INVASIVE HEMODYNAMICS MANUAL
FOR
ADULT CARDIAC CATHETERIZATION
(CATH. LAB)

Akram M. Al Ajmi
CCT, CRAT, RCIS, FSICP, MSc. (HCM)
2013

Sponsored
By
Saudi Group of Cardiac Technologists SGCT
P.O. BOX 295275 Riyadh 11351
sgct.info@gmail.com/ akram.alajmi@gmail.com
“There is no future in any job. The future lies in the man who holds the job”

George W. Grane
This manual is to be used as a reference within the Adult Cardiac Catheterization Laboratory. It is not designed to be the only source of information, but to be used in conjunction with information gained from The Adult Supervisor, Clinical Instructors, and other Senior Staff.

I hope that this manual will be of good help and useful to anyone applying for the RCIS registry exams, Staff working in CICU, CCU, and any Critical Care area with Invasive Hemodynamic Monitoring.

Good Luck to all
Acknowledgment ...

I have been working on this Manual since 2007 and it has been a dream of mine to do something for my profession, to help in a way that will make Invasive Hemodynamic easier and more recognizable.

I tried to review, collect, modify, write my own experience, and simplify the most relative information about Invasive Hemodynamic Monitoring in Cath Lab and Critical Areas.

This Manual is not for sale, and/or republishing. It meant to be used as a quick reference to Hemodynamic Monitoring in the Cath Lab and/or Critical Areas.

On page 60 you will find the lists of references and books where I relied on to formulate this Manual.

Thank you

Akram M. Al Ajmi CCT, CRAT, RCIS, FSICP, MSc. (HCM)
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>TOPICS</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Hemodynamics</td>
<td>6</td>
</tr>
<tr>
<td>Methodology of Hemodynamic Measures</td>
<td>6</td>
</tr>
<tr>
<td>Cardiac Performance</td>
<td>8</td>
</tr>
<tr>
<td>Potential Etiologies of Left Ventricular End Diastolic Pressure Elevation</td>
<td>16</td>
</tr>
<tr>
<td>Normal Hemodynamic Values</td>
<td>17</td>
</tr>
<tr>
<td>Pulmonary Artery Catheter</td>
<td>18</td>
</tr>
<tr>
<td>Normal Pressure and Waveform Characteristics as PA Catheter is Advanced</td>
<td>20</td>
</tr>
<tr>
<td>The Physiology of Central Venous Catheters</td>
<td>24</td>
</tr>
<tr>
<td>Normal Oxygen Saturation</td>
<td>34</td>
</tr>
<tr>
<td>Valvular Hemodynamics</td>
<td>35</td>
</tr>
<tr>
<td>Pressure Gradients Across Stenosis</td>
<td>37</td>
</tr>
<tr>
<td>Valvular Stenosis</td>
<td>38</td>
</tr>
<tr>
<td>Aortic Stenosis</td>
<td>41</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>44</td>
</tr>
<tr>
<td>Valvular Area Formulas</td>
<td>46</td>
</tr>
<tr>
<td>Hypertrophic Obstructive Cardiomyopathy</td>
<td>47</td>
</tr>
<tr>
<td>Intra-Cardiac Pressure Wave Forms</td>
<td>49</td>
</tr>
<tr>
<td>Constrictive Versus Restrictive Physiology</td>
<td>51</td>
</tr>
<tr>
<td>Shunt calculation</td>
<td>53</td>
</tr>
<tr>
<td>Right-To-Left Shunts</td>
<td>55</td>
</tr>
<tr>
<td>Intra-Aortic Balloon Pump (IABP)</td>
<td>56</td>
</tr>
<tr>
<td>Common Complications Associated with Intra-Aortic Balloon Pump Counterpulsation</td>
<td>59</td>
</tr>
<tr>
<td>References</td>
<td>60</td>
</tr>
</tbody>
</table>
NORMAL HEMODYNAMICS

Hemodynamic data are an important part of every diagnostic catheterization, particularly in patients with cardiomyopathies, valvular disorders, and pericardial disease. The measurement of hemodynamics utilizes pressure, oximetry, and temperature differences to derive functional information about the heart. To fully understand hemodynamics, one must first learn how to make proper measurements, calculate derived values, and interpret the results in relation to specific disease conditions.

METHODOLOGY OF HEMODYNAMIC MEASUREMENTS

1. Pressure Measurements

The most accurate method for measuring pressures in the heart is to utilize a system with the pressure transducer (usually a strain-gauge type) located at the exact location of interest. However, while catheters with a pressure transducer at the tip are available, they are too expensive to be used routinely and are generally used for research purposes only. Thus, the most common method of measuring pressure in the cardiac catheterization lab utilizes a system incorporating a fluid-filled (i.e. BD Transducer) catheter connected through a manifold to a pressure transducer. This system, however, has several characteristics that influence its fidelity and accuracy.

Preparation of Fluid-Filled Transducer:

**Items:**
1. 500ml Normal Saline 0.9 % w/v Sodium Chloride.
2. Pressure Cuff.
3. 500 unit of Heparin
4. Disposable Transducer (i.e. BD Transducer)

**Preparation:**

Add 500 unit of Heparin to the 500ml 0.9% N.S, so each ml of normal saline will contain 1 unit of Heparin. Prime your Transducer line and make sure it is free of any air bubbles. Place the Heparinized saline bag in the Pressure Cuff and inflated to 300 mmHg. **Noted that with 300mmHg the Transducer will deliver 3ml/h**
2. Oximetry Measurements

Oximetry measurements are most commonly performed to measure cardiac output by utilizing the Fick method and to rule out a left-to-right shunt. Oximetry measures the oxygen saturation of blood. The oxygen content of blood can then be calculated.

\[ \text{Hgb O}_2 \text{ Capacity} = 1.36 \times 100\% \text{sat} \times \text{Hgb} \]

\[ \text{Oxygen Content} = \text{Hgb (g/dL)} \times 1.36 \times (\text{mLO}_2/\text{g Hgb}) \times \text{Sat} \]

\[ \text{O}_2 \text{ Content} = \text{Hb} \times 1.36 \times \frac{\text{O}_2 \text{ Sat}}{100} = \text{ml/100ml} \]

Where Hgb is hemoglobin in grams per deciliter, 1.36 is the oxygen-carrying capacity of blood in milliliters of oxygen per gram of hemoglobin, and Sat is the oxygen saturation of the blood. The dissolved oxygen in blood is generally negligible in these calculations and is usually ignored.

3. Temperature Measurements

A thermistor is mounted at the tip of the pulmonary artery catheter to measure the temperature of the fluid as it passes through the pulmonary artery. Temperature is most commonly used to calculate cardiac output by using the thermodilution technique, which is a variant of indicator dilution.
CARDIAC PERFORMANCE

Cardiac Output

\[ \text{CO} = \text{SV} \times \text{HR} \]

Normal Value of CO is about 3 to 7 L/min

The primary purpose of the heart is to deliver oxygenated blood to the peripheral tissue. Cardiac output is measured clinically in two ways: the Thermodilution Method and the Fick Method. Cardiac output can also be indirectly estimated with left ventriculography. Cardiac output is affected by several different factors, including age, body size, and metabolic demands. To normalize resting cardiac output among different body sizes, the cardiac index is used:

\[
\text{Cardiac Index (L/min/m}^2\) = \frac{\text{Cardiac Output (L/min)}}{\text{Body Surface Area (BSA) (m}^2\)}
\]

\[
\text{BSA} = \sqrt{\left[\text{Height (cm)} \times \text{Weight (kg)}/ 3,600\right]}
\]

Thermodilution Method

(Remember, \(\text{CO(L/min)} = \text{stroke volume} \times \text{heart rate}\).) This can be a vital piece of information to help with a diagnosis, make decisions concerning therapeutic interventions and assess prognosis. The PAC (Pulmonary Artery Catheter) measures cardiac output with a technique called thermodilution.

The thermodilution technique is based on indicator dilution methods. This method utilizes a bolus injection of a known amount of a substance, and followed by measurement of the concentration of this substance downstream as a function of time. The concentration – time curve can then be used to determine the cardiac output. While this technique has been used with several different indicators, e.g. indocyanine green, the most common indicator used today is room temperature saline, with the temperature difference between saline and blood measured in lieu of concentration. The temperature is measured with a thermistor, usually at the level of the injectate bag and at the tip of the catheter. While this usually provides a reasonably accurate result, if the temperature of the saline is increased between the injectate bag and the injection port the measurement can be safely elevated.
This most commonly occurs because the saline is heated by the operator's hand while on the injection syringe. In low-output states, the saline gets warmed by the blood and heart prior to reaching the thermistor, which may result in inaccurate calculations. Valvular abnormalities, such as tricuspid or pulmonic regurgitation, or intracardiac shunting will also affect the thermodilution cardiac output.

**How is this performed?**

- A small quantity (2.5-10ml) of cool fluid is injected into the right atrium from the proximal opening in the PAC.

