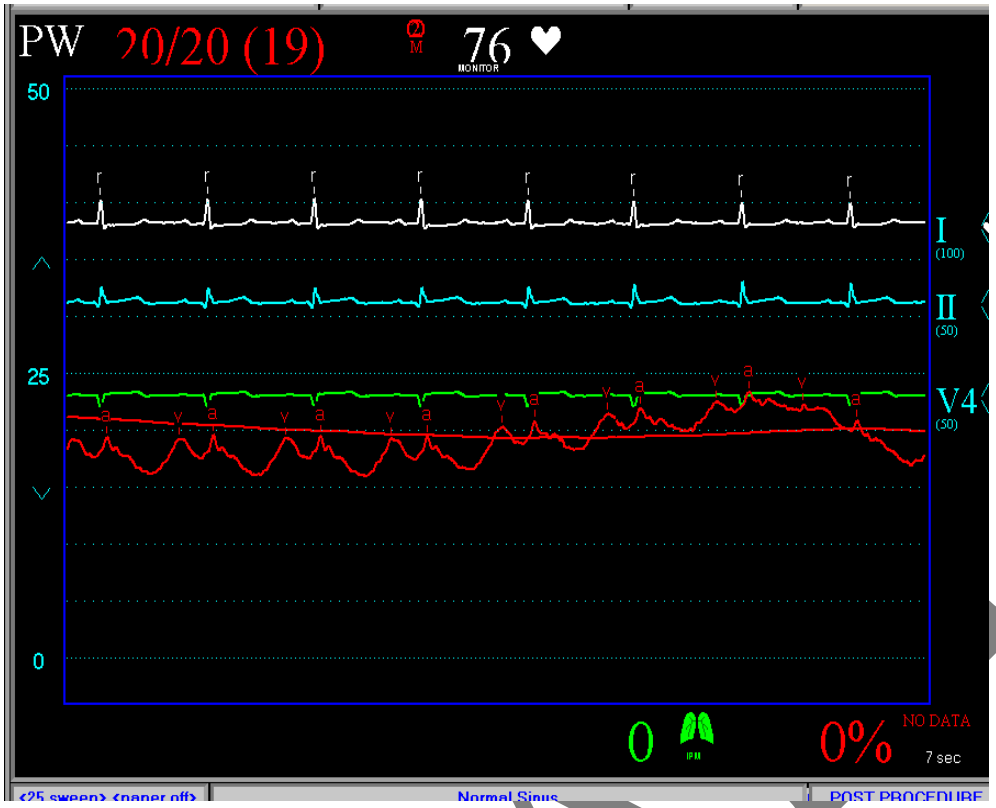

Before we move on to ventricular pressures, we should discuss the relationship of the central venous pressure (or right atrial pressure) and the pulmonary venous (of left atrial pressure). The left atrial pressure is higher than the right atrium during normal conditions. This is largely due to the lower compliance of the left ventricle (Figure XXX).

Figure XXX- Simultaneous tracing of the CVP and LA pressure

Pathology:
If the left atrial pressure exceeds the right atrial pressure the left atrium is not being filled appropriately. Conditions which may cause this include acute or chronic pulmonary emboli (CTPHTN), right ventricular failure, pneumothorax, focal tamponade

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Ventricular Pressure Tracings

We know that the same relative events occur on both the right and left sides of the heart during the phases of the cardiac cycle. We have looked at the atrial events including the "a", "c", and "v" waves. Now we will look at the pressure tracings in the ventricle.

The ventricles fill with blood during diastole. This results in an increase in pressure. The diastolic pressure in the right ventricle is generally approximately 6 (the rule of "6's").

