Cardiac output and Venous Return

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Objectives

- Define cardiac output and venous return
- Describe the methods of measurement of CO
- Outline the factors that regulate cardiac output
- Follow up the cardiac output curves at different physiological states
- Define venous return and describe venous return curve
- Outline the factors that regulate venous return curve at different physiological states
- Inter-relate Cardiac output and venous return curves
Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m²).

- CO is proportional to tissue \(O_2\) use.
- CO is proportional to \(1/TPR\) when AP is constant.

- \(F=\Delta P/R\) (Ohm’s law)
- \(CO = (MAP - RAP) / TPR\), (RAP=0) then
- \(CO=MAP/TPR\) ; \(MAP=CO*TPR\)
Magnitude & Distribution of CO at Rest & During Moderate Exercise
## Variations in Tissue Blood Flow

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Per cent</th>
<th>ml/min</th>
<th>ml/min/100 gm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain</td>
<td>14</td>
<td>700</td>
<td>50</td>
</tr>
<tr>
<td>Heart</td>
<td>4</td>
<td>200</td>
<td>70</td>
</tr>
<tr>
<td>Bronchi</td>
<td>2</td>
<td>100</td>
<td>25</td>
</tr>
<tr>
<td>Kidneys</td>
<td>22</td>
<td>1100</td>
<td>360</td>
</tr>
<tr>
<td>Liver</td>
<td>27</td>
<td>1350</td>
<td>95</td>
</tr>
<tr>
<td>Portal (21)</td>
<td></td>
<td>(1050)</td>
<td></td>
</tr>
<tr>
<td>Arterial (6)</td>
<td></td>
<td>(300)</td>
<td></td>
</tr>
<tr>
<td>Muscle (inactive state)</td>
<td>15</td>
<td>750</td>
<td>4</td>
</tr>
<tr>
<td>Bone</td>
<td>5</td>
<td>250</td>
<td>3</td>
</tr>
<tr>
<td>Skin (cool weather)</td>
<td>6</td>
<td>300</td>
<td>3</td>
</tr>
<tr>
<td>Thyroid gland</td>
<td>1</td>
<td>50</td>
<td>160</td>
</tr>
<tr>
<td>Adrenal glands</td>
<td>0.5</td>
<td>25</td>
<td>300</td>
</tr>
<tr>
<td>Other tissues</td>
<td>3.5</td>
<td>175</td>
<td>1.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100.0</strong></td>
<td><strong>5000</strong></td>
<td><strong>---</strong></td>
</tr>
</tbody>
</table>
Control of Cardiac Output

Cardiac output

Heart rate

- ↑ Parasympathetic activity

+ ↑ Sympathetic activity (and epinephrine)

Stroke volume

+ ↑ End-diastolic volume

Intrinsic control

Extrinsic control

- ↑ Venous return
Factors that affect the Cardiac Output

- Increased end diastolic volume (stretches the heart)
  - Increased PRELOAD
    - Within limits, cardiac muscle fibers contract more forcefully with stretching (Frank-Starling law of the heart)
- Positive inotropic agents such as increased sympathetic stimulation; catecholamines, glucagon, or thyroid hormones in the blood; increased Ca²⁺ in extracellular fluid
  - Increased CONTRACTILITY
    - Positive inotropic agents increase force of contraction at all physiological levels of stretch
- Decreased arterial blood pressure during diastole
  - Decreased AFTERLOAD
    - Semilunar valves open sooner when blood pressure in aorta and pulmonary artery is lower

- Increased STROKE VOLUME

- Increased HEART RATE
- Increased sympathetic stimulation and decreased parasympathetic stimulation

- NERVOUS SYSTEM
  - Cardiovascular center in medulla oblongata receives input from cerebral cortex, limbic system, proprioceptors, baroreceptors, and chemoreceptors

- CHEMICALS
  - Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca²⁺

- OTHER FACTORS
  - Infants and senior citizens, females, low physical fitness, increased body temperature
Ventricular Stroke Work Output

Left Atrial Mean Pressure (mm Hg)

Right Atrial Mean Pressure (mm Hg)
Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output
IPP = INTRAPLEURAL PRESSURE

CARDIAC OUTPUT (L/min)

RIGHT ATRIAL PRESSURE (mmHg)

IPP = -5.5 mmHg
IPP = -4 mmHg
IPP = -2 mmHg
IPP = 2 mmHg

CARDIAC TAMPONADE
The Cardiac Output Curve

- Plateau of CO curve determined by heart strength (contractility + HR)
  - Sympathetics $\Rightarrow$ plateau
  - $\downarrow$ Parasympathetics (HR) $\Rightarrow$ (? plateau)
  - Plateau
  - Heart hypertrophy’s $\Rightarrow$ plateau
  - Myocardial infarction $\Rightarrow$ (? plateau)
  - $\downarrow$ Plateau
The Cardiac Output Curve (cont’d)

