Advanced Hemodynamic Monitoring
Dynamics of Critical Care 2015

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Outline

Pressure Monitoring Basics

How can we measure pressure?

What errors exist in our measurements?

What pressures can we measure?

What options do we have to monitor hemodynamics?
How did we first measure blood pressure?
Auscultatory Blood Pressure measurements

- Traditional method using stethoscope and sphygmomanometer
- Observer dependent, calibration, severe atherosclerosis, too rapid deflation, limb ischemia
Oscillometric Automated NIBP

Based on amplitude of pressure change in the cuff during deflation after occlusion of artery.

Fig. 1 Oscillometric technique to measure systolic, mean, and diastolic blood pressures. (Adapted from Dinamap Models 845/847, Operation Training Manual, Applied Medical Research, Tampa, Florida, 1979.)
Issues with NIBP

- Limb ischemia, pain
- Cuff Size
  - Too large or too long a cuff underestimates pressure
- Movement and Shivering
- Irregular rhythm

What number is the most reliable?
Invasive Pressure Monitoring

- Arterial line
- Pressure bag
- Pressure transducer & automatic flushing system
- Saline filled non-compressible tubing
Water is a non-compressible substance.

Therefore any pressure change on one side of a column of water is propagated down the length of the column of water.

The transducer needs to be level.

Changing the height of the column of water will change the number on the monitor.
Where do we level the transducer?

The transducer is usually placed at the level of the right atrium of the heart.
Waves

- A wave is a disturbance that travels through a medium, transferring energy but not matter (Sine wave is one of the simplest).

- Any waveform can be broken down into a series of sine waves that add together (Fourier Analysis).
Natural Frequency and Resonance
Frequency of Pressure Transducing Systems

If any of the component sine waves of an arterial waveform are at the same frequency of the system, it will cause amplification.

This will cause wide pulse pressures and elevated systolic pressures.
Natural Frequency

- The Natural Frequency can be increased by reducing length of tubing or compliance of the system.

- Natural Frequency can be decreased by addition of stopcocks, bubbles, additional tubing.
Fast Flush test for testing the natural frequency of a system

Figure 215–2 Pressure–time waveforms during fast-flush testing of dynamic response of arterial pressure monitoring systems. Top: optimal dynamic response showing true intra-arterial blood pressure. Middle: overdamped system showing spuriously low systolic pressure. Bottom: hyperresonant, underdamped system showing spuriously elevated systolic pressure. Inset: method of determining amplitude ratio (A2 ÷ A1) and resonant waveform period (τ). A1 and A2 = successive amplitudes.
Over and Under

(A) Observed waveform

(B) Observed waveform

(C) Observed waveform

Physics of Invasive Pressure Monitoring

Aortic Root
Subclavian
Axillary
Brachial
Radial
Femoral
Dorsalis Pedis
Arterial Waveform
Anacrotic Limb

End Diastole to Systole

Ventricle ejects blood into arterial tree

As pressure reaches max, wave levels off (y = anacrotic notch)
Steepness of ascending phase affected by

- HR (higher HR causes steeper upstroke)
- changed SVR (more steep if vasopressor, less steep if vasodilated)
- Contractility (impaired contractility is less steep)
Dicrotic Limb

- Descending limb as arterial pressure drops to end diastolic pressure
- Dicrotic notch occurs at any point there is fluctuation in pressure (usually aortic closure)
Dicrotic Notch

- A flat or non-existent notch can be a sign of dehydration
- A low notch can be due to a high pulse pressure or low diastolic pressure
- A flattened notch can be present in valve insufficiency
Dicrotic Limb

- Dicrotic “fall-off” changes in relation to SVR
- If SVR is low, the fall off is very rapid as there is reduced pressure in the arterial tree (tracing looks thin and pointed)
- If SVR is high, the fall off is increased. There is an increased time to return to end-diastolic pressure (tracing looks fat)
Pulsus Alternans
Indicative of left ventricular failure

Collapsing Pulse
May be seen in patients with aortic regurgitation or a hyperdynamic circulation

Pulsus Bisferiens
Associated with aortic regurgitation & hypertrophic cardiomyopathy

Anacrotic Pulse
Classically associated with aortic stenosis
Arterial Pulse Contour Analysis

\[ P \text{ [mm Hg]} \]

\[ P(t) = \frac{\text{SVR}}{\text{HR}} + C(p) \cdot \frac{dP}{dt} \int_{\text{Systole}} \text{dt} \]

- Patient-specific calibration factor (determined by thermodilution)
- Heart rate
- Area under pressure curve
- Aortic compliance
- Shape of pressure curve

SV max
SV min
SV mean
SVV, PPV and SPV

- **Stroke Volume Variation** - based on arterial waveform
- **Pulse Pressure Variation** - based on pulse pressure
- **Systolic Pressure Variation** - based on systolic pressure
Increases in these assessments suggest that the patient may be on the steep part of the Frank-Starling Curve.

