WHAT IS “HEMODYNAMIC MONITORING?”

- We assess all patients using the five “vital signs”
  - Temperature, blood pressure, heart rate, respiratory rate, arterial oxygen saturation
- Critically ill patients demonstrate pathophysiologic changes in organ function that require close observation and scrutiny
  - Cardiac
  - Pulmonary
  - Renal
  - Hepatic
  - Cerebral

WHAT IS “HEMODYNAMIC MONITORING?”

- A variety of advanced monitoring techniques exist that allow assessment of such end-organ function
  - Pulmonary artery catheterization
  - Arterial waveform pulse contour analysis
  - Esophageal Doppler
  - Echocardiography
  - Bioimpedance
  - Carbon dioxide excretion
- Each of these are discussed in a separate lecture
- First, we need to cover some general principles

WHY DO WE MONITOR PATIENTS?

- Monitor
  - “to keep watch over”
  - “to keep track of by an electronic device”
  - “to scrutinize or check systematically with a view to collecting certain specified categories of data”

The American Heritage Dictionary

IN THEORY, WE MONITOR PATIENTS TO...

- Generate physiologic data
- Guide therapeutic interventions
- Avoid progression of organ dysfunction to multiple organ failure
- Allow early detection of problems
- Identify the need for changes in treatment strategy

BUT DO WE REALLY MONITOR PATIENTS?

- A 31 question multiple-choice examination of invasive hemodynamic monitoring knowledge was administered to attending and resident physicians
  - 77% residents / fellows
  - 23% attending physicians
- Mean score: 67% (range 19-100%)
  - 47% of physicians could not derive basic information from a pulmonary artery catheter (PAC)

Iberti et al. 1990
BUT DO WE REALLY MONITOR PATIENTS?

- The same examination was subsequently given to 216 critical care nurses at a nursing convention
  - Mean score: 49%
    - 42% of nurses could not identify a pulmonary artery occlusion pressure (PAOP) or “wedge” tracing correctly

HOW ABOUT CRITICAL CARE PHYSICIANS?

- Another similar multiple choice examination was administered to critical care specialists to assess their understanding of hemodynamic monitoring
  - The mean score was higher (83%, range 10-100%)
    - HOWEVER
      - 33% could not identify a PAOP tracing
      - 33% could not determine how to increase a patient’s oxygen delivery
      - 54% admitted they use the data from a patient’s PAC 5 or fewer times per day

DO WE REALLY MONITOR PATIENTS?

Conclusions
1. Significant deficits exist regarding fundamental concepts of hemodynamic monitoring and pulmonary artery catheterization
2. Pulmonary artery catheters are diagnostic tools and not therapeutic interventions
3. The outcome of a patient with a pulmonary artery catheter is altered only if management is guided by the data obtained
4. These same conclusions apply to any monitoring technology

WHEN WE DECIDE TO MONITOR...

Hemodynamic monitoring CANNOT have a positive impact on patient outcome without:
1) Knowledgeable interpretation of the data
   and
2) Institution of appropriate therapy based on the data obtained

THE IDEAL MONITOR WOULD BE...

- Accurate
- Reliable
- Simple
- Continuous
- Safe
- Informative
- Useful
- Cost-effective

A IDEAL MONITOR IS ACTUALLY...

- Subject to variability
- Prone to artifact
- Complex
- Intermittent data
- Significant risks
- Can be erroneous
- May change therapy
- Expensive
HOW DO WE MONITOR PATIENTS?

- Noninvasive
  - Sphygmomanometric “cuff” blood pressure
  - Electrocardiography
  - Urinary output
- Invasive
  - Arterial pressure monitoring
  - Central venous pressure monitoring
  - Pulmonary artery catheterization
  - Intracranial pressure monitoring
  - Intra-abdominal pressure monitoring

WHEN SHOULD WE MONITOR PATIENTS?