- A thermistor embedded in the tip of the PAC lying in the pulmonary artery detects a temperature change as the cooler blood flows by. The degree of change in the temperature is inversely proportional to the cardiac output.
  - Increased blood flow (and C.O.) = Minimal temperature change
  - Decreased blood flow (and C.O.) = Pronounced temperature change

- Plotting this temperature change against the time it took for the cooler fluid to reach the thermistor gives us a **thermodilution curve**. The equation for this curve is given by the **Stewart-Hamilton equation**:

\[
Q = \frac{V_1 (T_b - T_1) K_1 K_2}{\int T_b(t) \, dt}
\]

Where:
- **Q** = cardiac output
- **V_1** = injectate volume
- **T_b** = blood temperature
- **T_1** = injectate temperature
- **K_1** = density factor
- **K_2** = a constant
- **T_b(t) dt** = change in blood temperature as a function of time
Thankfully, a computer built into the monitor calculates this for the anesthesiologist, integrates the area under the thermodilution curve, and gives a digital readout of the cardiac output in L/min. Isn’t technology great?

**TROUBLESHOOTING**

*Common pitfalls in Measuring Cardiac Output*

1. Warming the saline in the syringe with your hand prior to injection when using the thermodilution method.
2. Not measuring cardiac outputs the same time pressure is measured.
3. For reproducible and accurate Thermodilution CO-measurement make sure you inject rapidly and forcefully; throw out the first TDCO reading; Synchronize all injection to the patients expiratory cycle.
4. TDCO may be inaccurate in TR; Cardiac Shunt; Rapid IV infusion.
5. In the TDCO curve reliability indicator, the smallest curve – no hump indicates the LARGEST CO.
6. Injection **must** be timed with the respiratory cycle (optimally during expiration).
7. Essential to have a rapid and even injection technique.
8. Discard any irregular waveforms.
9. Low cardiac output (Slow prolonged down slope, large area under curve)
10. High cardiac output (Rapid shorter down stroke, small area under curve)

*TDCO Thermodilution Cardiac Output*

**References:**

Fick Method

In 1870, Adolph Fick developed a principle that demonstrated that the total uptake or release of any substance by an organ is the product of blood flow to the organ and the arteriovenous concentration difference of the substance.

In common clinical practice, the organ to which this is applied is the lungs, and the substance is oxygen. This method calculates pulmonary blood flow, which in the absence of intracardiac shunts, equals systemic blood flow. Thus, systemic blood flow equals oxygen consumption divided by the pulmonary arteriovenous oxygen difference.

The arteriovenous oxygen difference is calculated by subtracting the oxygen content of mixed venous blood, usually pulmonary arterial blood in most clinical settings, from pulmonary venous blood, which is estimated by systemic arterial blood. The oxygen content equals oxygen saturation (%) multiplied by 1.36 mL\textsubscript{O_2}/g Hgb (oxygen-carrying capacity of hemoglobin) multiplied by hemoglobin (g/100 mL blood). The term for dissolved oxygen in the blood is usually negligible and therefore dropped.

\[
\text{Arterio-Venous O}_2 \text{ Difference} = \frac{(\text{AO sat} - \text{PA sat})}{100}
\]

\[
\text{CO} = \frac{\text{Oxygen Consumption (mL/min)}}{\left(\frac{\text{Ao}\% \text{ Sat} - \text{PA}\% \text{sat}}{100}\right) \times 1.36 \times \text{Hgb (mg/dl)} \times 10}
\]

or

\[
\text{CO} = \frac{\text{Oxygen Consumption (mL/min)}}{\left[\left(\text{a} - \text{V dif}\right) \times 10\right]}
\]
O₂ consumption is measured using a technique such as **Douglas bag** or can be estimated as 3ml O₂/kg or 125ml/min/m², AVO₂ (arteriovenous O₂) difference is calculated from atrial – mixed venous (pulmonary artery) O₂ content,

\[
\text{Sa O₂} \times 1.36 \times \text{Hb}
\]

Where O₂ content = \[
\frac{3 (\text{SVC}) + 1 (\text{IVC})}{100}
\]

**Flamme’s formula Mix. V =** \[
\frac{3 (\text{SVC}) + 1 (\text{IVC})}{4}
\]

**Fick Cardiac Output (L/min) =**

\[
\frac{\text{Oxygen Consumption (mL/min)}}{(\text{Arterial – Venous O}^2 \text{ Sat}) \times 1.36 \times \text{Hgb (mg/dL)} \times 10}{100}
\]

The uptake of oxygen by the lungs can be measured directly by using a metabolic cart. Given the unwieldiness, time, and expense, oxygen consumption is often estimated by a formula or nomogram. This simplification can, however, introduce inaccuracies to the calculation, especially in patients with significantly higher or lower metabolic demands than usual.

**Factors Affecting Oxygen Consumption**

- Age
- Gender
- Hyper or Hypothyroidism
- Hyper or Hypothermia
- Exercise
- Sepsis
Other Calculation used

- **Effective Stroke Volume** = \( \frac{CO \text{ (ml/min)}}{HR \text{ (bpm)}} \)

- **Effective Stroke Index SI** = \( \frac{SV \text{ (ml/beat)}}{BSA \text{ (m}^2\text{)}} \)

- **Total Stroke Volume** = \((\text{end Diastolic Volume [EDV]} - \text{end systolic volume [ESV]})\)

- **Ejection Fraction** = \( \frac{(EDV - ESV)}{EDV} \) which is \( \frac{SV}{EDV} \)

- **HR = 60 / R-R Interval**

- **Hgb O\text{2 Content : CaO}_2 = 1.36 \times Hgb \times O2Sat/100**

- **Hgb O\text{2 Capacity : O2Cap.} = 1.36 \times 100\%Sat \times Hgb**

- **\%O\text{2 Saturation} : O\text{2 Content} / O\text{2 Capacity} \times 100\%**

- **Normal Co : TDCo = Co_{fic} = LVMF = PBF = SBF**

- **With 100% Oxygen the formula of CO_{Fick} will be modified as**

\[
MVO2 / (AO\% - PA\%) \times Hgb \times 10 \times 1.36 + (PO2 \times 0.03) - (PO2 \times 0.03)
\]

References:

Self-Assessment

Calculation of Cardiac Output and Cardiac Index

A 56-year-old man:
Height = 180 cm
Weight = 70 kg
Oxygen consumption = 250 mL/min
Arterial oxygen saturation = 98%
Venous oxygen saturation = 70%
Hemoglobin = 14 g/dL

Cardiac Output =

\[
\text{Oxygen Consumption (mL/min)} \div (\text{Arterial} - \text{Venous O}_2 \text{ Sat}) \times 1.36 \times \text{Hgb} \times 10
\]

\[
\text{CO} = \frac{250}{(0.98 - 0.70) \times 1.36 \times 14 \times 10} = 4.69 \text{ L/min}
\]

BSA = \sqrt{\left[ \frac{\text{Height (cm) x Weight (kg)}}{3,600} \right]} \]

\[
\text{BSA} = \sqrt{(180) \times 70 / 3,600} = 1.87 \text{ m}^2
\]

\[
\text{CI (L/min/m}^2\text{)} = \frac{\text{CO (L/min)}}{\text{BSA (m}^2\text{)}}
\]

\[
\text{CI} = \frac{4.69}{1.87} = 2.51 \text{ L/min/m}^2
\]
Angiographic Techniques

This method uses the left ventriculogram to estimate stroke volume based on geometric assumptions about the shape of the ventricle. The stroke volume is multiplied by the heart rate, which gives an estimate of cardiac output. This method usually is the least accurate, especially in ventricles that do not hold up to geometric assumptions (Table 1).

Left Ventricular Filling Pressures

The left ventricular filling pressures are often estimated by the PCW pressure. Differentiating between the PA pressure and PCW pressure can sometimes be difficult, especially in the setting of severe mitral regurgitation, but three criteria can be used: (a) the mean PCW pressure should be about 10mm Hg less than the mean PA pressure; (b) blood withdrawn from the catheter in the wedged position should have an arterial saturation; (c) if in the setting of fluoroscopy, the catheter tip is wedged when it is lodged and not moving in the distal PA.

The left ventricular end diastolic pressure (LVEDP) provides the best hemodynamic correlation with the volume status of the heart and can help guide diuretic and vasodilator therapy. The LVEDP in normal patients is 3 to 12 mmHg. This parameter may increase with pressure or volume overload and with decreased left ventricular compliance.

Table 1. - METHODS FOR DETERMINING CARDIAC OUTPUT AND WHEN THEY ARE MOST (OR LEAST) RELIABLE

<table>
<thead>
<tr>
<th>Method</th>
<th>Most Reliable For</th>
<th>Least Reliable For</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fick</td>
<td>Low Cardiac Output</td>
<td>High cardiac output</td>
</tr>
<tr>
<td>Thermodilution</td>
<td>High cardiac output</td>
<td>Low cardiac output, Pulmonic regurgitation, Tricuspid regurgitation, Intracardiac shunting</td>
</tr>
<tr>
<td>Angiographic</td>
<td>Normal-shaped ventricle</td>
<td>Extensive segmental wall motion abnormalities, Dilated ventricle, Aortic regurgitation, Mitral regurgitation</td>
</tr>
</tbody>
</table>

Pressure can sometimes be difficult, especially in the setting of severe mitral regurgitation, but three criteria can be used: (a) the mean PCW pressure should be about 10mm Hg less than the mean PA pressure; (b) blood withdrawn from the catheter in the wedged position should have an arterial saturation; (c) if in the setting of fluoroscopy, the catheter tip is wedged when it is lodged and not moving in the distal PA.

