Cardiac output and Venous Return

Faisal I. Mohammed, MD, PhD
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/m²).
- CO is proportional to tissue O₂ 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
- \( CO = \frac{MAP}{TPR} ; MAP = CO \times TPR \)
Magnitude & Distribution of CO at Rest & During Moderate Exercise

- At rest
  - Digestive tract, liver: 27% of total cardiac output (1,350 ml/min)
  - Kidneys: 20% of total cardiac output (1,000 ml/min)
  - Skin: 9% of total cardiac output (450 ml/min)
  - Brain: 13% of total cardiac output (650 ml/min)
  - Heart: 3% of total cardiac output (150 ml/min)
  - Skeletal muscle: 15% of total cardiac output (750 ml/min)
  - Bone, other: 13% of total cardiac output (650 ml/min)
  - Total cardiac output: 5,000 ml/min

- Moderate exercise
  - Digestive tract, liver: 367% increase (5,550 ml/min)
  - Kidneys: 45% increase (5,500 ml/min)
  - Skin: 370% increase (1,700 ml/min)
  - Brain: 5.2% increase (650 ml/min)
  - Heart: 4.4% increase (560 ml/min)
  - Skeletal muscle: 1068% increase (8,000 ml/min)
  - Bone, other: 30% increase (1,500 ml/min)
  - Total cardiac output: 12,500 ml/min
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<tr>
<th>Tissue</th>
<th>Per cent</th>
<th>ml/min</th>
<th>ml/min/100 gm</th>
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<td>14</td>
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<tr>
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<td>200</td>
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<tr>
<td>Bronchi</td>
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<td>1350</td>
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<td>Portal Arterial</td>
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<td>(1050)</td>
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<td>Muscle (inactive state)</td>
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<td>750</td>
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<tr>
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<td>250</td>
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<tr>
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<td>300</td>
<td>3</td>
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<td>160</td>
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<tr>
<td>Adrenal glands</td>
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<td>25</td>
<td>300</td>
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<tr>
<td>Other tissues</td>
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<tr>
<td><strong>Total</strong></td>
<td>100.0</td>
<td>5000</td>
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Control of Cardiac Output

Cardiac output

Heart rate

Stroke volume

↑ Parasympathetic activity

↑ 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 end diastolic volume (stretches the heart) → Increased STROKE VOLUME
- 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
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- Increased STROKE VOLUME → Increased CARDIAC OUTPUT

- Increased sympathetic stimulation and decreased parasympathetic stimulation → Increased HEART RATE
  - Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca^{2+}

- Infants and senior citizens, females, low physical fitness, increased body temperature

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

- CHEMICALS

- OTHER FACTORS
**Ventricular Stroke Work Output**

- **Left Atrial Mean Pressure (mm Hg)**
  - L.V. stroke work (gram meters)

- **Right Atrial Mean Pressure (mm Hg)**
  - R.V. stroke work (gram meters)
CARDIAC OUTPUT CURVES

RIGHT ATRIAL PRESSURE (mmHg)

CARDIAC OUTPUT (L/min)

HYPEREFFECTIVE

NORMAL

HYPOEFFECTIVE
Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output

Cardiac Output (L/min)

Right Atrial Pressure (mmHg)
$\text{CARDIAC OUTPUT (L/min)}$

$\text{RIGHT ATRIAL PRESSURE (mmHg)}$

$I\text{PP} = \text{INTRAPLEURAL PRESSURE}$
The Cardiac Output Curve

- Plateau of CO curve determined by heart strength (contractility + ↑HR)
- ↑ Sympathetics ⇒ ↑ plateau
- ↓ Parasympathetics (HR↑) ⇒ (?) plateau
- ↑ Plateau
- Heart hypertrophy’s ⇒ ↑ plateau
- Myocardial infarction ⇒ (?) plateau
- ↓ 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

Heart Rate

End-diastolic volume
End-systolic volume

Cardiac Output

Stroke Volume
Factors Affecting Stroke Volume

Contractility of Muscle cells

- Increased by sympathetic stimulation
- Increased by E, NE, glucagon, thyroid hormones
- Decreased by parasympathetic stimulation

End-systolic volume (ESV)

Afterload
- Increased by vasoconstriction
- Decreased by vasodilation

STROKE VOLUME (SV)

- 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 (Cont)
  - $\uparrow$ Cont = $\downarrow$ ESV
  - $\downarrow$ Cont = $\uparrow$ ESV

