Applied Cardiac Physiology

Presented by: F. Farah
Moderated by: Dr. S.Taha
Introduction

• Intraoperative arterial hypotension correlates directly with surgical mortality

• 2 factors have a major role in worsening prognosis:
  - The severity
  - The duration
Introduction

• In elderly

• Delayed or inadequate correction of hypotension → 40% of substandard intraoperative care → Postoperative myocardial ischemia and infarction.

• Need for fast diagnosis and management of intraoperative hypotension
MYOCARDIAL PERFORMANCE

Physiological Algorithm

Blood pressure

Systemic vascular resistance

Cardiac output

Heart rate

Stroke volume

End-diastolic volume

End-systolic volume

Afterload

Rhythm

Preload; compliance

Contractility; afterload
MYOCARDIAL PERFORMANCE

Physiological Algorithm

Systemic vascular resistance

Blood pressure

Cardiac output

- Thermodilution
- Arterial waveform contour analysis
- Echocardiography
- Esophageal Doppler
- $P_{et}CO_2$
Cardiac Output

- **Formulas:**

\[ SV = \frac{CO}{HR} \]

\[ O_2 \text{ delivery} = CO \times CaO_2 \]

\[ O_2 \text{ delivery} = \dot{V}O_2 + (CO \times \dot{C}VO_2) \]

\[ CO \times CaO_2 = \dot{V}O_2 + (CO \times \dot{C}VO_2) \]

\[ CO(CaO_2 - \dot{C}VO_2) = \dot{V}O_2 \]

\[ CO = \frac{\dot{V}O_2}{CaO_2 - \dot{C}VO_2} \]

\[ CI = \frac{CO}{BSA} \]

\[ SVI = \frac{CI}{HR} \]

\[ SVR = \frac{\text{Mean SAP} - \text{RAP}}{CO} \times 80 \]

\[ PVR = \frac{\text{Mean PAP} - \text{PCWP}}{CO} \times 80 \]
Cardiac Output

- Assessment of cardiac output is necessary after a sudden hypotension
Cardiac Output

- **Cardiac output estimated by**
- **Invasive techniques:**
  - Thermodilution
  - Dye dilution measurement
Cardiac Output

- Less invasive:
  - Arterial waveform contour analysis
  - Esophageal Doppler
  - Transthoracic impedance
  - TEE
Cardiac Output

- **Non invasive:**
  - End-tidal carbon dioxide measurement
  - To be reliable:
    - Constant minute ventilation
    - Constant patient temperature
    - No exogenous source of CO2 (laparoscopic surgery or exogenous sodium bicarbonate)

- 3-4 mmHg decrease end-tidal CO2 $\Leftrightarrow$ 1L/min/m² reduction in cardiac output
Cardiac Output

- If hypotension without end-tidal CO2 change =>

- Decrease in systemic vascular resistance

Treatment: α–agonist (phenylephrine, norepinephrine)
Cardiac output

- If hypotension + decrease in end-tidal CO2 =>
- cardiac output reduction (heart rate, stroke volume)

Treatment: Inotropes, anticholinergics

In the supine, anesthetized, intubated adult

- heart rate increase does not improve cardiac output (inadequate venous return), unless too low

- Stroke volume (difference between end-diastolic and end-systolic volumes) depends on preload, afterload, contractility

- However, The importance of a sinus rhythm cannot be overlooked
• Based on the previously cited algorithm, blood pressure can be increased by increasing Cardiac Output or increasing Systemic Vascular Resistance
**Afterload**

- **Afterload** Represents all forces opposing LV fiber shortening during ejection

- **Static indices**
  - Aortic impedance
  - Blood viscosity
  - Peripheral vascular resistance

- **Dynamic indices** ↔ Ventricular wall stress
  - Ventricular wall stress proportionnal \([(radius) \ (pressure)]/wall \ thickness\)
  - Ventricular shape
  - Ventricular size
  - Wall thickness
  - Intracavitary pressure
MYOCARDIAL PERFORMANCE

Afterload

Summation of all forces opposing ventricular fiber shortening during ejection:

- Ventricular shape and size
- Wall thickness
- Intracavitary pressure
- Aortic impedance (resistance)
- Ventricular resistance (inertia)

\[ \text{Law of Laplace} \]
\[ \text{stress} = \frac{P_r}{h} \]

\[ \text{SVR} = \text{MAP} - \frac{CVP}{CO} \]
Heart rate

- During exercise, tachycardia can increase cardiac output 5-fold. (sympathetic response)
Heart rate

