Ancillary Factors

Ancillary Factors Affect the Venous System and Cardiac Output

- Gravity
  - Venous pooling may significantly reduce CO
- Muscular Activity and Venous Valves
- Respiratory Activity

Gravity

- Mostly venous due to high compliance
- Preload decreases
- CO and arterial pressure fall
- Baroreceptor reflex
  - HR increases
  - Vasodilation

Venous Return

Factors Affecting Venous Return

1. Blood Volume
2. Transmural pressure
   - Muscle pumps
3. SNS activity
   - T SNS activity → vasoconstriction of peripheral veins → blood volume stored in veins → venous return

Effects of Gravity on the Venous System and Cardiac Output

Gravity

- Venous pooling may significantly reduce CO
Effect Of Gravity on Venous Pressure

Venous Return

Skeletal Muscle Pump (Increase Venous Return)

Muscular Activity and Venous Valves

Effect of Venous Valves
Effects of Respiration

- Spontaneous respiration
  - Decreased intra-thoracic pressure results in a decreased right atrial pressure which enhances venous return

- Mechanical ventilation
  - Increased intra-thoracic pressure during positive-pressure lung inflation causes increased right atrial pressure which decreases venous return

- Valsalva Maneuver
  - Causes a large increase in intra-thoracic pressure which impedes venous return to the right atrium

Increase Pleural Negative Pressure (Increase Venous Return)

• Intrathoracic pressure (which decreases during inspiration and increases during expiration) inversely affects venous return.

Venous Return

- Blood volume and venous pressure (which increases during venoconstriction [constriction of the veins]) directly affect venous return.
  - What effect will blood loss have on EDV?
**Factors that Facilitate Venous Return**

- Venous return
  - Central venous pressure (Pcv)
    - Pressure in the vena cava and right atria
      - i.e., central venous pressure = right atrial pressure
    - Venous return \( \propto \frac{1}{P_{cv}} \)
      - If \( P_{cv} \) increases, VR decreases

**Cardiac Function Curve**

- Reflects dependence of CO on central venous pressure \( \sim \) End Diastolic Volume (Frank-Starling Mechanism)
- Is a function of only the heart
  - Measured in hearts isolated from the vascular system

**Vascular Function Curve**

- Is a function of only the vascular system
  - Can be measured by replacing the heart with a mechanical pump
- Influenced by:
  a. Peripheral resistance
  b. Arterial and venous compliance
  c. Blood volume

**CO = VR**

Concept:
- Central Venous Pressure (Pcv)
  - Pressure in the vena cava and right atria
    - i.e., central venous pressure = right atrial pressure
  - Venous return \( \propto \frac{1}{P_{cv}} \)
    - If \( P_{cv} \) increases, VR decreases

![Cardiac Function Curve](image)

- Normal curve (Frank-Starling Mechanism)
  - Hypovolemic
  - Normovolemic
  - Tachycardia
  - SNS
  - Tachycardia

![Vascular Function Curve](image)

- Normal VR (L/min)
  - Increase blood volume or vasodilation
  - Decrease blood volume or vasoconstriction
Plateau of CO curve determined by heart strength (contractility + ↑ HR)
↑ Sympathetics ⇒ ↑ plateau
↓ Parasympathetics (HR↑) ⇒ (?) plateau
↑ Plateau
Heart hypertrophy ⇒ ↑’s plateau
Myocardial infarction ⇒ (?) plateau
↓ Plateau

• Valvular disease ⇒ ↓ plateau
  (stenosis or regurgitation)
• Myocarditis ⇒ ↓ plateau
• Cardiac tamponade ⇒ (?) plateau
  ↓ Plateau
• Metabolic damage ⇒ ↓ plateau

Cardiac Output – Venous Return Curves
• Since CO=VR (with small variation) we can graph the cardiac function and vascular function curves together

Cardiac and Vascular function curves
- Point of equilibrium ⇒ predicts cardiac output and central venous pressure
- Steady state of this particular system
Changes in contractility

- **Digoxin:**
  - Inhibits Na-K ATPase
  - \( \text{Ca}^{++} \) builds up

\[ \text{Ejection fraction} = \frac{\text{SV}}{\text{EDV}} \]

