CV Lecture - 3

Cardiac cycle
Heart as a pump – cardiac performance
Polygram - analysis of cardiac activity

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The Cardiac Cycle

- definitions:
  the sequence of mechanical and electrical events that repeats with every heartbeat
  OR the period of time from the beginning of one heartbeat to the beginning of the next one
  OR a sequence of filling and pumping
  ▫ the duration of the cardiac cycle is the reciprocal of heart rate:
    \[
    \text{Duration (s/beat)} = \frac{60 \text{ (s/min)}}{\text{Heart rate (beats/min)}}
    \]

- length: frequency – duration relation

- 2 phases:
  ▫ systole (contraction)
  ▫ diastole (relaxation)
    ▫ atrial
    ▫ ventricular

Obs: normally, A and V do not contract and relax at the same time
The Cardiac Cycle

Note the valves (blue) which control the one way direction of blood flow; also, the tendons prevent the AV valves from turning inside-out.

As the animation starts, the atria fill, then contract (atrial systole), pumping blood via the AV valves into the ventricles. Then the ventricles contract (ventricular systole), causing the AV valves to shut and the semicircular valves to open, allowing blood out of the heart.

This is followed by relaxation (diastole) of the ventricles, and the semilunar valves shut. The cycles then repeats itself...
The Cardiac Cycle

The closing and opening of the cardiac valves define four phases of the cardiac cycle.

1. AS: pumping the blood into V during the last part of the VD
2. VS: isovolumic contraction
3. VS: rapid ventricular ejection
4. VS: reduced ventricular ejection
5. VD: isovolumic ventricular relaxation
6. VD: rapid ventricular filling
7. VD: slow/reduced ventricular filling (diastasis)
8. AD: during all the VS and part of the VD
<table>
<thead>
<tr>
<th>Valvular Events</th>
<th>Cardiac Chamber Events</th>
<th>Phase of the cardiac cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening of AV valves (tricuspid and mitral)</td>
<td>Rapid ventricular filling</td>
<td>1 Diastole</td>
</tr>
<tr>
<td></td>
<td>Decreased ventricular filling; diastasis</td>
<td>1 Diastole</td>
</tr>
<tr>
<td></td>
<td>Atrial contr. (additional ventricular filling)</td>
<td>1 Diastole</td>
</tr>
<tr>
<td>Closing of AV valves (tricuspid and mitral)</td>
<td>Isovolumetric ventricular contraction (with all valves closed)</td>
<td>2 Systole</td>
</tr>
<tr>
<td>Opening of semilunar valves (pulmonary and aortic)</td>
<td>Rapid ventricular ejection (fast muscle shortening)</td>
<td>3 Systole</td>
</tr>
<tr>
<td></td>
<td>Decreased ventricular ejection (slower muscle shortening)</td>
<td>3 Systole</td>
</tr>
<tr>
<td>Closing of semilunar valves (pulmonary and aortic)</td>
<td>Isovolumetric ventricular relaxation (with all valves closed)</td>
<td>4 Diastole</td>
</tr>
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<td></td>
</tr>
</tbody>
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The Cardiac Cycle

**Isovolumic ventricular relaxation**—as ventricles relax, pressure in ventricles drops, blood flows back into cups of semilunar valves and snaps them closed.

**Late diastole**—both sets of chambers relaxed. Passive ventricular filling.

**Atrial systole**—atrial contraction forces a small amount of additional blood into ventricles.

**Ventricular ejection**—as ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.

**Isovolumic ventricular contraction**—first phase of ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.

**EDV** = end-diastolic volume. The maximum amount of blood in ventricles occurs at the end of ventricular relaxation. EDV $\approx 135$ mL.
A RIGHT HEART

Pressures (mm Hg) recorded simultaneously from:

- Tricuspid valve closes
- Pulmonary valve closes
- Pulmonary valve opens
- Tricuspid valve opens

B LEFT HEART

- Aortic valve closes
- Mitral valve opens

Ventricular filling
Isovolumetric contraction
Ventricular ejection
Isovolumetric relaxation
Ventricular filling