- Valvular disease $\Rightarrow$ ↓ plateau  
  (stenosis or regurgitation)
- Myocarditis $\Rightarrow$ ↓ plateau
- Cardiac tamponade $\Rightarrow$ (plateau)
- ↓ Plateau
- Metabolic damage $\Rightarrow$ ↓ plateau
Factors Affecting Cardiac Output

- Autonomic innervation
- Hormones
- End-diastolic volume
- End-systolic volume

HEART RATE

STROKE VOLUME

CARDIAC OUTPUT
Factors Affecting Stroke Volume

**Venous return (VR)**
- $\uparrow VR = \uparrow EDV$
- $\downarrow VR = \downarrow EDV$

**Filling time (FT)**
- $\uparrow FT = \uparrow EDV$
- $\downarrow FT = \downarrow EDV$

**End-diastolic volume (EDV)**

**Contractility of Muscle cells**
- $\uparrow Cont = \downarrow ESV$
- $\downarrow Cont = \uparrow ESV$

**Increased by sympathetic stimulation**

**Increased by E, NE, glucagon, thyroid hormones**

**Decreased by parasympathetic stimulation**

**End-systolic volume (ESV)**

**Afterload**
- $\uparrow AL = \uparrow ESV$
- $\downarrow AL = \downarrow ESV$

**STROKE VOLUME (SV)**
- $\uparrow EDV = \uparrow SV$
- $\downarrow EDV = \downarrow SV$
- $\uparrow ESV = \downarrow SV$
- $\downarrow ESV = \uparrow SV$
A Summary of the Factors Affecting Cardiac Output

(a) Factors affecting heart rate

- Atrial reflex
- Autonomic innervation
- Hormones
- HEART RATE
- Filling time

(b) Factors affecting stroke volume

- Venous return
- Blood volume
- Changes in peripheral circulation
- Hormones
- Autonomic innervation
- Preload
- Contractility
- Afterload
- End-diastolic volume
- End-systolic volume

CARDIAC OUTPUT
REGULATION OF STROKE VOLUME: PRELOAD

- increased venous pressure
- increased venous return
- increased ventricular filling
- increased preload
- increased ventricular stretch
  Frank-Starling mechanism
- increased force of contraction
- increased stroke volume
- increased cardiac output

- decreased heart rate
- increased length of diastole
- increased ventricular filling
REGULATION OF STROKE VOLUME: CONTRACTILITY

- increased sympathetic activity
- increased epinephrine
- other factors

→ increased contractility

→ increased force of contraction

→ increased stroke volume

→ increased cardiac output
Best is to measure the C.O. curve, but this is nearly impossible in humans.
dP/dt is not an accurate measure because this increases with increasing preload and afterload.
(dP/dt)/P_{ventricle} is better. P_{ventricle} is instantaneous ventricular pressure.
Excess K^+ decreases contractility.
Excess Ca^{++} causes spastic contraction, and low Ca^{++} causes cardiac dilation.
REGULATION OF STROKE VOLUME: AFTERLOAD

- Increased arterial pressure
  - Increased afterload
    - Decreased blood volume ejected into artery
      - Decreased stroke volume
        - Decreased cardiac output
Measurement of Cardiac Output

- Electromagnetic flowmeter
- Indicator dilution (dye such as cardiogreen)
- Thermal dilution
- **Oxygen Fick Method**
  
  \[ CO = \frac{O_2 \text{ consumption}}{A-V O_2 \text{ difference}} \]
Electromagnetic flowmeter
\[ q_1 = CO \times C_{VO2} \]
\[ q_2 = \text{amount of Oxygen uptake by the lungs} \]
\[ q_3 = CO \times C_{AO2} \text{ and equals } = CO \times C_{VO2} + \text{O}_2 \text{ uptake} \]
\[ \text{Oxygen uptake} = CO \{C_{AO2} - C_{VO2}\} \]
\[ CO = \text{Oxygen uptake/} \{C_{AO2} - C_{VO2}\} \]
Spirometer
Swan-Ganz catheter
O₂ Fick Problem

- If pulmonary vein O₂ content = 200 ml O₂/L blood
- Pulmonary artery O₂ content = 160 ml O₂ /L blood
- Lungs add 400 ml O₂ /min
- What is cardiac output?
- Answer: \( \frac{400}{(200-160)} = 10 \text{ L/min} \)
THE INDICATOR DILUTION PRINCIPLE

\[ \text{Area} = \int_{t_2}^{t_1} dc \, dt \]

\[ \text{Area} = \bar{C} \times (t_2 - t_1) \]  
(Rectangular)

\[ \bar{C} = \frac{\text{Area}}{(t_2 - t_1)} \]