• In patient under anesthesia, tachycardia has a negligible effect on cardiac output (can decrease cardiac output by decreasing filling)

• In patients undergoing coronary revascularization before and after cardiopulmonary bypass, sequentially increasing heart rate from 80 to 110 beats/min causes a proportional reduction in RV end-diastolic volume, and consequently stroke volume, so that cardiac output remains unchanged (Johnston et.al Ann Thorac Surg 1991; 52:797–804)
Heart rate

Exercise
- Increase venous return
- Decrease resistance
- Increase contractility
- Increase heart rate
- Increase cardiac output

General Anesthesia
- Decrease venous return
- Decrease resistance
- Decrease contractility
- Positive-pressure ventilation
- Increase heart rate
- No change cardiac output
Heart rate

- On the other hand, increasing heart rate increases myocardial oxygen consumption (decreases diastole) and may precipitate heart failure in patients with coronary artery disease.
Heart rate

• However, heart rhythm is an important component of blood pressure particularly in patients with reduced ventricular compliance.
  - LV hypertrophy
  - Aortic stenosis
  - Diastolic dysfunction

• In these patients the contribution from a properly timed atrial contraction may represent 25 to 40% of end-diastolic volume
HEART RHYTHM

• Normal atrial kick 10–15% EDV
• Noncompliant heart (hypertension, AS, age) may be 25–40% EDV
• Diagnose by
  ✓ Electrocardiogram
  ✓ Transesophageal echo
  ✓ Central venous pressure waveform
Heart rate

- Example:
- Significant reduction in blood pressure in a patient with aortic stenosis that accompanies the conversion from sinus rhythm to nodal rhythm
MYOCARDIAL PERFORMANCE

Stroke Volume

Blood pressure

Systemic vascular resistance
Cardiac output

Heart rate

Stroke volume
End-diastolic volume
End-systolic volume

Afterload
Rhythm
Preload; compliance
Contractility; afterload
Assessment of stroke volume variability

- Means the change in stroke volume after fluid administration
- Adequate if 10-15% increase in SV
Assessment of stroke volume variability

- Stroke volume change assessed by:
  - **Static indices:**
    - CVP
    - PCWP
  - **Dynamic indices:**
    - Arterial blood pressure tracing,
    - Pulse oximeter plethysmographic tracing with positive pressure ventilation

- 12-14% variability in dynamic indices ⇔ 10-15% change in SV.
DYNAMIC INDICES
Fluid Volume Responsiveness

Positive pressure ventilation

PPV = \frac{PP_{\text{max}} - PP_{\text{min}}}{(PP_{\text{max}} + PP_{\text{min}}) / 2} 
> 12%
MYOCARDIAL PERFORMANCE

Preload

Blood pressure

Systemic vascular resistance

Cardiac output

Heart rate

End-diastolic volume

End-systolic volume

Stroke volume

Rhythm

Contractility; afterload

preload; compliance

afterload
Preload

• Preload ⇔ The load or force stretching the ventricular myofibrils at end-diastole
Preload

• Preload recruitable function

• The intrinsic ability of the heart to increase the stroke volume at a greater end-diastolic volume (Frank-Starling)
Preload

- The above theory was explained by the sliding of actin-myosin and the overlap between the myofibrils.
- However, new evidence showed no correlation between actin-myosin crossbridges and the force of contraction.
- New evidence put forth the role of calcium activation.
CURVILINEARITY
Frank-Starling Relationship

D Glower, Circulation 1985;71:994-1009
Contractility

- Inotropic drugs increase cardiac contractility by stimulating the heart to eject to a lower LV end-systolic volume

- Ideally, Contractility
  - Increases Stroke Volume
  - Improves coronary perfusion pressure and oxygen delivery.
Contractility

• However, the net effect of an inotrope on myocardial oxygen consumption depends on baseline ventricular size

• With a high ventricular size (high end-diastolic volume + pressure) increased inotropy = increased stroke volume
  - => smaller end-systolic volume and smaller end-diastolic volume
  - => decrease wall stress and oxygen consumption

• This may be true if there is no change in heart rate (increased oxygen consumption)
Contractility