Atrial systole
1 (late) 2 3 4 1 (early)

Ventricular volume (mL)

Atrial Systole

- 0.1 sec
- changes in intra-atrial pressure:
  - 0 → 4 - 6 mm Hg - **right** atrium
  - 0 → 7 - 8 mm Hg – **left** atrium
- functional significance:
  - contributes to, but is not essential for, ventricular filling
  - A = primer pump for the ventricle
  - venous pulse: *a wave*
- causes the 4\textsuperscript{th} heart sound
Polygram: events of the cardiac cycle for left ventricular function

- Isovolumic contraction
- Ejection
- Isovolumic relaxation
- Rapid inflow
- Diastasis
- Atrial systole

- Aortic pressure
- Atrial pressure
- Ventricular pressure
- Ventricular volume
- Electrocardiogram
- Phonocardiogram

Graph showing:
- Volume (ml) vs Pressure (mmHg)
- Various cardiac events and pressures

Key:
- Ao : Aorta
- Mo : Mitral valve opening
- MC : Mitral valve closure
- AoC : Aortic valve closure

Events:
- S1, S2, S3, S4 : Heart sounds
- a, c, v : Pressure waves
Atrial Diastole

- 0.7 sec
- changes in diastolic intra-atrial pressure:
  - physiological changes: c and v waves
  - pathological changes (valves pathology)
Polygram: events of the cardiac cycle for left ventricular function
Ventriculare systole

- atrio-ventricular delay in impulse conduction
- 0,27 s;
- phases:
  1. isovolumic contraction – 0,05 s
  2. rapid ejection - 0,09 s; 70% ejection
  3. reduced/slow ejection - 0,13 s; 30% ejection
Isovolumic Contraction
Ventricular ejection

When $P_{LV} > 80 \text{ mmHg}$

$P_{RV} > 8 \text{ mmHg}$

Rapid (1/3; 70%) ejection

Slow (2/3; 30%) ejection
Polygram: events of the cardiac cycle for left ventricular function
Ventricular diastole

- 0,53 s
- phases:
  - protodiastole – 0,04 s
  - isovolumic relaxation - 0,08 s
  - rapid filling – 0,11 s
  - slow filling – 0,19 s
  - atrial systole – 0,11 s
Isovolumic Relaxation

Ventricular changes:
- Wall tension increasing
- Apex-to-base shortening
- Circumferential elongation
Ventricular filling
Polygram: events of the cardiac cycle for left ventricular function
EDV = End-diastolic volume
ESV = End-systolic volume

Aortic valve closes; ESV
Aortic valve opens
Mitral valve opens
Mitral valve closes; EDV

Isovolumic relaxation
Isometric contraction

Ventricular ejection
Ventricular diastole + filling

A→B: Passive filling and atrial contraction
B→C: Isovolumic contraction
C→D: Ejection of blood into aorta
D→A: Isovolumic relaxation
The Cardiac Cycle

![Diagram of the Cardiac Cycle]

**Phase of cardiac cycle:**

<table>
<thead>
<tr>
<th></th>
<th>1 = VFP (ventricular filling)</th>
<th>2 = ICP (ventricular volume unchanged)</th>
<th>3 = VEP (ventricular ejection)</th>
<th>4 = IRP (ventricular volume unchanged)</th>
</tr>
</thead>
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<tr>
<td>AV valves</td>
<td>Open</td>
<td>Closed</td>
<td>Open</td>
<td>Closed</td>
</tr>
<tr>
<td>Semilunar valves</td>
<td>Closed</td>
<td>Open</td>
<td>Closed</td>
<td>Open</td>
</tr>
</tbody>
</table>
C  TIMING OF EVENTS IN CARDIAC CYCLE

D  RIGHT VENTRICULAR PRESSURE AND FLOW

E  LEFT VENTRICULAR PRESSURE AND FLOW

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The Heart as a Pump – Cardiac Performance
Chemical Energy Required for Cardiac Contraction

derives mainly from *oxidative metabolism* of *fatty acids* and, to a lesser extent, of lactate and glucose
- is measured by the *rate of oxygen consumption* in the heart
- is used to provide the work of contraction.