- Increased by $\uparrow$ EDV = $\uparrow$ SV
  - ESV = $\downarrow$ SV
- Decreased by $\downarrow$ EDV = $\downarrow$ SV
  - ESV = $\uparrow$ SV
A Summary of the Factors Affecting Cardiac Output

(a) Factors affecting heart rate
- Autonomic innervation
- Hormones
- Atrial reflex
- Skeletal muscle activity

Venous return
- Blood volume
- Changes in peripheral circulation

(b) Factors affecting stroke volume
- End-diastolic volume
- End-systolic volume
- Preload
- Contractility
- Afterload
- Hormones
- Autonomic innervation

Changes in peripheral circulation

Cardiac output
- Filling time

Heart rate

Graphical representation of factors affecting heart rate and stroke volume.
REGULATION OF STROKE VOLUME: PRELOAD

- Increased venous pressure
- Decreased heart rate
  - Increased length of diastole
- Increased venous return
- Increased venous pressure
- Increased venous return
  - Increased ventricular filling
- Increased ventricular filling
  - Increased preload
- 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.
- \( \frac{dP}{dt} \) is not an accurate measure because this increases with increasing preload and afterload.
- \( \left( \frac{dP}{dt} \right)/P_{\text{ventricle}} \) 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

\[ CO = \left( \frac{O_2 \text{ consumption}}{A-V O_2 \text{ difference}} \right) \]
Electromagnetic flowmeter
\[ q_1 = CO \times C_{VO2} \]
\[ q_2 = \text{amount of Oxygen uptake by the lungs} \]
\[ q_3 = CO \times C_{AO2} \text{ and equals } = CO \times C_{VO2} + O_2 \text{ uptake} \]
\[ \text{Oxygen uptake} = CO \{ C_{AO2} - C_{VO2} \} \]
\[ CO = \text{Oxygen uptake} / \{ C_{A02} - C_{VO2} \} \]
Spirometer

A spirometer

Floating drum
Air
Water
Expired air
Inspired air

Recording paper advancing with time
Spirogram
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: 400/(200-160) = 10 L/min
THE INDICATOR DILUTION PRINCIPLE

Area = \( \int_{t_1}^{t_2} dc/dt \)

Area = \( \bar{C}^* \) (t_2 - t_1) (Rectangular)

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

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

\[ \text{AREA} = \int_{t1}^{t2} dT \, dt \]
**VENOUS RETURN**

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

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

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

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

(b) Pooling of blood in distended veins
Venous pressure = 100 mm Hg
Capillary blood pressure = 137 mm Hg
↓ Venous return
↓ Filtration → swelling of ankles and feet
Vessel Structure and Function

Diagram showing normal vein with blood flow and closed valve, and varicose vein with 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

1. Cardiac output
2. Stroke volume
3. End-diastolic volume
4. Venous valves (mechanically prevent backflow of blood)
5. Cardiac-suction effect (↓ pressure in heart → ↑ pressure gradient)
6. Pressure imparted to blood by cardiac contraction (↑ venous pressure → ↑ pressure gradient)
7. Sympathetic vasoconstrictor activity (↑ venous pressure → ↑ pressure gradient)
8. Respiratory pump (↓ pressure in chest veins → ↑ pressure gradient)
9. Blood volume (↑ venous pressure → ↑ pressure gradient)
10. Passive bulk-flow shift of fluid from interstitial fluid into plasma
11. Salt and water retention

Legend:
- ■ = Short-term control measures
- □ = Long-term control measures
The Venous Return Curve

MSFP = Mean Systemic Filling Pressure

MSFP = 4.2

MSFP = 7

MSFP = 14
VENOUS RETURN (L/min/m)

RIGHT ATRIAL PRESSURE (mmHg)

-4 0 4 8

NORMAL RESISTANCE

1/2 RESISTANCE

2 X RESISTANCE

MSFP = 7

VENOUS RETURN (L/min/m)

RIGHT ATRIAL PRESSURE (mmHg)
Venous Return (VR)

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

Normal Cardiac

Spinal Anesthesia

Maximal Sympathetic Stimulation

Spinal

Sympathetic Stimulation

VR Curve Normal

Anesthesia

MAX

0  4  8  12  16  20  25

0  5  10  15  20  25

-4  0  4  8  12  16

Right Atrial Pressure (mmHg)
Thank You