Hemodynamic Monitoring in the CCU

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Figure 1.1 Panel A: Simultaneous recording of left ventricular (LV) pressure and pulmonary artery (PA) pressure made in the Cardiac Catheterization Laboratory. At end diastole (arrow), the pulmonary artery pressure and the left ventricular pressure are equal (28 mmHg) in this patient with normal pulmonary vascular resistance.

Panel B: Simultaneous recording of LV pressure and wedge pressure from the same patient. The mean wedge pressure (25 mmHg), the pulmonary artery diastolic pressure (28 mmHg) and the left ventricular end diastolic pressure (28 mmHg) are approximately equal (pressure difference ≤ 5 mmHg).

Scale = 0-40 mmHg; Paper speed = 50 mm/sec.
Normal left ventricular pressures are:

- Systolic 100 to 140 mm of mercury
- End-diastolic 3 to 12 mm of mercury

Left ventricular pressure cannot be measured directly using bedside monitoring techniques. Nevertheless, it is possible to accurately estimate the left ventricle or pressure in the following way:

- The left ventricular systolic pressure equals the aortic systolic pressure in the absence of left ventricular outflow obstruction.
- The left ventricular end diastolic pressure equals the mean wedge pressure in the absence of mitral valve disease.
Left Ventricular Pressure

• The end of left ventricular diastole coincides with the onset of the electrocardiographic QRS complex.

• Measurement of the left ventricular end diastolic pressure allows the clinician to use the Frank Starling principle to access and manipulate left ventricular performance.

• Myocardial or pericardial disease significantly alters the relation between left ventricular end diastolic pressure and volume.
Left Ventricular Pressure

- As a rule, cardiac disease causes a decrease in compliance; the result is a higher filling pressure to achieve the same degree of filling volume.
- At the same time, cardiac disease diminishes the response of left ventricular performance to an increase in the end diastolic pressure.
- The left ventricular end diastolic pressure for normal hearts is 3 to 12 mm of mercury.
- With left ventricular disease (acute myocardial infarction, cardiomyopathy), the optimal filling pressure increases to 20 to 25 mm of mercury.
- The need to maintain a higher left ventricular filling pressure comes with a price since an increase in the diastolic pressure eventually leads to pulmonary congestion.
Figure 1.2 Relation between left ventricular end-diastolic pressure (LVEDP) and left ventricular end-diastolic volume (LVEDV). In normal hearts the left ventricular end-diastolic pressure is low (10-12 mmHg). Optimal left ventricular filling occurs at a hydrostatic pressure well below that associated with pulmonary congestion.

Figure 1.3 Relation between left ventricular end-diastolic pressure (LVEDP) and stroke volume (SV) in normal and abnormal hearts. In abnormal hearts, the response of stroke volume to an increase in the LVEDP is diminished. With disease, optimal filling pressures occurs at an LVEDP of 20-25 mmHg.
Figure 1.7 Normal right atrial pressure waveform. The rhythm is sinus. The mean right atrial pressure is 6 mmHg. The A wave (a) is the dominant positive pressure wave. The peak of the right atrial A wave follows the peak of the electrocardiographic P wave by about 80 msec. The right atrial C wave (c) occurs immediately following the QRS complex. The right atrial V wave (v) occurs on the downslope of the electrocardiographic T wave. The X (x) and Y (y) descents are similar in amplitude. Note that the C-wave interrupts the X descent yielding an X and X’ (x’) descent.

Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Figure 1.8 Influence of inspiration on the normal right atrial pressure waveform. The rhythm is sinus. The mean right atrial pressure (RA) is 6 mmHg at end-expiration. With inspiration, the mean right atrial pressure declines 2-3 mmHg. Note that the A and V waves and the X (x) and Y (y) descents are accentuated on inspiration. Scale = 0-25 mmHg; Paper speed = 25 mm/sec.
Figure 1.5 Normal pulmonary artery (PA) and wedge pressure waveforms. Sinus rhythm is present. The pulmonary artery pressure (left) is 31/19 mmHg. The pulse pressure is 12 mmHg. The pulmonary artery systolic wave (S) coincides with the T wave of the electrocardiogram. The dicrotic notch (N) marks pulmonic valve closure. Its crisp character signifies a high quality recording. The mean wedge pressure (right) is 15 mmHg at end-expiration. The pulmonary artery diastolic pressure (D) is within 5 mmHg of the mean wedge pressure in this patient with normal pulmonary vascular resistance. The A (a) and V (v) waves of the wedge pressure waveform are similar in amplitude. Compare the timing of the wedge pressure A and V waves here with the right atrial pressure A and V waves from the same patient (Figure 1.11). Note also that the wedge pressure V wave occurs significantly later in time than the pulmonary artery systolic wave. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
The normal wedge pressure is 2-12 mm Hg and is twice the mean right atrial pressure. \( \text{RA/Wedge} = 0.5 \)

The wedge pressure A wave follows the electrocardiographic P wave by \( \geq 200 \) msec and represents atrial systole.

The A wave magnitude is increased in such conditions as mitral stenosis and left ventricular noncompliance.
• The C wave is caused by closure of the mitral valve and marks the onset of left ventricular systole.

• The C wave is visible in the right atrial pressure recording but is often not seen in the PAOP waveform because of damping.
PAOP Waveform Review

- The V wave represents venous filling of the left atrium when left ventricular systole has closed the mitral valve.
- In some normal patients, the V wave is the dominant positive wave in the PAOP waveform.
- Left atrial volume overload from mitral regurgitation or a ventricular septal defect will magnify the V wave.
- The peak of the V wave occurs after the T wave of the ECG and is noticeably later than the pulmonary artery systolic wave. This difference in timing is important when interpreting hemodynamic data from patients with a giant V wave.
PAOP Waveform Review

• The X and Y descents follow the A and V waves respectively.
• The X descent represents left atrial relaxation combined with the sudden downward motion of the atrioventricular junction during early left ventricular systole.
• Mitral regurgitation can attenuate or obliterate the X descent.
• The Y descent is caused by the rapid exit of blood from the left atrium into the left ventricle at the moment of mitral valve opening.

• The Y descent marks the onset of left ventricular diastole. The Y descent is blunted with mitral stenosis.

• Coincident with the X and Y descents there is a surge of pulmonary venous return to the left atrium.
Clinical Use of the R Atrial Pressure Measurement

• The mean Right Atrial pressure is used clinically in the following ways:
  – To assess the adequacy of right ventricular filling volume
  – To determine the hydrostatic pressure in the systemic veins

• The mean right atrial pressure is a reliable measure of the right ventricular end diastolic pressure if significant tricuspid stenosis or regurgitation is absent.
Clinical Use of the R Atrial Pressure Measurement

• In the normal heart measurement of the right atrial pressure can be used to predict the left atrial pressure.

• In the presence of cardiac disease the right atrial pressure is a poor predictor of the left atrial pressure.

• Significant cardiac disease mandates measurement of the wedge pressure to assess the left atrial pressure and the left ventricular filling pressure.
Clinical Use of the R Atrial Pressure Measurement

• The mean **right atrial pressure** provides a measurement of the hydrostatic pressure in the **systemic veins**.

• This is an important variable in the formation of **peripheral edema**.

• Elevation of the right atrial pressure causes **visceral** congestion.

• The right atrial pressure waveform itself produces valuable clinical information.
• Conditions such as pericardial tamponade, pericardial constriction, right ventricular infarction and tricuspid regurgitation can be suspected by careful analysis of the right atrial pressure waveform.

• The right atrial pressure waveform is equally valuable in the assessment of cardiac arrhythmias.

• Finally knowledge of the right atrial pressure relative to the wedge pressure is helpful. Elevations of the right atrial pressure out of proportion to the wedge pressure points to conditions such as pulmonary embolism and right ventricular infarction. (RAP/WP > 0.5)
Pulmonary Artery Pressure

- Normal pulmonary artery pressures are:
  - Systolic 15 to 30 mm of mercury
  - Diastolic 4 to 12 mm of mercury
  - Mean 9 to 18 mm of mercury

The normal pulmonary artery pulse pressure is approximately 15 mm of mercury.

The upstroke of the pulmonary artery pressure waveform reflects the onset of right ventricular ejection.

The dicrotic notch is due to pulmonic valve closure and marks the end of right ventricular ejection.
Pulmonary Artery Pressure

• The peak of the pulmonary artery systolic pressure wave occurs within the electrocardiographic T-wave.

• Note that the peak pulmonary artery systolic pressure wave occurs earlier in time than the peak wedge pressure V wave.

Figure 1.5 Normal pulmonary artery (PA) and wedge pressure waveforms. Sinus rhythm is present. The pulmonary artery pressure (left) is 31/19 mmHg. The pulse pressure is 12 mmHg. The pulmonary artery systolic wave (S) coincides with the T wave of the electrocardiogram. The diastolic notch (N) marks pulmonic valve closure. Its crisp character signifies a high quality recording. The mean wedge pressure (right) is 15 mmHg at end-expiration. The pulmonary artery diastolic pressure (D) is within 5 mmHg of the mean wedge pressure in this patient with normal pulmonary vascular resistance. The A (a) and V (v) waves of the wedge pressure waveform are similar in amplitude. Compare the timing of the wedge pressure A and V waves here with the right atrial pressure A and V waves from the same patient (Figure 1.11). Note also that the wedge pressure V wave occurs significantly later in time than the pulmonary artery systolic wave. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Pulmonary Artery Pressure

- In patients with normal pulmonary artery vascular resistance and no mitral valve obstruction the pulmonary artery diastolic pressure is very close (2-4 mm Hg) to both the mean wedge pressure and to the left ventricular end diastolic pressure.

- When the pulmonary artery diastolic pressure **exceeds** the mean wedge pressure by \( \geq 5 \) mm of mercury, conditions known to increase pulmonary vascular resistance (for example pulmonary embolism) should be considered.
Pulmonary Artery Pressure

• The pulmonary artery diastolic pressure does not correlate well with the mean wedge pressure in the following situations:
  – Abnormal pulmonary vascular bed. The pulmonary artery diastolic pressure overestimates the mean wedge pressure.
  – Mitral regurgitation with a large V wave. The pulmonary artery diastolic pressure underestimates the mean wedge pressure.
Pressure Waveform Analysis

• The following steps are recommended for proper pressure data and analysis:
  – Check that the pressure transducer has been properly zeroed to the estimated level of the heart.
  – Check the dynamic pressure response of the system using the fast flush test, alternatively a crisp dicrotic notch on the pulmonary artery tracing indicates a properly responsive system.
  – Choose the pressure scale which best accommodates the intracardiac pressure being monitored.
Pressure Waveform Analysis

- Choose an electrocardiographic lead which best illustrates atrial activity.
- Record the single lead electrocardiogram together with the pressure waveform at a paper speed of 25 mm per second.
- Include two to four respiratory cycles and measure the intracardiac pressure at end expiration.
- Identify the A wave and the V wave in the right atrial and the wedge pressure waveforms by drawing a vertical line from the positive pressure waves to the electrocardiogram.
Pressure Waveform Analysis

• Identify the X descent and the Y descent.
• Assess the effect of spontaneous inspiration on the mean right atrial pressure.
• If indicated, perform the hepatojugular reflux test while recording the right atrial pressure.
• Identify the systolic pressure and the diastolic pressure in the pulmonary artery and the aortic pressure waveforms and measure the respective pulse pressures; identify the dicrotic notch on each arterial pressure waveform.
Pressure Waveform Analysis

• Measure the pressure gradient between the pulmonary artery diastolic pressure and the mean wedge pressure. This should be $\leq 5$ mm Hg.

• Measure the ratio of the mean right atrial pressure/mean wedge pressure. Normally this is approximately 0.5.
• The mechanical action of the heart is governed by the cardiac rhythm. An arrhythmia will therefore have an immediate impact on hemodynamic parameters. When analyzing this effect, it is important to consider the following:

• What is the arrhythmia rate?
• What is the effect of the arrhythmia on coordinated atrial ventricular contraction (A-V synchrony)?
• Has the arrhythmia compromised the efficiency of atrial or ventricular systole?
Sinus Tachycardia

• With an increase in the heart rate, diastole progressively shortens.
• As a consequence, the A wave initiating a cardiac cycle begins to encroach on the V wave of the preceding cycle.