The left ventricular end diastolic pressure (LVEDP) provides the best hemodynamic correlation with the volume status of the heart and can help guide diuretic and vasodilator therapy. The LVEDP in normal patients is 3 to 12 mmHg. This parameter may increase with pressure or volume overload and with decreased left ventricular compliance.

References:
- Ronnier J. Aviles, Adria W. Messerli – Introductory Guide to Cardiac Catheterization, Lippincott Williams & Wilkins 2004/ Cardiac output – angiographic Techniques
POTENTIAL ETIOLOGIES OF LEFT VENTRICULAR END DIASTOLIC PRESSURE ELEVATION

Aortic insufficiency  
Mitra Regurgitation  
Intracardiac Shunts  
High-output congestive heart failure  
Hypertension  
Hypertrophic cardiomyopathy  
Aortic stenosis  
Cardiomyopathy (Ischemic or Nonischemic)  
Restrictive cardiomyopathy  
Infiltrative cardiomyopathy

Vascular resistance

Resistance is defined as the ration of the pressure gradient across a vascular bed divided by the flow through that bed. Clinically, the two commonly calculated resistances are the systemic vascular resistance and the pulmonary vascular resistance.

**Systemic Vascular Resistance [ Wood units (mm Hg/L/min) ] =**

\[
\frac{\text{Mean Aortic Pressure} - \text{Mean Right Arterial Pressure}}{\text{Cardiac Output}}
\]

* (SBF used in shunts only) or, Qs or, Cardiac Output

**Pulmonary Vascular Resistance [ Wood units (mm Hg/L/min) ] =**

\[
\frac{\text{Mean PA Pressure} - \text{Mean Left Arterial Pressure}}{\text{Cardiac Output}}
\]

* (PBF used in shunts only) or, Qp or, Cardiac Output

Units: 80 dynes = 1 Wood unit (or mm Hg/L/min)

In most patients, changes in vascular resistance reflect changes in arteriolar tone or changes in the viscosity of blood (often secondary to anemia). In patients who are hypotensive or in shock, systemic vascular resistance (SVR) calculations help to differentiate between certain etiologies, and may help guide therapy. For example, a hypotensive patient with a low SVR may have sepsis, while a patient in cardiogenic shock often has hypotension with an elevated SVR.

Where:

- **Mean Arterial Pressure = Diastolic pressure+ 1/3 Pulse pressure**
- **Pulse Pressure = Systolic pressure – Diastolic pressure**
- **Ohm’s law (Fluid) = Pressure drop / Blood flow**
# NORMAL HEMODYNAMIC VALUES

## Flows
- **Cardiac Index**: 2.6 - 4.2 (L/min/m²)
- **Stroke Volume Index**: 35 - 55 (mL/m²)

## Pressure (mm Hg)

**Aorta/systemic artery**
- Peak systolic/end diastolic: 100 - 140 / 60 - 90
- Mean: 70 - 105

**Left Ventricle**
- Peak systolic/end diastolic: 100 - 140 / 3 - 12

**Left Atrium (Pulmonary Capillary Wedge)**
- Mean: 1 - 10
- \(a\) wave: 3 - 15
- \(v\) wave: 3 - 15

**Pulmonary Artery**
- Peak systolic/end diastolic: 16 - 30 / 2 - 8
- Mean: 10 - 16

**Right Ventricle**
- Peak systolic/end diastolic: 16 - 30 / 2 - 8

**Right Atrium**
- Mean: 2 - 8
- \(a\) wave: 2 - 10
- \(v\) wave: 2 - 10

## Resistances

**Systemic Vascular Resistance**
- Wood units: 10 - 20
- Dynes\(^a\) (dynes/sec/cm\(^5\)): 770 – 1500

**Pulmonary Vascular Resistance**
- Wood units: 0.25 - 1.50
- Dynes (dynes/sec/cm\(^5\)): 20 - 120

**Oxygen Consumption**: 110 – 150 (mL/min/m²)

**AVO\(_2\) difference**: 3.0 – 4.5 (mL/dL)

---

\(^a\) The amount of force needed to move a unit weighing 1 g (when acted on continuously) to an acceleration of 1 cm \(\cdot \) s\(^2\).

AVO, arteriovenous oxygen difference.
PULMONARY ARTERY CATHETER

- Provides Measurement of RA Pressure and RV Pressure as it passes through the chambers
- **PCWP** reflects LAP / LVEPP / LV preload
  PCWP is not a perfect reflection of Left heart preload
- Measurement of CO and mixed venous sat (SVO2)
- As PA inserted, distal port should be transuded so practitioner can identify where tip is located.

**Inserting Pulmonary Artery Catheter**

1. Obtain vascular accesses, typically with an 8 or 7 French (F) catheter sheath, allowing passage of the 7F or 6F pulmonary artery (PA) catheter. Typically, if the PA catheter is guided solely by pressure tracings to advance it to wedge position, the right internal jugular and left subclavian veins provide the most direct anatomic routes to the pulmonary artery that matches the natural curve of the catheter.

2. Inflated the balloon at the tip of the catheter under water to ensure there is no air leakage.

3. Make sure all lumens of the PA catheter are flushed.

4. Zero the pressure transducer at the level of the mi-right atrium.

5. Connect the PA catheter’s distal (PA) port to the pressure transducer. Make sure there are no air bubbles in the tubing or catheter.

6. Advance the catheter 20 cm through the sheath prior to balloon inflation to ensure the catheter tip clears the sheath. Do not advance if any resistance is met.

7. Beware of arrhythmias, primarily premature ventricular contractions (PVCs) and nonsustained ventricular tachycardia, especially after the catheter crosses the tricuspid valve. In the setting of underlying left bundle branch block, the catheter may induce complete heart block. In the setting of myocardial infarction, the catheter may induce ventricular fibrillation.

8. Monitor pressure as the catheter is being advanced through the RA, RV, and PA to wedge position. Be careful not to over-wedge.

9. Do not pull back the catheter with the balloon inflated. Damage to valves, either pulmonary or tricuspid may result.
COMPLICATIONS

- Cardiac arrhythmias
- Valvular damage
- Myocardial puncture
- Catheter knots
- PA injury or rupture
- Catheter malposition in right ventricle
- Pulmonary infarction
- Misinterpretation of information
- RV Injury or Rupture

General rules for Hemodynamic Measurements:

- Measure all Pressures at End-Expiration
- At the top curve with spontaneous respiration respiration (patient-peak)
- At the bottom curve with mechanical ventilation (vent-valley)
NORMAL PRESSURE and WAVEFORM CHARACTERISTICS as PA CATHETER is ADVANCED

<table>
<thead>
<tr>
<th>Location</th>
<th>Pressure</th>
<th>Waveform Characteristics</th>
</tr>
</thead>
</table>
| RA       | RAP = 6 mm Hg | Low amplitude, continuous baseline oscillation  
a wave = RVEDP  
v wave = RA filling |
| RV       | RVSP = 15-30 mmHg  
RVEDP = 2-8 mmHg | Steep upstroke to peak  
RVSP  
Sharp downstroke to  
PVEDP  
No dicrotic notch |
| PA       | PASP = 15.30 mmHg  
= RVSP | Steep upstroke to peak  
PASP  
Sharp downstroke to PADP  
Dicrotic notch is present |
| PADP     | = 8-15 mmHg  
> RVEDP | |
| PAMP     | = 10-15 mmHg  
= \( \frac{\text{PASP} + 2\text{PADP}}{3} \) | |
| PAWP     | = 4-12 mmHg | Low amplitude, oscillation (LA waveform)  
a wave = LVEDP  
v wave = LA filling  
= \( \frac{\text{LAP}}{\text{LVEDP}} \) |
DESCRIPTION

The pulmonary artery catheter is a flexible balloon tipped, flow directed catheter. There are two types, the Swan-Ganz catheter for Thermodilution output studies and Berman catheter, of which there are two types. The Berman Angio is for taking angiograms and there is also the type that we are concerned with here, the Berman Wedge.

USES

- Assessment of ventricular function
- Guide for therapy
- Means of evaluating therapy
LUMENS

Swan Ganz Catheter

Distal Lumen - Opens at the tip of the catheter and measures pulmonary artery and pulmonary artery wedge pressures.

Balloon Lumen - Ends 1 cm proximal to the distal lumen. The balloon is made of thin latex material; it surrounds but does not cover the catheter tip. The inflated balloon allows the catheter to advance in a forward direction.

Thermistor Lumen - Terminates at the thermosensitive bead on the surface of the catheter about 4 cm from the distal tip. It contains two fine insulated wires used to measure cardiac output using the thermodilution method.

