Pulmonary Artery Catheters and their Waveforms

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June 11th, 2014
Overview

- History
- Description
- Indications
- Placement
  - Waveforms and their meanings
- Physiologic theory
- Complications
- Controversies
History

- In 1929, Dr. Forssmann self cathed his R atrium establishing feasibility of the procedure

- PAC developed by Swann and Ganz in 1970

- Built on prior work of pulmonary artery catheterization and flow directed catheterization

- First balloon flotation, flow directed catheter that could be placed at bedside without fluoro
Indications

- monitoring periop high risk patients
- diagnosis of shock & mgmt of fluids and vasoactive meds (eg. mixed shock states, burns)
- diagnosis & mgmt of complex patients with pulmonary hypertension
- monitoring and mgmt of complicated AMI
- diagnosis and mgmt of valvular dysfct, intracardiac shunt, PE, cardiac tamponade
- Contraindications: tricuspid or pulm valve prosthesis or endocarditis, R heart thrombus or tumor; caution with LBBB, coagulopathy
Placement

- Will need 9F sheath, two monitoring setups (CVC, PA), pulmonary artery catheter

- Cannulate the access vein (typically RIJ) via Seldinger, dilated to 9F; verify location with CXR

- Clean site, obtain maximal sterile conditions, open catheter and flush with saline to prevent air embolism

- Have assistant hook up catheter, ensure proper function

- Thread PAC through sheath utilizing plastic sleeve to maintain sterility, at ~30cm inflate balloon
Figure 1. Standard Pulmonary-Artery Catheter.

The pulmonary-artery catheter is generally 110 cm long and 7 to 8 French in diameter. An air-filled syringe (A) is used to inflate the balloon at the catheter tip (inset). An accessory infusion port (B) is present in most catheters and connects to a lumen 30 cm from the catheter tip (arrow). The distal port (C) connects to a lumen at the catheter tip and is used to measure all pressures during catheter insertion. The proximal port (D) connects to an additional lumen 30 cm from the catheter tip and is used to monitor right atrial pressures once the catheter tip is in the pulmonary artery. A thermistor wire extends from the catheter tip to an electronic connector (E) and is used to measure cardiac output by means of thermodilution.
Balloon Inflation Volume
- Appropriate inflation volume is 1.25 – 1.5 cc

VIP Port
- 30 cm from tip
- Located in RA/SVC

Proximal Injectate Port
- 26 cm from tip
- Located in RA
- Transduce Proximal Injectate Lumen – proper waveform is RA

PA Distal Port
- Transduce distal lumen – proper waveform is PA

Thermistor
- 4 cm from tip

Pulmonic Valve

Thermal Filament
- 14 – 25 cm from tip
- Rests between RA and RV
- Should be free floating and avoid endocardial surface
- Should not be in PA

Tricuspid Valve
<table>
<thead>
<tr>
<th>Pressure</th>
<th>Right atrium</th>
<th>Right ventricle</th>
<th>Pulmonary artery</th>
<th>Pulmonary artery wedge</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 mm Hg</td>
<td></td>
<td></td>
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<tr>
<td>10 mm Hg</td>
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<tr>
<td>0 mm Hg</td>
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</tbody>
</table>

20-25cm  30-35cm  40-45cm  50-55cm
Placement

• If difficulty with waveform progression: deflate balloon, withdraw catheter 10cm, re-inflate balloon, and re-advance

• Once a pulmonary artery waveform has been obtained, inflate balloon to obtain a wedge pressure (if wedge appears with <1.25mL, withdraw catheter- it is too deep)

• wedge pressure should be taken at end expiration, regardless of whether the patient is mechanically ventilated or breathing spontaneously

• deflate balloon, PA tracing should appear; if not, withdraw catheter until it does
Calibrations

- once catheter is in appropriate position, calibrate:

  - aspirate sample from distal port - send for SvO$_2$

  - after thermistor is attached, calibrate CO by injecting 10mL crystalloid aliquot via proximal port

  - repeat process for three samples - <10% variation confirms validity

- modern catheters have a thermistor which warms surrounding blood and provides continuous CO (CCO)
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Abbreviation</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central Venous Pressure</td>
<td>CVP</td>
<td>0 – 5 mm Hg</td>
</tr>
<tr>
<td>Pulmonary Artery Wedge Pressure</td>
<td>PAWP</td>
<td>6 – 12 mm Hg</td>
</tr>
<tr>
<td>Cardiac Index</td>
<td>CI</td>
<td>2.4 – 4.0 L/min/m²</td>
</tr>
<tr>
<td>Stroke Index</td>
<td>SI</td>
<td>20 – 40 mL/m²</td>
</tr>
<tr>
<td>Systemic Vascular Resistance Index</td>
<td>SVRI</td>
<td>25–30 Wood Units †</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance Index</td>
<td>PVRI</td>
<td>1–2 Wood Units †</td>
</tr>
<tr>
<td>Oxygen Delivery (Index)</td>
<td>DO₂</td>
<td>520 – 570 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen Uptake (Index)</td>
<td>VO₂</td>
<td>110 – 160 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen Extraction Ratio</td>
<td>O₂ER</td>
<td>0.2 – 0.3</td>
</tr>
</tbody>
</table>