- One should consider invasive hemodynamic monitoring when patients:
  - Fail to respond to resuscitation as documented by
    - Ongoing or worsening shock
    - Progressive or refractory lactic acidosis
    - Develop organ dysfunction / failure
    - Have pre-existing cardiopulmonary dysfunction
  - May benefit from preoperative assessment based upon an increased risk for perioperative morbidity and mortality

EVOLUTION OF RESUSCITATION GOALS

<table>
<thead>
<tr>
<th>Decade</th>
<th>Definition of Shock</th>
<th>Resuscitation Goals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1970's</td>
<td>Inadequate cardiac output</td>
<td>Blood pressure, heart rate, urinary output, central venous pressure</td>
</tr>
<tr>
<td>1980's</td>
<td>Inadequate oxygen transport balance</td>
<td>Mixed venous oxygen saturation (SvO₂), pulmonary artery occlusion pressure</td>
</tr>
<tr>
<td>1990's-2000's</td>
<td>Inadequate end organ perfusion &amp; oxygenation</td>
<td>Lactate, pH, RVEDVI, oxygen delivery, oxygen consumption</td>
</tr>
</tbody>
</table>

PRIMARY MONITORING QUESTIONS

- There are four questions that should be asked of any critically ill patient:
  - Is intravascular volume or “preload” adequate?
  - Is blood flow adequate?
  - Is vascular resistance appropriate?
  - Is oxygen transport balance adequate?

MEASURED VS. CALCULATED VARIABLES

- The hemodynamic parameters used in clinical practice are either
  - Measured
    - Directly determined from a monitoring device
  - Calculated
    - Mathematically determined from measured variables
    - More subject to measurement error
      - Operator error
      - Dynamic response artifacts
      - Measuring device errors

MEASURED PRESSURE VARIABLES

<table>
<thead>
<tr>
<th>Variable</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td>SBP</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>DBP</td>
</tr>
<tr>
<td>Systolic pulmonary artery pressure</td>
<td>PAS</td>
</tr>
<tr>
<td>Diastolic pulmonary artery pressure</td>
<td>PAD</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure</td>
<td>PAOP</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>CVP</td>
</tr>
<tr>
<td>Intra-abdominal pressure</td>
<td>IAP</td>
</tr>
<tr>
<td>Intracranial pressure</td>
<td>ICP</td>
</tr>
</tbody>
</table>
### Calculated Pressure Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure</td>
<td>MAP</td>
</tr>
<tr>
<td>Mean pulmonary artery pressure</td>
<td>MPAP</td>
</tr>
<tr>
<td>Cerebral perfusion pressure</td>
<td>cerebral PP</td>
</tr>
<tr>
<td>Coronary perfusion pressure</td>
<td>coronary PP</td>
</tr>
<tr>
<td>Abdominal perfusion pressure</td>
<td>APP</td>
</tr>
</tbody>
</table>

### Mean Arterial Pressure and Mean Pulmonary Artery Pressure

- Mean pressures
  - Remain fairly constant despite “dynamic response artifacts”
  - Should be used to titrate vasoactive infusions and other resuscitative therapies

\[
MAP = \frac{SBP + 2 \times DBP}{3}
\]

\[
MPAP = \frac{PAS + 2 \times PAD}{3}
\]

### The Circulatory System

- The body consists of two circuits in series
  - Systemic circulation
  - Pulmonary circulation
- Each circuit has a...
  - Pump
    - Left ventricle
    - Right ventricle
  - Resistance
    - Systemic
    - Pulmonary

### Preload Assessment

- Starling defined “preload” as end-diastolic volume
  - Actually myofibril length, but we can’t measure this
- PAOP and CVP have traditionally been used as surrogate estimates of preload as volumes could not be easily measured
- The use of such pressures assumes that...
  - Ventricular compliance is unchanging
  - Pressure accurately reflects end-diastolic volume

\[
\text{Compliance} = \frac{\Delta \text{Pressure}}{\Delta \text{Volume}}
\]
CENTRAL VENOUS PRESSURE (CVP)

• CVP can be measured using any catheter whose tip is in the superior or inferior vena cava
• CVP is assumed to estimate ventricular filling volumes
  – May correlate with right ventricular volumes in the absence of measurement errors or changing compliance
  – Does not correlate with left ventricular volumes
• Although widely used, CVP alone should not be relied upon to guide fluid resuscitation
  – Measurement errors are common in the critically ill patient

