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

Diagram showing heart sounds and pressure changes in the aorta and left atrium. The sounds are classified as:
- \(S_1\) (Lubb)
- \(S_2\) (Dupp)
- \(S_3\)
- \(S_4\)

Key points:
- Semilunar valves open
- Semilunar valves close
- AV valves open
- AV valves close

Diagram labeled (a) and (b) with anatomical locations of valve sounds and pressure changes.
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 (ml/min) = HR (75 beats/min) \times SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)
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
  = 4900 ml/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%
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.
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.
(a) Preload

(b) Afterload
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 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

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

- **OTHER FACTORS**
  - CHEMICALS
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.

Cardiac cycle

START

800 msec
0 msec
100 msec
370 msec
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
  - $\text{Ca}^{2+}$ and some drugs
Extrinsic Factors Influencing Stroke Volume

- Agents/factors that decrease contractility include:
  - Acidosis
  - Increased extracellular $K^+$
  - Calcium channel blockers
Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system.
Potential Energy (PE)
LEFT VENTRICULAR PRESSURE/VOLUME P/V LOOP

SEMILUNAR VALVES CLOSE

A-V valves Open

SEMILUNAR VALVES OPEN

A-V valves Close

A-V valves Close

A-V valves Open

LEFT VENTRICULAR PRESSURE (mmHg)

LEFT VENTRICULAR VOLUME (ml)
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).
Work Output of the Heart

Intraventricular Pressure (mmHg)

Left Ventricular Volume (ml)

- Period of Filling
- Isovolumic Relaxation
- Period of Ejection
- Isovolumic Contraction
- End systolic Volume
- End diastolic Volume

Diagram shows the relationship between intraventricular pressure and left ventricular volume, highlighting the different phases of the cardiac cycle.
Increased preload
Increased afterload
Increased contractility
PRESSURE/VOLUME RELATIONSHIPS UNDER DIFFERENT CONDITIONS

A

B

C

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)

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

Normal resting length

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

Increase in EDV

Increase in SV

A, B
Regulation of Heart Rate

- Positive chronotropetic factors increase heart rate
- Negative chronotropetic factors decrease heart rate
Regulation of Heart Rate: Autonomic Nervous System

- 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 = $3L/min/m^2$ (surface area in $m^2$)).

- $CO$ is proportional to tissue $O_2$ use.
- $CO$ is proportional to $1/TPR$ when AP is constant.
- $CO = (MAP - RAP) / TPR$
Cardiac Contractility

- Best is to measure the CO curve, but this is nearly impossible in humans.
- dP/dt is not an accurate measure because this increases with increasing preload and afterload.
- \(\frac{dP}{dt}/P_{\text{ventricle}}\) is better. \(P_{\text{ventricle}}\) is instantaneous ventricular pressure.
- Excess \(K^{+}\) decreases contractility.
- Excess \(Ca^{++}\) causes spastic contraction, and low \(Ca^{++}\) causes cardiac dilation.

Thank You