†mm Hg/L/min/m²
Waveforms

- a wave = R atrial contraction
- c wave = closure of tricuspid valve
- v wave = ventricular contraction against closed tricuspid
- x descent is caused by atrial relaxation
- y descent is caused by tricuspid valve opening
Waveform Pathologies

A) Atrial Fibrillation

B) Atrial Flutter

C) Complete AV Block

D) Tricuspid Regurgitation

E) Pericardial Tamponade

F) Constrictive Pericarditis
Waveform Pathologies

- A-fib: no a wave, pronounced y descent
- A-flutter: multiple, sawtoothed a waves
- complete AV block: “cannon” a waves caused by atrial contraction against a tricuspid valve closed by higher ventricular pressures
- tricuspid regurg: steep y descent
- tamponade: exaggerated x, loss of y descent due to diastolic equalization
- constrictive pericarditis: sharp a & v waves with sharp x & y descents
- large v waves seen in mitral regurg, CHF and VSD 2/2 increased atrial pressures
WARNING: at NO point should the PAC be withdrawn with the balloon inflated

Similarly, the balloon should never remain inflated as it can cause pulmonary necrosis
Some Physiology
- "closed pipe" analogy: with balloon inflated, flow = 0
- without flow, a continuous column of blood connects the pulmonary artery and left atrium
- assumes that the balloon has followed flow into zone where pulmonary capillary pressure > alveolar pressure
Assumption 1 (closed pipe assumption)

- if catheter does not migrate into “zone 3” and alveolar pressure > capillary pressure, elevating PAOP

- identified if PA diastolic pressure > PAOP or respiratory variation
Assumption 2 (mitral valve assumption)

- Additionally, we assume LAP = LVEDP
  - holds so long as no obstruction btw LA and LV
  - mitral stenosis, myxoma, mitral regurg will overestimate PAOP
  - PAOP will underestimate LVEDP in aortic regurg
  - LAP will also be > LVEDP when LV is not compliant (LVH, ischemia, restrictive cardiomyopathy)
Assumption 3 (compliance assumption)

- $LVEDP = LVEDV$

- depends on the compliance of the LV
  - compliance changes in LVH, ischemia, infarct
  - changes with increased juxta-cardiac pressure
    - eg. pericardial tamponade, high PEEP
Frank Starling Curves

- Increasing preload creates increased CO
- Giving volume pushes up the LVEDP increasing SV
- Dependent on ventricular compliance
- Addition of inotropes increases the shape of the curve
Cardiac Output

Modified Stewart-Hamiltonon Equation:

\[ CO = \frac{(T_b - T_i) \times V_i \times K}{\int \Delta T_b \times dt} \]

Where \( T_b \) is temp of blood, \( T_i \) is temp of inject ate, \( V_i \) is volume of inject ate and \( K \) is a constant
Cardiac output = \frac{\text{Quantity of Indicator}}{\int_0^\infty \text{Concentration of Indicator} \cdot dt}

Figure 29

Normal Cardiac Output
4.33 l/min

Figure 30

Low Cardiac Output
2.50 l/min

Figure 31

High Cardiac Output
8.21 l/min
Measuring CO

- Historically, cold (0-5°C) injectates were used (typically crystalloid solutions)
  - can cause reflex bradycardia
- Sensitivity now allows use of room temperature injectate with equal fidelity
  - signal-to-noise ratio still highest with cold injectates, may have a role in hypothermic patients
Measuring CO

- Falsely low CO readings (increase in time, decrease in temp difference)
  - injectate is warmer than measured, more dye is injected than anticipated, dye is injected too rapidly, injection occurs during PPV, left to right shunt
- Falsely elevated CO reading (decrease in time, increase in temp difference)
  - injectate is cooler than measured, less dye is injected than allotted, catheter has migrated distally, right to left shunt
Mixed Venous $O_2$ Saturation ($SvO_2$)

**Low $SvO_2$:**
- Decreased delivery
  - low CO
  - low Hgb
  - low $SaO_2$
- Increased consumption
  - activity
  - fever
  - reperfusion
  - hyperthyroidism

**High $SvO_2$:**
- Decreased consumption
  - hypothermia
  - analgesics/ paralysis
  - hypothyroidism
  - late sepsis
  - AV shunting
  - cyanide poisoning

Sampling error occurs if $Pc$ blood taken; avoided by withdrawing catheter if wedges with <1.25mL and withdrawing 1mL of blood over 20s from distal port. Send for oximeter analysis not ABG.
Complications