THE PAOP ASSUMPTION

• It didn’t take long to recognize the limits of PAOP
  – 1974
    • Negative effect of positive pressure ventilation on PAOP accuracy is demonstrated
  – 1981
    • Negative effect of changing ventricular compliance on PAOP described
  – 1992
    • Advantages of volumetric measurements over PAOP and CVP described
    • PAOP found to incorrectly predict response to fluid challenge in 52% of critically ill patients

PULMONARY ARTERY OCCLUSION PRESSURE (PAOP)

• PAOP was described as a measurement of left atrial pressure (LAP) by Swan & Ganz in 1970
  – It soon became the gold standard for preload assessment
• Requires a more invasive pulmonary artery catheter rather than simply a central venous catheter
  – “Optimal” PAOP is widely considered to be 12-18 mmHg regardless of patient physiology
• Commonly misunderstood and misused

POTENTIAL SOURCES OF ERROR IN THE PAOP ASSUMPTION

Preload = LVEDV = LVEDP = LAP = PAOP

PAOP and CVP are accurate ONLY when these potential sources of error have been eliminated

CORONARY PERFUSION PRESSURE

Coronary Perfusion Pressure = DBP − PAOP

• A primary goal in any resuscitation
  – Especially in patients with myocardial disease
• Calculated as the perfusion pressure across the coronary artery during maximal blood flow (diastole)
  – DBP = coronary artery inflow
  – PAOP = coronary artery outflow during diastole
• Goal: To maintain coronary PP above 50 mmHg
  – Achieved by increasing MAP or reducing PAOP

CARDIAC FILLING PRESSURES

• The assumptions upon which PAOP and CVP are based may not necessarily be true in patients with...
  1) Changing ventricular compliance
    • Most critically ill patients
  2) Increased intrathoracic pressure
    • Patients with acute lung injury
  3) Increased intra-abdominal pressure
    • Patients with intra-abdominal hypertension
• As a result, PAOP and CVP must be used with caution
Hemodynamic Calculations I - M. L. Cheatham, MD, FACS, FCCM

**CEREBRAL PERFUSION PRESSURE**

Cerebral Perfusion Pressure = MAP – ICP

- Important in the head-injured patient with elevated ICP
- Calculated as the perfusion pressure across the brain
  - MAP = cerebral inflow
  - CVP or ICP (whichever is higher) = cerebral outflow
- Goal: To maintain cerebral PP above 60 mmHg
  - Achieved by increasing MAP or decreasing ICP
  - Associated with improved long-term outcome

**ABDOMINAL PERFUSION PRESSURE**

Abdominal Perfusion Pressure = MAP – IAP

- Elevated intra-abdominal pressure (IAP) is commonly present in the critically ill
  - An important cause of multi-system organ failure
- Calculated as the perfusion pressure across the abdomen
  - MAP = abdominal inflow
  - IAP = abdominal outflow
- Goal: To maintain APP above 60 mmHg
  - Achieved by increasing MAP or decreasing IAP
  - Associated with improved survival

**FLOW-RELATED VARIABLES**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output</td>
<td>CO</td>
</tr>
<tr>
<td>Right ventricular ejection fraction</td>
<td>RVEF</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>CI</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>SV</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>SVI</td>
</tr>
<tr>
<td>Stroke volume variation</td>
<td>SVV</td>
</tr>
<tr>
<td>Right ventricular end-diastolic volume index</td>
<td>RVEDVI</td>
</tr>
</tbody>
</table>

**FLOW-RELATED VARIABLES**

- Blood flow-related variables are used with pressure variables to calculate
  - Vascular resistance
  - Cardiac work
- Must be indexed to body surface area (BSA) to assign a “normal range”

**INDEXED VARIABLES**

- Both patients have the same cardiac output (CO), but their cardiac index (CI) varies greatly due to the difference in their weight