Proximal Lumen - Opens 30 cm from the distal tip and measures right atrial pressure and/or to infuse solutions. It is the port used for injection when performing cardiac outputs by thermodilution.

Pacing Lumen - Found in the 8.5 french catheter. It is a lumen that can incorporate pacing wires, infuse IV solutions, or monitor right ventricular function.

Berman Wedge

Distal Lumen - Opens at the tip of the catheter and measures pulmonary artery and pulmonary artery wedge pressures.

Balloon Lumen - Ends 1 cm proximal to the distal lumen. The balloon is made of thin latex material; it surrounds but does not cover the catheter tip. The inflated balloon allows the catheter to advance in a forward direction.
## PORTS

### Swan Ganz Catheter

<table>
<thead>
<tr>
<th>Lumen</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal Lumen / Pulmonary Artery</td>
<td>Connected to pressure tubing and a transducer by which pulmonary artery systolic, diastolic and wedge pressure Port can be measured. Mixed venous blood samples are withdrawn from this lumen.</td>
</tr>
<tr>
<td>Balloon Lumen</td>
<td>Connects to a syringe for inflating air into the balloon, then Port be locked closed</td>
</tr>
<tr>
<td>Thermistor Lumen / Coupling port</td>
<td>Connects thermistor wires to a bedside computer (HP monitor) and is used to measure cardiac output and blood temperature.</td>
</tr>
<tr>
<td>Proximal Lumen / Right Atrial Port</td>
<td>Connects via pressure tubing to a transducer to measure central venous pressure (CVP). Injectates to measure cardiac output are installed through this port. It can also serve as a line for IV infusions.</td>
</tr>
</tbody>
</table>

### Berman Wedge

<table>
<thead>
<tr>
<th>Lumen</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal Lumen / Pulmonary Artery</td>
<td>Connected to pressure tubing and a transducer by which pulmonary artery systolic, diastolic and wedge pressure Port can be measured. Mixed venous blood samples are withdrawn from this lumen.</td>
</tr>
<tr>
<td>Balloon Lumen</td>
<td>Connects to a syringe for inflating air into the balloon.</td>
</tr>
</tbody>
</table>
THE PHYSIOLOGY OF CENTRAL VENOUS CATHETERS

- **Central venous pressure (CVP)** is a good approximation of **right atrial pressure**, which in turn is a major determinant of **right ventricular end diastolic volume** (or the *preload* of the right ventricle.) Remember that the *preload* is a reflection of the resting myocardial fiber length, which is also the ventricular end diastolic volume.

- So...

**CVP ~ right atrial pressure ~ right ventricular end diastolic volume (*preload*)**

Recall that right atrial pressure (and thus central venous pressure) is a reflection of:
1. Cardiac function
2. Venous return to the heart

Knowing the status of these two variables is very important when taking care of critically ill patients. However, these two variables are interrelated and influence one another, so this can make it *very difficult* to interpret how one (or both) may be influencing the central venous pressure we are looking at!

Let’s go back to the FRANK-STARLING CURVE (just for a minute...)

This curve looks at right atrial pressure (or *preload*) in relation to cardiac output. As you can see from the graph, there can be vastly different cardiac output for the same right atrial pressure. How is this possible? Recall the 4 major determinants of cardiac function:

1. Preload
2. Afterload
3. Heart rate
4. Contractility
If the right atrial pressure (preload) stays constant, cardiac output can still be influenced by changes in the other three factors. So while CVP alone cannot tell the whole story, it can give a good approximation of the hemodynamic status and right-sided cardiac function of the patient. Furthermore, if the patient is healthy and has good heart and lung function, the central venous pressure can provide a good estimate of the cardiac function of the left side of the heart as well.

**Normal CVP (RA) Waveforms**

The central venous waveform seen on the monitor reflects the events of cardiac contraction; the central venous catheter “sees” these slight variations in pressure that occur during the cardiac cycle and transmits them as a characteristic waveform. There are three positive waves (a, c, and v) and two negative waves (x and y), and these correlate with different phases of the cardiac cycle and ECG.
• **+ a wave**: This wave is due to the increased atrial pressure during right atrial contraction. It correlates with the P wave on an ECG.

• **+ c wave**: This wave is caused by a slight elevation of the tricuspid valve into the right atrium during early ventricular contraction. It correlates with the end of the QRS segment on an ECG.

• **- x descent**: This wave is probably caused by the downward movement of the ventricle during systolic contraction. It occurs before the T wave on an ECG.

• **+ v wave**: This wave arises from the pressure produced when the blood filling the right atrium comes up against a closed Tricuspid Valve. It occurs as the T wave is ending on an ECG.

• **- y descent**: This wave is produced by the tricuspid valve opening in diastole with blood flowing into the right ventricle. It occurs before the P wave on an ECG.

1. "a" Positive wave – follow ECG "P" wave
2. "x" Negative wave – follow ECG "P" wave
3. "c" Positive wave – follow ECG "QRS" wave
4. "v" Positive wave – follow ECG "T" wave
5. "y" Negative wave – follow ECG "T" wave

### Pathologic CVP Waveforms

Variations on the normal central venous waveform can provide information about cardiac pathology. For example.....

• **In atrial fibrillation**, a waves will be absent, and in **atrioventricular disassociation**, a waves will be dramatically increased ("cannon waves") as the atrium contracts against a closed tricuspid valve.

• **In tricuspid regurgitation**, the c wave and x descent will be replaced by a large positive wave of regurgitation as the blood flows back into the right atrium during ventricular contraction. This can elevate the mean central venous pressure, but it is not an accurate measurement. A better way of estimating CVP in this case would be to look at the pressure between the regurgitation waves for a more accurate mean.

• **In cardiac tamponade**, all pressure will be elevated, and the y descent will be nearly absent.
ATRIAL PRESSURE

1. (a) wave:
   - Small pressure rise that is produced by the action of atrial systole
   - After (p) wave.

2. (x) descent:
   - A decline in pressure, reflects a decrease in atrial volume during atrial relaxation (immediately following systole).

3. (c) wave:
   - Appear as a distinct wave, as a notch on the (a) wave, or may be absent altogether.
   - Reflects a slight increase in atrial pressure due to (TV, MV) closure.
   - Corresponds to (RS-T junction).

4. (x) descent:
   - Is produced by a downward pulling of the septum during ventricular systole.

5. (v) wave:
   - Is produced by an increase in atrial pressure by atrial filling + ventricular systole, which causes the closed (TV, MV) leaflets to bulge back into the atrium.
   - In the (T-P interval).

6. (y) descent:
   - Is produced by the opening of the (TV, MV) + emptying of the atrium into the ventricle.

Pressure Ranges: mmHg

<table>
<thead>
<tr>
<th>RA:</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>6</td>
<td>1-5</td>
</tr>
<tr>
<td>v</td>
<td>5</td>
<td>2-8</td>
</tr>
<tr>
<td>m</td>
<td>3</td>
<td>1-5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LA:</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>10</td>
<td>4-16</td>
</tr>
<tr>
<td>v</td>
<td>13</td>
<td>6-21</td>
</tr>
<tr>
<td>m</td>
<td>8</td>
<td>2-12</td>
</tr>
</tbody>
</table>
VENTRICULAR PRESSURE

Systole ⇒ (QT Interval)

1. Isovolumic Contraction:
   - (TV + PV, MV + AV) are closed.
   - Refers to the increase in pressure due to ventricular muscle contraction without change in ventricular volume.

2. Rapid Ejection:
   - Occurs when (PV + AV) are opened due to ventricular pressure rise.

3. Reduced Ejection:
   - Caused by a drop in ventricular pressure.
   - Some blood is still being pumped into PA, AO.

Diastole ⇒ (TQ Period)

4. Isovolumic Relaxation:
   - Sharp decline in pressure due to muscle fibers relaxing + losing tension.
   - (TV + PV, MV + AV) are closed.

5. Early Diastole:
   - Ventricular diastolic pressure falls below (RA, LA) pressure.
   - (TV, MV) open + passive filling occur.

6. Atrial Systole:
   - Follows early diastole + forces an additional volume of blood into the ventricle.
   - It is seen by as increase in pressure, termed (atrial kick, or “a” wave).
7. End-Diastole:

- The period of time following (a wave), just before systolic pressure rise occurs.
- It is the ventricular end-diastolic volume that determines the extent of the fiber shortening (FS) + the subsequent stroke volume (SV) according to Starling’s Law.
- With (QRS Complex).

Pressure Ranges: mmHg

<table>
<thead>
<tr>
<th></th>
<th>RV:</th>
<th></th>
<th>LV:</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>25</td>
<td>15-30</td>
<td>130</td>
</tr>
<tr>
<td>edp</td>
<td>4</td>
<td>1-8</td>
<td>9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>edp</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>RV:</td>
<td>4</td>
<td>1-8</td>
</tr>
<tr>
<td>LV:</td>
<td>9</td>
<td>5-12</td>
</tr>
</tbody>
</table>
ARTERIAL PRESSURE

1. Systolic:

- Begins with the opening of the (PV, AV), resulting in the ejection of blood into the (PA, AO).
- On the pressure tracing, this is seen as a sharp rise in pressure, followed by a decline in pressure as the volume decreases.
- When the (RV, LV) pressure falls below the level of the (PA, AO) pressure, the (PV, AV) snap shut. This sudden closure of the valve leaflets produces a small rise on the downslope of the (PA, AO) pressure + is termed the (dicrotic notch).
- Correspond to ventricular depolarization (QT interval).