Figure 4.1 (Panels A-C)
Right atrial pressure waveforms from a patient at 3 different heart rates. The PR interval is normal.
Panel A: Sinus rhythm at 85 beats/min. Normal A(a) and V (v) waves with X (x) and Y descents (y) present.
Panel B: Sinus rhythm at 102 beats/min. The A wave (a) begins to blend with the V wave (v) of the preceding cardiac cycle. As a result, the Y descent (y) is attenuated. This should not be confused with the absent Y descent of pericardial tamponade.
Panel C: Sinus rhythm at 123 beats/min. The A wave (a) has now fused with the V wave (v) of the preceding cardiac cycle. The Y descent is absent.
HR = heart rate;
Scale = 0-30 mmHg;
Paper speed = 25 mm/sec.
Sinus Tachycardia

• Eventually the two waves summate to generate a single wave and the Y descent is obliterated.

• It is important to remember the influence of a heart rate on the Y descent because pericardial tamponade also causes disappearance of the Y descent.

• First-degree AV block can cause the A and V ways to summate in the same way as does sinus tachycardia. Therefore both the heart rate and the PR interval must be considered when evaluating the atrial pressure waveforms.
Sinus Bradycardia

• As diastole lengthens during sinus bradycardia, the time interval lengthens between the V wave of one cardiac cycle and the A wave of the next cycle.

• The Y descent is easily seen. Often an additional positive wave (the H wave) is present after the Y descent when the heart rate is less than 60 beats/min.

• This wave is most prominent in the right atrial pressure waveform especially when the right atrial pressure is elevated.

• The origin of the H wave is unclear and is not associated with any mechanical cardiac event.
Figure 4.2 Right atrial pressure waveform from a patient with sinus bradycardia at 49 beats/min. An H wave (h) is present due to the long diastole. The A-C interval is slightly prolonged due to first degree AV block. The right atrial pressure is elevated (21 mmHg) in this patient with inferior/right ventricular infarction. The H wave is accentuated when the right atrial pressure is elevated. HR = heart rate; Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Atrial Fibrillation

• The hallmarks of atrial fibrillation are disappearance of the atrial systole and variation in the length of the diastole.
• The A wave disappears from the atrial pressure waveform and is sometimes replaced by atrial fibrillation waves.
• The fibrillation waves are most evident during a long R-R interval.
• These waves are sometimes visible in the jugular veins and can produce enough mechanical activity to move the mitral and tricuspid valves.
Atrial Fibrillation

• The fibrillation waves are associated with coarse atrial fibrillation on the electrocardiogram.

• The C and V waves are dominant features of the atrial pressure waveform.

• The C and V waves are separated by the X descent. The X descent is usually shallower than the Y descent.
Figure 4.3 (Panels A & B) Right atrial pressure waveforms from a patient during atrial fibrillation and after spontaneous conversion to sinus rhythm.

Panel A: During atrial fibrillation, the C (c) and V waves (v) are dominant due to loss of the A wave. Fibrillation waves can be seen during the diastole accompanying a long R-R interval. The Y descent (y) is steeper than the X descent (x) as is typical of atrial fibrillation. The right atrial pressure is elevated (10 mmHg) in this patient with inferior/right ventricular infarction.

Panel B: Right atrial pressure waveform from the same patient after conversion to sinus rhythm. The A wave (a) has returned.

Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Atrial Fibrillation

• Many patients with atrial fibrillation have coexisting myocardial or pericardial disease and the atrial pressure waveform may also be influenced by these pathological conditions.

• During atrial fibrillation, the ventricular stroke volume varies directly with the electrocardiographic R-R interval. As a result, the pulse pressure in the aorta and the pulmonary artery will be greatest following a long R-R interval.
Figure 4.4  Systemic arterial pressure waveform during atrial fibrillation. The arterial pulse pressure varies directly with the R-R interval. Following long R-R intervals, the ventricular stroke volume and therefore the arterial pulse pressure increase (arrow). Scale = 0-120 mmHg; Paper speed = 25 mm/sec.
Atrial Flutter

- As with atrial fibrillation, the A wave of the atrial pressure waveform is absent.
- During atrial flutter, the atria continue to contract at a rate of approximately 300 beats per minute.
- This mechanical atrial activity generates flutter waves in the atrial pressure waveform.
- This regular mechanical activity may partly explain why the systemic embolization rate during atrial flutter is lower than during atrial fibrillation.
Atrial Flutter

• In the presence of 2:1 AV block, every other flutter wave often occurs coincident with ventricular systole.

• The flutter waves occurring during ventricular systole maybe slightly enhanced because the right atrium is contracting against a closed tricuspid valve.
**Atrial Flutter**

**Figure 4.5** Right atrial pressure waveform from a patient with atrial flutter demonstrates typical mechanical flutter waves (f). The atrial rate is somewhat less than 300 beats/min because of treatment with a type IA antiarrhythmic drug. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.

**Figure 4.6** Right atrial pressure waveform from a patient with atrial flutter and 2:1 AV conduction. At left, alternate flutter waves are exaggerated (arrows) because they occur during ventricular systole. At right, an adenosine bolus transiently induces complete heart block. All flutter waves (f) are now monomorphic because of absent ventricular systole. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Premature Ventricular Contractions

• A premature ventricular contraction sets the stage for a mechanical cannon wave (Cannon A wave).

• Cannon waves are the result of an atrial systole occurring when ventricular systole has already closed the mitral and tricuspid valves.

• That is, atrial and ventricular systole are either simultaneous or reversed from their normal timing sequence.
Premature Ventricular Contractions

• The Cannon wave causes a transient reversal in the normal systemic and pulmonary venous return.

• The ventricles are not properly filled at the onset of systole.

• Isolated premature ventricular contractions rarely disturb overall cardiac function.

• A Cannon wave in the atrial pressure waveform is a helpful marker that the normal sequence of atrial and ventricular systole has been disturbed.

• Cannon waves can be seen with a variety of arrhythmias.
Figure 4.7 Right atrial pressure waveform during sinus rhythm with a single premature ventricular contraction (PVC). The sinus P wave (p) is clearly visible in the ST segment of the PVC. The normal sequence of atrial-ventricular systole is reversed and a cannon wave results.
Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
AV Junctional (Nodal) Rhythm

• During a nodal rhythm, atrial systole can either precede or follow ventricular systole.
• AV dissociation may also occur.
• When the sequence of atrial and ventricular systole is reversed, Cannon waves will be present on the atrial pressure waveform.

Figure 4.8 (Panels A & B) Right atrial pressure waveforms from a patient during nodal rhythm.
Panel A: The sinus rate and the nodal rate are nearly the same (isorhythmic dissociation). The P wave precedes the QRS complex and the normal sequence of atrial-ventricular systole is maintained. A normal right atrial A wave (a) is present.
Panel B: The P wave now occurs within the QRS complex. Atrial and ventricular systole are nearly simultaneous which generates cannon waves (arrows) in the right atrial pressure tracing. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
AV Nodal Reentrant Tachycardia

- Reentry within the AV node is one of the most common causes of paroxysmal supraventricular tachycardia.
- Each time the electrical impulse travels the reentrant loop, there is retrograde activation of the atria and antegrade activation of the ventricles.
- In the majority of patients with this arrhythmia, the retrograde P wave occurs either within or after the QRS complex.
- When ventricular systole is coincident with atrial systole, the A and V waves fuse and Cannon waves occur. The Cannon waves are regular because there is 1:1 AV association.
AV Nodal Reentrant Tachycardia

Panel A: Reentrant supraventricular tachycardia at a rate of 150 beats/min. Retrograde P waves (p) are visible at the end of the QRS complex. The normal sequence of atrial-ventricular systole is reversed which generates regular cannon waves (v/a) in the right atrial pressure tracing. The mean right atrial pressure is elevated at 19 mmHg.

Panel B: Intravenous adenosine restores sinus rhythm. Normal A (a), C (c), and V waves (v) are now present. The mean right atrial pressure has abruptly dropped to 13 mmHg.

HR = heart rate; Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
AV Nodal Reentrant Tachycardia

- The Cannon waves also abruptly elevate the right atrial mean pressure. This abrupt increase in right atrial pressure can trigger the release of atrial natriuretic factor and may be responsible for polyuria in some of these patients.
- The forward stroke volume, aortic systolic blood pressure, and aortic pulse pressure are often reduced during this tachycardia because of the shortened diastole coupled with the loss of the normal atrial contribution to ventricular filling.
- In some patients, Cannon waves may trigger a vasodepressor reflex further aggravating the fall in blood pressure.
AV Nodal Reentrant Tachycardia

Figure 4.10 Graphic presentation of the change in heart rate (HR) and mean central venous pressure (CVP M) that occur with the onset of reentrant supraventricular tachycardia. At baseline, the heart rate is 76 beats/min and the mean central venous pressure is 10 mmHg. During tachycardia, the heart rate is 165 beats/min and the mean central venous pressure is 23 mmHg.

Figure 4.11 Graphic representation of the change in heart rate (HR) and arterial blood pressure (ABP) systolic (S), mean (M), diastolic (D) after conversion of reentrant supraventricular tachycardia to sinus rhythm. The systolic blood pressure rises 39 mmHg and the pulse pressure rises 36 mmHg due to an increase in the stroke volume with restoration of sinus rhythm.
Automatic Atrial Tachycardia

- This arrhythmia is due to enhanced atrial automaticity. The atrial rate is usually less than 200 beats per minute and generates rapid regular A waves in the atrial pressure waveform.

- It is common to observe 2:1 nodal block.

- In this circumstance, the blocked P wave usually occurs within the QRS-T interval.

- The A wave of the blocked P wave sums with the V wave of the QRS complex creating a single larger wave. This “summation” wave does not have the appearance of a typical cannon wave perhaps because it occurs at the very end of ventricular systole near the time when tricuspid and mitral valves opening occur.
Automatic Atrial Tachycardia

Figure 4.12 Panel A: Right atrial pressure waveform and lead II electrocardiogram resemble sinus rhythm. However, the right atrial V wave (V) is noticeably higher than the A wave (A).

Panel B: Administration of adenosine reveals that the rhythm is actually an automatic atrial tachycardia at a rate of about 200 beats/min. The right atrial pressure waveform in Panel A is actually distorted. The V waves seen in Panel A are being augmented by the A waves of the atrial tachycardia.

RA = right atrium;
Scale = 0-30 mmHg;
Paper speed = 25 mm/sec.
Ventricular Tachycardia

- Ventricular tachycardia arises within the ventricles.
- Atrial activation occurs either by coexisting sinus rhythm (AV dissociation) or by retrograde VA conduction to the atrial (VA association).
- The type of atrial electrical activation has an important influence on the hemodynamic consequences of ventricular tachycardia.
Ventricular Tachycardia

• With AV dissociation, the relation between atrial and ventricular systole is random. On some cycles, ventricular systole precedes atrial systole and Cannon waves occur in the atrial pressure waveform.

• The beats generate a reduced stroke volume and therefore a reduced aortic pulse pressure because of absent atrial filling of the ventricles.
Figure 4.13 Right atrial (RA) and aortic (AO) pressure waveforms from a patient with ventricular tachycardia and AV dissociation. Irregular cannon waves due to a reversed sequence of atrial-ventricular contraction are present in the right atrial tracing. Because of poor ventricular filling, these beats produce a lower stroke volume and therefore a lower pulse pressure (arrows). Occasionally, the P waves (p) precede the QRS complex and the normal sequence of atrial-ventricular systole occurs. Cannon waves are absent on these beats and the aortic pulse pressure is wider because of improved stroke volume. HR = heart rate; Scale = 0-180 mmHg (AO) and 0-30 mmHg (RA); Paper speed = 25 mm/sec.

Figure 4.14 Arterial (ABP) and pulmonary artery (PAP) pressure waveforms from a patient with ventricular tachycardia and AV dissociation. Spontaneous conversion to sinus rhythm occurred during the recording. During ventricular tachycardia the arterial pulse pressure is reduced reflecting a low stroke volume. In addition, there is variation in the arterial pulse pressure due to AV dissociation. With conversion to sinus rhythm, the arterial pulse pressure immediately increases due to an improved stroke volume. V. tachycardia = ventricular tachycardia; Scale = 0-180 mmHg (ABP) and 0-30 mmHg (PAP); Paper speed = 25 mm/sec.
Ventricular Tachycardia

• On other cycles, atrial systole precedes ventricular systole (mimicking normal physiology) and Cannon waves are absent on the atrial pressure waveform.

