Heart Pump and Cardiac Cycle

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Objectives

- To understand the volume, mechanical, pressure and electrical changes during the cardiac cycle
- To understand the inter-relationship between all these changes
- To describe the factors that regulate Cardiac output and Stroke volume.

Resources: Textbook of Medical Physiology By Guyton and Hall
Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
  - Systole – contraction of heart muscle
  - Diastole – relaxation of heart muscle
Cardiac Cycle

- Atrial systole 0.1 second
- Atrial diastole 0.7 second
- Ventricular systole 0.3 second
  - Isovolumic contraction 0.01 seconds
  - Rapid ejection period
  - Slow ejection period
- Ventricular diastole 0.5 seconds
  - Isovolumic relaxation 0.02 seconds
  - Rapid filling
  - Slow filling (Diastasis)
  - Atrial contraction
Cardiac cycle …cont

- End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)
- SV X heart rate (HR) = cardiac output (CO)
- Ejection fraction = SV/EDV
- Inotropic vs. Chronotropic
- Autonomic control of cardiac cycle (pump)
Phases of the Cardiac Cycle

- Ventricular filling – mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - AV valves are open, then atrial systole occurs
Phases of the Cardiac Cycle

- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase
  - Ventricular ejection phase opens semilunar valves
Phases of the Cardiac Cycle

- **Isovolumetric relaxation – early diastole**
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves

- **Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves**
Changes during Cardiac cycle

- Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.
- Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic
- Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.
- Atrial pressure: A wave = atrial systole, C wave = ventricular contraction (AV closure), V wave = ventricular diastole (Av opening)
- Heart sounds: $S_1$ = turbulence of blood around a closed AV valves, $S_2$ = turbulence of blood around a closed semilunar valves.
Heart Sounds

(a) Sounds heard:
- Aortic valve location
- Pulmonary valve location
- Left AV valve location
- Right AV valve location

(b) Pressure (mm Hg):
- Semilunar valves open
- Semilunar valves close
- AV valves close
- AV valves open

Heart sounds:
- S4
- S1
- S2
- S3
- S4

"Lubb" and "Dupp"
Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves
Heart sounds

- Auscultation – listening to heart sound via stethoscope
- Four heart sounds
  - $S_1$ – “lubb” caused by the closing of the AV valves
  - $S_2$ – “dupp” caused by the closing of the semilunar valves
  - $S_3$ – a faint sound associated with blood flowing into the ventricles
  - $S_4$ – another faint sound associated with atrial contraction
Cardiac Output (CO) and Reserve

- CO is the amount of blood pumped by each ventricle in one minute.
- CO is the product of heart rate (HR) and stroke volume (SV).
- HR is the number of heart beats per minute.
- SV is the amount of blood pumped out by a ventricle with each beat.
- Cardiac reserve is the difference between resting and maximal CO.
Cardiac Output: Example

- \[ CO \text{ (ml/min)} = HR \text{ (75 beats/min)} \times SV \text{ (70 ml/beat)} \]
- \[ CO = 5250 \text{ ml/min (5.25 L/min)} \]
Regulation of Stroke Volume

- SV = end diastolic volume (EDV) minus end systolic volume (ESV)
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction
Factors Affecting Stroke Volume

- **Preload** – amount ventricles are stretched by contained blood
- **Contractility** – cardiac cell contractile force due to factors other than EDV
- **Afterload** – back pressure exerted by blood in the large arteries leaving the heart
Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume.
- Slow heartbeat and exercise increase venous return to the heart, increasing SV.
- Blood loss and extremely rapid heartbeat decrease SV.
(a) Preload

(b) Afterload
Phases of the Cardiac Cycle

(a) Atrial systole begins: Atrial contraction forces a small amount of additional blood into relaxed ventricles.

(b) Atrial systole ends; atrial diastole begins

(c) Ventricular systole—first phase: Ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.

(d) Ventricular systole—second phase: As ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.

(e) Ventricular diastole—early: As ventricles relax, pressure in ventricles drops; blood flows back against cusps of semilunar valves and forces them closed. Blood flows into the relaxed atria.

(f) Ventricular diastole—late: All chambers are relaxed. Ventricles fill passively.
Extrinsic Factors Influencing Stroke Volume

- Contractility is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca\(^{2+}\) and some drugs
Extrinsic Factors Influencing Stroke Volume

- Agents/factors that decrease contractility include:
  - Acidosis
  - Increased extracellular $K^+$
  - Calcium channel blockers
Contractility and Norepinephrine

- Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system

![Diagram of norepinephrine and cyclic AMP signaling]

1. Extracellular fluid
   - Norepinephrine

2. β₁-adrenergic receptor
   - G protein
   - Adenylate cyclase
   - ATP → cAMP

3. cAMP → Inactive protein kinase → Active protein kinase

4. Active protein kinase → Sarcoplasmic reticulum
   - Ca²⁺ → Enhanced actin–myosin interaction → Troponin
   - Troponin → Ca²⁺ binds to

Cardiac contractile force and velocity
Potential Energy (PE)
LEFT VENTRICULAR PRESSURE/VOLUME P/V LOOP