**Efficiency of Cardiac Contraction**
- most of the expended chemical energy is converted into *heat* (75-80%)
- a much smaller portion is converted into *work output* (20-25%).

*Efficiency of cardiac contraction* = \( \frac{WO}{\text{total chemical energy expenditure}} \)

Maximum efficiency of the normal heart ~ 20-25 %.
In heart failure, it decreases to as low as 5 -10 %.
Cardiac Work Output

• **Stroke work output** of the heart - amount of energy converted to work/heartbeat.

• **Minute work output** - total amount of energy converted to work/1 minute (stroke work output × HR)

• **Work output (WO) of the heart** is used:

  1) to move the blood from the low-pressure veins to the high-pressure arteries - *volume-pressure work or external work* (WO$_{RV} \sim 1/6$ WO$_{LV}$, given the different systolic press. in the 2 pumps).

  2) a minor proportion of energy is used to accelerate the blood to its velocity of ejection through the aortic and pulmonary valves – *kinetic energy of blood flow*: $\sim$mass of blood ejected $\times v_{\text{ejection}}^2$.

    normally 1% of WO, up to 50% in Aortic Stenosis
Volume-pressure curves for the LV. "Volume-Pressure Diagram" during the cardiac cycle.

- **diastolic pressure curve:**
  * shows gradual filling of LV up to the end-diastolic pressure (EDP)
  * pressure greatly rises after 150 ml ventricular filling …
    (no more stretch, pericardial limit)

- **systolic pressure curve:**
  * shows systolic pressure during LV contraction at each volume of filling;
  * increases even at low ventricular volumes
  * reaches a maximum (250-300 mmHg for the LV, and 60-80 mmHg for the RV) at 150 -170 ml.
  * for volumes > 170 ml, the systolic pressure actually decreases
    (actin and myosin filaments interrelation)
Graphical Analysis of Ventricular Pumping. Relationship between LV volume and intraventricular pressure during diastole and systole.

Red lines show the "volume-pressure diagram", demonstrating changes in intraventricular volume and pressure during the normal cardiac cycle. EW, external work (the area subtended by the volume-pressure diagram).
The 4 phases of the "volume-pressure diagram", during the normal cardiac cycle.

Phase I: *Period of filling*.

- initial ventricular volume ~45 ml (*end-systolic volume*),
  diastolic pressure ~0 mm Hg.

- ventricular volume normally increases with 70 ml, up to ~115 ml (*end-diastolic volume*), and the diastolic pressure rise to about 5 mm Hg.

Phase II: *Period of isovolumic contraction*.

- volume of the ventricle constant (all valves closed) ~115 ml, the pressure inside the ventricle increases to equal the pressure in the aorta, at ~80 mm Hg.

Phase III: *Period of ejection*.

- systolic pressure rises higher during contraction of the ventricle (up to ~120 mmHg), while the volume of the ventricle decreases during ejection.

Phase IV: *Period of isovolumic relaxation*.

- aortic valve closes, no change in volume (~45 ml ESV), decrease of ventricular pressure back to diastolic pressure (~0 mm Hg).
Note the change in External Work.
Preload and Afterload

**Preload**
- the degree of tension on the muscle when it begins to contract.
- is usually considered to be the end-diastolic pressure when the ventricle has become filled.
- depends on the incoming blood in the RA = *venous return*

**Afterload**
- the load against which the muscle exerts its contractile force.
- is the systolic pressure in the artery leading from the ventricle, (relation with the vascular resistance).
Cardiac activity regulation

A. Intrinsic/local regulation of the heart
B. Systemic regulation (nervous, humoral, integrated)

CV Integration

Heart activity

Vascular tone
A. Intrinsic/local regulation

Heart activity regulation
1. Ca\(^{2+}\) role
   - Ca\(^{2+}\) homeostasis
   - Ca\(^{2+}\) regulation factors
2. Frank-Starling low of the heart