• These beats generate an improved stroke volume and therefore a higher aortic pulse pressure because each atrial systole augments ventricular filling.

• Physical examination of these patients reveals irregular cannon waves in the jugular venous pulse as well as a variable carotid artery pulse volume despite a regular cardiac rhythm.
Ventricular Tachycardia

• With 1:1 VA conduction during ventricular tachycardia, the normal sequence of atrial and ventricular contraction is reversed on every cycle.

• Regular Cannon waves appear in the atrial pressure waveform and the aortic pulse pressure remains constant from beat to beat.

• In these patients regular Cannon waves are present in the jugular venous pulse and the carotid artery pulse volume is constant.
Figure 4.15  Right atrial pressure waveform from a patient with ventricular tachycardia and 1:1 VA conduction. Regular cannon waves (v/a) are present on the right atrial pressure tracing.

HR = heart rate; Scale = 0-25 mmHg; Paper speed = 25 mm/sec.
• Acute mitral valve regurgitation is a catastrophic event occurring as a result of ruptured chordae tendinae, ruptured papillary muscle, or bacterial destruction of the mitral valve.

• The severity and time course of the valvular insufficiency both have a major impact on the hemodynamic consequences of acute mitral regurgitation.

• Chronic mitral regurgitation maybe severe with little or no change in the bedside hemodynamic measurements and will not be discussed.
Acute Mitral Regurgitation and the V Wave

- Wedge pressure and pulmonary artery pressure.
  - With acute mitral valve regurgitation, the left ventricle ejects blood into the left atrium during systole.
  - The left atrium is subjected to an acute volume overload because the high pressure regurgitant volume is added to the normal pulmonary venous return.
  - When the left ventricle is ejecting blood into a normal sized and relatively unyielding left atrium, the wedge pressure (left atrial pressure) rises dramatically during ventricular systole.
Acute Mitral Regurgitation and the V Wave

• Wedge pressure and pulmonary artery pressure.
  - Mitral regurgitation begins with the onset of ventricular systole (marked by the C wave in the PAOP waveform) and continues until the end of systole (marked by the peak of the V wave in the PAOP waveform).
  - The hallmark of acute mitral regurgitation is a giant “C-V” wave in the wedge pressure tracing.
  - The X’ descent which normally separates the C wave from the V wave disappears or is attenuated.
  - This “C-V” wave is therefore commonly referred to as simply the V wave.
  - The large V wave causes a striking increase in the mean wedge pressure. The mean wedge pressure frequently exceeds 25 to 30 mm of mercury resulting in acute pulmonary edema.
Acute Mitral Regurgitation and the V Wave

![Wedge pressure and pulmonary artery pressure waveforms](image)

**Figure 5.1** Wedge pressure and pulmonary artery pressure waveforms from a patient with acute severe mitral regurgitation. The wedge pressure waveform is shown first and is dominated by a large V wave (v). The mean wedge pressure is 54 mmHg and the V wave peak is 95 mmHg. The giant V wave causes the wedge pressure waveform to resemble that of an artery. The balloon is deflated (**large arrow**) to reveal the pulmonary artery (**PA**) pressure waveform. Severe pulmonary hypertension (91/34 mmHg) is present. The pulmonary artery systolic waveform is bifid and composed of the pulmonary artery systolic wave (s) followed shortly by the retrogradely transmitted giant V wave. The V wave in this patient is higher than the pulmonary artery systolic wave. This retrograde V wave should not be confused with the dicrotic wave of pulmonic valve closure. The timing of the wedge pressure V wave is identical to that of the pulmonary artery V wave. Note also that the pulmonary artery diastolic pressure (34 mmHg) underestimates the mean wedge pressure (54 mmHg). The best method to estimate the left ventricular filling pressure in this patient would be to measure the wedge pressure at the end of the A wave (40 mmHg).

Scale = 0-120 mmHg; Paper speed = 25 mm/sec.
Acute Mitral Regurgitation and the V Wave

- The giant V wave of acute mitral regurgitation may be transmitted retrogradely into the pulmonary artery. This yields a biphasic pulmonary artery systolic waveform composed of the pulmonary artery systolic wave followed shortly by the V wave.

- As the catheter moves from the pulmonary artery position into the wedge position, the pulmonary artery systolic wave disappears and only the V wave remains.
Acute Mitral Regurgitation and the V Wave

- The wedge pressure V plays may be so striking as to resemble the pulmonary artery systolic pressure waveform and the operator may not realize that the catheter has moved from the pulmonary artery into the wedge position.
- This problem can be avoided by carefully examining the pulmonary artery pressure waveform and its relation to the electrocardiogram.
- The timing of the peak pulmonary artery systolic way and the peak V wave are significantly different.
- The pulmonary artery systolic wave occurs at the peak of the electrocardiographic T-wave; the V wave occurs after the T-wave.
- The transient reversal of pulmonary blood flow that accompanies the giant V wave can result in highly oxygenated blood entering the main pulmonary artery resulting in the mistaken diagnosis of a left to right shunt.
Acute Mitral Regurgitation and the V Wave

Figure 5.1 Wedge pressure and pulmonary artery pressure waveforms from a patient with acute severe mitral regurgitation. The wedge pressure waveform is shown first and is dominated by a large V wave (V). The mean wedge pressure is 54 mmHg and the V wave peak is 95 mmHg. The giant V wave causes the wedge pressure waveform to resemble that of an artery. The balloon is deflated (large arrow) to reveal the pulmonary artery (PA) pressure waveform. Severe pulmonary hypertension (91/34 mmHg) is present. The pulmonary artery systolic waveform is bifid and composed of the pulmonary artery systolic wave (S) followed shortly by the retrogradely transmitted giant V wave. The V wave in this patient is higher than the pulmonary artery systolic wave. This retrograde V wave should not be confused with the dicrotic wave of pulmonic valve closure. The timing of the wedge pressure V wave is identical to that of the pulmonary artery V wave. Note also that the pulmonary artery diastolic pressure (34 mmHg) underestimates the mean wedge pressure (54 mmHg). The best method to estimate the left ventricular filling pressure in this patient would be to measure the wedge pressure at the end of the A wave (40 mmHg). Scale = 0-120 mmHg; Paper speed = 25 mm/sec.
Acute Mitral Regurgitation and the V Wave

- Cardiac output and aortic pressure:
- The cardiac output is decreased and shock is frequently present.
- The left ventricular forward stroke volume is decreased.
- Sinus tachycardia compensates to some degree for the decreased forward stroke volume.
- The total left ventricular stroke volume may be normal.
- The aortic systolic pressure is usually low.
- The aortic pulse pressure is usually narrow reflecting a decreased left ventricular forward stroke volume.
Acute Mitral Regurgitation and the V Wave

- Cardiac output and aortic pressure:
- The thermodilution cardiac output method measures the pulmonary blood flow which is the same as the forward flow across the aortic valve.
- The thermodilution method therefore ignores the volume of blood ejected into the left atrium.
- This cannot be measured at the bedside with hemodynamic techniques.
General comments on the V wave

• The V wave is a normal finding on the wedge pressure tracing and is often higher than the A wave.
• Therefore the definition of a “large” V wave is subjective.
• Furthermore, a large V wave commonly occurs in conditions other than acute mitral regurgitation.
• They are often observed with left ventricular failure from any cause (i.e., dilated cardiomyopathy, ischemic cardiomyopathy).
• These prominent V waves may occur in the absence of significant mitral regurgitation and are usually a marker for a distended and noncompliant left atrium.
General comments on the V wave

Figure 5.2 Wedge pressure waveform from a patient with dilated cardiomyopathy. The mean wedge pressure is elevated at 20 mmHg. A large V wave (v) is present and measures 32 mmHg. Doppler echocardiography revealed only mild mitral regurgitation. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
General comments on the V wave

• An acute ventricular septal defect (complicating myocardial infarction) can cause a large V wave because of the increased pulmonary blood flow and increased pulmonary venous return to the left atrium.

• It should be apparent that a large V wave in the wedge pressure waveform must be interpreted carefully and in the context of the patient’s clinical status.

• Mitral regurgitation is often a dynamic event and the magnitude of the V wave may therefore vary considerably over time.

• This is especially true during episodes of acute myocardial infarction.
General comments on the V wave

- The degree of mitral regurgitation is sensitive to left ventricular afterload. Afterload reduction with nitroglycerin or nitroprusside can significantly reduce the amount of mitral regurgitation and the size of the wedge pressure V wave.

Figure 5.3 (Panels A & B): Pulmonary artery and wedge pressure waveforms from a patient with acute severe mitral regurgitation before and after treatment with intravenous nitroglycerin.

Panel A: Before nitroglycerin, severe pulmonary hypertension (95/41 mmHg) is present. The pulmonary artery (PA) systolic waveform is blunted and composed of the pulmonary artery systolic wave (S) and the retrograde V wave (V). The retrograde V wave is slightly less than the pulmonary artery systolic wave. The balloon is inflated (large arrow) to reveal the wedge pressure waveform. The wedge pressure waveform is dominated by the large V wave. The timing of the wedge pressure V wave is identical to that of the pulmonary artery V wave. The mean wedge pressure is 57 mmHg while the V wave peak is 80 mmHg. The pulmonary artery diastolic pressure (41 mmHg) underestimates the mean wedge pressure (57 mmHg).

Panel B: Minutes after treatment with intravenous nitroglycerin, the pulmonary artery pressure has dropped to 47/23 mmHg and the mean wedge pressure has dropped to 24 mmHg. The large V wave has disappeared from both the pulmonary artery and the wedge pressure waveforms. A normal diastolic notch (N) and diastolic wave are visible in the pulmonary artery pressure waveform. The pulmonary artery diastolic pressure (23 mmHg) now correlates closely with the mean wedge pressure (24 mmHg). The heart rate has decreased from 96 to 84 beats/min. Scale = 0-60 mmHg; Paper speed = 25 mm/sec.
General comments on the V wave

• A large V wave disrupts the normal close correlation between the pulmonary artery diastolic pressure and the mean wedge pressure.

• The pulmonary artery diastolic pressure is a measurement made in a single point in time (end diastole), while the wedge pressure is a mean pressure recorded over the entire cardiac cycle.

• The peaks and valleys of a normal wedge pressure waveform are minor, therefore the pulmonary artery diastolic pressure usually correlates closely with the mean wedge pressure.
General comments on the V wave

• A large V wave distorts the wedge pressure waveform so that the pulmonary artery diastolic pressure now overestimates the mean wedge pressure.

• Consequently, the pulmonary artery diastolic pressure cannot be used as an estimate of the mean wedge pressure in the presence of a large V wave.

• As a corollary to this, a large V leave causes the mean wedge pressure to overestimate the left ventricular end diastolic pressure.

• For the best estimate of the left ventricular end diastolic filling pressure in the presence of a large V wave, measure the wedge pressure at a single time point (end diastole).
General comments on the V wave

• The end of the wedge pressure A wave (post A wave pressure) coincides with the end of left ventricular diastole.

• In the presence of a large V wave, measurement of the post A wave wedge pressure allows a reliable estimate of the left ventricular filling pressure.

Figure 5.1 Wedge pressure and pulmonary artery pressure waveforms from a patient with acute severe mitral regurgitation. The wedge pressure waveform is shown first and is dominated by a large V wave (v). The mean wedge pressure is 54 mmHg and the V wave peak is 95 mmHg. The giant V wave causes the wedge pressure waveform to resemble that of an artery. The balloon is deflated (large arrow) to reveal the pulmonary artery (PA) pressure waveform. Severe pulmonary hypertension (91/34 mmHg) is present. The pulmonary artery systolic waveform is bifid and composed of the pulmonary artery systolic wave (s) followed shortly by the retrograde transmitted giant V wave. The V wave in this patient is higher than the pulmonary artery systolic wave. This retrograde V wave should not be confused with the dicrotic wave of pulmonic valve closure. The timing of the wedge pressure V wave is identical to that of the pulmonary artery V wave. Note also that the pulmonary artery diastolic pressure (34 mmHg) under-estimates the mean wedge pressure (54 mmHg). The best method to estimate the left ventricular filling pressure in this patient would be to measure the wedge pressure at the end of the A wave (40 mmHg). Scale = 0-120 mmHg; Paper speed = 25 mm/sec.
General comments on the V wave

• For clinical purposes, the mean wedge pressure reflects the hydrostatic force in the pulmonary capillary bed.