<table>
<thead>
<tr>
<th>Patient A</th>
<th>Patient B</th>
</tr>
</thead>
<tbody>
<tr>
<td>60 year old male</td>
<td>60 year old male</td>
</tr>
<tr>
<td>Weight = 50 kg</td>
<td>Weight = 150 kg</td>
</tr>
<tr>
<td>CO = 4.0 L/min</td>
<td>CO = 4.0 L/min</td>
</tr>
<tr>
<td>BSA = 1.66</td>
<td>BSA = 2.64</td>
</tr>
<tr>
<td>CI = 2.4 L/min/m²</td>
<td>CI = 1.5 L/min/m²</td>
</tr>
</tbody>
</table>

**CARDIAC INDEX**

- A measure of both preload status and myocardial contractility
- Calculated as the total blood flow from the heart in liters per minute per meter squared BSA

Cardiac Index = Heart rate × Stroke volume index

\[ CI = HR \times SVI \]
CARDIAC INDEX

• Causes of decreased CI
  – Intravascular depletion
  – Increased vascular resistance
• Causes of increased CI
  – Early distributive or “septic” shock
  – Cirrhosis
  – Pregnancy
  – High performance athletes

VASCULAR RESISTANCE

• An indication of ventricular afterload
• Calculated using Ohm’s Law
  – resistance = voltage difference / current
  = pressure change / total blood flow

VASCULAR RESISTANCE INDICES

Systemic Vascular Resistance Index =
Change in systemic pressure / total blood flow
SVRI (dynes*sec*cm⁻⁵) = (MAP-CVP)(80)/CI

Pulmonary Vascular Resistance Index =
Change in pulmonary pressure / total blood flow
PVRI (dynes*sec*cm⁻⁵) = (MPAP-PAOP)(80)/CI
80 converts mm Hg-min-m²/filters to dynes*sec/*cm²

SYSTEMIC VASCULAR RESISTANCE INDEX

• Causes of increased SVRI
  – Various shock states
  • Obstructive, hypovolemic, late distributive, cardiogenic
  – Pheochromocytoma
• Causes of decreased SVRI
  – Various shock states
  • Neurogenic, early distributive

PULMONARY VASCULAR RESISTANCE INDEX

• Causes of decreased PVRI
  – Various shock states
• Causes of increased PVRI
  – Pulmonary hypertension
  – Acute respiratory distress syndrome
  – Intra-abdominal hypertension
  – Mitral stenosis
  – Aortic stenosis
  – Left heart failure

VENTRICULAR STROKE WORK INDICES

• Describes ventricular work and performance
• Dependent upon preload status
• Work = force x distance
  = Δ pressure x Δ volume
VENTRICULAR STROKE WORK INDICES

Ventricular work = $\Delta$ pressure x $\Delta$ volume

$\text{LVSWI} = (\text{MAP-PAOP}) \cdot (\text{SVI}) \cdot (0.0136) \text{ (g m/m}^2\text{)}$

$\text{RVSWI} = (\text{MPAP-CVP}) \cdot (\text{SVI}) \cdot (0.0136) \text{ (g m/m}^2\text{)}$

0.0136 converts mm Hg-liters/beat-m² to g*m/m²

VENRICULAR STROKE WORK INDICES

• Causes of decreased LVSWI / RVSWI
  – Inadequate intravascular volume
  – Increased vascular resistance
  – Decreased contractility

• Causes of increased LVSWI / RVSWI
  – Ventricular hypertrophy
  – Physiologic conditioning

CONCLUSIONS

• We monitor patients to
  – Generate physiologic data
  – Guide therapeutic interventions
  – Avoid organ dysfunction and failure
  – Allow early detection of problems
  – Identify the need for changes in treatment strategy

• Hemodynamic monitoring CANNOT improve patient outcome unless the data is utilized

CONCLUSIONS

• Cardiac performance can be assessed and therapeutic interventions directed using simple physiologic equations

• By calculating the hemodynamic parameters outlined above, a critically ill patient’s current physiology can be identified and appropriate therapy instituted to correct any deficits present

• While the parameters are commonly calculated automatically by the monitors, knowing HOW the parameters are determined explains what must be done to bring about the desired change