• Practically, we should take into consideration if an inotropic drug could adversely affect the balance between myocardial oxygen supply and demand

• Another important consideration is whether the addition of an inotropic drug can worsen the severity of ischemic myocardial injury
Contractility

• In experimental animals,
• before ischemia
  - Addition of an inotrop => increases oxygen demand => increase of injury and size of infarction
• After ischemia (coronary occlusion)
  - Addition of inotrop => minimal effect

• Myocardial consumption at the time of coronary occlusion determines the magnitude of ischemic injury
Contractility

• If the inotrops are administered chronic
  • => Level of catecholamines increases
  • => calcium overload (sarcolemmal permeability changes and alterations in the sarcoplasmic reticulum)
  • => Cardiotoxic effect
Contractility

• The transition from acute beneficial effects of inotropic therapy to chronic cardiotoxicity in patients has not been clearly defined.

• These detrimental effects may be secondary to the oxidative metabolites of catecholamines

• Furthermore, long-term catecholamine administration can induce pump dysfunction (β-adrenoreceptor downregulation, increased myocardial norepinephrine release, and LV dilatation)
Contractility

- A potential exception to inotrop cardiotoxicity is levosimendan
- ECG stabiliser
- Increases sensitivity of troponin C to intracellular calcium
Afterload–Preload Matching

- An acute increase in afterload causes a transient reduction in stroke volume
  - => end-systolic volume increases.

- In turn, end-systolic volume increases => a greater end-diastolic volume

- greater end-diastolic volume => recovery of stroke volume by the Frank–Starling mechanism
Afterload–Preload Matching

- In a normal heart
- Stroke volume is maintained in the face of increasing afterload by preload recruitable
- The increase in afterload is matched by an increase in preload so the force of contraction is augmented to maintain stroke volume.
Afterload–Preload Matching

• In contrast, in a failing heart with reduced contractility
  • Afterload increase will decrease stroke volume due to reduced preload
  • recruitable function.

• Thus, a normal heart appears insensitive to an increase in afterload due to preload sensitivity, whereas a failing heart is more sensitive to an increase in afterload due to preload insensitivity.
AFTERLOAD - PRELOAD

Ross J, Prog Cardiovasc Dis 1976;18:255-64
Afterload- Preload Matching

• The Afterload- Preload Matching has clinical relevance in the use of Vasopressors and vasodilators
Afterload- Preload Matching

• Phenylephrine

• Increases systemic blood pressure

• If the patient has coronary disease and good LV function:
  - 1 mcg/kg phenylephrine doubles end-systolic wall stress + reduces shortening of fibers.

• The rise in BP after phenylephrine administration is due to:
  - Increased preload (2/3)
  - Increased SVR (1/3) due to splanchnic and mesenteric vasoconstriction
Afterload- Preload Matching

- The hemodynamic response to phenylephrine can be explained by this interrelationship between afterload and preload.

- Phenylephrine increases afterload
- The maintenance of stroke volume with an increase in preload means that LV ejection fraction decreases (ejection fraction equals stroke volume/end-diastolic volume).

- Similarly, both end-systolic wall stress and myocardial oxygen consumption increase with phenylephrine (increases in LV end-systolic pressure and volume)

- However, coronary perfusion pressure (equal to diastolic arterial pressure LV end-diastolic pressure) may not change or may even decrease due to the increment in LV preload

- Increasing blood pressure with phenylephrine can provoke myocardial ischemia in anesthetized patients and cause myocardial perfusion defects proportional to the severity of diseased coronary vessels.
Afterload- Preload Matching

• Norepinephrine

• Increases Blood pressure

• However, it has:
  - No effect on fiber shortening
  - No effect on heart rate
  - No effect in end-systolic wall stress

• Vasopressor response is derived from:
  - Increased inotropy
  - Increase in SVR
Afterload- Preload Matching

• **Vasodilators**
  • if there are no dynamic indicators of a reduced end-diastolic volume (preload),

  • Afterload reduction helps ejecting to a lower end-systolic volume and increases contractility

  • Using a vasodilator to reduce afterload could restore blood pressure only if the net increase in stroke volume exceeds the net reduction in vascular resistance.
Afterload- Preload Matching

• Afterload reduction (vasodilators) could effectively treat hypotension under the following conditions
  - Severe aortic regurgitation
  - Severe mitral regurgitation
  - Atrial septal defect
  - Ventricular septal defect
  - Left to right shunt
  - Pulmonary hypertension with right ventricular (RV) failure
  - Severe left ventricular (LV) systolic failure.