Figure XXX- Simultaneous LV and Ao pressure demonstrating cardiac events

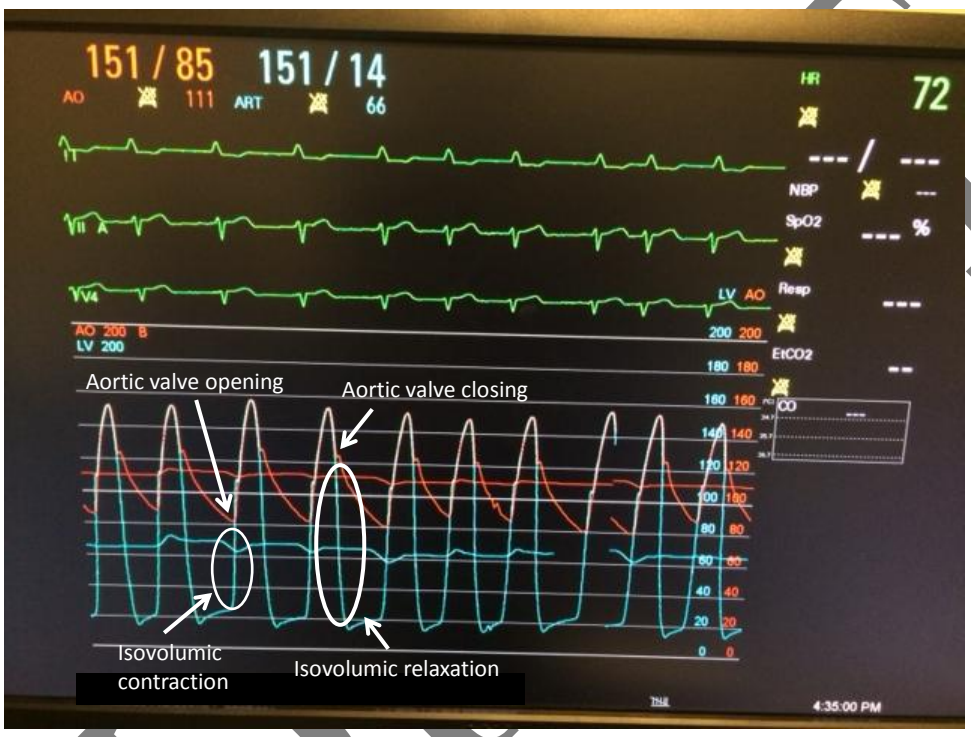

Figure XXX- RV pressure tracings during both systole and diastole

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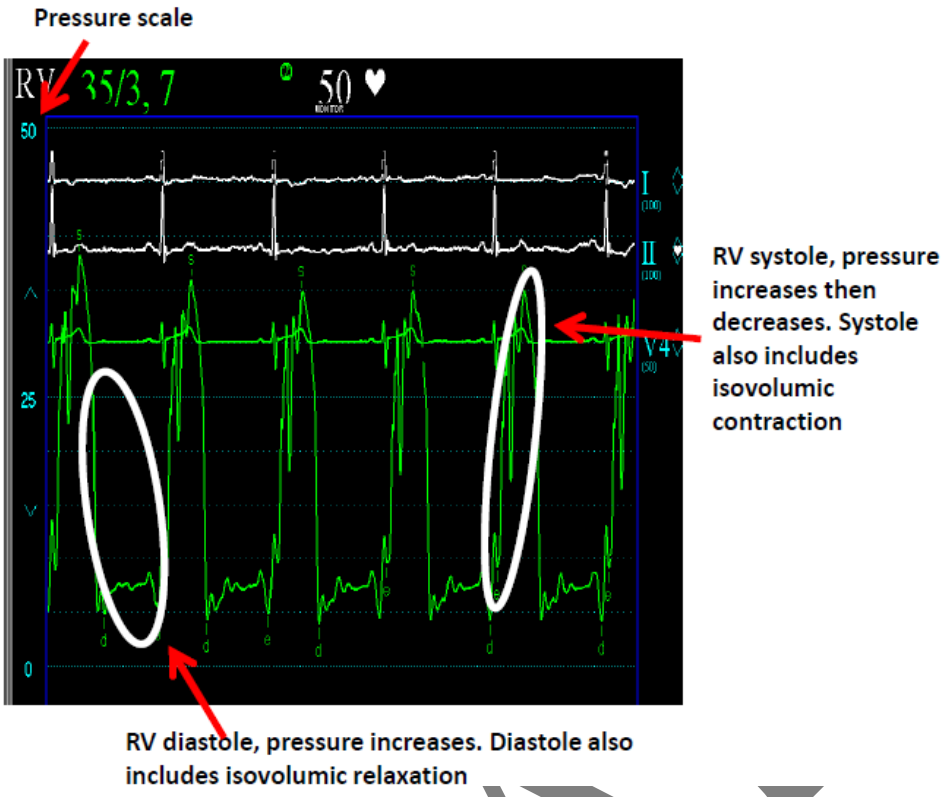

The morphology of the pressure tracing of the left ventricle is the same as the right but the pressure scale is obviously much higher. The pressure increases during diastole as the ventricle fills. Pressure increases rapidly associated with contraction during systole. Initially pressure increases without ejection of blood (isovolumic contraction) and is followed by ejection.

Figure XXX-Pressure tracing of the left ventricle

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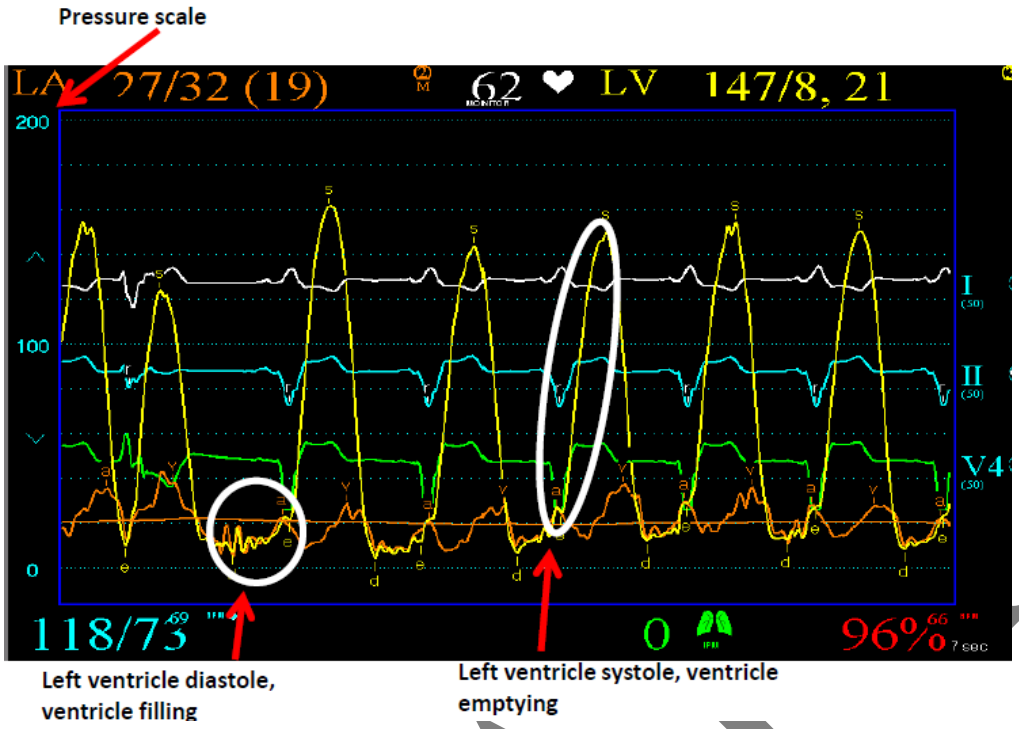