• A large V wave will raise the mean wedge pressure and promote pulmonary edema formation.

• If the patient’s primary problem is respiratory failure due to pulmonary congestion, then the effort should be directed at lowering the mean wedge pressure.

• On the other hand, if the patient’s primary problem is a low cardiac output, attention should be directed at maintaining an adequate left ventricular filling pressure (post A wave pressure in the wedge waveform).
Tricuspid Regurgitation

- Tricuspid regurgitation is a chronic condition caused by a right ventricular failure and dilatation.
- The right ventricular failure can often be traced to long-standing pulmonary artery hypertension.
- Tricuspid regurgitation changes the right atrial pressure waveform, raises the right atrial mean pressure, and may invalidate the thermodilution method of measuring cardiac output.
- Furthermore, advancing the balloon tipped catheter from the right atrium into the right ventricle is often challenging in these patients because of the regurgitant jet of blood.
Tricuspid Regurgitation – R Atrial Pressure

• The classic pressure waveform of tricuspid regurgitation is a large broad C-V wave followed by a steep Y descent.
• The tricuspid valve begins to leak with the onset of right ventricular systole.
• The onset of right ventricular systole is marked by the C wave in the right atrial pressure waveform.
• As the tricuspid regurgitation progresses during ventricular systole the right atrial pressure progressively rises.
Tricuspid Regurgitation – R Atrial Pressure

• The X’ descent is therefore attenuated or obliterated. The result is a fusion of the C and V ways into a single broad positive wave (the so called C-V wave).

Figure 6.1 Right atrial pressure waveform from a patient with severe tricuspid regurgitation after long standing pulmonary hypertension. The rhythm is sinus. The mean right atrial pressure is elevated at 22 mmHg. The A wave (a) and the X descent (x) are normal. The X’ descent is obliterated by the tricuspid regurgitation. The result is a broad positive C-V wave (cv) that is higher than the A wave. Note that inspiration (INS) magnifies the peak of the C-V wave and the nadir of the Y descent (y). As a result, the mean right atrial pressure shows little respiratory variation. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Tricuspid Regurgitation – R Atrial Pressure

• As the degree of tricuspid regurgitation increases, the right atrial C-V wave becomes more accentuated.

• The C-V wave of tricuspid regurgitation is never as striking as the C-V wave of acute mitral regurgitation because tricuspid regurgitation is a chronic condition that develops gradually.

• Furthermore, the left ventricle usually generates a much higher pressure than the right ventricle.
Tricuspid Regurgitation – R Atrial Pressure

• The Y descent is the dominant feature of the right atrial pressure waveform with significant tricuspid regurgitation.
• The Y descent is exaggerated because the high pressure within the right atrium is suddenly relieved as the tricuspid valve opens and the right atrial blood volume is delivered to the right ventricle at the beginning of diastole.
• During inspiration the C-V wave is augmented and the Y descent becomes more pronounced.
• As a result, the mean right atrial pressure remains constant or may even rise (Kussmaul’s sign).
Tricuspid Regurgitation – R Atrial Pressure

• The right atrial pressure waveform of tricuspid regurgitation will be modified by the **size** and **dispensability** of the right atrium.

• When the right atrium is very dilated and compliant, the characteristic C-V wave and steep Y descent may be attenuated or even absent despite severe tricuspid regurgitation.

*Figure 6.2* Right atrial pressure waveform from a patient with severe tricuspid regurgitation from chronic pulmonary hypertension due to cirrhosis of the liver. The mean right atrial pressure is minimally elevated at 6 mmHg. The A wave (a) is the dominant feature of the tracing. The C wave (c) is not visible. The diagnosis of tricuspid regurgitation cannot be made from this pressure tracing. Two-dimensional echocardiography revealed severe tricuspid regurgitation and marked right atrial enlargement. The thermodilution cardiac output curve was typical of severe tricuspid regurgitation. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Tricuspid Regurgitation – R Atrial Pressure

- In this setting, the characteristic thermodilution cardiac output curve may provide a helpful clue to the presence of significant tricuspid regurgitation.
- Doppler echocardiography is a particularly useful way to evaluate the severity of tricuspid regurgitation.
- With tricuspid regurgitation the mean right atrial pressure is elevated. In addition the ratio of right atrial/wedge pressure is increased. (RA/W > 0.5)
- The right atrial pressure may equal or exceed the wedge pressure, especially when the tricuspid regurgitation occurs in the absence of left heart disease.
- When the right atrial pressure exceeds the wedge pressure, right to left shunting or paradoxical embolization can occur through a patent foramen ovale.
Tricuspid Regurgitation – Cardiac Output

• Significant tricuspid regurgitation invalidates the thermodilution method because a portion of the indicator (cold) warms during its prolonged stay within the right atrium and right ventricle.

• Significant tricuspid regurgitation produces an easily identifiable thermodilution curve characterized by very slow decay to baseline temperature. The computer will measure the area under this curve and generate a “cardiac output” number. This measurement is unreliable and should be discarded.

Figure 3.3 Thermodilution cardiac output curves from a patient with severe tricuspid regurgitation before (top) and after (bottom) tricuspid valve annuloplasty. At top, the upstroke of the curve is abnormally slow and the decay is prolonged due to the tricuspid regurgitation. The computer generated cardiac output would not be accurate. After repair of the tricuspid valve, the curve is normal. Paper speed = 5 mm/sec. Calibration artifact = -0.5 °C.
Tricuspid Regurgitation – Pulmonary Artery Pressure

• Pulmonary artery hypertension is the rule and may be severe.

• An important exception to this rule can be observed with a right ventricular infarction where right ventricular dilatation is caused by ischemic injury and not pulmonary hypertension.

• When present, pulmonary hypertension may be caused by either left heart disease or primary pulmonary hypertension.

• The wedge pressure may be normal or elevated depending on whether left heart disease is present.
Acute Left Ventricular Infarction

• The hemodynamic consequences of an acute myocardial infarction encompass the entire spectrum.
• The size and location of the infarction, the mitral valve function, the heart rate and rhythm, and the pre-existing left ventricular function are all variables which influence the hemodynamic measurements.
• Right ventricular infarction complicating an inferior left ventricle or infarction is associated with unique hemodynamic findings.
• The hemodynamic abnormalities of acute Left ventricular infarction are confined largely to the wedge pressure, the cardiac index, and the arterial blood pressure.
Acute Left Ventricular Infarction

• The hallmark of acute infarction is a sudden loss of regional myocardial systolic and diastolic dysfunction. This regional contractile dysfunction is compensated by enhanced contraction of available normal myocardium.

• In the 1970s, investigators reported the relation between infarct size and parameters of left ventricular function.
Acute Left Ventricular Infarction

- Abnormal left ventricular compliance can be measured with an infarction involving only 8% of the left ventricle.
- When the infarction exceeds 10% of the left ventricle, the ejection fraction is reduced;
- With a 15% infarction, the left ventricular end diastolic pressure is increased.
- When the infarct exceeds 25% of the left ventricle, clinically evident congestive heart failure occurs.
- Cardiogenic shock, the most extreme form of heart failure, appears when acute infarction involves 40% or more of the left ventricle.
Acute Left Ventricular Infarction

• Hemodynamic consequences of an acute left ventricular infarction are confined mainly to a variable increase in the left ventricular end diastolic pressure and a variable decrease in the stroke volume.

• Acute infarction alters left ventricular compliance causing a shift in the Frank Starling relationship.

• Therefore patients with acute myocardial infarction will often require a higher than normal left ventricular end diastolic pressure to achieve optimal stroke volume and cardiac output.

• In patients with acute infarction, optimal left ventricular stroke volume occurs with a left ventricular and diastolic pressure of 20 to 25 mm Hg.
Acute Left Ventricular Infarction

- The normal close correlation between the mean wedge pressure and the left ventricular end diastolic pressure is disrupted by an acute myocardial infarction.
- In normal hearts, left atrial systole raises the left ventricular diastolic pressure by only 1 to 2 mm Hg. With acute infarction, left atrial contraction augments the left ventricular diastolic pressures by an average of 8 mm Hg.
- The several fold increase in the A wave is caused by reduced left ventricular compliance.
- The mean wedge pressure significantly underestimates the left ventricular end-diastolic pressure (on average by 8-10 mm Hg) because of the large A wave. This fact explains the important observation that the optimal mean wedge pressure for patients with an acute MI is 14-18 mm Hg which corresponds to a LVEDP of 20-25 mm Hg.
Figure 7.1 Noncompliant left ventricle disrupting the relationship of mean wedge pressure to left ventricular end diastolic pressure (LVEDP). Recordings made in the Catheterization Lab from a patient with acute myocardial infarction. The LVEDP is markedly elevated at 34 mmHg. The mean wedge pressure (PCW) of 20 mmHg seriously underestimates the LVEDP because of a very large A wave. The A wave amplitude is nearly 16 mmHg. Scale = 0-40 mmHg; Paper speed = 50 mm/sec.

Figure 7.2 Optimal wedge pressure in acute myocardial infarction. Recording of right atrial pressure (RA), wedge pressure (PCW), and cardiac index (CI) from a patient with acute anterior myocardial infarction and clinical low output state. Panel A: At baseline (left), the cardiac index is significantly reduced at 1.9 L/min/m² with normal right atrial and wedge pressures. Panel B: (right) after 500 mL saline bolus, the wedge pressure increases to 17 mmHg (near optimum for an acute infarction) with a dramatic improvement in the cardiac index to 3.0 L/min/m². The stroke volume index improved from 18 mL/beat/m² to 30 mL/beat/m². Note that the saline bolus had little effect on the right atrial pressure in this patient with acute infarction of the left ventricle. Note also that the A and V waves are easily visible in the wedge tracing after the volume challenge because the left atrial pressure now exceeds the alveolar pressure (allowing retrograde waveform transmission). Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
In patients with a very noncompliant infarction (and a very large A wave), the optimal mean wedge pressure may be below 15 mm Hg.

Thus the ideal mean wedge pressure during an acute MI varies with the individual.

In critically ill patients, the effect of increasing or decreasing the mean wedge pressure should be carefully assessed by measuring the response of the cardiac output and SV.

As a rule, there is little gain in increasing the wedge above 18-20 mm Hg.
Acute Left Ventricular Infarction

- Forrester, Swan and colleagues described the correlation of hemodynamic measurements with hospital mortality in patients with acute MI.
- Patients can be triaged into one of four hemodynamic subsets based on measurements of the mean wedge pressure and the cardiac index.

<table>
<thead>
<tr>
<th>Subset</th>
<th>Wedge Pressure &gt; 18 mmHg</th>
<th>Cardiac Index &lt; 2.2 L/min/m²</th>
<th>Observed Frequency</th>
<th>Hospital Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No</td>
<td>No</td>
<td>31%</td>
<td>3%</td>
</tr>
<tr>
<td>II</td>
<td>Yes</td>
<td>No</td>
<td>17%</td>
<td>9%</td>
</tr>
<tr>
<td>III</td>
<td>No</td>
<td>Yes</td>
<td>17%</td>
<td>23%</td>
</tr>
<tr>
<td>IV</td>
<td>Yes</td>
<td>Yes</td>
<td>35%</td>
<td>51%</td>
</tr>
</tbody>
</table>

Acute Left Ventricular Infarction

• A depressed CI confers a mortality increase of 5 to 15 fold depending on whether or not the wedge pressure is also increased.

• Likewise, an increased wedge pressure raises the mortality by 2 to 15 fold depending on whether or not the cardiac index is also decreased.