Cardiac output = \( \frac{q}{C} \times \frac{X}{\text{duration in seconds}} \times \frac{60}{\text{km}} \)
Thermodilution Method Curve

\[
\text{AREA} = \int_{t_1}^{t_2} dT \cdot dt
\]
VENOUS RETURN

- Definition: Volume of blood returns to either the left side or right side of the heart per minute

- \( VR = CO = \Delta P/R \)

- \( VR = \text{(Venous pressure - Rt. Atrial pressure)}/ \text{resistance to venous return} \)
Effect of Venous Valves
Effect of Venous Valves

(a) Contracted skeletal muscles
(b) Relaxed skeletal muscles
Venous Valves

- Deep vein
- Perforating vein
- Superficial vein
- Valve
Effect Of Gravity on Venous Pressure

Pressure = 100 mm Hg
90 mm Hg caused by gravitational effect
10 mm Hg caused by pressure imparted by cardiac contraction

Pooling of blood in distended veins

Venous return

Filtration → swelling of ankles and feet

Capillary blood pressure = 137 mm Hg
Vessel Structure and Function

Normal vein

Blood flow
Closed valve

Varicose vein

Incompetent valve

Dilated and twisted appearance of varicose veins in the leg
Venous Pressure in the Body

- Compressional factors tend to cause resistance to flow in large peripheral veins.

- Increases in right atrial pressure causes blood to back up into the venous system thereby increasing venous pressures.

- Abdominal pressures tend to increase venous pressures in the legs.
Pressure in the right atrium is called central venous pressure.

Right atrial pressure is determined by the balance of the heart pumping blood out of the right atrium and flow of blood from the large veins into the right atrium.

Central venous pressure is normally 0 mmHg, but can be as high as 20-30 mmHg.
Factors affecting Central Venous Pressure

- Right atrial pressure (RAP) is regulated by a balance between the ability of the heart to pump blood out of the atrium and the rate of blood flowing into the atrium from peripheral veins.

- Factors that increase RAP:
  - increased blood volume
  - increased venous tone
  - dilation of arterioles
  - decreased cardiac function
Factors that Facilitate Venous Return

- **↑ Cardiac output**
  - **↑ Stroke volume**
  - **↑ End-diastolic volume**

- **Venous valves** (mechanically prevent backflow of blood)

- **Cardiac-suction effect** (↓ pressure in heart → ↑ pressure gradient)

- **Pressure imparted to blood by cardiac contraction** (↑ venous pressure → ↑ pressure gradient)

- **↑ Sympathetic vasoconstrictor activity** (↑ venous pressure → ↑ pressure gradient)

- **↑ Blood volume** (↑ venous pressure → ↑ pressure gradient)

- **Passive bulk-flow shift of fluid from interstitial fluid into plasma**

- **Salt and water retention**

- **Respiratory pump** (↓ pressure in chest veins → ↑ pressure gradient)

- **Skeletal muscle pump** (↑ venous pressure → ↑ pressure gradient)

- **Short-term control measures**
- **Long-term control measures**
The Venous Return Curve

MSFP = Mean Systemic Filling Pressure

Venous Return (l/min) vs. Right Atrial Pressure (mmHg)

- MSFP = 4.2
- MSFP = 7
- MSFP = 14
VENOUS RETURN (L/min/m)

RIGHT ATRIAL PRESSURE (mmHg)

MSFP = 7

NORMAL RESISTANCE

1/2 RESISTANCE

2 X RESISTANCE
Venous Return (VR)

- Beriberi - thiamine deficiency ⇒ arteriolar dilatation ⇒ \( \downarrow \) RVR
- (RVR= resistance to venous return)
  because VR = (MSFP - RAP) / RVR
  (good for positive RAP’s)
- A-V fistula ⇒ (? RVR)
- \( \downarrow \) RVR
- C. Hyperthyroidism ⇒ (? RVR)
- \( \downarrow \) RVR
Anemia $\Rightarrow$ ↓ RVR (why?)

Sympathetics $\Rightarrow$ MSFP

Blood volume $\Rightarrow$ MSFP + small ↓ in RVR

↓ Venous compliance (muscle contraction or venous constriction) $\Rightarrow$ (?) MSFP

MSFP
Factors Causing ↓Venous Return

- ↓ Blood volume ⇒ ↓ MSFP
- ↓ Sympathetics ⇒ (? v. comp. and MSFP)
- Venous compliance and ↓MSFP
- Obstruction of veins ⇒ (? RVR)
- RVR
CARDIAC OUTPUT AND VENOUS RETURN (L/min/m)

RIGHT ATRIAL PRESSURE (mmHg)

- MAX
- SPINAL ANESTHESIA
- SPINAL
- VR CURVE NORMAL
- SYMPATHETIC STIMULATION
- MAXIMAL SYMPATHETIC STIMULATION

NORMAL CARDIAC
Thank You