2. Diastolic:

- Follows closure of (PV, AV).
- During this time, run off to the system occurs without any further blood flow from the ventricle until the next systole.

Pressure Ranges: mmHg

<table>
<thead>
<tr>
<th></th>
<th>PA:</th>
<th>AO:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>15-30</td>
<td>100-140</td>
</tr>
<tr>
<td>D</td>
<td>5-12</td>
<td>60-90</td>
</tr>
<tr>
<td>M</td>
<td>9-16</td>
<td>70-105</td>
</tr>
</tbody>
</table>
1. Pulsus Tardus:

- Aortic stenosis slows the upstroke (making it tardy "Tardus").

2. Corrigan's Pulse:

- Corrigan's pulse is seen in Aortic regurgitation. It is termed water-hammer because it feels bounding. It shows a rapid upstroke followed by rapid collapse early in diastole. No dicrotic notch is seen, because the Aortic valve does not close completely.

3. Pulsus bisferiens:

- It is a double humped arterial pulse. Both percussion and tidal waves are distinct.
- Bisferiens pulse is associated with HOCM/IHSS and AR.

4. Pulsus Alternans:

- Pulsus alternans is alternating systolic BP, even though heart rate is regular.
- In alternating pulse; the heart is so weak that it seems to rest every 2nd beat. It is an Ominous sign; can be seen with heart failure, myocardial depression.

5. Pulsus bigeminus:

- It is caused by ventricular bigeming.
Pathologic Right Atrial Pressure

Causes of decreased pressure:
1. Hypovolemia
2. Venodilation
3. Decreased venous return.

Causes of increased pressure:
1. Hypervolemia
2. Impedance to left atrial emptying:
   a. Mitral stenosis / regurgitation
   b. LV failure
   c. Aortic stenosis
   d. Pericardial tamponade
   e. Systemic hypertension
3. Increased intrathoracic pressure (PEEP, tension pneumothorax)

Pathologic Right Ventricular Pressure

Causes of decreased pressure:
1. Hypovolemia
2. Venodilation
3. Decreased venous return

Causes of increased pressure:
1. Hypervolemia
2. Right ventricular failure
3. Impedance to RV emptying:
   a. Pulmonic stenosis
   b. Pulmonary hypertension
   c. Mitral stenosis
   d. LV failure
   e. Aortic stenosis
   f. Pericardial tamponade
4. Increased intrathoracic pressure (PEEP, tension pneumothorax)
Pathologic Pulmonary Artery Pressure

Causes of decreased pressure:
1. Hypovolemia
2. Venodilation
3. Decreased venous return

Causes of Increased pressure:

1. Hypervolemia
2. Hypoxic pulmonary vasoconstriction.
3. Impedance to pulmonary blood flow.
   a. Interstitial pulmonary edema
   b. Mitral stenosis / regurgitation
   c. LV failure
   d. Aortic stenosis
   e. Pericardial tamponade
4. Increased intrathoracic pressure (PEEP, tension pneumothorax)

Pathologic Pulmonary Capillary Wedge Pressure

PCWP

Causes of decreased pressure:
1. Hypovolemia
2. Venodilation
3. Decreased venous return

Causes of increased pressure:
1. Hypervolemia
2. Impedance to left atrial emptying:
   a. Mitral stenosis / regurg
   b. LV failure
   c. Aortic stenosis
   d. Pericardial tamponade
   e. Systemic hypertension
3. Increased intrathoracic pressure (PEEP, tension pneumothorax)
# Normal Oxygen Saturation

<table>
<thead>
<tr>
<th>Site</th>
<th>Average</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVC</td>
<td>74%</td>
<td>67 – 83%</td>
</tr>
<tr>
<td>IVC</td>
<td>78%</td>
<td>65 – 87%</td>
</tr>
<tr>
<td>RA</td>
<td>75%</td>
<td>65 – 87%</td>
</tr>
<tr>
<td>RV</td>
<td>75%</td>
<td>67 – 84%</td>
</tr>
<tr>
<td>PA</td>
<td>75%</td>
<td>67 – 84%</td>
</tr>
<tr>
<td>LA</td>
<td>95%</td>
<td>92 – 98%</td>
</tr>
<tr>
<td>LV</td>
<td>95%</td>
<td>93 – 98%</td>
</tr>
<tr>
<td>SA</td>
<td>95%</td>
<td>92 – 98%</td>
</tr>
</tbody>
</table>

Have you noticed that the SVC saturation is lower than IVC saturation?

**Why?**
VALVULAR HEMODYNAMICS

Normal Values for Valve Areas

MITRAL VALVE

The normal adult mitral valve orifice area is approximately 4 to 6 cm$^2$.

A decrease in valve area causes an increase in left atrial pressure and a diastolic pressure gradient between the left atrium and the left ventricle. The gradient is determined by the valve area and the flow through the valve. The left atrial pressure and transvalvular pressure gradient are only minimally elevated when the valve area exceeds 2 cm$^2$.

However, a valve area of 1 cm$^2$ or less is usually considered to be a severe stenosis as it is associated with a significant pressure gradient across the valve and a pressure overload of the left atrium, pulmonary vasculature and the right ventricle.

AORTIC VALVE

The normal adult aortic valve orifice area is 3 to 4 cm$^2$.

A reduction in area is accompanied by a progressive increase in left ventricular systolic pressure. Elevation of the systolic pressure imposes a pressure overload on the left ventricle, which compensates by an increase in ventricular wall thickness.

Symptoms of aortic stenosis occur only when the valve area has reached an area of 1.0 cm$^2$ and smaller.
TRICUSPID VALVE

The normal adult tricuspid valve orifice area is 7.0 cm$^2$.

The area of the tricuspid valve in significant stenosis is usually less than 1.5 cm$^2$ at which time impairment of right ventricular filling occurs. In severe stenosis the valve area is less than 1.0 cm$^2$.

Elevation of the mean right atrial pressure above 10 mmHg usually produces peripheral oedema.

PULMONARY VALVE

The definitive diagnosis for pulmonary valve stenosis is the measurement of systolic differences between the pulmonary artery and right ventricle and the cardiac output.

The degree of stenosis is frequently classified on the basis of the peak-to-peak pressure differences: less than 50 mmHg is mild; 50 to 100 mmHg is moderate; and 100 mmHg is severe.
PRESSURE GRADIENTS ACROSS STENOSIS

Measuring pressure gradients across stenotic valves is an important process in determining the need for surgical intervention, particularly when the hemodynamics, as measured by noninvasive means, are in question. The valve orifice area can often be estimated by a formula that was developed by Dr. Richards Gorlin if the mean pressure gradient, the cardiac output, and the systolic ejection time are known, and if the patient is not in low cardiac-output state:

\[
\text{Valve Orifice Area (VOA) in cm}^2 = \frac{\text{Cardiac Output (L/min)}}{44.3 \times (C) \times \text{Heart Rate} \times (\text{SEP or DFP}) \times \sqrt{\Delta P \text{ (mm Hg)}}}
\]

Where SEP is the systolic ejection period in aortic stenosis (length of time blood is ejected from LV every beat); DFP is the diastolic filling period in mitral stenosis (length of time blood fills LV every beat); and DP is the mean pressure gradient. The constant \( C = 0.85 \) is factored into the equation in mitral stenosis.

The Hakki formula can be substituted if the heart rate is within normal range:

Hakki x 1.35 if HR \(<\) 75bpm in MS
Hakki x 1.35 if HR \(\geq\) 90bpm in AoS

\[
\frac{\text{Cardiac Output (L/min)}}{(\text{Hakki}) \text{ Valve Orifice Area (cm}^2)} = \frac{\text{\sqrt{pressure gradient (mm Hg)}}}{\text{ }}
\]

The Angel correction mandates that the above result be divided by 1.35 for a heart rate of less than 75 beats per minute in the setting of mitral stenosis, or greater than 90 beats per minute in the setting of aortic stenosis.
VALVULAR STENOSIS

A stenotic cardiac valve predominantly affects the chamber immediately upstream from it.

Although other cardiac chambers further upstream may also be affected, these effects are indirect, resulting from altered conditions in the chamber immediately upstream from the stenotic valve.

The reduced orifice areas of a stenotic valve presents an obstruction to normal forward blood flow requiring the generation of an abnormally increased pressure gradient across the valve. This, in turn, requires an increased cavity pressure in the chamber upstream from the stenosed valve.

The stenosed valve therefore, places a pressure load on the upstream chamber, requiring the upstream chamber to generate an increased pressure.