Microcirculation regulation
1. Myogenic autoregulation
2. Metabolic factors
3. Chemical messengers

B. Systemic regulation

Short-term regulation
- Nervous reflex regulation

Long-term regulation
- Humoral regulation

Integrated regulation
A. Intrinsic/Local Regulation

Heart activity regulation

1. Ca\(^{2+}\) role
   - Ca\(^{2+}\) homeostasis
     - excitation-contraction coupling, intracellular calcium
     - membrane transport systems for Ca\(^{2+}\)
Ca$^{2+}$ regulation factors

- **Sympathetic stimulation** via β1 receptors $\rightarrow$ cAMP $\rightarrow$ intracellular protein phosphorilation: L-type Ca channels, phospholamban (a protein that inhibit Ca pump on the SR) $\rightarrow$ $\uparrow$ [Ca$_{2+}$]$_i$

- "garden-hose" effect - the coronary artery pressure itself influences ventricular function by distending the heart from within its walls, and so invokes Starling’s law of the heart (Arnold Katz, Physiology of the Heart). Changing perfusion pressure also modify calcium release during excitation-contraction coupling (Kitakaze & Marban, 1989).

- $\uparrow$ heart rate – positive (Bowditch) staircase:
  
  cumulative increase in [Ca$_{2+}$]$_i$ $\rightarrow$ $\uparrow$ Ca stores $\rightarrow$ $\uparrow$ contractility

- **cardiac glycosides** (digitalis):
  
  Na/K pump, Na/Ca exchanger $\rightarrow$ $\uparrow$ [Ca$_{2+}$]$_i$

- **pH effect**: intracell H$^+$ competes with Ca$^{2+}$ for binding on troponine complex
Epinephrine from adrenal medulla
Norepinephrine from sympathetic neurons

bind to

$b_1$ receptors on myocardial contractile cell

that activate

cAMP second messenger system

resulting in phosphorylation of

Voltage-gated $Ca^{2+}$ channels

Open time increases

$\downarrow Ca^{2+}$ entry from ECF

$Ca^{2+}$ stores in SR

$Ca^{2+}$ released through $Ca^{2+}$-induced $Ca^{2+}$ release

More forceful contraction

Phospholamban

$Ca^{2+}$ removed from cytosol faster

Time of $Ca$-troponin binding shorter

Shorter duration of contraction
2. Frank-Starling low of the heart:

*Within physiological limits, the heart pumps all the blood that returns to it.*

- **Preload**: the wall tension that corresponds to ED pressure
  - venous return - skeletal mm pump & respiratory pump
  - sympathetic constriction of veins
  - EDV - length of sarcomere at beginning of contraction;

- **length-tension relationship** in cardiac muscle
  - optimal sarcomere lengths – max. no. of A-M cross-bridges,
  - troponin affinity for Ca
  - increase Ca uptake from ECF and release from SR

- **Afterload** – arterial blood pressure
- **Inotropic state** of the heart
- **Stretch of the right atrial wall** directly increases the heart rate by 10-20 % → increase the amount of blood pumped each minute
Frank-Starling law of the heart

More blood in the ventricle at the beginning of contraction (EDV), the greater the stroke volume.

Stroke volume is proportional to force.
The tension generated (force) is directly proportional to the initial length of the muscle fiber.

Length-Tension Relationship

Factors that influence this relationship:
- Intracellular Ca^{2+}
- Changes in force due to fiber length
- Changes in force created by catecholamines discharges

The ability of stretched muscle, up to an optimal length, to contract with increased work output is characteristic of all striated muscle.
Pressure-Volume curve for the left ventricle during one cardiac cycle.