• It is important to note that these observations were made prior to the era of emergency reperfusion therapy for acute myocardial infarction.
Wedge Pressure & Pulmonary Congestion
As the left ventricular end-diastolic pressure increases with acute myocardial infarction, so does the mean wedge pressure. The increased hydrostatic pressure promotes pulmonary congestion and ultimately pulmonary edema. Clinically, this is manifest as dyspnea and respiratory failure. As a rule, the degree of elevation of the mean wedge pressure correlates with the degree of pulmonary congestion as follows: 10, 11

<table>
<thead>
<tr>
<th>Mean Wedge Pressure</th>
<th>Degree of Pulmonary Congestion</th>
</tr>
</thead>
<tbody>
<tr>
<td>18 - 20 mmHg</td>
<td>Onset of pulmonary congestion</td>
</tr>
<tr>
<td>21 - 25 mmHg</td>
<td>Moderate congestion</td>
</tr>
<tr>
<td>26 - 30 mmHg</td>
<td>Severe congestion</td>
</tr>
<tr>
<td>&gt; 30 mmHg</td>
<td>Acute pulmonary edema</td>
</tr>
</tbody>
</table>
Cardiac Index and Tissue Perfusion

Cardiac Index
- 2.5-3.5 L/min/m²
- 2.0-2.2 L/min/m²
- 1.8-2.0 L/min/m²

Severity of Tissue Hypoperfusion
- Normal range
- Onset of peripheral hypoperfusion
- Onset of cardiogenic shock
Arterial Blood Pressure

• The arterial blood pressure is normal in the majority of patients with acute myocardial infarction.
• It is common to observe moderate hypertension greater than 160/90 mm Hg even in previously normotensive patients due to the sympathetic discharge accompanying myocardial infarction.
• Hypotension (< 90 mm Hg) does not always signify the presence of cardiogenic shock.
• Activation of the Bezold-Jarisch reflex may result in profound peripheral vasodilation and hypotension. Stimulation of this reflex is more common in patients with inferior infarction. The reflex can also be stimulated by administration of nitroglycerin.
• Patients with hypotension mediated by high vagal tone usually appear warm and well perfused. The vagus nerve action also promotes bradycardia in these patients.
Figure 7.4 Lead III rhythm strip demonstrates activation of the Bezold-Jarisch reflex by nitroglycerin. The patient has an acute inferior infarction. At baseline (08:36) sinus rhythm is present with ST-segment elevation. Administration of intravenous nitroglycerin resulted in profound sinus bradycardia and hypotension. This episode resolved quickly with administration of atropine and discontinuation of the nitroglycerin.
Mechanical Complications of Acute MI

• Cardiogenic shock carries a mortality exceeding 70% and is the leading cause of hospital death in patients with acute MI. These patients have pathological evidence for infarction involving 40% or more of the LV myocardium.

• Clinical diagnosis defined by the triad:
  – Hypotension: SBP ≤ 90 mm Hg (prior to inotropic or IABP support)
  – Poor tissue perfusion
  – Pulmonary congestion

• Forrester Class IV.
Intracardiac Pressures in Cardiogenic Shock

- RA, PA, and PAOP pressures are all elevated.
- With shock, the ratio of the mean RA pressure to the mean WP is usually 0.5.
- This ratio will be closer to 1.0 when cardiogenic shock complicates RV infarction.
- The RA waveform may demonstrate summation of the A and V waves due to pronounced sinus tachycardia.
Intracardiac Pressures in Cardiogenic Shock

Figure 7.5 (Panels A-D) Intracardiac pressure recordings from a patient with an acute anterior myocardial infarction, right bundle branch block, and cardiogenic shock. Sinus tachycardia (129 beats/min) is present. Panel A: Modest pulmonary artery (PA) hypertension (33/24 mmHg) is present. The pulmonary artery pulse pressure is reduced (9 mmHg) because of reduced stroke volume. Panel B: The mean pulmonary capillary wedge pressure (PCW) is elevated at 25 mmHg. Pulmonary edema was present on chest x-ray. The mean wedge pressure correlates well with the pulmonary artery diastolic pressure. Panel C: The mean right atrial pressure (RA) is elevated at 11 mmHg. The ratio of right atrial pressure to wedge pressure is 0.4. The A wave and V wave have begun to merge because of sinus tachycardia. As a result, the y descent is blunted. Panel D: Thermocatheter cardiac output curve recorded from this patient. The cardiac index is significantly reduced at 1.7 liters/min/m² (body surface area = 1.8 m²). The stroke volume is markedly reduced at 23 mL/beat. The cardiac output is maintained at the level of 3.0 liters/min by sinus tachycardia together with intravenous dopamine and an intraaortic balloon pump. Without intraaortic balloon pump support, the cardiac output and stroke volume were 2.6 liters/minute and 20 mL/beat respectively.
Intracardiac Pressures in Cardiogenic Shock

• Mean WP is usually elevated to a level that causes clinical pulmonary congestion or overt pulmonary edema.

• Diagnosis of shock requires that the patient has received adequate volume expansion (mean WP $\geq$ 12 mm Hg).

• Remember that optimal cardiac performance occurs with mean WP of 14-18 mm Hg.

• The A and V waves are usually of similar magnitude.

• A large V wave suggest the presence of acute mitral regurgitation.
Intracardiac Pressures in Cardiogenic Shock

Figure 7.6 Continuous recording of mixed venous oxygen saturation (SV O₂) from a patient with acute anterior myocardial infarction and shock. At baseline, the cardiac index (Cl) was 1.9 L/min/m² and the wedge pressure (PCW) was 10 mmHg. The mixed venous saturation at this point was 40-45%. After volume expansion with blood and albumen, the cardiac index rose to 3.2 L/min/m². The rapid rise of the mixed venous saturation to 70-75% reflects the improved cardiac index. In this patient, the change in the mixed venous oxygen saturation provided nearly instant confirmation that volume expansion was beneficial.
Cardiac Index in Cardiogenic Shock

• Clinical Cardiogenic Shock is associated with a CI $\leq 1.8$ liter/m/min. The CI is critically dependent on Heart Rate.

• It is crucial to examine the SV since a change in CI may be caused simply by a change in the heart rate and not the intrinsic cardiac performance.
Arterial Blood Pressure in Cardiogenic Shock

• The cuff blood pressure is notoriously inaccurate in patients with cardiogenic shock.
• Cuff pressures can underestimate the actual intraarterial pressure by as much as 160 mm Hg.
• Intraarterial pressure measurement is mandatory.
• Moderate to severe systolic hypotension $\leq 90$ mm Hg is the rule.
Intraaortic Balloon Pump in Cardiogenic Shock

• An intraaortic balloon pump is often used to support the circulation in patients with cardiogenic shock.

• The balloon pump inflation/deflation cycle occurs during diastole and produces a predictable effect on the arterial pressure, the mean wedge pressure, and the stroke volume.

• It is programmed to inflate at the moment of aortic valve closure (dicrotic notch) and to deflate prior to the onset of aortic ejection (aortic pressure upstroke).
Figure 7.7 Arterial blood pressure recording demonstrating the effects of an intraaortic balloon pump (IABP) on the aortic pressure waveform. The IABP inflation/deflation (arrows) occurs every third beat (1:3). Inflation occurs at aortic valve closure (dicrotic notch). Deflation occurs before the onset of ejection of the next beat. The IABP inflation augments the early diastolic pressure of beat “a”. Note that IABP deflation lowers the aortic end-diastolic pressure so that beat “b” begins ejection against a lower “afterload.” The stroke volume of beat “b” is therefore improved. This results in a higher aortic pulse pressure (compare the pulse pressure of beat “a” with that of beat “b”). Scale = 0-120 mmHg; Paper speed = 25 mm/sec.
IABP

- Balloon pump inflation causes a sudden augmentation of the early aortic diastolic BP. This promotes tissue perfusion and increases the diastolic coronary artery blood flow velocity.
- Balloon pump deflation lowers the aortic end-diastolic pressure and provides a mechanical advantage (decreased afterload) for the next LV ejection.
- As a result, the SV of the damaged LV rises and contributes to improved CO.
- This is especially true when significant mitral valve regurgitation is present.
Figure 7.8 Effect of intraaortic balloon pump (IABP) support on the cardiac output (CO liters/min) and stroke volume (SV, mL per beat) in a patient with cardiogenic shock. The thermodilution cardiac output curve recorded with the IABP on (solid line) is superimposed with the cardiac output curve recorded with the IABP off (dashed line). The improved cardiac output is due solely to an improved stroke volume.
Mitral Regurgitation and Pericardial Tamponade

• These complications of an acute MI are uncommon especially since the advent of reperfusion therapy.

• Acute severe mitral regurgitation is the result of infarction of one of the papillary muscles and adjacent ventricular myocardium.

• Cardiac tamponade is the result of post-infarction pericarditis or sub-acute rupture of the left ventricular free wall.
Ventricular Septal Rupture

• Can occur as a consequence of either anterior or inferior MI.
• The result is a ventricular septal defect with a left to right shunt and a pulmonary to systemic blood flow ratio usually greater than 2:1.
• Can be confirmed by demonstrating a significant increase (10% or more) in the oxygen saturation between the right atrium and the pulmonary artery.

Table 7.2: Hemodynamic Parameters and Oxygen Saturations in a Patient with Acute Ventricular Septal Defect

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure</td>
<td>18 mmHg</td>
</tr>
<tr>
<td>Pulmonary artery pressure</td>
<td>54/17 mmHg</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>22 mmHg</td>
</tr>
<tr>
<td>Cardiac output (thermodilution)</td>
<td>4.2 liters/minute</td>
</tr>
<tr>
<td>Right atrial oxygen saturation</td>
<td>49%</td>
</tr>
<tr>
<td>Pulmonary artery oxygen saturation</td>
<td>61%</td>
</tr>
</tbody>
</table>
Ventricular Septal Rupture

• The RA SaO2 must be interpreted carefully; this chamber receives blood from the inferior vena cava, the superior vena cava, and the coronary sinus.

• The RA SaO2 can be artificially decreased if the proximal catheter lumen is adjacent to the coronary sinus (venous blood flow).

• The RA SaO2 can be artificially increased if significant TR further complicates the ventricular septal rupture. Oxygenated blood is shunted across the septal defect into the RV and then refluxes across the tricuspid valve into the RA.

• This unusual scenario is most likely to occur when septal rupture complicates acute inferior MI with concomitant RV infarction and tricuspid papillary muscle dysfunction.
Ventricular Septal Rupture

• With acute VSD, the mean RA pressure, wedge, and pulmonary artery pressures are all significantly elevated.

• A large V wave is often present in the wedge pressure tracing.

• With acute septal rupture, the systemic blood flow averages only one-half to one-forth of the thermodilution determined cardiac output. Thus a “normal” thermodilution CO in a patient with acute septal rupture usually reflects a severe reduction in systemic blood flow.
Right Ventricular Infarction

• RV infarction is almost always complicated by inferior LV infarction since the right coronary artery usually also supplies the inferior (diaphragmatic) wall of the left ventricle.

• The hemodynamic findings of RV infarction are governed by the infarct size, the degree of RV dilatation, the function of the ventricular septum, the contractile state of the right atrium and the cardiac rhythm.

![Electrocardiogram](image-url)

*Figure 8.1* Twelve-lead electrocardiogram from a patient with an acute inferior myocardial infarction. ST segment elevation is present in the inferior limb leads 2, 3, and aVF. The precordial leads are right sided. Significant ST segment elevation in precordial leads V3R - V6R is diagnostic of coincident right ventricular infarction.
Right Ventricular Infarction

• The RV is a thin walled structure with a muscle mass of only 1/6 that of the LV.

• Consequently, RV infarction leads to acute RV dilatation. The degree of dilatation is limited by the unyielding nature of the normal pericardium resulting in a form of acute pericardial constriction.

• The RV shares the interventricular septum with the LV. With RV free wall infarction, the IVS can lend contractile support to the RV, thus limiting the hemodynamic consequences of the infarction.

• When the infarction also involves the IVS, the consequences are more serious.

• The right coronary provides blood supply to a variable portion of the IVS through the posterior descending coronary artery. Therefore occlusion can lead to coincident RV and IVS infarction.
Right Ventricular Infarction

• RA pressure is elevated to 10 mm Hg or greater. The X and Y descents are prominent. This pattern is also seen with pericardial constriction and restrictive cardiomyopathy.

• The prominent X and Y descents cause the RA waveform to resemble the letter W or M.

• Either the X descent or the Y descent may be the dominant negative wave.