Relationship of the Atrial and Ventricular Pressures during diastole

The pressure relationships of the atrium and the ventricle are most important during the diastolic phase of the cardiac cycle. Pressure gradients are what move blood forward. Blood can only move from the atrium to the ventricle when the pressure in the atrium exceeds that in the ventricle. This is of course during diastole. The minute the pressure in the ventricle drops below the atrium, the atrioventricular valve opens and the ventricle fills. When the pressure in the ventricle exceeds that in the atrium, at the end of diastole, the atrioventricular valve closes. The increased ventricular pressure is a combination of both filling and contraction (Figure XXX).

Figure XXX- Relationship between the atrial and ventricular pressures

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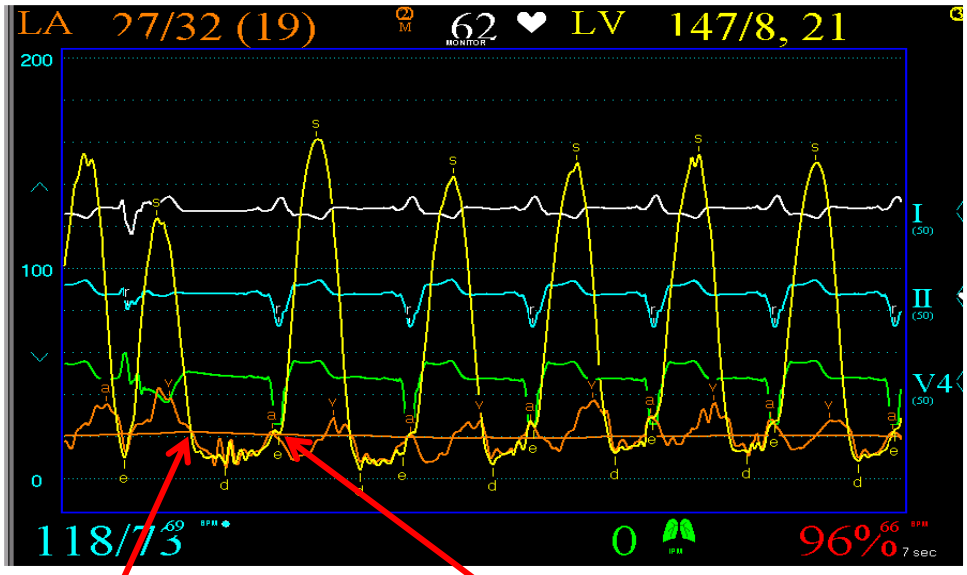

Pressure in atrium (orange tracing) exceeds that in the ventricle (diastolic filling begins)

Pressure in ventricle (yellow) exceeds that in the atrium (diastole ends)

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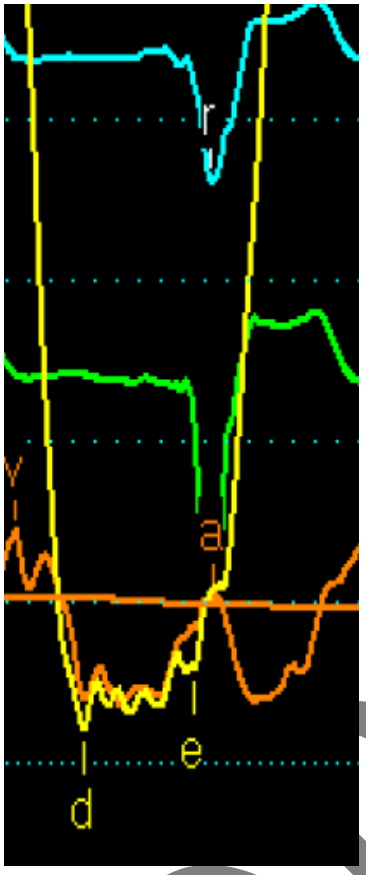
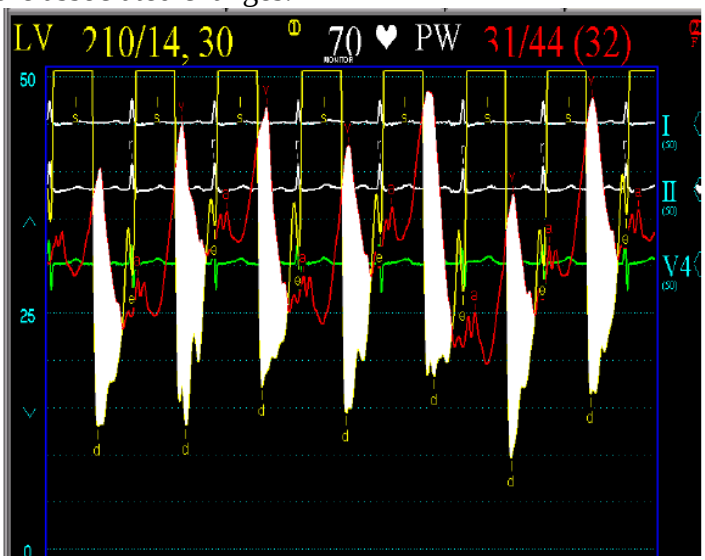
Figure XXX- Close up of pressure relationship between the left atrium and left ventricle during diastole where pressure in the left atrium (orange) is higher than the ventricle (yellow) throughout diastole but when the pressure in the atrium falls below the left ventricle, the mitral valve closes and diastole ends