Stroke volume is determined by the preload, afterload (arterial pressure) and the intrinsic inotropic state of the myocardium.
Frank-Starling law of the heart

End-systolic pressure-volume relation

↑ EDV
Normal EDV
↓ EDV
B. Systemic Regulation

- Short-term regulation
  - Nervous reflex regulation: fast response
- Long-term regulation
  - Humoral regulation

- Integrated regulation
Nervous reflex regulation: Autonomic Nervous Regulation

S and PS branches of the autonomic nervous system influence HR and AV node conduction through antagonistic control.

PS: \( \leq 70 \) beats/min - [SAN: intrinsic rate of 70-100/min] - S: \( >100/\text{min} \)
80-200/min

(Ach, muscarinic rec) (NE, \( \beta_1 \) rec)

**PS tone** - decrease HR and AV conduction; vagal ‘escape’
- strong vagal stimulation can decrease the strength of heart muscle contraction by 20 -30 %
- nitric oxide (NO) \( \rightarrow \) vasodilatation

**S tone** - increase HR, AV conduction and contractility (\( \beta_1 \) Rec)
- normally S discharge continuously at a slow rate \( \rightarrow \) 30% CO ↑
- determine vasoconstriction by \( \alpha_1 \) Rec (NE)
- vasodilatation by \( \beta_2 \) Rec (E in heart, skeletal mm \( \rightarrow \) fight or flight response)
Cardiac output can be increased more than 100% by sympathetic stimulation, and can be decreased to almost zero by vagal (parasympathetic) stimulation.
Effect on the cardiac output of different degrees of sympathetic or parasympathetic stimulation.

The picture shows relation between RA pressure at the input of the right heart and CO from the LV into the aorta. CO changes caused by nerve stimulation result both from changes in heart rate and from changes in contractile strength of the heart.
Effect of NE on contractility of the heart
Factors that affect cardiac output

Cardiac output

is a function of

Heart rate

determined by

Rate of depolarization in autorhythmic cells

is slowed by

Parasympathetic innervation

is made faster by

Sympathetic innervation

Epinephrine from adrenal medulla

Stroke volume

determined by

Force of contraction in ventricular myocardium

is influenced by

Contractility

Length-tension relationship of muscle fibers

which varies with

Venous return

aided by

Skeletal muscle pump

Respiratory pump
Polygram - Analysis of Cardiac activity

Electrical activity – measured by electrocardiography

Mechanical activity – evaluated by:

1. Atrial pressure curve and venous pressure: recorded at jugular vein level (jugulogram)
2. Aortic pressure curve: recorded at carotid artery level (carotidogram)
3. Phonocardiography: record of the heart sounds
4. Ventricular volume: evaluated by apexocardiogram
Mechanical, electrical, acoustic, and echocardiographic events in the cardiac cycle.

(1) the cardiac cycle begins with atrial contraction;

(2) phase 1 of the cardiac cycle has three subparts: rapid ventricular filling, decreased ventricular filling, and atrial systole;

(3) phase 3 has two subparts: rapid and decreased ventricular ejection.
Pressure Waves in Veins

- **systemic veins** have pressure waves - **venous pulse**: 
  1. retrograde action of the heartbeat during the cardiac cycle,
  2. the respiratory cycle
  3. the contraction of skeletal muscles.

- **Jugular vein**, has a complex pulse wave synchronized to the cardiac cycle:
  1. 3 peaks, labeled $a$, $c$, and $v$
  2. 3 minima, labeled $av$, $x$, and $y$. 

![Jugular Venous Pressure Changes](image)
Pressure transients in the jugular vein pulse reflect events in the cardiac cycle:

- **a peak** - caused by the contraction of the right atrium.
- **av minimum** is due to relaxation of the right atrium and closure of the tricuspid valve.
- **c peak** reflects the pressure rise in the right ventricle early during systole and the resultant bulging of the tricuspid valve-which has just closed-into the right atrium.
- **x minimum** occurs as the ventricle contracts and shortens during the ejection phase, later in systole. The shortening heart-with tricuspid valve still closed-pulls on and therefore elongates the veins, lowering their pressure.
- **v peak** is related to filling of the right atrium against a closed tricuspid valve, which causes right atrial pressure to slowly rise. As the tricuspid valve opens, the v peak begins to wane.
- **y minimum** reflects a fall in right atrial pressure during rapid ventricular filling, as blood leaves the right atrium through an open tricuspid valve and enters the right ventricle. The increase in venous pressure after the y minimum occurs as venous return continues in the face of reduced ventricular filling.
Effect of the Respiratory Cycle