Figure 8.2 (Panels A & B) Right atrial pressure waveform from a patient with right ventricular infarction. **Panel A:** The mean right atrial pressure is elevated at 9 mmHg. The X (X) and Y (Y) descents are prominent causing the waveform to resemble the letter W. The A wave (A) is prominent likely representing augmented right atrial systole. The C wave is trivial. Note the inspiratory exaggeration of the A wave and the X and Y descents present on the first cardiac cycle of this figure. Scale = 0-12.5 mmHg; Paper speed = 25 mm/sec. **Panel B:** Right atrial pressure waveform from a patient with right ventricular infarction. An inspiratory increase in the mean right atrial pressure (Kussmaul’s sign) is present. Scale = 0-25 mmHg; Paper speed = 10 mm/sec.
Right Ventricular Infarction

• RA systolic dysfunction may complicate RV infarction, especially when the coronary artery occlusion is proximal and compromises RA blood supply.
• Severe hemodynamic compromise can occur due to the decreased force of RA systole.
• The magnitude of the right atrial A wave (relative to the mean right atrial pressure) provides some information about the atrial contractile function.
• Patients with small amplitude A waves tend to fare worse than those with augmented A waves. (Implies decreased atrial filling)

Figure 8.4 Right atrial pressure waveform from a patient with severe right ventricular infarction. The mean right atrial pressure is elevated at 15 mmHg. The A wave (α) is attenuated possibly due to right atrial ischemia. The X’ (x’) and Y (y) descents are prominent and vary with respiration. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Right Ventricular Infarction

• Heart block is yet another cause of hemodynamic deterioration during right ventricular infarction. The worsening in hemodynamic status is due primarily to the loss of AV synchrony (not bradycardia) further emphasizing the importance of effect right atrial systole.

• Tricuspid regurgitation can also occur with RV infarction and will alter the RA pressure waveform and further raise RA pressure.
Right Ventricular Infarction

- Wedge pressure is usually elevated because of concomitant inferior-septal left ventricular infarction.
- The increase in RA pressure is usually disproportionately greater than the increase in wedge pressure.
- The ratio of RA/wedge (normal ≤ 0.5) often exceeds 0.75 and may even exceed 1.0 during RV infarction.
- The increase RA pressure relative to LA (wedge) can promote R to L shunting across a patent foramen ovale.
- Serious arterial desaturation can occur.

Figure 8.5 (Panels A & B): Wedge pressure and right atrial pressure waveforms from a patient with right ventricular infarction.  
Panel A: The mean wedge pressure is elevated at 23 mmHg due to inferior left ventricular infarction.  
Panel B: The mean right atrial pressure is elevated at 23 mmHg. The ratio of right atrial to wedge pressure is 1.0. Prominent X'(x') and Y (y) descents are present. An H wave is visible following the Y descent because of sinus bradycardia. Scale = 0.30 mmHg; Paper speed = 25 mm/sec.
Right Ventricular Infarction

• Pulmonary Artery Pressure and Cardiac Output

  – PA pressure is commonly elevated and parallels the increased wedge pressure.
  – RV stroke volume is decreased causing a decrease in pulmonary artery pulse pressure.
  – With severe RV infarction, the PA pulse pressure is so narrowed that it resembles a venous waveform.

Figure 8.6 Pulmonary artery (PA), wedge, and right atrial (RA) pressure recordings from a patient with severe right ventricular infarction. The pulmonary artery pulse pressure is narrow because of reduced right ventricular stroke volume and the waveform resembles that of a venous tracing. The mean wedge pressure is elevated at 20 mmHg because of inferior wall left ventricular infarction. The mean right atrial pressure is elevated at 16 mmHg and the ratio of right atrial to wedge pressure is 0.8. The mean pressures in the pulmonary artery, wedge, and right atrium are therefore quite similar. In this situation, catheter placement can be very difficult without fluoroscopy. Scale = 0.25 mmHg; Paper speed = 5 mm/sec.
Right Ventricular Infarction

• This can make bedside catheter placement difficult. Changing the pressure scale to expand the waveform is helpful.

Figure 8.7 (Panels A & B): Pulmonary artery and wedge pressure waveforms from a patient with right ventricular infarction recorded on different pressure scales.

Panel A: The pressure scale is 0-60 mmHg. The pulmonary artery pulse pressure is reduced because of decreased right ventricular stroke volume. It is difficult to appreciate the change from pulmonary artery (PA) to wedge (PCW) in this tracing. S = pulmonary artery systolic wave; Arrow = balloon inflation; Paper speed = 25 mm/sec.

Panel B: The pressure scale is now 0-30 mmHg. The pulmonary artery (PA) pressure waveform and transition to wedge (PCW) waveform are much easier to recognize. The A and V waves can now be identified in the wedge waveform. S = Pulmonary artery systolic wave; Arrow = balloon inflation; Paper speed = 25 mm/sec.
Right Ventricular Infarction

• It is a widely held misconception that volume loading is always beneficial for patients with RV infarction and hemodynamic compromise.
• In fact, volume loading does not uniformly produce an increase in the cardiac output in these patients.
• While volume loading can certainly lead to an increase in both RA pressure and the wedge pressure, this may not translate into an improved SV.
• The increase in the wedge pressure is not associated with an increase in LV volume because of geometric changes in the LV. In fact, volume loading may be harmful if it results in severe peripheral or pulmonary edema.
• Therefore, it is important to quantitate the effect of volume loading on the SV and CO in these patients.
Acute Left Ventricular Ischemia

- Myocardial ischemia can complicate many serious illnesses since coronary artery disease is so common in the intensive care unit population.
- It can be difficult to recognize the presence of myocardial ischemia; it is often painless and short-lived.
- In the intensive care unit, intermittent left ventricular ischemia may manifest itself clinically as congestive failure.
- Recurrent painless ischemia is one of the causes of refractory respiratory failure.
- Myocardial ischemia is evanescent and continuous recording of hemodynamic parameters is necessary to detect its presence.
Acute Left Ventricular Ischemia

- Acute left ventricular ischemia causes immediate impairment of both systolic and diastolic myocardial function.
- The hemodynamic changes occur in both painful and painless ischemia.
- The diastolic dysfunction leads to an increase in the left ventricular end diastolic pressure.
- The increase in the left ventricular end diastolic pressure is transmitted to the left atrium causing an increase in the wedge pressure.
- Eventually the elevated left ventricular filling pressure leads to pulmonary congestion.
- When myocardial ischemia causes an elevation of the wedge pressure to ≥ 25 mm Hg, overt pulmonary edema occurs.
Acute Left Ventricular Ischemia

Figure 9.1 Left ventricular pressure recording at baseline (left) and during spontaneous myocardial ischemia (right). At baseline, the left ventricular end diastolic pressure is 25 mmHg (arrow). A modest A wave (A) is present on the pressure tracing. During ischemia, the left ventricular diastolic pressure increases significantly. The left ventricular end diastolic pressure (arrow) is now 42 mmHg due in part to an exaggeration of the A wave. Note also that the left ventricular systolic pressure increases during ischemia in this patient who is experiencing significant chest pain.

Scale = 0-100 mmHg; Paper speed = 50 mm/sec.
Acute Left Ventricular Ischemia

- The rate the formation of interstitial and alveolar pulmonary edema may be very rapid during periods of elevated pulmonary capillary wedge pressure.

- In contrast, removal rate of the edema fluid is often relatively slow once the elevated wedge pressure has returned to normal. As a result, the clinical and radiographic effects of the pulmonary edema may linger long after hemodynamic measurements have returned to normal.

- The pulmonary artery pressure increases during acute ischemia because of the sudden increase in the left ventricular and diastolic pressure and the wedge pressure.
Acute Left Ventricular Ischemia

Figure 9.2 Recording of pulmonary artery pressure (PA), wedge pressure (wedge) and Lead II electrocardiogram at baseline (top) and during ischemia (bottom). At baseline, the heart rate is 96 beats/min, and the Lead II ST-segment is isoelectric. The pulmonary artery pressure is 50/20 mmHg and the mean wedge pressure is 15 mmHg. During spontaneous ischemia, the heart rate is 110 beats/min and there is 2 mm (0.2 mV) ST-segment depression in Lead II. The pulmonary artery pressure is 62/26 mmHg and the mean wedge pressure is 34 mmHg. During ischemia, the pulmonary artery diastolic pressure significantly underestimates the mean wedge pressure because of the presence of a large V wave. Scale = 0-60 mmHg.
Acute Left Ventricular Ischemia

• Baseline measurements of the pulmonary artery pressure and the wedge pressure are deceiving and may be normal.

• During acute ischemia striking increases in the heart rate, pulmonary artery pressure and wedge pressure may occur.

<table>
<thead>
<tr>
<th>Table 9.1 Hemodynamic Parameters at Baseline &amp; During Ischemia*</th>
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<tbody>
<tr>
<td>Parameter</td>
</tr>
<tr>
<td>--------------------</td>
</tr>
<tr>
<td>HR (beats/min)</td>
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<tr>
<td>PAP systolic (mmHg)</td>
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<tr>
<td>PAP diastolic (mmHg)</td>
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<tr>
<td>PCW (mmHg)</td>
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</tbody>
</table>

*The above data was collected from patients presenting with ischemia mediated acute pulmonary edema.

Data presented are mean and (range). HR = heart rate; PAP = pulmonary artery pressure; PCW = pulmonary capillary wedge pressure.
Acute Left Ventricular Ischemia

- Continuous recording of the pulmonary artery pressure can be used to detect ischemic mediated increases in the left ventricular end diastolic pressure.
- At the same time, measurements of the pulmonary artery diastolic pressure provides an assessment of the physiologic consequences of such episodes with respect to pulmonary congestion.
- Transient pulmonary artery hypertension can occur with stresses other than ischemia. It is therefore necessary to continuously record the ST segment of the electrocardiogram to prove that myocardial ischemia is the cause of observed increases in the pulmonary artery pressure.
Acute Left Ventricular Ischemia

Figure 9.3 Continuous recording of heart rate and pulmonary artery pressures from a patient with recurrent acute pulmonary edema. **Top:** Before angioplasty (PTCA), multiple episodes of painless pulmonary artery hypertension and sinus tachycardia are present. Each episode was associated with ST-segment depression in leads V₁ - V₆.

**Bottom:** After PTCA, the episodic pulmonary hypertension is gone. The pulmonary edema resolved and the patient was extubated. HR = heart rate (scale is in beats/min); PAP = pulmonary artery pressure; S = systolic; M = mean; D = diastolic (scale is in mmHg). Time displayed is military time. (From Sharkey SW, Aberg NB, Am Heart J 1995; 129:189, with permission.)

Figure 9.4 Continuous recording of heart rate, pulmonary artery pressure, and ST-segment deviation in lead V₃ over 2 hours. An episode of spontaneous ischemia occurs at about 05:30. At baseline (05:15), the heart rate is 94 beats/min, the pulmonary artery pressure is 46/21 mmHg, and ST-segment depression of 0.5 mm (0.05 mV) is present. During peak ischemia, the heart rate is 112 beats/min, the pulmonary artery pressure is 78/44 mmHg and ST-segment depression of 2.1 mm (0.21 mV) is present. Careful inspection of this recording reveals that the change in the pulmonary artery pressure and the ST-segment precede the increase in the heart rate. HR = heart rate (scale is 90 to 114 beats/min); PAP = pulmonary artery pressure; S = systolic and D = diastolic (scale is 10 to 78 mmHg); ST3 = ST-segment in Lead V₃ (scale is -0.3 to -2.2 mm, 1 mm = 0.1 mV).
Acute Left Ventricular Ischemia

• Wedge pressure and pulmonary artery pressure
  - During acute ischemia, both the A and V waves of the wedge pressure waveform are accentuated because the increased left atrial pressure distends the pulmonary venous channels allowing more effective transmission of all left atrial mechanical events.
  - Even in the absence of significant mitral regurgitation, the V wave and the wedge pressure is often increased relative to the A wave because of ischemia mediated noncompliance of the left heart.
  - The magnitude of the increase in the wedge pressure depends on the duration of the ischemia, the baseline left ventricular function, and the amount of myocardium involved.
Acute Left Ventricular Ischemia

- Wedge pressure and pulmonary artery pressure

- Capillary muscle ischemia can cause a profound increase in the mean wedge pressure because of transient or severe mitral regurgitation. In this setting, it is common to observe a mean wedge pressure exceeding 30 mm Hg together with a giant V wave.
Acute Left Ventricular Ischemia

• The increase in the wedge pressure is transmitted to the pulmonary circulation causing an increase in the pulmonary artery systolic and diastolic pressures.

• The pulmonary artery diastolic pressure may significantly underestimate the mean wedge pressure if a large V wave is present in the wedge waveform.

• In general, painful ischemia produces a greater hemodynamic derangement than does painless ischemia.