Pathology:
Mitral stenosis results in a high pressure gradient across the mitral valve throughout diastole. The pressure in the atrium remains high leading to pulmonary edema (especially in situations of high cardiac output such as exercise) and ultimately pulmonary hypertension and the associated changes.



The Aortic and Pulmonary Artery Pressure Tracings

The aortic and pulmonary artery tracings represent the results of the effectiveness of the atrium and ventricles. The aortic pressure should be nearly the same as the femoral pressure.

The peak pressure generated by the ventricle should be the same as the peak pressure in the aorta in the absence of any aortic stenosis.

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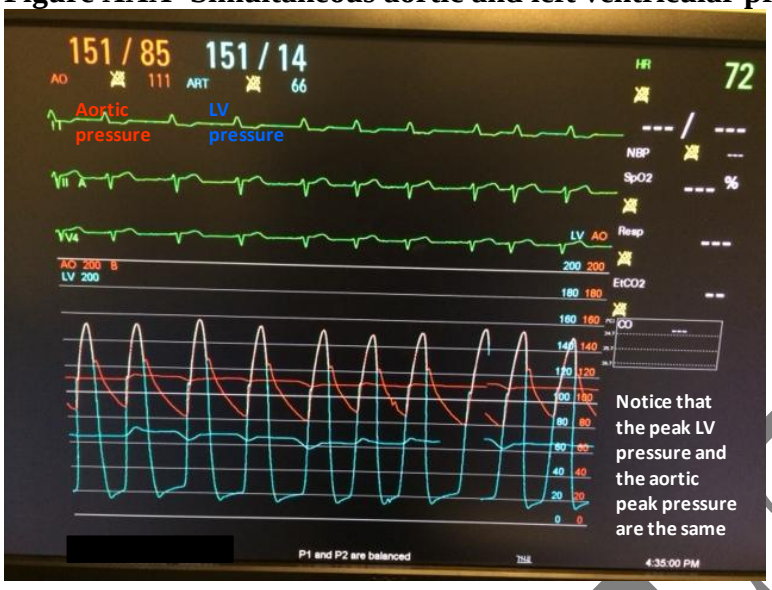
Figure XXX- Simultaneous aortic and left ventricular pressure



Pathology:
The peak pressure in the left ventricle will be much higher than that in the aorta when aortic stenosis is present.

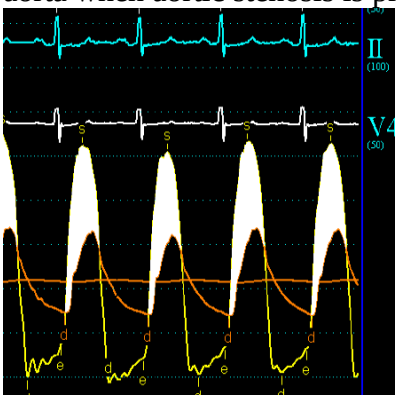
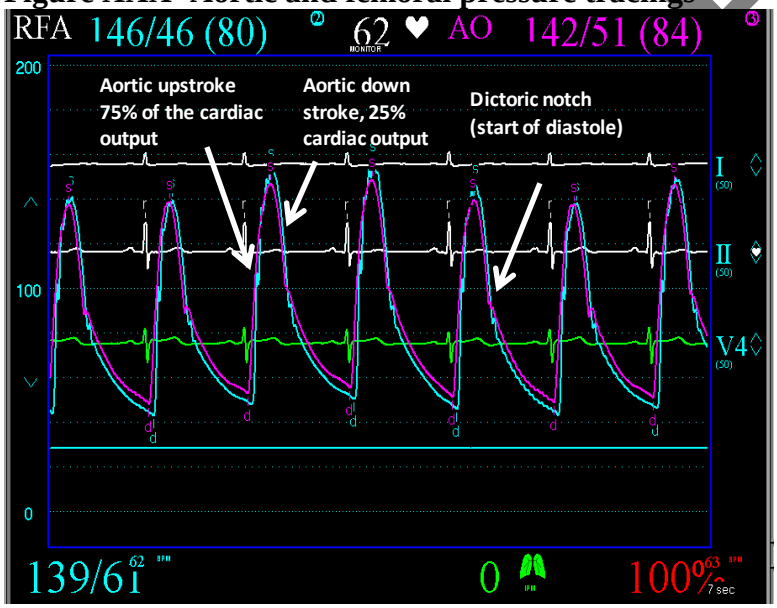


Figure XXX- Aortic and femoral pressure tracings



Echocardiography and Hemodynamics

So far we have discussed the ECG and normal pressure tracings. The third component of our journey through global hemodynamics is echocardiography. We are going to limit ourselves primarily to transesophageal echocardiography (TEE) but the same principles apply to transesophageal echocardiography (TEE). We are going to also leave discussions of advanced assessments of pressures with TEE for another venue. Some of these are quite simple while others are more complicated.