- During inspiration, the diaphragm descends, causing intrathoracic pressure (and therefore the pressure inside the thoracic vessels) to decrease and intra-abdominal pressure to increase → the venous return from the head and upper extremities transiently increases, as low-pressure vessels literally suck blood into the thoracic cavity.
- Simultaneously, the venous flow decreases from the lower extremities because of the relatively high pressure of the abdominal veins during inspiration.
- Therefore, during inspiration, pressure in the jugular vein falls while pressure in the femoral vein rises.
Polygram - Analysis of Cardiac activity

**Electrical activity** – measured by *electrocardiography*

**Mechanical activity** – evaluated by:

1. Atrial pressure curve and venous pressure: recorded at jugular vein level (jugulogram)
2. Aortic pressure curve: recorded at carotid artery level (carotidogram)
3. Phonocardiography: record of the heart sounds
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Cardiac Cycle causes flow waves in aorta and peripheral vessels

With the closing & opening of pulmonary and aortic valves, **blood flow and blood velocity** across these valves oscillate from near zero, when the valves are closed, to high values, when the valves are open. Blood flow in the aortic arch actually oscillates between slightly negative and highly positive values.

**Pressure** in the aortic arch typically oscillates between ~ 80 - 120 mm Hg. Phasic changes in pressure and flow also occur in the peripheral arteries. Arterial pressure is usually measured in a large artery, such as the brachial artery → the measured **systolic** and **diastolic** arterial pressures, as well as the pulse pressure and mean arterial pressure, closely approximate the corresponding aortic pressures.

Flow (A) and pressure (B) profiles in the aorta and smaller vessels.
Comparison of the dynamics of the left and right ventricles.
Polygram - Analysis of Cardiac activity

**Electrical activity** – measured by electrocardiography

**Mechanical activity** – evaluated by:

1. Atrial pressure curve and venous pressure: recorded at jugular vein level (jugulogram)
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4. Ventricular volume: evaluated by apexocardiogram
Heart sounds and phonocardiography

Heart sounds are relatively brief, discrete auditory vibrations of varying intensity (loudness), frequency (pitch), and quality (timbre).

The first heart sound identifies the onset of ventricular systole, and the second heart sound identifies the onset of diastole.

These two auscultatory events establish a framework within which other heart sounds and murmurs can be placed and timed.

Listening to the sounds of the body with the aid of a stethoscope is called auscultation. The stethoscope can detect leaks in the valves that permit jets of blood to flow backward across the valvular orifice (i.e., regurgitation) as well as stenotic lesions that narrow the valve opening, forcing the blood to pass through a narrower space (i.e., stenosis). During certain parts of the cardiac cycle, blood passing through either regurgitant or stenotic lesions makes characteristic sounds that are called murmurs.

Phonocardiogram: the recording of the auscultatory cardiac activity, using a transducer placed on the thorax.

The movement of the valve leaflets can be detected by echocardiography.
The **primary aortic area**: 2\(^{nd}\) right intercostal space, adjacent to the sternum.

The **secondary aortic area**: 3\(^{rd}\) left intercostal space, adjacent to the sternum (known as Erb area).

The **pulmonary area**: 2\(^{nd}\) left intercostal space

The **tricuspid area**: 4\(^{th}\) & 5\(^{th}\) intercostal spaces, adjacent to the left sternal border.