This will cause a pressure drop (gradient) between the proximal chamber or vessel beyond the stenosed valve. This gradient will only be present when blood is flowing across the valve; during systole at the aortic and pulmonary valves, and during diastole at the mitral and tricuspid valves.

Valvular stenosis, which can be either congenital or acquired, does not develop acutely. Therefore, the affected cardiac chambers can adapt to the haemodynamic load over time.

The severity of the stenosis is largely assessed by the size of the gradient. However, this requires quantification.

The variables that govern the magnitude of the gradient are:

a.) The volume of blood flowing during each cardiac cycle (i.e. the cardiac output divided by the heart rate);

b.) The time available for blood flow during each cycle (i.e. the proportion of each cycle occupied by diastole – for the mitral valve, or systole – for the aortic valve);

c.) The area of the valve orifice – which is the variable we want to know.

These variables are interrelated by the following mathematical expression:

\[ \text{Flow} = \text{Area} \times \text{Velocity} \]
This relationship, which is merely an expression of conservation of volume, has two practical consequences:

1. At a given valve orifice area, an increase in flow rate requires a **direct** increase in flow velocity.
2. At a constant flow rate, a change in orifice area requires an **inverse** change in flow velocity.

Thus, either a decrease in orifice area or an increase in flow rate requires an increase in the flow velocity.

These factors are allowed for in the “Gorlin” formula, which is used to calculate valve area.

**Valve Area Calculation**

\[
\text{Area (cm}^2\text{)} = \frac{\text{Valve flow (ml/sec)}}{K \times C \times \sqrt{\text{MVG}}}
\]

\( \text{(MVG = Mean Valve Gradient [mmHg])} \)

Where \( K = 44.3 \) which is a derived constant by Gorlin and Gorlin

\( C = \) is an empirical constant that is 1 for **semilunar** valve and the tricuspid valve and 0.85 for the **mitral** valve.

Valve flow = is counted in ml/sec during diastolic or systolic flow period.

Therefore:

**Mitral Valve Flow**

\[
\text{CO (ml / min)} = \frac{\text{MVF}}{\text{Diastolic filling period (sec / min)}}
\]

Where:

Diastolic filling period (sec / min) = diastolic period (sec / beat) x heart rate (HR).
**Aortic Valve Flow**

\[
\text{CO (ml/min)} = \quad \text{Systolic filling period (sec/min)}
\]

Where systolic filling period (sec/min) = systolic period (sec/beat) x heart rate (HR).

**We can now calculate the valve areas:**

**Mitral Valve Area**

\[
\text{Mitral Valve Flow} = \quad \text{Mitral Valve Flow} = 0.85 \times 44.3 \times \sqrt{\text{gradient}}
\]

**Aortic Valve Area**

\[
\text{Aortic Valve Flow} = \quad \text{Aortic Valve Flow} = 1.0 \times 44.3 \times \sqrt{\text{gradient}}
\]

The above formulae are the classical formulae for calculating the area of stenotic valves as derived by Gorlin.

Due to the somewhat tedious nature of calculating flow across the valve and the time available for flow to take place, a simplified formula, the ‘Hakki’ formula is used. It gives reasonable results for calculating both mitral and aortic valve areas and has the merit of simplicity.

It is:

\[
\text{Valve Area} = \frac{\text{Cardiac output in liters/min. divided by the square root of the pressure gradient (the mean mitral gradient and either the peak systolic gradient or the mean gradient across the aortic valve)}}{\text{\sqrt{Pressure Gradient}}}
\]
AORTIC STENOSIS

The normal orifice area of the aortic valve is 3 to 4 cm$^2$. The aortic valve can become significantly narrowed prior to the onset of symptoms or even prior to hemodynamic significance.

### Aortic Valve Orifice Areas

<table>
<thead>
<tr>
<th>Orifice Area</th>
<th>Area (cm$^2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal aortic valve</td>
<td>3-4 cm$^2$</td>
</tr>
<tr>
<td>Area</td>
<td></td>
</tr>
<tr>
<td>Mild stenosis</td>
<td>1.1-1.3 cm$^2$</td>
</tr>
<tr>
<td>Moderate stenosis</td>
<td>0.8-1.0 cm$^2$</td>
</tr>
<tr>
<td>Severe stenosis</td>
<td>&lt;0.8 cm$^2$</td>
</tr>
</tbody>
</table>

The Gorlin formula can be used to estimate the valve orifice area, but may be inaccurate in severe aortic stenosis with low-output states. The accuracy of the formula is flow dependent and will result in small orifice areas, even with low gradients if the flow across the aortic valve is low. To distinguish between “pseudostenosis” and true stenosis, maneuvers to increase the cardiac output can be employed, such as exercise, dobutamine, or nitroprusside. In patients with pseudostenosis, the valve orifice area will increase as flow increases, but it will not change significantly in patients with severe aortic stenosis.

The most accurate method for measuring aortic valve gradients is by obtaining simultaneous pressure measurements from the left ventricle (LV) and the ascending aorta. This method allows for the calculation of the mean gradient by direct measurement from both recordings. The two catheters required for this, however, necessitate dual arterial access. A double-lumen pigtail catheter may also be used but is not commonly available. Alternatively, a long arterial sheath can be placed in the descending thoracic aorta, and the pressure measured from the side descending thoracic aorta, and the pressure measured from the side port. The femoral artery pressure is also often substituted by this measurement. The peak femoral artery pressure is usually higher than the peak aortic root pressure due to reflected pressure waves seen in the periphery, thus using the femoral artery results in underestimation of the pressure gradient. This can be somewhat compensated by measuring the pressure difference between the catheter at the ascending aorta and the side arm of the femoral artery sheath, and subtracting the difference.

A more commonly utilized method involves pullback of the catheter from the LV into the ascending aorta. This technique yields a “peak-to-peak” gradient between the maximum aortic pressure and the maximum LV pressure. Each of these peaks occurs at different points in time, however, and this measurement is only an estimate of the mean gradient. In addition, in patients with severe aortic stenosis, the catheter itself may take up a significant fraction of the orifice area, resulting in worsened stenosis and increased gradients.
Self-Assessment

Calculating Valve Area in Aortic Stenosis

68-year-old male:

Cardiac output (CO) = 4,800 mL/min
Heart Rate (HR) = 80 beats per minute
Systolic ejection period (SEP) = 0.35
Mean AO-ventricular gradient (P) = 80 mmHg
**Gorlin formula:**

\[ \text{Valve Orifice Area (VOA)} = \frac{\text{CO}}{(\text{HR})(\text{SEP})} \times 44.3 \left( C \right) \sqrt{ \Delta P } (\text{mm Hg}) \]

\[ 4,800 / (80) (0.35) \]

\[ \text{VOA} = \frac{4,800}{(80)(0.35)} = 0.4 \text{ cm}^2 \]

\[ 44.3 (\sqrt{\Delta 80}) \]

**Hakki formula:**

\[ \text{CO (L/min)} \]

\[ \text{VOA} = \frac{\text{CO}}{\sqrt{\Delta P}} \]

\[ 4.8 \]

\[ \text{VOA} = \frac{4.8}{\sqrt{80}} = 0.5 \text{ cm}^2 \]

\[ (\sqrt{\Delta 80}) \]
MITRAL STENOSIS

The normal mitral VOA is 4 to 6 cm\(^2\). Significant narrowing can occur prior to hemodynamic compromise. When the valve area falls to approximately 2.0 cm\(^2\), the left atrial pressures will start increasing to maintain cardiac output. Valve areas less than 1.0 cm\(^2\) may require some intervention.

Mitral Valve Orifice Areas

<table>
<thead>
<tr>
<th>Normal mitral valve orifice area</th>
<th>4-6 cm(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild stenosis</td>
<td>1.6-2.0 cm(^2)</td>
</tr>
<tr>
<td>Moderate</td>
<td>1.1-1.5 cm(^2)</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;1.0 cm(^2)</td>
</tr>
</tbody>
</table>

The gradient across the mitral valve is typically measured with simultaneous measurements of left ventricular (LV) and pulmonary capillary wedge (PCW) pressures as a surrogate for left atrial (LA) pressure. The Gorlin equation is also used in this instance, except that the diastolic filling period is used in lieu of the systolic ejection period used in aortic stenosis (AS), and that an empiric constant of 0.85 is added to the equation. Concomitant mitral regurgitation with mitral stenosis will affect this calculation, and usually will underestimate the true orifice area.
Self-Assessment

Calculating Valve Area in Mitral Stenosis

37-year-old male:
- Cardiac Output (CO) = 5,000 mL/min
- Heart rate (HR) = 76 beats per minute
- Diastolic filling pressure (DFP) = 0.4
- Mean mitral valve gradient (P) = 20 mmHg

**Gorlin formula:**

Valve Orifice Area (VOA) =

\[
\frac{\text{CO}}{(\text{HR})(\text{DFP})} \quad \frac{44.3}{(C)} \sqrt{\Delta P}
\]