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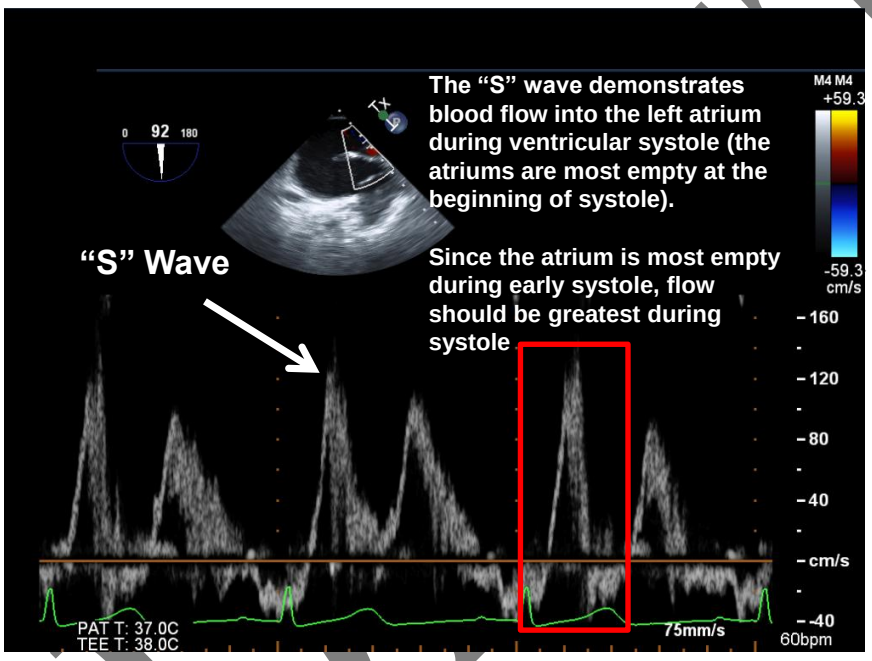
TEE can help understand the events that occur throughout the cardiac cycle. The use of **pulse wave Doppler** allow us to determine flow velocities at certain points in the cardiac cycle. **Continuous wave Doppler** allows measurements of pressure gradients.

We have discussed that the atrium primarily fills during systole (the atrium is most empty during early systole since it has just emptied its volume in the ventricle). The highest rush of blood into the atrium generally occurs in early systole. Echocardiography can demonstrate this flow with a prominent "s" wave when one looks at the pulmonary veins which obviously conduct blood to the left atrium (Figure XXX).

Figure XXX- Demonstration of high flow velocity into the left atrium during early systole ("s" wave).

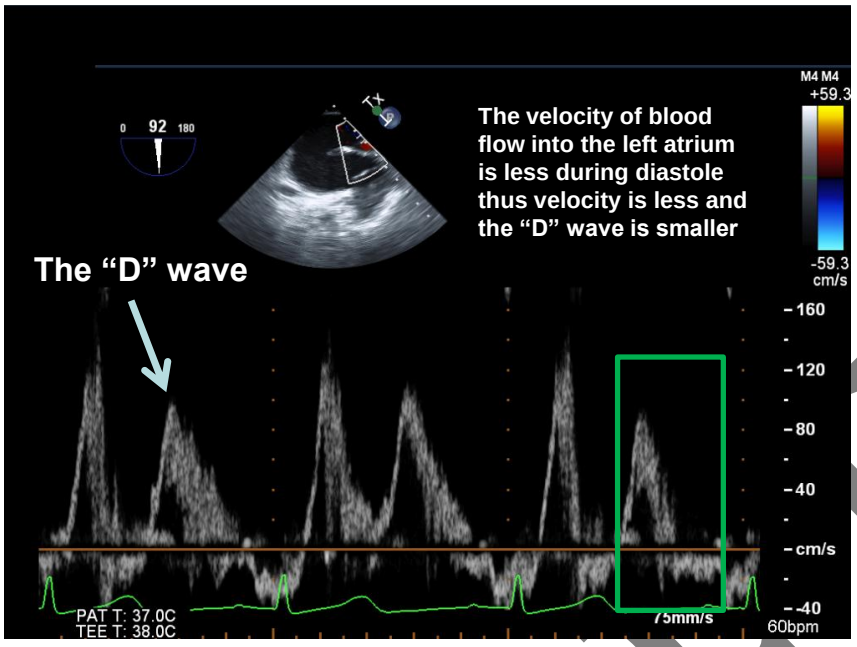


Pathology:
The "s" wave occurs in systole as the atrium is filling. The "s" wave is blunted in conditions where the atrium is filling rapidly from another source, ie mitral regurgitation (think of the prominent "V" wave during systole.