Figure 9.2 Recording of pulmonary artery pressure (PA), wedge pressure (wedge) and lead II electrocardiogram at baseline (top) and during ischemia (bottom). At baseline, the heart rate is 96 beats/min, and the lead II ST-segment is isoelectric. The pulmonary artery pressure is 50/20 mmHg and the mean wedge pressure is 15 mmHg. During spontaneous ischemia, the heart rate is 110 beats/min and there is 2 mm (0.2 mV) ST-segment depression in lead II. The pulmonary artery pressure is 62/26 mmHg and the mean wedge pressure is 34 mmHg. During ischemia, the pulmonary artery diastolic pressure significantly underestimates the mean wedge pressure because of the presence of a large V wave. Scale = 0-60 mmHg.
Chronic Congestive Heart Failure

• Congestive heart failure is the unfortunate final outcome for a number of heart diseases.

• In contrast to patients with acute heart failure, the physical examination and chest x-ray are of limited value in acutely predicting the hemodynamic status of patients with chronic congestive heart failure.

• In one study, physical examination evidence specific for pulmonary congestion was absent in 44% of patients with pulmonary capillary wedge pressures greater than or equal to 35 mm of mercury.
Chronic Congestive Heart Failure

• Similarly, chest x-ray evidence of an increased wedge pressure (interstitial or alveolar edema) may be masked by the increased lymphatic drainage which occurs in patients with chronic heart failure.

• Hemodynamic monitoring is often necessary to guide therapy in patients admitted to the hospital with refractory heart failure.

• The hemodynamic findings discussed pertain to patients with chronic congestive heart failure in the setting of a dilated heart with poor systolic function.
Chronic Congestive Heart Failure

• Right atrial pressure, wedge pressure and pulmonary artery pressure
  – Typically, all intracardiac pressures are elevated to a varying degree.
  – The RA pressure and the mean wedge pressure are subject to the influence of any coexisting tricuspid or mitral regurgitation respectively.
  – Atrial and ventricular arrhythmias are common in these patients and will alter the right atrial and wedge pressure waveforms.
Chronic Congestive Heart Failure

• The mean right atrial pressure in patients hospitalized with severe heart failure is 9 to 12 mm of Hg. (range 2-38 mm Hg.)
• The wedge pressure is 21 to 30 mm Hg (range 8-44 mm Hg.)
• The mean pulmonary artery pressure is 33 mm of mercury.

Figure 10.1 Pulmonary artery (PA), wedge, and right atrial (RA) pressure waveforms from a patient with chronic congestive heart failure. The pulmonary artery pressure is 61/33 mmHg; the mean wedge pressure is 31 mmHg; and the mean right atrial pressure is 19 mmHg (note: the right atrial pressure scale is 0-30 mmHg). The ratio of the mean right atrial pressure/mean wedge pressure is nearly normal (0.6) in this patient without significant tricuspid regurgitation. The pulmonary artery diastolic pressure and the mean wedge pressure are nearly identical. The wedge pressure A (a) and V (v) waves are of similar amplitude. Mitral regurgitation was absent on echocardiography. Despite a mean wedge pressure of 31 mmHg, the patient was comfortable lying flat in bed. Scale = 0-60 mmHg (PA) and 0-30 mmHg (RA); Paper speed = 25 mm/sec.
Chronic Congestive Heart Failure

• Patients with chronic heart failure generally have higher intracardiac pressures than do patients with acute heart failure.

• In one study mean wedge pressure was $\geq 35$ mm Hg in 36% of patients hospitalized with severe chronic congestive heart failure.

• In comparison, the mean wedge pressure of patients with acute myocardial infarction and cardiogenic shock is typically 8-28 mm Hg.
Chronic Congestive Heart Failure

- It is important to note the relation between the mean right atrial pressure and mean wedge pressure.
- In many patients with chronic heart failure, the usual ratio of RA/PAOP of $\leq 0.5$ is observed.
- However it is not uncommon for the ratio to exceed 0.5 because of RV dilatation and severe TR.
- In some patients, right heart failure may predominate resulting in a right atrial pressure greater than the wedge pressure.
- The right atrial pressure waveform will have the features typical of tricuspid regurgitation in this subset of patients.
- It is rare for the mean RA pressure to actually exceed the mean wedge pressure unless a complication such as a pulmonary embolism has occurred.
Chronic Congestive Heart Failure

Figure 10.2 Hemodynamic data recorded from a patient with refractory heart failure due to ischemic heart disease and severe mitral regurgitation. Sinus tachycardia 100 beats/min is present. The pulmonary artery (PA) pressure is markedly elevated (83/35 mmHg); the mean wedge pressure is 32 mmHg with a V (v) wave of 47 mmHg; the mean right atrial (RA) pressure is 26 mmHg. The ratio of mean RA pressure/mean wedge pressure is increased (0.7) because of severe tricuspid regurgitation. The right atrial pressure waveform is typical of tricuspid regurgitation with a prominent CV wave and steep Y descent (y). Note that inspiration (insp) accentuates both the positive waves (A, C, V) and the negative waves (X, X', Y).

The wedge pressure waveform is dominated by the V wave in part because of significant mitral regurgitation. With a large V wave, the pulmonary artery diastolic pressure should be significantly less than the mean wedge pressure (Chapter 5). In this patient, the pulmonary artery diastolic pressure exceeds the mean wedge pressure because of increased pulmonary vascular resistance. A lung scan revealed no evidence for pulmonary embolism. Despite a mean wedge pressure of 32 mmHg, the patient was comfortable lying flat in bed. The thermodilution cardiac output (CO) curve is distorted by tricuspid regurgitation. The computer generated cardiac output (3.4 L/min) is probably inaccurate. Scale = 0-60 mmHg; Paper speed = 25 mm/sec.
Chronic Congestive Heart Failure

- The wedge pressure waveform is dominated by the V wave. The V wave is prominent because of noncompliance of the LV, although it is common to find some degree of MR in these patients.

- Moderate pulmonary hypertension is the rule.

- If the PA diastolic pressure exceeds the mean wedge pressure by $\geq 5$ mm Hg, the presence of a complication such as pulmonary embolism should be considered.

- The PA artery pulse pressure may be narrow in the presence of a low stroke volume.
Chronic Congestive Heart Failure

• Aortic pressure may be normal or even high. A decrease in the aortic pulse pressure correlates with a decrease in the cardiac index.

• Occasionally, pulsus alternans occurs in the final stages of CHF.

Figure 10.3 Aortic and pulmonary artery (PA) pressure waveforms from a patient with severe congestive heart failure. A premature ventricular contraction (PVC) initiates pulsus alternans (arrows) which is visible in both the aortic and pulmonary artery tracings. Scale = 0-125 mmHg (aorta) and 0-25 mmHg (PA); Paper speed = 25 mm/sec.
Chronic Congestive Heart Failure

- Cardiac Output/Index
  - Are usually reduced with the average being 3.0 L/min and 1.6 L/min/m² respectively.
  - The low CO is due largely to a significant reduction in the SV.
  - An occasional patient will have a marked reduction in the CI to levels as low as 1.0 to 1.5 L/min/m².

Figure 10.4 Superimposed cardiac output curves before and after therapy for refractory heart failure. At baseline (dashed line) the cardiac output is 1.6 L/min due to a profound decrease in the stroke volume (21 mL/beat). After treatment with intravenous inotropic drugs (solid line), the cardiac output is now 3.0 L/min. The improved cardiac output is primarily due to doubling of the stroke volume (42 mL/beat). Calibration artifact is -0.5°C.
Chronic Congestive Heart Failure

• Patients with chronic CHF adapt to a low CI primarily by increasing the tissue extraction of oxygen from hemoglobin, resulting in a decrease in the mixed venous (pulmonary artery) oxygen saturation.

• CO measurement is susceptible to error.
  – The presence of TR renders the method inaccurate.
  – Arrhythmias are another source of potential error. The thermodilution method samples blood flow during only a few heartbeats and extrapolates this measurement to a 1 min period. If a ventricular or atrial arrhythmia occurs during the injection and sampling period, the CO may not be representative.
  – Atrial fibrillation is a major offender, especially when the R-R intervals vary widely.
Chronic Congestive Heart Failure

• Alternatively, continuous monitoring of the pulmonary artery (mixed venous) oxygen saturation is clinically useful in these patients.
• In patients with chronic CHF, changes in the pul O2 sat parallel changes in CO. Measurement of pulmonary artery oxygen saturations over time and can be used to gauge the effects of therapy on CO.

Figure 10.5 Effect of nitroprusside therapy for congestive heart failure on the pulmonary artery oxygen saturation ($SV_2O_2$). Before nitroprusside, the $SV_2O_2$ is 55%. During nitroprusside infusion (NP on), the $SV_2O_2$ rises abruptly to 70% reflecting an increase in the forward cardiac output. After infusion (NP off), the $SV_2O_2$ drops reflecting a sudden decrease in the cardiac output.
Chronic Congestive Heart Failure

• In some patients, aggressive medical management can worsen heart failure.
• This is especially true in patients with ischemic cardiomyopathy.
• Therapy with IV inotropic drugs or powerful vasodilator drugs can trigger episodes of myocardial ischemia thus worsening the heart failure.
Pericardial Tamponade

- Uncommon but life-threatening condition.
- The astute clinician can establish the diagnosis of tamponade by a careful examination of a RA pressure waveform.
- Continuum with the degree of hemodynamic abnormality determined largely by the degree of fluid compression present.
- The rate of pericardial fluid accumulation, the volume of fluid, the tensile properties of the pericardium, and the volume status of the patient are all variables which will affect the hemodynamic parameters.
- The classic features of extreme tamponade include elevation and equalization of intracardiac pressures, pulsus paradoxus, and arterial hypotension.
Pericardial Tamponade—Physiology

- As fluid accumulates within the pericardial sac, the intrapericardial pressure rises.
- Initially, the intrapericardial pressure is less than either the right atrial pressure or the wedge pressure. At this stage, pulsus paradoxus is absent and the CO is unchanged.
- As more fluid accumulates, the intrapericardial pressure rises to equal the RA pressure, but is still less than the wedge pressure.
- Tamponade of the right heart now exists and the SV is compromised. Pulsus paradoxus may now appear.
Pericardial Tamponade - Physiology

• With a further increase in the pericardial pressure the intracardiac pressure rises to the level of the wedge pressure.

• Fluid compression of both the R and L heart now exists and the pericardial pressure, RA pressure, and wedge pressure are now equal. Pulsus paradoxus is magnified and SV is significantly decreased.
Pericardial Tamponade-RA, PAOP, PAP

- Mean RA pressure is elevated ~ 10-15 mm Hg.
- The X descent is prominent while the Y descent is markedly attenuated and often absent.

![Image of ECG waveforms]

**Figure 11.1** Pulmonary artery, wedge, and right atrial pressure waveforms from a patient with cardiac tamponade. Sinus rhythm is present. The pulmonary artery pressure is 31/13 mmHg; the mean wedge pressure is 15 mmHg; and the mean right atrial pressure is 13 mmHg. Note that the pulmonary artery diastolic pressure, the mean wedge pressure, and the mean right atrial pressure are essentially equal. The mean right atrial pressure is elevated with a prominent X descent (x) and an absent Y descent (arrow). This constellation of findings is highly specific for cardiac tamponade. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Figure 11.2 Right atrial pressure waveform from a patient with cardiac tamponade before (left) and after (right) pericardiocentesis. Sinus rhythm is present. During tamponade (left), the mean right atrial pressure is markedly elevated (23 mmHg). The X descent is prominent, but the Y descent is absent (arrow). After successful pericardiocentesis (right), the mean right atrial pressure has fallen (18 mmHg) and the Y descent has returned (arrow). The right atrial pressure remains elevated because of an underlying cardiomyopathy. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Pericardial Tamponade-Aortic Pressure

• Alfred Kussmaul is responsible for describing pulsus paradoxus in patients with pericardial tamponade. A widely accepted definition states that pulsus exits when a normal inspiration is accompanied by a drop in the systolic arterial pressure $\geq 10$ mm Hg.

![Graph showing arterial pressure and respiration](image)
Although pericardial constriction and restrictive cardiomyopathy are very different diseases, both share several clinical and hemodynamic features. With both constriction and restriction, the principle physiologic abnormality is impaired cardiac filling. In both conditions, the atrial pressures are elevated and the atrial pressure waveforms have steep X and Y descents.