Changes in volume: mean systemic pressure

- Decreased blood volume
- Decreased venous compliance

**Using CO-VR Curves**

**Scenario 1**

Sudden increase in blood volume by 20% on venous side (e.g., 1L infusion of blood or saline)

*Initial Effects*
- \( \uparrow \text{BV} \rightarrow \uparrow \text{venous pressure} \)
**Using CO-VR Curves**

Scenario 2
Increase in SNS activity

**Initial effects**
1. ↑ SNS → ↑ HR and ↓ TSV → ↑ CO
2. ↑ SNS → Vasconstriction → ↑ peripheral resistance
3. ↑ SNS → Venocconstriction → ↑ venous pressure

**Changes in Total Peripheral Resistance**

- Constrict arterioles
  - Increased afterload
  - Decreased venous return

**Pressure-Volume Loop**

- Special case of exercise:
  - Large ↑ SNS activity,
  - Plus:
    - Huge dilation of skeletal muscle arterioles – due to local metabolic factors – decrease resistance
    - Big increase in muscle pump activity

**Exercise**

- ↑ Preload
- ↑ Afterload
- ↑ Contractility
Pressure-Volume Loop

- ↑Preload
- ↑Afterload
- ↑Contractility

<table>
<thead>
<tr>
<th>Stroke Volume</th>
<th>= EDV - ESV</th>
</tr>
</thead>
</table>
| Preload       | Contractility
| Systolic      | Afterload   |

Pressure-Volume Loop

- ↑Preload
- ↑Afterload
- ↑Contractility

Summary of Factors That Influence Cardiac Output and Mean Arterial Pressure

- Sympathetic
  - Stimulate: increase HR and increase vasoconstriction
  - Inhibit: decrease HR and decrease vasoconstriction

- Parasympathetic (vagus)
  - Stimulate: decreases HR and causes vasodilation
Control of Cardiac Output

Factors that affect the Cardiac Output

Cardiac Output Concept Map

Myocardial Oxygen Consumption
Myocardial Oxygen Consumption

- Oxygen consumption is defined as the volume of oxygen consumed per minute (usually expressed per 100 grams of tissue weight)

Factors Increasing Myocardial Oxygen Consumption

- Increased Heart Rate
- Increased Inotropy (Contractility)
- Increased Afterload
- Increased Preload
  - Changes in preload affect myocardial oxygen consumption less than do changes in the other factors

In Summary...

- Heart rate and stroke volume are the two factors that determine cardiac output.
- Each of these is affected by many factors.
- Chronotropic agents affect heart rate while inotropic agents affect contractility, which affects stroke volume.
- Some factors (e.g., epinephrine and norepinephrine) affect both.
Answers to Questions

• If HR increases, what will happen to cardiac output?
  - Cardiac output increases.

• If SV decreases, what will happen to cardiac output?
  - Cardiac output is expected to decrease (note that heart rate can be increased to compensate).
  - What effect will sympathetic nerve impulses have on heart rate?
    - The norepinephrine released will increase heart rate.
  - What effect will parasympathetic nerve impulses have on heart rate?
    - The ACh released will decrease heart rate.

Answers to Questions

• What effect will increased heart rate have on stroke volume (if other factors stay the same)?
  Stroke volume will increase to some extent and if further increases will lead to reduced filling time and SV will decrease (note that SV may be maintained if the cause of the increased heart rate also increases contractility).

• What effect will increased venous return have on EDV?
  EDV will increase.

• What effect will blood loss have on EDV?
  EDV will decrease (note that the body has compensatory mechanisms to initially maintain SV when blood is lost).

Answers to Questions

• What effect will inhaling more deeply have on venous return?
  Venous return will increase because deeper inhalation lowers thoracic pressure more than normal.

• What effect will epinephrine have on stroke volume?
  Stroke volume will increase due to the increased contractility.

Answers to Questions

• What effect will blocking calcium channels have on stroke volume?
  Stroke volume will decrease.

• What effect will hypertension have on afterload?
  Afterload will increase.

• What effect will hypertension have on stroke volume?
  Stroke volume will decrease (and the heart will have to work harder to eject blood).

The End