The "S' wave may even appear "reversed" if mitral regurgitation is so severe that blood is forced back into the pulmonary veins.

The blood does enter the left atrium during diastole but at a slower rate than during systole, thus the wave demonstrated with Doppler ("D" wave), is of less magnitude than the "S" wave (Figure XXX).

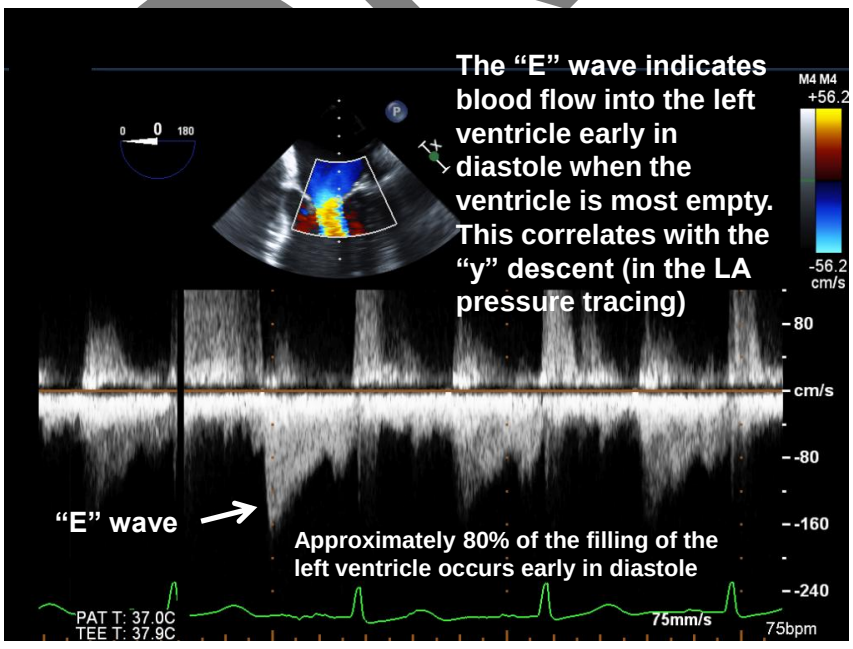
Figure XXX- The "D" wave demonstrating left atrial filling during diastole



Pathology:
The "D" wave is generally of less magnitude than the "S" wave but conditions such as mitral regurgitation or elevated left atrial pressures due to another condition may result in the "D" wave appearing prominent.

The ventricle fills during diastole while the atrium is emptying. The left ventricle is most empty early in diastole, thus this is when filling is easiest and blood generally rushes in the fastest. Early diastolic filling is demonstrated with pulse wave Doppler by a tall "E" wave (Figure XXXa). A smaller wave at the end of diastole represents atrial contribution to LV filling (atrial "kick") Figure XXXb).

Figure XXXa- "E" wave demonstrated early in diastole by pulse wave Doppler interrogation



Pathology:
Conditions which produce a high pressure or volume in the left ventricle during diastole will result in less vigorous blood flow and the "E" wave will appear "blunted" with reduced magnitude. Such conditions include left ventricular hypertrophy or restrictive cardiomyopathy

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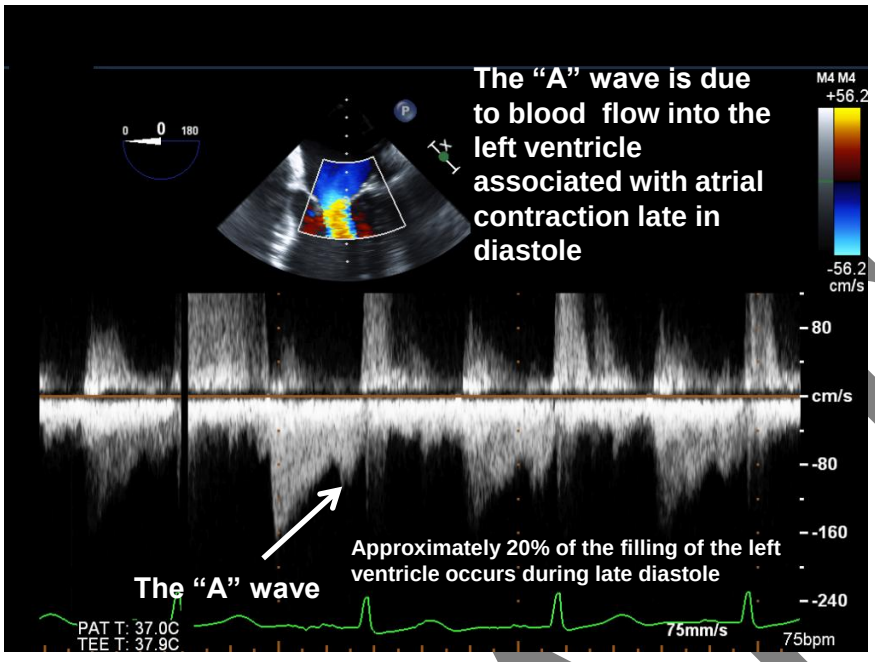
Figure XXXb-