• Otherwise, a positive inotropic drug would be indicated to improve stroke volume and consequently blood pressure
VASODILATORS
Indications for Treating Hypotension

To improve BP: \( \uparrow \uparrow \) SV \( >> \downarrow \downarrow \) SVR

- Mitral or aortic regurgitation
- Atrial or ventricular septal defect
- Pulmonary hypertension; RV failure
- Dilated cardiomyopathy
CHALLENGE
Basic Principle of Fluid Administration

Cost
- Tissue/pulmonary edema
- Intra-abdom pressure
- Coronary perfusion pressure

Stroke volume vs. filling pressure:
- Volume responsive
- Volume unresponsive

End-diastolic volume vs. filling pressure:
- Low cost
- High cost

Graphs show the relationship between stroke volume, filling pressure, and end-diastolic volume, highlighting the responsive and unresponsive volumes, and the associated costs.
Response to Fluid Challenge

- Classically, response to fluid challenge was defined as:
  - Responsive = Stroke Volume increases more than filling pressure
  - Unresponsive = Stroke Volume increases less than filling pressure
In fact, patients remain sensitive to fluid challenges as demonstrated by progressive increases in stroke volume at greater end-diastolic volumes.
Response to Fluid Challenge

• Sarcomeres become maximally stretched to 2.25 mm at LV end-diastolic pressures between 15 and 18 mmHg.
• Any increase in contractile force with further ventricular distention is attributed to recruitment of unaligned muscle fibers and stretch-induced calcium activation of myocytes.
Response to Fluid Challenge

- It is the cost of volume expansion (tissue and pulmonary edema, intraabdominal hypertension, and decreased coronary perfusion pressure) that defines the upper limit of fluid administration.

- The steep portion of the diastolic curve is due to the restraining effects of the pericardium, cardiac cytoskeleton, and embedded collagen between sarcomeres.
Response to Fluid Challenge

• In critically ill patients, any leftward shift of the diastolic pressure/volume relationship due to various influences such as:
  - Myocardial edema
  - Extrinsic compression
  - Myocardial ischemia
  - only increases the relative cost of volume expansion.
Practical Approach to Hypotension

• A sudden drop in blood pressure warrants assessing whether the end-tidal CO2 tension decreases as well.

• If not, an a-agonist would be appropriately administered and possibly reduce the inspired concentration of volatile agent.

• If the endtidal CO2 tension decreases, sinus rhythm should be verified by examining the electrocardiogram or the central venous pressure tracing.

• Any arrhythmia (i.e., nodal rhythm) should be promptly treated.
Practical Approach to Hypotension

• At that point, some assessment of preload should be made, preferably using dynamic indices such as pulse pressure variability or stroke volume variability from the arterial pressure tracing or the pulse oximeter tracing.

• Greater than 12 to 14% variability in these parameters predicts a positive response to improve stroke volume with colloid infusion.

• Static measures such as filling pressures or changes in filling pressures with fluid volume expansion do not accurately reflect ventricular preload.
Practical Approach to Hypotension

• In the absence of dynamic indicators of reduced filling, attention should be focused on ejecting to a lower end-systolic volume either by afterload reduction or by increased contractility.

• Afterload reduction could improve blood pressure only if the increase in stroke volume exceeds the relative decrease in systemic vascular resistance.

• Clinical settings where this might occur include aortic or mitral regurgitation, atrial or ventricular septal defect, pulmonary hypertension with RV failure, or severe LV systolic failure.

• Otherwise, at this point, an inotropic drug infusion would be indicated to improve stroke volume and consequently blood pressure.
Practical Approach to Hypotension

Blood pressure $\rightarrow \downarrow P_{etCO_2}$?

- Yes
  - NSR?
    - Yes
      - Dynamic indices (SVV; PPV; PVI > 12-14%?)
        - Yes
          - Inotrope (vasodilator)
        - No
          - Fluids (colloid)
    - No
      - Treat dysrhythmia
        - Yes
          - Inotrope (vasodilator)
        - No
          - Fluids (colloid)
Thank You!!

That wasn't chicken