Where \(C = 0.85\) (for the mitral valve)

\[
\frac{5,000}{(76)} (0.4) = \frac{5,000}{(76)} (0.4) = 1.0 \text{ cm}^2
\]

\[
44.3 (0.85) (\sqrt{20})
\]

**Hakki formula:**

\[
\text{VOA} = \frac{\text{CO (L/min)}}{\sqrt{\Delta P}}
\]

\[
\text{VOA} = \frac{5.0}{\sqrt{20}} = 1.1 \text{ cm}^2
\]
Simpler Way to Calculate Gorlin / Hakki

**VALVULAR AREA FORMULAS:**

- Gorlin AVA Formula = flow / $44.5 \sqrt{\Delta P}$
  
  where AO Flow = $\frac{C_o}{(SEP \times HR)}$

- Gorlin MVA Formula = flow / $38 \sqrt{\Delta P}$
  
  where MV Flow = $\frac{C_o}{(DFP \times HR)}$

- **Ao** or **PA** Hakki Formula = $\frac{C_o}{\sqrt{\text{Peak to Peak Gradient}}}$

- **MV** or **TV** Hakki Formula = $\frac{C_o}{\sqrt{\Delta P}}$

- **K for semilunar values** = 44.5

- **K for values atrioventricular** = 38

References:

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

In hypertrophic obstructive cardiomyopathy (HOCM), the obstruction lies below the aortic valve and may be dynamic, with little or no resting gradient. The pullback to measure the peak-to-peak pressure should start at the ventricular apex and proceed through the left ventricular outflow tract (LVOT) and through the valve. In typical HOCM, there will be a gradient from the ventricular apex to the LVOT, but no gradient across the valve. In Yamaguchi variant, no gradient will be noted, although the classic spade-like appearance may be observed with left ventriculography.

![Hypertrophic Obstructive Cardiomyopathy](image)

Pullback from left ventricular apex to left ventricular outflow tract demonstrating an intraventricular gradient consistent with hypertrophic obstructive cardiomyopathy. L. V. In, left ventricular cavity pressure; L. V. Out, left ventricular outflow tract pressure; AO, ascending aortic pressure.

If a significant resting gradient is not appreciated, then provocative maneuvers can be performed to unmask an intraventricular gradient. The most common maneuvers include Valsalva, nitroglycerin administration, isoproterenol administration, amyl nitrite inhalation, or PVC induction. The Brockenbrough sign helps differentiate HOCM from valvular aortic stenosis. In a patient with HOCM, the systemic pressure of the sinus beat following a PVC is lower than that of the sinus beat before the PVC.
Brockenbrough sign. The gradient across the aortic valve is increased in the post-extrasystolic beat, with a reduction in aortic or systemic pressure. This accentuation of the gradient is either small or absent in a fixed obstruction/valvular aortic stenosis. LV, left ventricle; PVC, premature ventricular contraction.

- **Brockenbrough's**, HOCM; PVC”s are usually followed by a compensatory pause. This increases the preload and makes the post PVC beat harder. So this post PVC beat should normally have higher systolic pressure (normal).

- **But in HOCM the opposite happens as shown**

- In As the relationship between LV systole and AO systole is constant. It is not dynamic. The AO systolic will be a constant % of the Peak LV. Post PVC beats in AS will increase the AO pressure just like in normal physiology.
MITRAL REGURGITATION

The hemodynamic hallmarks of mitral regurgitation are increased left atrial pressure and reduced cardiac output. A prominent \( v \) wave is suggestive of, but not specific to, mitral regurgitation.

Severe mitral regurgitation. The \( v \) wave in the pulmonary capillary wedge (PCW) pressure tracing is very prominent in this case, and the \( y \) descent is sharp. LV, left ventricle. (From Topol EJ, ed. *Textbook of cardiovascular medicine*. 2\(^{nd}\) ed. Philadelphia: Lippincott Williams & Wilkins; 2002, with permission.)
Cardiac Tamponade

A pericardial effusion that results in hemodynamic compromise causes tamponade. With regard to hemodynamic measurements, there is eventual diastolic equalization of pressures (right atrial pressure = right ventricular end-diastolic pressure = left atrial pressure = left ventricular end-diastolic pressure) in all cardiac chambers usually associated with pulsus paradoxus (exaggerated inspiratory fall in arterial pressures of greater than 10mmHg), a fall in cardiac output and hypotension. Pulsus paradoxus, however, is neither sensitive nor specific for tamponade, and may be found in constrictive pericarditis, pulmonary embolism, and COPD. On the pressure tracings, there may be a prominent \( \chi \) descent with a blunted \( \gamma \) descent.

Cardiac tamponade, with a large pericardial effusion. Note the diastolic equalization of pressures in the right ventricular and right atrial positions.

**In Cardiac Tamponade will:**

- Shows the elevated venous pressure with a prominent "\( \chi \)" descent. No "\( \gamma \)" descent or square root sign \( \sqrt{\text{ }} \) is usually seen on atrial pressure. Inspiration dramatically reduces with inspiration termed pulsus paradoxus.
- Brachial pulses may disappear or reduce with inspiration termed pulsus paradoxus.
- Tamponade can be either effusive or hemorrhagic. The termed tamponade means that the heart is compressed enough to restrain diastolic filling.
- On Echo the RV and RA chamber may actually collapse during diastolic due to this pericardial compression.
CONSTRUCTIVE VERSUS RESTRICTIVE PHYSIOLOGY

Making the diagnosis of constrictive pericarditis may be difficult. Differentiating between constrictive pericarditis and restrictive cardiomyopathy is even harder. While the usual features of constrictive pericarditis include elevation and equalization of the diastolic pressures in all four cardiac chambers, a "dip and plateau" pattern in the Ventricular pressure tracings, an "M or W" pattern in the atrial tracing, and an elevation of the mean right atrial pressure during inspiration, none of these features are either sensitive or specific to the diagnosis of constriction.

One finding that may help differentiate between constrictive and restrictive pericarditis is respiratory discordance between the left and right ventricular systolic pressures. This would require simultaneous pressure recordings in the left and right ventricles.

Constrictive pericarditis. A classic "W" or "M" pattern is seen in the right atrial pressure tracing

- In Constrictive pericarditis venous pressure will be elevated with prominent "Y" descent is seen on the diastolic portion of the RA, RV and LV pressure.
- This is usually seen in LA also. There is an elevated diastolic plateau on these pressures.
- Rt. and Lt. Sided diastolic pressures equilibrate so that
  RA mean = RV – End diastolic pressure = LV - End diastolic pressure usually = LA mean
• This pattern of dip and plateau is termed a "square root sign" ( √ ).

• Inspiration does not change the systolic pressure, since the rigid case around the heart prevents transmission of the negative intrathoracic pressure.

• Inspiration results in no change in venous pressure or an actual increase, termed (kussmaul's sing)

Constrictive pericarditis. Ventricular interdependence and respiratory variation are seen in this example. LV, left ventricle; PCW, pulmonary capillary wedge; RV, right ventricle.

References:

SHUNT CALCULATION

Shunts can be localized and quantified by using oximetry, indocyanine green dye, and angiography. The most common method used clinically is oximetry. In the location of the shunt, blood usually flows from the left-sided (higher-pressure) chamber to the right-sided (lower-pressure) chamber (left-to-right shunt), with abnormally high oxygen saturation in that chamber and all chambers distal. The level where this "step-up" is detected identifies where the shunt exists. **A step-up is considered significant if it is greater than 7% from the vena cava to the right atrium and greater than 5% from the right atrium to the right ventricle, or greater than 5% from the right ventricle to the pulmonary artery.**

The quantification of shunting is determined by calculating the shunt fraction, which in left-to-right shunts is the ratio of pulmonary to systemic blood flow (Qp/Qs). Oxygen saturations should be drawn in the superior vena cava (SVC), inferior vena cava (IVC), right atrium (three sites), right ventricle (three sites), pulmonary artery, and aorta.

\[
Qp/Qs = \frac{O_2 \text{ Sat (systemic arterial)} - O_2 \text{ Sat (systemic mixed venous)}}{O_2 \text{ Sat (pulmonary venous)} - O_2 \text{ Sat (pulmonary arterial)}}
\]

Multiplying the SVC saturation by 3, adding it to the IVC saturation, then dividing the sum by 4 calculates the mixed venous saturation.

\[
* \text{Mix. V} = \frac{3 \text{ (SVC)} + 1 \text{ (IVC)}}{4}
\]

* The closest value sample of mix. V sat is SVC sat

**Minimal Qp/QS Reliably Detected**

- 1.5 – 1.9 L/min/m² at level of atrium
- 1.3 – 1.5 L/min/m² at level of ventricle
- 1.3 L/min/m² at level of great vessels

Shunts are small if the Qp/Qs is less than 1.5, moderate if the Qp/Qs is between 1.5 to 2.0 L/min/m², and large if the Qp/Qs is greater than 2.0
Self-Assessment

Shunt Calculation

61-year-old male with an atrial septal defect and the following oxygen saturations:

Femoral artery = 98% (systemic arterial and pulmonary venous)
Superior vena cava = 69%
Inferior vena cava = 73%
PA = 80%

\[
\text{MVO}_2 = \frac{(3)(0.69) + (0.73)}{4} = 0.70 \text{ (systemic mixed venous)}
\]

\[
\text{Qp/Qs} = \frac{\text{O}_2 \text{ Sat (systemic arteria)} - \text{O}_2 \text{ Sat (systemic mixed venous)}}{\text{O}_2 \text{ Sat (pulmonary venous)} - \text{O}_2 \text{ Sat (pulmonary arterial)}}
\]

\[
\text{Qp/Qs} = \frac{0.98 - 0.70}{0.98 - 0.80} = 1.55
\]

Remember, Qp/Qs:

< 1.5 = small
1.5 – 2.0 = medium
> 2.0 = large
RIGHT-TO-LEFT SHUNTS

Right-to-left shunts cannot be localized or quantified by using a standard right heart catheterization. Sampling must be done at the pulmonary vein level as well as in the chamber of interest (e.g., left atrium) to determine if a "step-down" in saturation is found. This technique usually necessitates a transseptal puncture.

* L to R shunt flow = ( Qp – Qs) or ( PBF – SBF )

* If the result in ( - ) that means it is R to left shunt

• **PBF** = Pulmonary Blood Flow, PBF= 5 L/min
• **SBF** = Systematic Blood Flow, SBF= 5 L/min
• **SBF** 5: **PBF** 5, so the ration **1:1**
• **L** = Left
• **R** = Right
• \[ \frac{Q_p}{Q_s} = \frac{(A_o \%- S_VC \% \text{ or } M_V \%)}{(P_V \%- P_A \%)} \]

• **PBF** = O₂ Consumption / [ ( PV% - PA% ) x 1.36 x Hgb x 10 ]
• **SBF** = O₂ Consumption / [ ( Ao% - SVC% ) x 1.36 x Hgb x 10 ]
• Shunt Percentage % (PBF-SBF / PBF)

For Bidirectional Shunts use \( Q_{eff} \) \( MVO2/ (PV%-MV\%) \) \( Hgb \times 10 \times 1.36 \)

to be as it is shown (L to R = Q_P - Q_{eff} and R to L = Q_S - Q_{eff})

We will use **SBF** as a replacement of Cardiac Output \( CO_{Fick} \) in shunts

- O₂ Consumption Can be calculated as:
  1. La-Farge equation (138.1 – K1 x (age) + .378 x HR ) x BSA
  2. BSA x 125
  3. 3ml X patient Wight in Kg
  4. O₂ Consumption data table

References:
INTRA-AORTIC BALLOON PUMP (IABP)

Indications and contraindications of IABP placement are listed below. When first introduced in 1962, IABPs were surgically placed. Beginning in 1980, however, the percutaneous approach via the femoral artery replaced the surgical approach as the primary means of insertion. **Balloon size is selected based on the patient's height. Most patients receive a 40-cc balloon. Patients shorter than 64 inches, or taller than 72 inches, require smaller (34cc) or larger (50cc) balloons, respectively.** The IABP can be inserted either through a sheath (8F or 9.5F catheter) or via a sheathless technique.

### INDICATIONS AND CONTRAINDICATIONS FOR INTRA-AORTIC BALLOON PUMP PLACEMENT

<table>
<thead>
<tr>
<th>Indications</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock</td>
<td>Moderate aortic insufficiency (&gt;2+)</td>
</tr>
<tr>
<td>Severe mitral regurgitation</td>
<td>Abdominal aortic aneurysm</td>
</tr>
<tr>
<td>Decompensated aortic stenosis</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>Bilateral lower-extremity PVD</td>
</tr>
<tr>
<td>Refractory ischemia</td>
<td>Significant arteriovenous shunts</td>
</tr>
<tr>
<td>High-Risk PCI</td>
<td>Severe coagulopathy</td>
</tr>
<tr>
<td>Bridge to definitive therapy</td>
<td>Sepsis</td>
</tr>
<tr>
<td></td>
<td>No planned, definitive therapy</td>
</tr>
</tbody>
</table>

PCI, percutaneous coronary intervention; PVD, peripheral vascular disease.

After vascular access has been obtained, the IABP is inserted into the descending thoracic aorta over a guide wire. Fluoroscopic guidance is essential to achieving optimal placement in the aorta. The proximal radiopaque tip should be located just below the subclavian artery or at the level of the carina (Fig.1.) and the distal end should be above the renal arteries (usually at the level of L1-2) and completely out of the sheath. The central lumen is aspirated and flushed with heparinized saline and connected to a pressure transducer. The balloon is then connected to the pump and filled to one-half the volume. Adequate filling and location should be confirmed by fluoroscopy.
Once location is confirmed, the IABP is filled completely and then secured with sutures. Patients are routinely placed on systemic anticoagulation to prevent thromboembolic complications resulting from an indwelling intravascular device. However, manufacturers of IABPs insist systemic anticoagulation is optional.

**Fig. 1.** Optimal positioning of the IABP. **A.** Diagram demonstrating the optimal positioning of the intraaortic balloon pump (IAB) pump (IABP) approximately 2 cm distal to the left subclavian artery. **B:** The radiopaque tip of the IABP is located approximately 2 cm cranial to the left mainstem bronchus at the level of the carina (**double arrowheads**).

**References:**
Optimal adjustments of the timing and triggers results in maximum hemodynamic effects (Fig. 2). Timing of inflation should correlate with the onset of diastole.

**Fig. 2.** Timing of intraaortic balloon pump (IABP) inflation and deflation. **A:** Correct timing. With correct timing of IABP inflation and deflation an augmentation of diastolic and systolic blood pressures is seen. Inflation occurs at the onset of diastole (arrow) and deflation should occur prior to the onset of systole (arrowhead) resulting in decreased aortic end-diastolic and systolic pressures. **B:** Premature inflation. Inflation of IABP prior to dicrotic notch (arrow). This may result in premature aortic valves closure, increased left ventricular (LV) wall stress, aortic regurgitation and increased myocardial oxygen demand. **C:** Delayed inflation. Inflation of IABP following the dicrotic notch (arrow) may result in inadequate accentuation of coronary perfusion. **D:** Premature deflation. Observed as a precipitous drop in the pressure waveform following diastolic augmentation (arrow). Other observations include a suboptimal diastolic augmentation and an assisted systole that is equal or greater than the unassisted systole (arrowhead). This may result in suboptimal after load reduction and increased myocardial oxygen demand. **E:** Delayed deflation. Observed as an assisted end diastolic pressure equal to or greater than the unassisted end diastolic pressure, a prolonged rate of rise of the assisted systole (arrowhead) and a widened diastolic augmentation (double arrow). A lack of after load reduction and an increase in myocardial oxygen demand occurs.

References:
COMMON COMPLICATIONS ASSOCIATED with INTRA-AORTIC BALLOON PUMP COUNTERPULSATION

To properly adjust timing of inflation, the IABP should be placed on an inflation ratio 1:2 to observe augmented and unaugmented beats. The central pressure waveforms is used to guide proper timing. Ideally, the balloon should inflate with the closure of the aortic valve, identified by the dicrotic notch of the central pressure waveform tracing. Deflation should occur with aortic valve opening, which can be timed with the onset of the R wave by ECG tracing. When timed appropriately, the central aortic waveform should have an augmentation pressure greater than the systolic pressure, and a post deflation pressure 10 to 15 mm Hg below the unaugmented diastolic blood pressure.

After IABP insertion, patients should have daily checks of hemoglobin, hematocrit, platelet count, white blood cell count, renal function, and a chest x-ray for placement. Meticulous attention to peripheral pulses and access site are of paramount importance in detecting early vascular complications.

Complications can be divided into vascular and nonvascular. (Table 1) Vascular complications occur in 6% to 25% of cases. Specific complications include limb ischemia, arterial dissection, vascular laceration (requiring surgical repair) and major hemorrhage.

<table>
<thead>
<tr>
<th>Vascular (a6%-25%)</th>
<th>Nonvascular (a 4%-15%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematoma formation</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Arterial dissection</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Vascular laceration</td>
<td>Hemolysis</td>
</tr>
<tr>
<td>Limb ischemia</td>
<td>Groin infection</td>
</tr>
<tr>
<td>Thromboembolic complications</td>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Spinal cord ischemia</td>
<td></td>
</tr>
</tbody>
</table>

aIncidence.

References:
REFERENCES

- Ronnier J. Aviles, Adria W. Messerli – Introductory Guide to Cardiac Catheterization, Lippincott Williams & Wilkins 2004
- Michael D. Gisenhauer & Morton Kern – Invasive Hemodynamic in the Catheterization Laboratory; Remedica 2002

Akram M. Al Ajmi CCT, CRAT, RCIS, FSICP, MSc. (HCM)

For any inquiry and/or ordering a free copy, please do not hesitate to email me at akram.alajmi@gmail.com, or mailing address P.O Box 295275 Riyadh 11351

Follow us at:
- Twitter: @sgctsha, Saudi Arabia
- Facebook: Hemodynamic Manual
- Mobile: +966 544008784