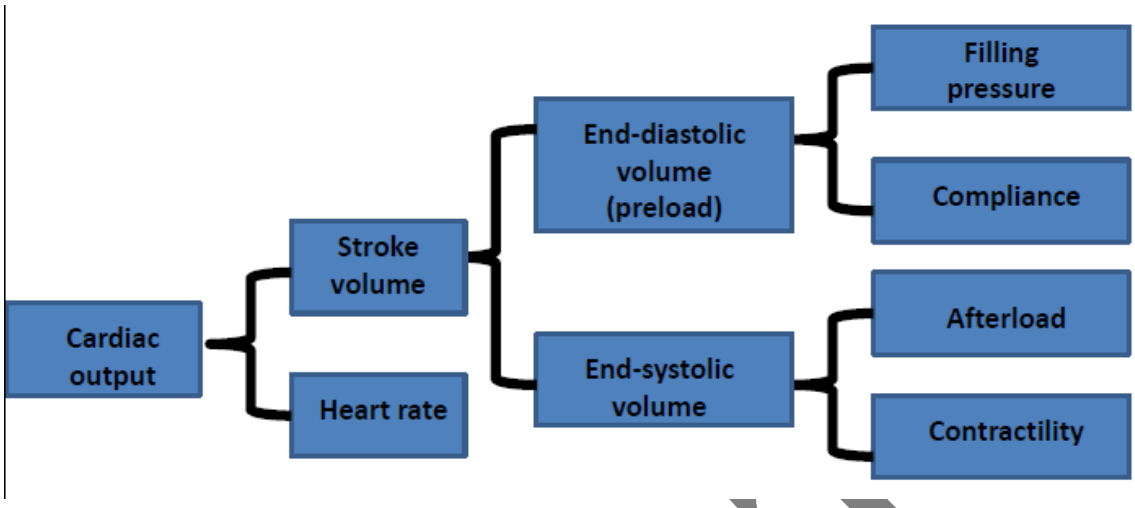
Pathology:
The “A” wave will be absent with atrial fibrillation. It will be exacerbated in conditions where the atrial lick contributes more to filling of the left ventricle such as left ventricular hypertrophy

Hemodynamic Mathematical Formulas

There are many formulas that reflect the status of the cardiopulmonary system. Understanding the simple as well as the more complex will allow a more complete assessment of the condition of the patient before surgery as well as where to target interventions throughout the perioperative course. For the purpose of this discussion, we will focus on the left ventricle although the same principles apply to the right.

The first place we should start is the simplest which is determining **cardiac output**. Cardiac output is the simplest of the equations (**CO = heart rate x stroke volume**).

Figure XXX-Components of Cardiac Output



Heart rate is quite simple to assess and plug into our consideration. Lower heart rates allow more diastolic filling and when ventricular function in preserved stroke volume will be higher. The higher stroke volume though is unlikely to offset the decrease in rate. Higher heart rates will increase cardiac output but at very high levels will limit diastolic filling.

Stroke volume is simply the difference between the end diastolic volume (LVEDV) and the end systolic volume (LVESV), thus **SV = LVEDV – LVESV**.

The real factors though are the conditions that affect the filling and function of the left ventricle (Table XXX).

Table XXX- Factors affecting myocardial performance

Ventricular pressure	Compliance	Afterload	Contractility
Pulmonary emboli Hypovolemia Tension PTX Venodilators Pulmonary vein stenosis	Amyloidosis Restrictive cardiomyopathy Constrictive pericarditis Tamponade Septal shift	Sepsis/SIRS ACE-I Vasodilators Hypertension	Ischemia Inotropic agents Hypothermia Hypocalcemia Sympathetic discharge Acidosis Hypercapnia Anoxia

Pressure-Volume (PV) Curves and Ventricular Function Curves

We are not going to spend much time on pressure-volume (PV) curves in the basic hemodynamic talk but I will introduce the topic as both are used in discussions and educational about the changes which occur with different physiologic and pathologic conditions.

The “X” or horizontal axis reflects the volume of the ventricle (generally the left ventricle). The “y” or vertical axis indicates the pressure within the ventricle. The normal left ventricular end diastolic volume (LVEDV) is 106 ± 22 cc in a male. The normal left ventricular end systolic volume (LVESV) is 41±10cc. The PV curve does imply that certain physiologic events occur including opening and closure of the aortic and mitral valves. The PV curve is an effective means to represent ventricular events in pathologic states such heart failure and valve conditions.