Need to test this with a fluid challenge.
Limitations of SPV/PPV/SVV

- Most studies have used mechanically ventilated patients who are under anesthesia.
- 8 ml/kg tidal volume, PEEP 0-5 cmH₂O
- Sinus Rhythm, chest closed, normal intra-abdominal pressures
- Unclear effect in LV failure or ARDS
Plethysmography
CVP Tracing

- Three Peaks (a, c, v)
- Two Descents (x, y)
Measuring CVP

- The peak of the “a” wave coincides with the point of maximal filling of the right ventricle.
- Therefore, this is the value which should be used for measurement of RVEDP.
- Machines just “average” the measurement.
- Should be measured at end-expiration.
Tachycardia and CVP

- A short PR interval can cause the “a” and “c” waves to fuse
- Tachycardia reduces the time spent in diastole, causing a short “y” descent
- This can make the “v” and “a” waves appear to merge
Bradycardia and CVP

- Causes each wave to become more distinct
- "h" wave may become evident - plateau wave in mid- or late diastole
- The "h" wave has very little clinical significance
Cannon “a” waves

- Atrial contraction during systole (closed TV) causes the CVP to rise significantly.
- Seen with AV dissociation, junctional rhythm, ventricular pacing.
Tricuspid Regurgitation

- The right atrium gains volume during systole – so the “c” and “v” wave is much higher.
- The right atrium “sees” right ventricular pressures and the pressure curve becomes “ventricularized”
Tricuspid Stenosis

- Problem with atrial emptying and a barrier to ventricular filling on the right side of the heart
- Mean CVP is elevated
- “a” wave is usually prominent as it tries to overcome the barrier to emptying
- “y” descent muted as a result of decreased outflow from atrium to ventricle
Pericardial Constriction

- Limited venous return to heart, elevated CVP, end-diastolic pressure equalization in all cardiac chambers
- Prominent “a” and “v” waves, steep “x” and “y” descents
- Characteristic M or W pattern, dip and plateau (square root sign)
Cardiac Tamponade

- Changes in atrial and ventricular volumes are coupled, so total cardiac volume does not change when blood goes from atrium to ventricle.
- CVP becomes monophasic with a single, prominent “x” descent with a muted “y” descent.
- Similar to pericardial constriction but not exactly the same.
Pulmonary Artery Catheters

Very Controversial

Do they save lives or cause complications?

Do we really know what the numbers mean?
Sandham et al - 2000 surgical patients (ASA class 3 or 4) aged 60 years or older. No difference in mortality rate (8%), length of stay, or organ dysfunction.

Richard et al - 700 patients with early shock, ARDS, or both. No significant difference in mortality or morbidity was noted.

Rhodes et al - 201 patients no difference in 28-day mortality, ICU, or hospital length of stay.

The PACMAN trial - 1000 patients. No difference in hospital mortality (primary outcome) was noted.

The ESCAPE trial - 400 patients with severe heart failure. No difference was noted in overall mortality or hospitalization.

Shah et al - large meta-analysis of 13 RCTs and more than 5000 patients (including the above mentioned trials). Neither an increase in mortality or length of hospital stay nor a significant benefit.

In 2006, the ARDSNET group looked at the role of the PAC in acute lung injury patients. No difference in mortality (primary outcome), ICU length of stay, or lung function was appreciated.
First intracardiac catheter

- Werner Forsmann (1904–1979)

- 1929 – Surgical Resident in Eberswald, Germany

- Inserted catheter into his antecubital vein and then went down to basement X-ray room. X-ray showed catheter in right atrium

- Fired on the spot, but won a Nobel Prize in 1956
H. J. C. Swan was watching a sailboat at the beach when he had the idea of allowing blood flow to carry a catheter.

William Ganz developed the thermodilution method of measuring cardiac output.
What can we measure?

- Pressures in the RA, RV and PA
- PCWP (wedge)
- Cardiac output using thermodilution
- Central temperature
Additional Information

Derived Information

- Systemic Vascular Resistance (SVR)
- Cardiac Index (CI)

Direct Information

- mixed venous oxygenation (MvO₂)
- oxygen “steps”
How do we measure wedge pressure?
West Zone III is where the alveolar pressure is always lower than arterial or venous pressure. There is blood flow throughout the respiratory cycle. This gives the most reliable readings of wedge pressure.
Errors with the PAC

- Pressure measurements - level, assumptions of the wedge pressure
- Cardiac Output - temperature differential, regurgitation, septal defects
- Mixed venous oxygen - arterialization of sample
What is the Wedge Pressure?

- Identify a waves (follow QRS) and v waves (follow T)
- Identify end exhalation.
- Measure A wave at peak and nadir and average results
Identify the waves
Other methods of hemodynamic monitoring

- Doppler Ultrasound Methods
- Echocardiography, Transcutaneous or Transesophageal Doppler
- Pulse Contour Analysis
- Plethysmography, Arterial Line
- Clinical Examination
Echocardiography

Very easy to perform a qualitative evaluation, technically hard to perform a quantitative evaluation
Clinical Cardiac Output Monitoring

- Urine
- Level of Consciousness
- Peripheral pulses
- Skin perfusion
- End-organ function
Application of this information
Cardiac Output = 5–8 l/min
Cardiac Index = 2–4 l/min/m²
Systemic Vascular Resistance = 900–1300 dynes*sec/cm²
Mixed Venous $O_2$ = 30–55 mmHg
Central Venous $O_2$ = 35–65 mmHg
BP = 80/50
HR = 130
CI = 1.8
MvO2 = 37
SVR = 1800

Diagnosis?

Hypovolemic Shock
BP = 80/40
HR = 115
CI = 1.9
MvO2 = 30
SVR = 1500
Diagnosis?

Left Heart Failure
BP = 80/35
HR = 130
CI = 5.0
MvO2 = 40
SVR = 600
Diagnosis?

Anaphylactic Shock
BP = 70/30
HR = 140
CI = 1.8
MvO2 = 30
SVR = 1600
Diagnosis?

Cardiac Tamponade
BP = 90/40
HR = 130
CI = 5.0
MvO2 = 48
SVR = 600
Diagnosis?

Septic Shock
BP = 80/40
HR = 110
CI = 2.0
MvO2 = 35
SVR = 1500
Diagnosis?

Right Heart Failure
BP = 80/40
HR = 80
CI = 2.5
MvO₂ = 40
SVR = 500
Diagnosis?

Adrenal Insufficiency