LEFT VENTRICULAR PRESSURE (mmHg)

LEFT VENTRICULAR VOLUME (ml)

A-V valves Close

Semilunar Valves Close

A-V valves Open

Semilunar Valves Open

A-V valves Close
Valvular Function

- To prevent back-flow.
- Chordae tendineae are attached to A-V valves.
- Papillary muscle, attached to chordae tendineae, contract during systole and help prevent back-flow.
- Because of smaller opening, velocity through aortic and pulmonary valves exceed that through the A-V valves.
Valvular Function (cont’d)

- Most work is external work or pressure-volume work.
- A small amount of work is required to impart kinetic energy to the heart \( (1/2 \ mV^2) \).
- What is stroke-volume in previous figure?
- External work is area of Pressure-Volume curve.
- Work output is affected by “preload” (end-diastolic pressure) and “afterload” (aortic pressure).
Increased preload
Increased afterload
Increased contractility
PRESSURE/VOLUME RELATIONSHIPS UNDER DIFFERENT CONDITIONS

PRELOAD

AFTERLOAD

CONTRACTILITY
Intrinsic Control of Stroke Volume (Frank-Starling Curve)

- **Optimal length**
  - Descending limb of length-tension curve does not exist in normal heart.

**Normal resting length**

**End-diastolic volume (EDV) (ml)**
- (related to cardiac muscle fiber length)

**Stroke volume (SV) (ml)**
- (related to muscle tension)

- Increase in SV
  - $B^1$
  - $A^1$

- Increase in EDV
  - $A$ to $B$
Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
- Negative chronotropic factors decrease heart rate
Sympathetic nervous system (SNS) stimulation is activated by stress, anxiety, excitement, or exercise.

Parasympathetic nervous system (PNS) stimulation is mediated by acetylcholine and opposes the SNS.

PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone.
Atrial (Bainbridge) Reflex

- Atrial (Bainbridge) reflex – a sympathetic reflex initiated by increased blood in the atria
  - Causes stimulation of the SA node
  - Stimulates baroreceptors in the atria, causing increased SNS stimulation
Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate
- Intra- and extracellular ion concentrations must be maintained for normal heart function
Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m² (surface area in m²)).

CO is proportional to tissue O₂ use.

CO is proportional to 1/TPR when AP is constant.

\[ CO = \frac{MAP - RAP}{TPR} \]
CARDIAC OUTPUT CURVES

CARDIAC OUTPUT (L/min)

RIGHT ATRIAL PRESSURE (mmHg)
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 $\Rightarrow$ ’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 $\Rightarrow (?)$ plateau
- $\downarrow$ Plateau
- Metabolic damage $\Rightarrow \downarrow$ plateau
During the latter part of the ejection phase how can blood still leave the ventricle if pressure is higher in the aorta? Momentum of blood flow

- Total energy of blood \( = P + mV^2/2 \)
  \[ = \text{pressure} + \text{kinetic energy} \]
- Total energy of blood leaving ventricle is greater than in aorta.
Ejection Fraction

- End diastolic volume = 125 ml
- End systolic volume = 55 ml
- Ejection volume (stroke volume) = 70 ml
- Ejection fraction = 70ml/125ml = 56%
  (normally 60%)
- If heart rate (HR) is 70 beats/minute, what is cardiac output?
  - Cardiac output = HR * stroke volume
    = 70/min. * 70 ml
    = 4900ml/min.
Ejection Fraction (cont’d)

- If HR = 100, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is cardiac output?
  - C.O. = 100/min. * 160 ml = 16,000 ml/min.
  - Ejection fraction = 160/180% =~ 90%
Aortic Pressure Curve

- Aortic pressure starts increasing during systole after the aortic valve opens.
- Aortic pressure decreases toward the end of the ejection phase.
- After the aortic valve closes, an *incisura* occurs because of sudden cessation of back-flow toward left ventricle.
- Aortic pressure decreases slowly during diastole because of the elasticity of the aorta.
Frank-Starling Mechanism

- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.
- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.
Ventricular Stroke Work Output

L.V. stroke work (gram meters)

Left Atrial Mean Pressure (mm Hg)

R.V. stroke work (gram meters)

Right Atrial Mean Pressure (mm Hg)
Autonomic Effects on Heart

- Sympathetic stimulation causes increased HR and increased contractility with HR = 180-200 and C.O. = 15-20 L/min.
- Parasympathetic stimulation decreases HR markedly and decreases cardiac contractility slightly. Vagal fibers go mainly to atria.
- Fast heart rate (tachycardia) can decrease C.O. because there is not enough time for heart to fill during diastole.
Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output

Cardiac Output (L/min)

Right Atrial Pressure (mmHg)
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.
- $(dP/dt)/P_{ventricle}$ is better. $P_{ventricle}$ is instantaneous ventricular pressure.
- Excess $K^+$ decreases contractility.
- Excess $Ca^{++}$ causes spastic contraction, and low $Ca^{++}$ causes cardiac dilation.
Thank You