With pericardial constriction, an unyielding pericardium is the culprit; with restrictive cardiomyopathy, the problem is an abnormal myocardium. In some patients, there is overlap between the two conditions. For example, with pericardial constriction, the inflammation may also invade the epicardium. Nonetheless, pericardial constriction can usually be differentiated from restrictive cardiomyopathy with careful attention to the hemodynamic findings.
Pericardial Constriction

- Infection, inflammation, and neoplasm can each cause the pericardium to become thickened, scarred, and noncompliant.
- The diastolic volume of the heart is reduced and the ventricular and atrial filling pressures are elevated.
- Since the constricting process is usually uniform, all four cardiac chambers are involved equally (in contrast to restrictive cardiomyopathy).
- RV infarction mimics constriction because the sudden dilation of the RV overdistends an otherwise normal pericardium.
Pericardial Constriction—Intracardiac Pressures

• RA and Wedge pressures are elevated; the magnitude of the atrial pressure elevation is determined by the degree of constriction.

• With moderate constriction the RA pressures are between 12-15 mm Hg.

• With severe constriction, the atrial pressures reach 20-25 mmHg.

• In pure constriction, the RA and wedge pressures are nearly identical.
Pericardial Constriction-Intracardiac Pressures

- Rapid filling of the ventricles is limited to early diastole (the ventricular size is smallest at the start of diastole, therefore the ventricular constriction is least at this time).
- The exaggerated early ventricular filling results in a steep Y descent in the atrial pressure waveform.
- Following atrial systole, the atrial volume is reduced (the atrial contents having been transported into the ventricles).
- At this time, constriction of the two atria transiently lessens resulting in a steep X descent.
- The combination of a steep X and steep Y descent causes the atrial pressure waveform to resemble the letter W (or M depending on your preference).
- This pattern can be observed in conditions other than pericardial constriction.
Figure 12.2 Right atrial (RA) pressure waveform typical of pericardial constriction. The mean right atrial pressure is elevated at 13 mmHg. The X' (x') and Y (y) descents are both prominent with a pattern of $X' < Y$. The waveform resembles the letter W. This pattern can also occur with restrictive cardiomyopathy. Note that a well-developed C wave (c) interrupts the X descent (x) generating two components, the X and X' descents. Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Pericardial Constriction-Intracardiac Pressures

• The presence of a noncompliant pericardium limits transmission of intrathoracic pressure to the heart.

• The normal inspiratory increase in superior and inferior vena cava flow is reduced or absent. Therefore the RA pressure often shows no respiratory change.

• With severe constriction, an inspiratory increase in the RA pressure (Kussmaul’s sign) may be present, but this is the exception, not the rule.
Pericardial Constriction-Intracardiac Pressures

Figure 12.3 Pulmonary artery (PA), wedge (PCW), and right atrial (RA) pressure recordings from a patient with pericardial constriction. The pulmonary artery pressure is moderately elevated (32/19 mmHg). The mean wedge pressure is 17 mmHg while the mean right atrial pressure is 16 mmHg. Although the mean atrial pressures are nearly equal, the waveforms are not identical. Note the absence of respiratory variation in the right atrial pressure tracing (In constriction, absence of respiratory variation is more common than is Kussmaul's sign). Scale = 0-30 mmHg; Paper speed = 6.25 mm/sec.

Figure 12.4 Right atrial (RA) pressure tracing from a patient with severe pericardial constriction. With inspiration (insp), the mean right atrial pressure increases (Kussmaul's sign). Scale = 0-30 mmHg; Paper speed = 6.25 mm/sec.
Pericardial Constriction-Intracardiac Pressures

• The PA pressures are modestly elevated.
• The pulmonary artery systolic pressure is typically 35-45 mm Hg.
• Severe pulmonary hypertension suggests coexisting myocardial or valvular heart disease.
• The pulmonary artery diastolic pressure should equal both the RA pressure and PAOP.
• Aortic pressure is usually maintained.
• Pulsus paradoxus is observed in only about 1/3 of patients with pericardial constriction, whereas in pericardial tamponade pulsus paradoxus is nearly universal.
• The SV is reduced, but tachycardia can maintain CO in all but the most severe cases. With severe constriction the SVI may be as low as 15-25 mL/m².
Effusive-Constrictive Pericarditis

• In some patients, pericardial inflammation leads to the combination of a pericardial effusion and pericardial constriction.

• In effusive-constrictive pericarditis, there is constriction of the heart by the visceral pericardium and pericardial fluid accumulation between the visceral and parietal pericardium.

• TB, mediastinal radiation, uremia, and pericardial malignancy are conditions known to cause effusive-constrictive pericarditis.

Figure 12.5 Right atrial (RA) pressure tracing from a patient with uremia and effusive-constrictive pericarditis. The mean right atrial pressure is markedly elevated at 31 mmHg. The $X$ descent ($x$) is dominant ($X > Y$ pattern). The $Y$ descent ($y$) is attenuated due to the presence of pericardial fluid compression. On inspiration ($\text{insp}$), the mean right atrial pressure rises (Kussmaul’s sign). Scale = 0-30 mmHg; Paper speed = 25 mm/sec.
Restrictive Cardiomyopathy

• A myocardial disease. Myocardial relaxation is restricted resulting in a hemodynamic picture closely resembling pericardial constriction.

• Uncommon. Its causes include rather obscure diseases, such as hemochromatosis, endomyocardial fibrosis, amyloidosis and myocarditis.
Restrictive Cardiomyopathy-Hemodynamics

- The RA pressure and wedge pressures are significantly elevated to levels observed with pericardial constriction (15-25 mm Hg).
- As with constriction, the X and Y descents are prominent with a pattern of $X = Y$ or $Y > X$.
- In contrast to constrictive pericarditis, the RA pressure and wedge pressure are usually not equal. The restrictive process involves both the L and R ventricle and causes a proportionate decrease in each chamber’s distensibility.
- As a result the R and L ventricular pressures are elevated but not equal.
- The wedge pressure usually exceeds the RA pressure.
Restrictive Cardiomyopathy-Hemodynamics

• Pulmonary hypertension is often more severe in restrictive cardiomyopathy than in constrictive pericarditis.

• In restriction, the pulmonary artery systolic pressure typically exceeds 50 mm Hg; in constriction this level of pulmonary hypertension is unusual.

• The CI may be normal, but maintained by tachycardia.
Constriction/Restriction

• The hemodynamic findings of pericardial constriction, effusive constrictive pericarditis, pericardial tamponade, and restrictive cardiomyopathy are compared:

![Schematic diagrams comparing pericardial constriction and tamponade.](image)

**Figure 12.6** Schematic demonstrating the effect of pericardial constriction on the heart. During ventricular systole (left), constriction is absent. The systolic venous return to the atria (X descent) is unimpaired. During early ventricular diastole (middle), constriction is still absent. The initial Y descent is unimpaired. In mid diastole (right), the heart volume suddenly equals the pericardial volume and constriction occurs. Diastolic filling is abruptly halted resulting in an abrupt halt to the Y descent. Adapted from Shabetai, R.¹

**Figure 12.7** Schematic demonstrating the effect of pericardial tamponade on the heart. The atrial pressures are governed by the elevated pericardial pressure. During early ventricular systole (left), the total heart size decreases as blood is ejected from the ventricles. Consequently, the pericardial pressure drops transiently resulting in an X descent in the atrial pressure waveform. During late systole, the heart size has started to increase because of venous return to the atria resulting in a progressive increase in the pericardial pressure. During early diastole (right), blood is transferred from the atria to the ventricles. The total cardiac volume remains constant and therefore the pericardial pressure also remains constant. The result is an absent Y descent. Adapted from Shabetai, R.¹
Dip and Plateau  Pressure Equalization
<table>
<thead>
<tr>
<th>Condition</th>
<th>Diastolic Equilibration</th>
<th>Dip and Plateau Physiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constrictive Pericarditis</td>
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<tr>
<td>Restrictive Cardiomyopathy</td>
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<tr>
<td>Cardiac Tamponade</td>
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## Constriction/Restriction

<table>
<thead>
<tr>
<th>Table 12.1</th>
<th>Comparison of Typical Hemodynamic Findings in Pericardial Constriction, Effusive-Constrictive Pericarditis, Restrictive Cardiomyopathy &amp; Pericardial Tamponade</th>
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<tbody>
<tr>
<td></td>
<td>Tamponade</td>
</tr>
<tr>
<td>RA Pressure Mean Range (mmHg)</td>
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<td>RA Waveform</td>
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</tr>
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<td>RA/Wedge Relation</td>
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<td>PA Systolic Pressure Normal or minimally elevated</td>
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<tr>
<td>Kussmaul's Sign Absent (RA pressure falls with inspiration)</td>
<td>Present in 1/3 of cases (especially with severe constriction)</td>
</tr>
<tr>
<td>Pulsus Paradoxus Almost always</td>
<td>Present in 1/3</td>
</tr>
</tbody>
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Pulmonary Embolism-Acute

- PA, Wedge, RAP and CO
  - Sinus tachycardia is the rule.
  - Pulmonary HTN is present in ~70% of patients.
  - In patients without prior cardiopulmonary disease, the mean PAP is consistently increased when the obstruction of the pulmonary vasculature exceeds 25-30%.
  - The mean PAP usually does not exceed 40 mm Hg because the normal right ventricle cannot generate a high pulmonary artery pressure acutely.
Pulmonary Embolism-Acute

Figure 13.1 Pulmonary artery (PA), wedge (PCW), and right atrial (RA) pressure waveforms from a patient with massive acute pulmonary embolism and shock. Sinus tachycardia (108 beats/min) is present. Modest pulmonary hypertension (42/24; mean 27 mmHg) is present. A significant pressure gradient between the pulmonary artery diastolic pressure (24 mmHg) and the mean wedge pressure (12 mmHg) is present reflecting an increased pulmonary vascular resistance. The elevated mean right atrial pressure (14 mmHg) signifies acute right heart failure. The ratio of mean right atrial pressure/mean wedge pressure (1.2) is increased. Scale = 0-25 mmHg; Paper speed = 10 mm/sec.
Pulmonary Embolism - Acute

• Higher levels of PAP suggest a chronic component to the PE or preexisting heart disease.
• The mean PAP correlates well with the degree of angiographic obstruction.
• With massive PE, pulmonary artery pulsus alternans may appear.
• The PAOP is usually normal or low unless the patient has underlying heart disease.
• When there is an obstruction in the pulmonary vasculature, a gradient between the PAD and the mean wedge is generated and the left atrial A and V waves are not transmitted retrogradely into the wedge pressure waveform.
Figure 13.2 Pulmonary artery (PA), wedge, and right atrial (RA) pressure waveforms from a patient with chronic pulmonary embolism. The rhythm is sinus (95 beats/min). Significant pulmonary hypertension (67/33; mean 45 mmHg) is present. The gradient between the pulmonary artery diastolic pressure (33 mmHg) and the mean wedge pressure (16 mmHg) is markedly increased. The abnormal pulmonary vasculature prevents retrograde transmission of the left atrial A and V waves into the wedge pressure waveform. The mean right atrial pressure (8 mmHg) is mildly elevated signifying early right heart failure. The right atrial pressure A wave (a) is prominent reflecting more forceful right atrial contraction. Scale = 0-60 mmHg (PA and wedge) and 0-30 mmHg (RA). Paper speed = 25 mm/sec.
Pulmonary Embolism-Acute

• There is concern that the wedge pressure may not reliably reflect the left atrial pressure in the presence of acute pulmonary embolism. The mean wedge pressure should be interpreted cautiously in these patients.

• The mean RA pressure is an important indicator of RV function in acute PE.

• The mean RA pressure increases in direct response to an increase in PAP.

• An elevation of mean RA pressure in a previously healthy patient usually indicates severe embolism with mean PAP ≥ 30 mm Hg and angiographic obstruction exceeding 35-40%.

• On the other hand, significant angiographic obstruction (up to 40%) may occur without elevation of the mean RA pressure.
Pulmonary Embolism-Acute

- The right atrial A wave is often prominent in response to the sudden elevation of right ventricular diastolic pressure.
- Tricuspid regurgitation may appear in response to RV dilation.
- The PAP and the mean RA pressure typically remain elevated for days after an acute PE.
- At 2-3 weeks, the mean PAP has frequently returned to normal, although persistent pulmonary hypertension occurs in some patients.
- With moderate PE, the CI is increased in large part to an increase in both the SV and HR from endogenous catecholamine release.
- With severe embolism and R heart failure, the CI and SV may be low and shock may occur.