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
Important Concepts About Cardiac Output (CO) Control

- Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m$^2$).
- CO is proportional to tissue O$_2$ use.
- CO is proportional to 1/TPR when AP is constant.
- $F=\frac{\Delta P}{R}$ (Ohm’s law)
- $CO = \frac{(MAP - RAP)}{TPR}$, (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 (Arterial)</td>
<td>21</td>
<td>1050</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

Stroke volume

† Sympathetic activity (and epinephrine)

† End-diastolic volume

† Venous return

Extrinsic control

Intrinsic control

Intrinsic control
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 Ca^{2+} 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 CARDIAC OUTPUT**

- **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
  - Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca^{2+}
  - Infants and senior citizens, females, low physical fitness, increased body temperature

- **CHEMICALS**
- **OTHER FACTORS**
Ventricular Stroke Work Output

L.V. stroke work (gram meters)

R.V. stroke work (gram meters)

Left Atrial Mean Pressure (mm Hg)

Right Atrial Mean Pressure (mm Hg)
CARDIAC OUTPUT CURVES

HYPOEFFECTIVE

HYPEREFFECTIVE

CARDIAC OUTPUT (L/min)

RIGHT ATRIAL PRESSURE (mmHg)
Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output
RIGHT ATRIAL PRESSURE (mmHg)

IPP = INTRAPLEURAL PRESSURE

CARDIAC OUTPUT (L/min)

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

CARDIAC TAMPOANDE
The Cardiac Output Curve

• Plateau of CO curve determined by heart strength (contractility + \( \uparrow HR \))
  
• \( \uparrow \) Sympathetics \( \Rightarrow \uparrow \) plateau

• \( \downarrow \) Parasympathetics (HR \( \uparrow \)) \( \Rightarrow \) (? plateau)

• \( \uparrow \) Plateau

• Heart hypertrophy \( \Rightarrow \uparrow \)’s plateau

• Myocardial infarction \( \Rightarrow \) (? plateau)

• \( \downarrow \) Plateau
The Cardiac Output Curve (cont’d)

- Valvular disease $\Rightarrow \downarrow$ plateau  
  (stenosis or regurgitation)
- Myocarditis $\Rightarrow \downarrow$ plateau
- Cardiac tamponade $\Rightarrow (? \text{ plateau})$
- $\downarrow$ Plateau
- Metabolic damage $\Rightarrow \downarrow$ plateau
Factors Affecting Cardiac Output

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

Heart Rate

Stroke Volume

Cardiac Output
Factors Affecting Stroke Volume

Contractility of Muscle cells
- Increased by sympathetic stimulation
- Increased by E, NE, glucagon, thyroid hormones
- Decreased by parasympathetic stimulation

Contractility (Cont) and End-systolic Volume (ESV)
- Increased Cont → Decreased ESV
- Decreased Cont → Increased ESV

Venous return (VR) and Filling time (FT)
- VR = EDV
- FT = EDV

End-diastolic volume (EDV) and End-systolic volume (ESV)

Stroke Volume (SV)
- EDV = SV
- ESV = SV
- EDV = SV
- ESV = SV

Afterload
- Increased AL → Increased ESV
- Decreased AL → Decreased ESV
A Summary of the Factors Affecting Cardiac Output
REGULATION OF STROKE VOLUME: PRELOAD

- Increased venous pressure
- Decreased heart rate

- Increased venous return
- Increased length of diastole

- Increased ventricular filling
- Increased ventricular filling

- Increased preload

- Increased ventricular stretch
  Frank-Starling mechanism

- Increased force of contraction

- Increased stroke volume

- Increased cardiac output
REGULATION OF STROKE VOLUME: CONTRACTILITY

- increased sympathetic activity
- increased epinephrine
- other factors

→ increased contractility

→ increased force of contraction

→ increased stroke volume

→ increased cardiac output
Cardiac Contractility

- 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.
- \((dP/dt)/P_{\text{ventricle}}\) is better. \(P_{\text{ventricle}}\) 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
\[ q_1 = CO \cdot C_{VO_2} \]
\[ q_2 = \text{amount of Oxygen uptake by the lungs} \]
\[ q_3 = CO \cdot C_{AO_2} \text{ and equals } = CO \cdot C_{VO_2} + O_2 \text{ uptake} \]
\[ \text{Oxygen uptake} = CO \{ C_{AO_2} - C_{VO_2} \} \]
\[ CO = \frac{\text{Oxygen uptake}}{C_{AO_2} - C_{VO_2}} \]
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

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

Area = C^* (t_2 - t_1)

(Rectangular)

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

Cardiac output = \frac{q}{C} \times \frac{X}{60} \times \frac{60}{\text{duration in seconds}}
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 = Δ P/R

• VR = (Venous pressure – Rt. Atrial pressure)/ 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

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

(b) Venous pressure = 100 mm Hg
Capillary blood pressure = 137 mm Hg
Filtration → swelling of ankles and feet

Pooling of blood in distended veins

↓ Venous return

1.5 m
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.
Central Venous Pressure

- Pressure in the right atrium is called \textit{central venous pressure}.

- \textit{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
- Passive bulk-flow shift of fluid from interstitial fluid into plasma
- Salt and water retention
- ↑ Blood volume (↑ venous pressure → ↑ pressure gradient)
- Respiratory pump (↓ pressure in chest veins → ↑ pressure gradient)
- Skeletal muscle pump (↑ venous pressure → ↑ pressure gradient)

- 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)

Diagram notes:
- □ = Short-term control measures
- □ = Long-term control measures
The Venous Return Curve

MSFP = Mean Systemic Filling Pressure

MSFP = 4.2

MSFP = 7

MSFP = 14
NORMAL RESISTANCE
1/2 RESISTANCE
2 X RESISTANCE

MSFP = 7

RIGHT ATRIAL PRESSURE (mmHg)
Venous Return (VR)

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

• Anemia ⇒ ↓ RVR (why?)
• ↑ Sympathetics ⇒ ↑ MSFP
• ↑ Blood volume ⇒ ↑ MSFP + small ↓ in RVR
• ↓ Venous compliance (muscle contraction or venous constriction) ⇒ (?) MSFP
• ↑ MSFP
Factors Causing ↓Venous Return

• ↓ Blood volume ⇒ ↓ MSFP
• ↓ Sympathetics ⇒ (? v. comp. and MSFP)
• ↑ Venous compliance and ↓MSFP
• Obstruction of veins ⇒ (? RVR)
• ↑ RVR
RIGHT ATRIAL PRESSURE (mmHg)

CARDIAC OUTPUT AND VENOUS RETURN (L/min/m)

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