The **mitral area** at the cardiac apex: 5\(^{th}\) left intercostal space, on the medioclavicular line.
The first heart sound (S1) – systolic sound

- the “lub”
- appears at 0.02 – 0.04 sec after the beginning of the QRS complex
- vibrations are low in pitch and relatively long-lasting
  - lasts ~ 0.12-0.15 sec;
- frequency ~ 30-100 Hz;
- produced, in this order, by: closing of the mitral valve, closing of the tricuspid valve, opening of the pulmonic valve, opening of the aortic valve.
The second heart sound (S2) – diastolic sound

- the “dub”
- appears in the terminal period of the T wave
- lasts 0.08 – 0.12s
- produced, in this order, by: closing of the aortic valve, closing of the pulmonic valve, opening of the tricuspid valve, opening of the mitral valve.
- heard like a rapid snap because these valves close rapidly, and the surroundings vibrate for a short period
- physiologic splitting that varies with respiration (wider splitting with inspiration)

Split S2

Normal or physiologic

<table>
<thead>
<tr>
<th>Inspiration</th>
<th>Expiration</th>
</tr>
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<tbody>
<tr>
<td>S₁ S₂ A P A P</td>
<td>S₁ S₂</td>
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</table>
Split S2

- Audible respiratory splitting means > 30 msec difference in the timing of the aortic ($A_2$) and pulmonic ($P_2$) components of the second heart sound.
- Splitting of $S_2$ is best heard over the 2nd left intercostal space.
- The normal $P_2$ is often softer than $A_2$ and rarely audible at apex.
- Inspiration accentuates the splitting of $S_2$.

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**Split S2**

Normal or physiologic
The third heart sound (S3)

- occurs in early diastole (at the beginning of the middle third of diastole) when rapid filling of the ventricles results in recoil of ventricular walls that have a limited distensibility
- lasts 0.02-0.04 sec
- protodiastolic sound or gallop

A gallop rhythm is a grouping of three heart sounds that together sound like hoofs of a galloping horse. The addition of an S3 to the physiological S1 and S2 creates a three-sound sequence, S1-S2-S3, that is termed a protodiastolic gallop or ventricular gallop.

- it is normal in children and individuals with a thin thoracic wall
- occasionally heard as a weak, rumbling sound
The fourth heart sound (S4) – presystolic sound:

- appears at 0.04 s after the P wave (late diastolic)
- lasts 0.04-0.10 s
- caused by the blood flow that hits the ventricular wall during the atrial systole.
- physiological only in small children, *if heard in other conditions it is a sign of reduced ventricular compliance.*
- addition of an S4 produces another three-sound sequence, S4-S1-S2, which is a *presystolic gallop rhythm or atrial gallop*
- during tachycardia S4-S1 can fuse, producing a *summation gallop*
EC=ejection click: most common early systolic sound; Results from abrupt halting of semilunar valves

OS=opening snap: high-frequency early diastolic sound (occurs 50-100 msec after A2) associated with mitral stenosis (stiffening of the mitral valve); sound due to *abrupt deceleration of mitral leaflets sound with associated murmur.*
The duration of S1, S2 is slightly more than 0.10 sec.

S1 ~ 0.14 sec
S2 ~ 0.11 sec.

(the semilunar valves are more taut than the A-V valves, so that they vibrate for a shorter time than do the A-V valves).

The audible range of frequency (pitch) in the first and second heart sounds:

~ 40 cycles/sec → up above 500 cycles/sec.
Polygram - Analysis of Cardiac activity

**Electrical activity** – measured by **electrocardiography**

**Mechanical activity** – evaluated by:

1. **Phonocardiography**: record of the heart sounds
2. **Atrial pressure curve**: recorded at jugular vein level (jugulogram)
3. **Ventricular volume**: evaluated by apexocardiogram
4. **Aortic pressure curve**: recorded at carotid artery level (carotidogram)
Polygram: events of the cardiac cycle for left ventricular function

- Isovolumic contraction
- Ejection
-Isovolumic relaxation
- Rapid inflow
- Diastasis
- Atrial systole

Atria pressure
Ventricular pressure
Aortic pressure
Ventricular volume
Electrocardiogram
Phonocardiogram

Volume (ml) vs. Pressure (mmHg)

- **Isovolumic contraction**
- **Ejection**
- **Isovolumic relaxation**
- **Rapid inflow**
- **Diastasis**
- **Atrial systole**

Polygram: events of the cardiac cycle for left ventricular function
ATRIAL SYSTOLE (The end of ventricular diastole)

Heart:
During atrial systole the atrium contracts and tops off the volume in the ventricle with only a small amount of blood. Atrial contraction is complete before the ventricle begins to contract.