- overall rate as high as 25%

- Catheter insertion:
  - bleeding, infection, PTX, thrombosis

- PA cath:
  - catheter knotting, arrhythmia (heart block), cardiac perforation with tamponade, PA rupture (higher in pHTN)

- complication rate goes up after 72 hours (infx, VTE)
“...perhaps the most dangerous complication of PA catheters is the misinterpretation of information”

–Mihae Yi, editor, Civetta Critical Care
Does PAC information improve patient outcomes?
Figure 2. Odds Ratio (PAC vs No PAC) for Mortality of RCTs Evaluating the Safety and Efficacy of the PAC

<table>
<thead>
<tr>
<th>Source</th>
<th>No. Deaths/Total No. of Patients</th>
<th>PAC</th>
<th>No PAC</th>
<th>Odds Ratio (95% CI)</th>
<th>Favors PAC</th>
<th>Favors No PAC</th>
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</thead>
<tbody>
<tr>
<td>Schultz et al., 1985</td>
<td>1/35</td>
<td>10/35</td>
<td>0.11 (0.02-0.63)</td>
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<tr>
<td>Shoemaker et al., 1988</td>
<td>11/58</td>
<td>7/30</td>
<td>0.76 (0.27-2.15)</td>
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<td>Isaacson et al., 1990</td>
<td>1/49</td>
<td>0/53</td>
<td>0.18 (0.02-1.42)</td>
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<td>Berlauk et al., 1991</td>
<td>1/66</td>
<td>2/21</td>
<td>1.10 (0.29-4.22)</td>
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<td>Guyatt, 1991</td>
<td>10/16</td>
<td>9/17</td>
<td>1.04 (0.11-9.95)</td>
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<td>Bender et al., 1997</td>
<td>1/51</td>
<td>1/53</td>
<td>2.38 (0.35-16.29)</td>
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<td>Valentine et al., 1998</td>
<td>3/60</td>
<td>1/60</td>
<td>1.01 (0.58-1.76)</td>
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<td>Bonazzi et al., 2002</td>
<td>0/50</td>
<td>0/50</td>
<td>1.06 (0.83-1.35)</td>
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<td>Rhodes et al., 2002</td>
<td>46/95</td>
<td>50/106</td>
<td>0.93 (0.68-1.26)</td>
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<td>Sandham et al., 2003</td>
<td>163/997</td>
<td>155/997</td>
<td>1.25 (0.78-2.02)</td>
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<tr>
<td>Richard et al., 2003</td>
<td>199/338</td>
<td>208/343</td>
<td>1.13 (0.87-1.47)</td>
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<tr>
<td>ESCAPE, 2005</td>
<td>45/215</td>
<td>38/218</td>
<td>1.11 (0.87-1.47)</td>
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<tr>
<td>Harvey et al., 2005</td>
<td>346/506</td>
<td>333/507</td>
<td>1.11 (0.87-1.47)</td>
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<tr>
<td>Combined</td>
<td>1/40</td>
<td>1/35</td>
<td>1.04 (0.90-1.20)</td>
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</tbody>
</table>

CI indicates confidence interval; NA, not available; PAC, pulmonary artery catheter; RCT, randomized clinical trial. P for heterogeneity = .36.
Is the lack of improvement caused by PAC complications?

- Complication rate the same but twice as many catheters placed in PAC group
- Increased arrhythmias with PAC but no deaths due to catheter insertion
Does PAC use affect therapy?

- Technically the PAC only monitors and is not a therapy
- Some studies have demonstrated increased use of vasodilators and inotropes with PAC use
- Fluid status and diuretic use was not statistically significant in PAC vs CVC trial
- Protocols for therapeutic intervention have not been well established or universalized
Do Physicians Know How to Use the PAC?

- Three studies published in the 90’s suggested that a significant number of physicians and nurses did not interpret the information from a PAC correctly.

- However, their use and insertion has persisted but declined (10.8% & 4.2% in 2001 -> 6% & 2.2% in 2008)
Is this conclusion universally applicable?

Pulmonary artery catheter use is associated with reduced mortality in severely injured patients: A National Trauma Data Bank analysis of 53,312 patients*

Randall S. Friese, MD; Shahid Shafi, MD; Larry M. Gentilello, MD

• PAC associated with higher mortality

• However, in a linear regression analysis, severely injured and high risk patients had improved survival with PAC

• Age 61-90, BD > -11, ISS > 25 had OR 0.33 (CI 0.17-0.62) mortality improvement with PAC
Continuing Controversies

• If we don’t know how to utilize PAC information, do we know how to utilize non-invasive hemodynamic data?

• How can we best capitalize on benefits of PAC when they are used so infrequently?

• Are there other populations besides sick trauma patients that would benefit from PAC usage? (septic shock, ARDS, acute decompensated HF)
References

• Layton, Gabrielli, Yu, Civetta Critical Care, 4th ed.

• Marino, The ICU Book, 4th ed.

• Vincent, Abraham, Moore, Textbook of Critical Care, 6th ed.