Figure XXX – LV Pressure volume curve

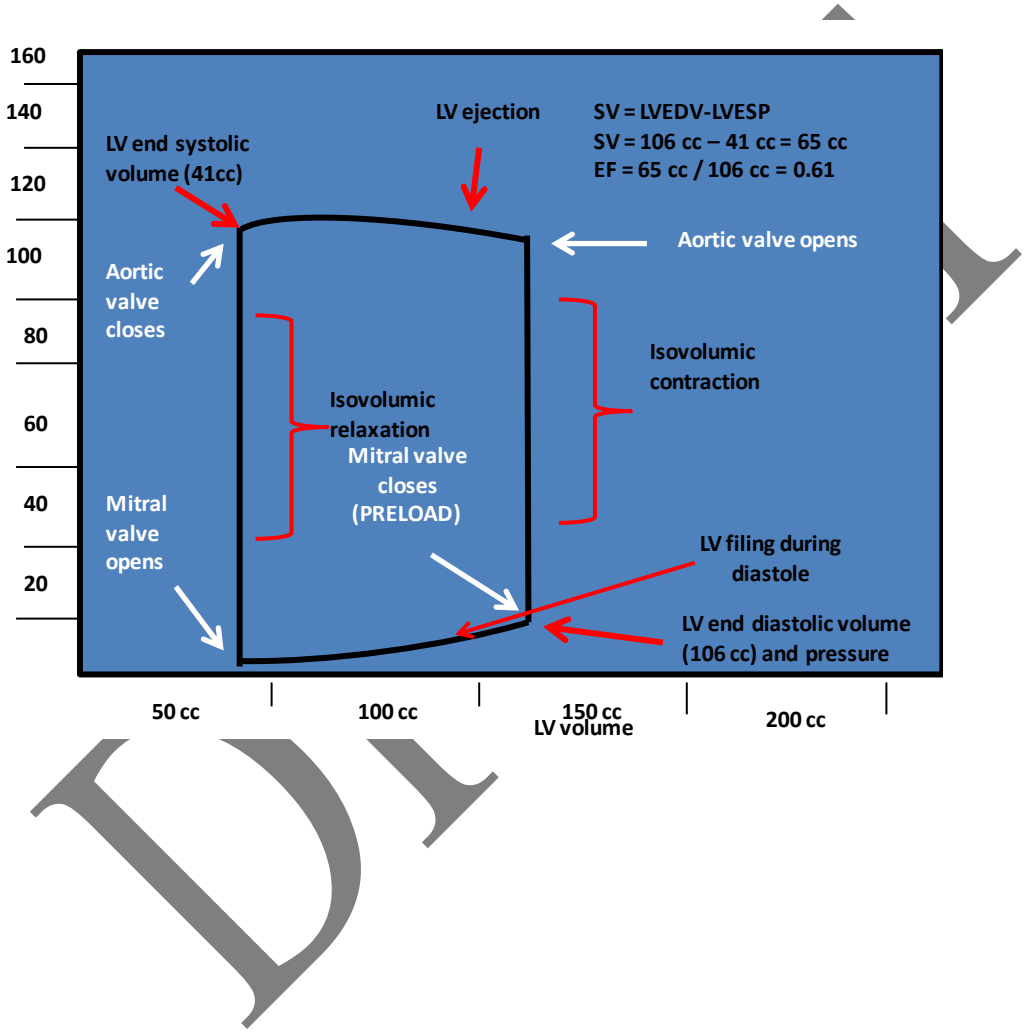


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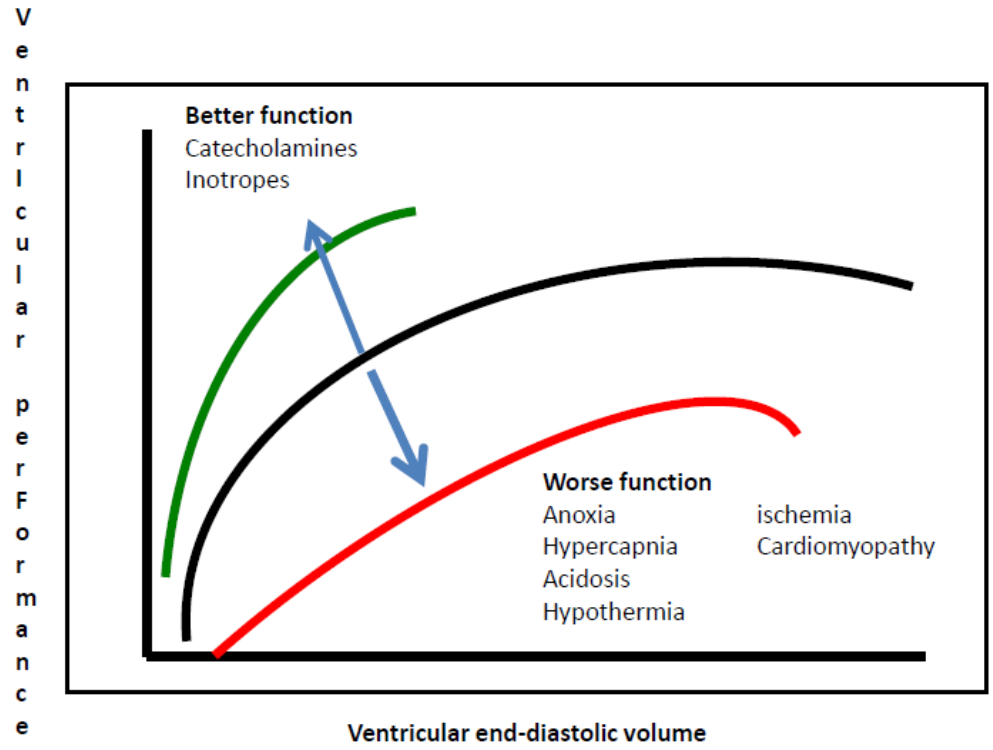
Figure XXX- Ventricular function curve



Systemic and Pulmonary vascular resistance

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References: