INTERPRETATION OF SWAN-GANZ CATHETER DATA

I. INTRODUCTION:

In a critically ill patient, cardiopulmonary dynamics can change quickly, often with grave consequences. Measurements of such changes can greatly improve our abilities to pinpoint a diagnosis, map out an effective treatment plan and follow the patient's response to treatment. The development and practical application of the flow directed, balloon tipped catheter or Swan-Ganz catheter grew out of advances in the knowledge of human physiology and cardiology and it allows us to more precisely measure the central hemodynamics of critically ill patients. The purpose of this presentation is to give housestaff and medical students a fundamental understanding in the interpretation of the data obtained from Swan-Ganz catheters.

II. INDICATIONS:

The principle indication for Swan-Ganz catheterization is the need for the physician to answer a specific question(s) about a patient that cannot be answered on the basis of the physical exam or other less invasive means. In most instances, the decision to catheterize the patient is precipitated by a failure of therapy chosen on the basis of the available clinical data. Five general indications for insertion are:

A. Evaluation of hypotension of unknown etiology.
B. Evaluation of volume status.
C. Determining cardiogenic from noncardiogenic pulmonary edema.
D. Monitoring the effects of potent vasoactive and inotropic drugs.
E. Evaluation of left and right ventricular function.

APPROACH TO THE PATIENT WITH HYPOTENSION:

\[ \text{BP} = \text{CO} \times \text{SVR} \]

- **HR** × **SV**
- **11 ABNL HR**
- **TACHYARRHYTHMIAS**
- **BRADYARRHYTHMIAS**
- **21 SVR**
- **SEPST**
- **NEUROGENIC**
- **ANAPHYLACTIC**
- **DRUGS**
- **31 CONTINUITY**
- **ACUTE M.I.**
- **MYOCARDITIS**
- **CARDIOMYOPATHY**
- **41 PRELOAD**
- **DEHYDRATION**
- **BLEEDING**
- **CARDIAC TAMponade**
- **MASSIVE PULM. EMBOLUS**
- **Tension P TX**
III. BASIC PRINCIPLES OF FLUID DYNAMICS:

One of the basic principles of fluid flow is that the pressure within a conduit connected by a series of pumps is at a maximum immediately after exiting a pump and progressively decreases until it reaches the next pump. As long as fluid flows in only one direction, the pressure within the conduit will never increase at any point downstream.

1) SERIES OF PUMPS:

![Diagram showing series of pumps and pressure vs distance.]

This principle can be applied to human cardiac physiology. As blood flows from one pump (e.g., RV) to another pump (e.g., LV), the pressure in the artery will be greater than in the capillaries which in turn will be greater than in the veins. An important exception to this rule occurs with severe MR due to transient backflow into the LA and PV. In this case, LA pressure can transiently exceed the pressures in the PV and capillaries. Clinically, this is seen only in severe MR, not mild to moderate MR.

2) NL HEART:

![Diagram showing normal heart with blood flow through the heart chambers and to the lungs.]

IV. MEASUREMENT AND IMPORTANCE OF PCWP:

When the S-G catheter is in the PA with the balloon deflated, the measured pressure is the PA pressure. When the balloon is inflated, blood distal to the catheter no longer flows and instead becomes a continuous column of fluid which transmits pressure from the PV to the catheter tip. This is approximately equivalent to placing the catheter through the lungs and into the PV to measure the pressure at that point.
directly. This pressure is known as the Pulmonary Capillary Wedge Pressure (PCWP). It is a misnomer because it is actually measuring LA pressure and not pulmonary capillary pressure. This is because pressure in the RV is very close to LA pressure. Therefore, one can assume PCWP ≈ LA pressure. An important point to remember is that this column of fluid between points A and B must be continuous. If collapse of the vessels occurs at any point between A and B (e.g., due to intra-alveolar pressure), the pressure at A does NOT equal the pressure at B.

1) PA PRESSURE:

![Diagram of PA Pressure]

2) PCWP:

![Diagram of PCWP]

\[ P_A \equiv P_B \equiv P_{LA} \]

In the absence of MV disease, one can further assume PCWP ≈ LA ≈ LVEDP. Using the Frank Starling curve, LV function can be assessed based on the knowledge of PCWP. This is why one measures PCWP because it is currently the most reliable and accurate method of assessing LV performance without a left heart catheterization.

Disease processes, in which the assumption that PCWP ≈ LA ≈ LVEDP is NOT true, include:

A. Mitral Stenosis - Due to the pressure gradient across
the stenotic valve, PCWP does equal LA pressure but both are > LVEDP.

B. Prolapsing Atrial Myxoma - Due to interference with LA flow.

C. Intra-alveolar Pressure - Usually due to high PEEP.

**FRANK STARLING CURVE**

![Frank Starling Curve Diagram]

V. NORMAL RIGHT HEART TRACING:

A. RA TRACING

1) NL. PRESSURES: RA

0 - 8 mm Hg (Mean)

2) VENT. DIASTOLE (VD):

- Y DESCENT - EARLY VD
  - Opening of TV and
  - Pressure emptying of RA

- A WAVE - LATE VD
  - Atrial contraction
  - After R

3) VENT. SYSTOLE (VS):

- C WAVE - EARLY VS
  - Closure of TV

- X DESCENT - MID. VS
  - Atrial relaxation

- V WAVE - LATE VS
  - Atrial filling by
  - Venous return
  - n.c.
B. **RV TRACING**

NL. PRESSURES:
SYS.: 15-25
DIAS.: 0-8

C. **PA TRACING**

NL. PRESSURES:
SYS.: 15-25
DIAS.: 8-15
MEAN: 10-20

D. **PCWP TRACING**

NL. PRESSURES:
MEAN: 6-12
- Similar to RA tracing because the PCWP tracing is essentially an indirect LA tracing (though the majority of the time, the ejection recording will not be nearly as distinct as in the RA).

**Mean PCWP should always be < PAD and < PAM, unless severe MR is present in which case, PCWP may transiently exceed PAD. **VERY IMPORTANT CONCEPT** **

1. Calculating PCWP value:
   a. It is usually measured as the mean of the peak systolic and diastolic excursions of the PCWP tracing, but this varies from ICU to ICU.
   b. The most important point is that the measurement be made consistently (i.e., in the same manner, everytime) because the trend, in response to therapy, is more important than the absolute value of the PCWP.
   c. Always measure PCWP from a tracing, never off the monitor or by the computer.

2. "Dicrotic Notch":
   a. This is the notch formed by the a-c-v waves. It is due to MV closure.
b. It is also present in the RA (TV closure) and the PA (PvV closure) tracings.

E. RIGHT HEART TRACING

VI. ZONES OF WEST:

Regional lung perfusion is determined by the relationship between pulmonary arterial ($P_A$), alveolar ($P_{ALV}$), and pulmonary venous ($P_V$) pressures. It is based on the fact that $P_{ALV}$ does not change in a vertical fashion within the lung [i.e., in an upright patient, $P_{ALV}$ (apical regions) = $P_{ALV}$ (basilar regions)] but $P_A$ and $P_V$ does. Vascular pressure ($P_A$ and $P_V$) are greatest in the dependent portions of the lungs. Based on these concepts, West divided the lung into 3 zones.

**ZONES OF WEST**

**UPRIGHT:**

- **ZONE 1:** $P_{ALV} > R.P.$
- **ZONE 2:** $P_A > P_{ALV} > P_V$
- **ZONE 3:** $P_A > P_V > P_{ALV}$

**SUPINE:**

When measuring PCWP, the catheter needs to be in zone 3 because, as previously noted, there needs to be a continuous column of blood to ensure PCWP = LA pressure. In zone 1 and zone 2, PCWP measures $P_{ALV}$ because the $P_{ALV}$ causes the blood vessels to collapse in this region.

To ensure proper catheter placement in an ICU patient, a supine, cross table lateral CXR (portable and slightly over penetrated) is useful. The catheter tip should be at or below the level of the LA.
VII. EFFECT OF RESPIRATORY VARIATION ON PCWP

1) SPONTANEOUS RESPIRATIONS:

<table>
<thead>
<tr>
<th>Exhalation</th>
<th>Inspiratory</th>
<th>PCWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pos.</td>
<td>Pos.</td>
<td>NEG</td>
</tr>
</tbody>
</table>

2) MECHANICAL VENTILATION (NO PEEP):

<table>
<thead>
<tr>
<th>Exhalation</th>
<th>Inspiratory</th>
<th>PCWP</th>
</tr>
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<tbody>
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<td>Pos.</td>
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<td>NEG</td>
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Always measure PCWP at END - EXPIRATION

The reason that PCWP is measured at end-expiration is because at end-expiration, the effect of respirations on pulmonary blood flow is at a minimum, regardless of the type of ventilation (spontaneous or mechanical). This is because PAoV and PPa return to zero (or atmospheric) at end-expiration in both forms of ventilation.

Respiratory variation on PCWP is the reason why PCWP should always be measured from a tracing and not from the monitor or from the computer's calculations. This is because the computer is unable to differentiate inspiration from expiration and simply calculates its value for PCWP from the entire tracing.

3) PEEP - PCWP is also measured at end-expiration with PEEP but two important guidelines need to be followed.

a. PEEP < 10 cmH₂O -Usually has no effect on CO and therefore should not PCWP. Calculate as one would for mechanical ventilation.

b. PEEP > 10 cmH₂O - Need to be VERY careful measuring PCWP with PEEP > 10 cmH₂O because PEEP > 10 not only decreases CO, but can convert zones 2 and 3 lung into zones 1 and 2. A good general rule to follow is:

- For every increase of 5 cmH₂O of PEEP over 10 should increase PCWP by a maximum of 2 - 3 cmH₂O (remember to convert cmH₂O to mmHg: 1 cmH₂O = 0.735 mmHg). If PCWP increases by more than 3.
cmH₂O, then it is probably measuring P<sub>ALV</sub>.

** Remember to use your PAD as a guide. If PCWP is suddenly > PAD after increasing PEEP, the PCWP is probably incorrect (usually = P<sub>ALV</sub>).**

4) If the PCWP measurement is crucial in the management of the patient but is difficult to assess due to excessive respiratory variation (e.g., due to severe COPD), it is OK to sedate or even paralyze the patient briefly while he/she is on the ventilator to make the measurement. However, NEVER TURN OFF PEEP to make PCWP measurements because:

a. Patient can develop profound hypoxia within seconds which may be very difficult to reverse.

b. The hemodynamics change after stopping PEEP. Therefore, any measurement off PEEP is invalid because it does not reflect the effect of PEEP on the hemodynamics, which is what one wanted to determine in the first place!

**ABNORMAL TRACINGS**

1) **TRICUSPID REGURGITATION:**

- RA TRACING
- Large V waves
- Can also see C-V waves (combined C and V waves)

2) **TRICUSPID STENOSIS:**

- RA TRACING
- Large A waves
- Increased RA pressure (Syst., Diast., and Mean)

3) **PULMONIC STENOSIS:**

- RV — PA TRACING
- Decrease in sys. pressure from RV to PA due to flow across stenotic valve.

4) **MITRAL REGURGITATION:**

- PA — PCWP TRACING
- Large Y waves in PCWP tracing
- Note that in the PA, peak pressure occurs with QRS complex, but in the PCWP, Y waves occur with or after T<sub>W</sub>.
5) CONstrictive PERICARDITIS:

6) PULMONARY EMBOLISM:

7) PERICARDIAL TAMponade:

- Elevation and equalization of pressures in: RA = RV0
- PAD = PCWP (primarily diastolic equalization).
- Equalization of right and left sided diastolic pressures.
- Decreased max. systolic and increased min. diastolic pressures.
- Pressure Plateau:
  - Y descent is decreased or absent.
  - No early diastolic dip in RV
  - This is unlike constrictive pericarditis which has a prominent Y descent and a RV diastolic dip.

IX. CALCULATING CO, CI, AND SVR:

A. CO - CARDIAC OUTPUT:

1. Thermodilution CO: This is performed by injecting
iced saline through the RA port. A thermister, near the end of the catheter, senses the change in the temperature of the blood. The computer then plots a thermodilution curve and computes the CO from this curve.

2. It is inaccurate in the presence of TR and intracardiac shunts (e.g., ASD or VSD).

3. NL. CO = 3.5 - 7.0 L/min

B. CI - CARDIAC INDEX:
1. CI = CO/BSA where BSA (Body Surface Area) = m². Obtained from a surface area nomogram.
2. NL. CI = 2.8 - 4.2 L/min/m

C. SVR - SYSTEMIC VASCULAR RESISTANCE:
1. \[ SVR = \frac{\text{MAP} - \text{CVP}}{\text{CO}} \times 80 \]
   \[ \text{MAP} = \frac{1}{3} \left[ \text{SBP} + (2 \times \text{DBP}) \right] \text{in mmHg} \]
   \[ \text{CVP} = \text{in mmHg} \]
   \[ \text{CO} = \text{in L/min} \]
2. NL. SVR = 800 - 1200 dyne-sec/cm

X. THE BOTTOM LINE:

\[ \text{PB} \]

\[ \text{RA} \]

\[ \text{RV} \]

\[ \text{PA} \]

\[ \text{PCW} \]

<table>
<thead>
<tr>
<th>TABLE 3-7. DIFFERENTIAL DIAGNOSIS USING A BEDSIDE BALLOON FLOW-DIRECTED (SWAN-GANZ) CATHETER</th>
</tr>
</thead>
<tbody>
<tr>
<td>DISEASE STATE</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td>Septic shock (early)</td>
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<tr>
<td></td>
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<tr>
<td>Volume overload</td>
</tr>
<tr>
<td>Volume depletion</td>
</tr>
<tr>
<td>Noncardiac pulmonary edema</td>
</tr>
<tr>
<td>Pulmonary heart disease</td>
</tr>
<tr>
<td>RV infarction</td>
</tr>
<tr>
<td>Pencardia tamponade</td>
</tr>
<tr>
<td>Papillary muscle rupture</td>
</tr>
<tr>
<td>Ventricular septal rupture</td>
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</tbody>
</table>

RA = right atrium; PCW = pulmonary capillary wedge; RV = right ventricle; PA = pulmonary artery; nl = normal; ↑ = increased; ↓ = decreased.
XI. GUIDELINES FOR THE SAFE USE OF SWAN-GANZ CATHETERS:

1. Keep "wedge" time to a minimum, especially in patients with pulmonary hypertension (preferably 10 to 15 sec).

2. When the balloon is reinflated for recording wedge pressure, the inflation medium (carbon dioxide or air) must be added slowly under continuous monitoring of the pulmonary artery pressure waveform. Inflation must be stopped immediately when the pulmonary artery pressure tracing shows a change in pulmonary wedge pressure.

3. If fluoroscopy is available (as in the cardiac catheterization laboratory), refloat the catheter tip from the central pulmonary artery for each wedge pressure measurement.

4. Careful note of the balloon inflation volume must be made. If "wedge" is recorded with a balloon volume significantly below that indicated on the catheter shaft, pull the catheter gradually into a position in which full or near full inflation volume produces a wedge tracing.

5. Anticipate spontaneous catheter tip migration toward the periphery of the pulmonary bed. To avoid possible damage to the pulmonary artery, monitor the pressure tracing during every balloon inflation.

6. Spontaneous catheter tip migration into wedge position may also induce pulmonary infarction. Continuous or frequent monitoring of the catheter tip pressure is therefore necessary.

7. Do not use liquids for balloon inflation; they may be irretrievable and may prevent balloon deflation.

8. Keep a syringe on the balloon lumen of the catheter to prevent accidental injection of liquids into the balloon.

XII. EXPLANATION OF ABBREVIATIONS:

ASD  - Atrial Septal Defect
BP   - Blood Pressure
BSA  - Body Surface Area
CO   - Cardiac Output
CI   - Cardiac Index
CVP  - Central Venous Pressure
HR   - Heart Rate
IVC  - Inferior Vena Cava
LA   - Left Atrium
LV   - Left Ventricle
LVEDP - Left Ventricular End-Diastolic Pressure
MAP  - Mean Arterial Pressure
MR   - Mitral Regurgitation
MV   - Mitral Valve
PA   - Pulmonary Artery
PV   - Pulmonary Vein
PA   - Arterial Pressure
PV   - Venous Pressure
PALV - Alveolar Pressure
PLE  - Pleural Pressure
PCWP - Pulmonary Capillary Wedge Pressure
PAD  - PA Diastolic Pressure
PAM  - PA Mean Pressure
PAS  - PA Systolic Pressure
PW   - P Wave
PVV  - Pulmonic Valve
PEEP - Positive End-Expiratory Pressure
RA   - Right Atrium
RV   - Right Ventricle
RVD  - RV diastolic Pressure
SV   - Stroke Volume
S-G  - Swan-Ganz
SVR  - Systemic Vascular Resistance
SVC  - Superior Vena Cava
TV   - Tricuspid Valve
T_W - T Wave
VSD - Ventricular Septal Defect

XII. CONCLUSION:

Hemodynamic monitoring involves far more than the ability to insert catheters. If it is to be of any value in patient management, careful attention must be paid to the methods of the measurement. Quality of the hemodynamic measurements is entirely dependent on the accuracy with which they are collected, interpreted, and recorded. It is difficult enough to interpret the meaning of the many physiologic abnormalities critically ill patients may have without adding the additional uncertainty of poorly collected data. And finally, even the best hemodynamic data is worthless if it is not correlated with changes, however subtle, in the clinical condition of the patient. Availability of bedside hemodynamic monitoring should never replace good clinical judgement.