Atrial pressure:
The "a" wave occurs when the atrium contracts, increasing atrial pressure. Blood arriving at the heart cannot enter the atrium so it flows back up the jugular vein, causing the first discernible wave in the jugular venous pulse. Atrial pressure drops when the atria stop contracting.

ECG:
An impulse arising from the SA node results in depolarization and contraction of the atria. The P wave is due to this atrial depolarization. The PR segment is electrically quiet as the depolarization proceeds to the AV node. This brief pause before contraction allows the ventricles to fill completely with blood.

Heart sounds:
A fourth heart sound (S4) is abnormal and is associated with the end of atrial emptying after atrial contraction. It occurs with hypertrophic congestive heart failure, massive pulmonary embolism or tricuspid incompetence.
ISOVOLUMETRIC CONTRACTION
The beginning of systole

Heart:
The atrioventricular (AV) valves close at the beginning of this phase. *Electrically,* ventricular systole is defined as the interval between the QRS complex and the end of the T wave (the Q-T interval). *Mechanically,* ventricular systole is defined as the interval between the closing of the AV valves and the opening of the semilunar valves (aortic and pulmonary valves).

Pressures & Volume:
The AV valves close when the pressure in the ventricles (red) exceeds the pressure in the atria (yellow). As the ventricles contract isovolumetrically - - their volume does not change white -- the pressure inside increases, approaching the pressure in the aorta and pulmonary arteries (green).

ECG:
The electrical impulse propagates from the AV node through the His bundle and Purkinje system to allow the ventricles to contract from the apex of the heart towards the base. The QRS complex is due to ventricular depolarization, and it marks the beginning of ventricular systole. It is so large that it masks the underlying atrial repolarization signal.

Heart sounds:
The first heart sound (S1, "lub") is due to the closing AV valves and associated blood turbulence.
RAPID EJECTION

Heart:
The semilunar (aortic and pulmonary) valves open at the beginning of this phase.

Pressures & Volume:
While the ventricles continue contracting, the pressure in the ventricles (red) exceeds the pressure in the aorta and pulmonary arteries (green); the semilunar valves open, blood exits the ventricles, and the volume in the ventricles decreases rapidly (white). As more blood enters the arteries, pressure there builds until the flow of blood reaches a peak. The "c" wave of atrial pressure is not normally discernible in the jugular venous pulse. Right ventricular contraction pushes the tricuspid valve into the atrium and increases atrial pressure, creating a small wave into the jugular vein. It is normally simultaneous with the carotid pulse.

ECG:

Heart sounds:
REDUCED EJECTION

The end of systole

Heart:
At the end of this phase the semilunar (aortic and pulmonary) valves close.

Pressures & Volume:
After the peak in ventricular and arterial pressures (red and green), blood flow out of the ventricles decreases and ventricular volume decreases more slowly (white). When the pressure in the ventricles falls below the pressure in the arteries, blood in the arteries begins to flow back toward the ventricles and causes the semilunar valves to close. This marks the end of ventricular systole mechanically.

ECG:
The T wave is due to ventricular repolarization. The end of the T wave marks the end of ventricular systole electrically.

Heart sounds:
ISOVOLUMETRIC RELAXATION

The beginning of diastole

Heart:
At the beginning of this phase the AV valves are closed.

Pressures & Volume:
Throughout this and the previous two phases, the atrium in diastole has been filling with blood on top of the closed AV valve, causing atrial pressure to rise gradually (yellow). The "v" wave is due to the back flow of blood after it hits the closed AV valve. It is the second discernible wave of the jugular venous pulse. The pressure in the ventricles (red) continues to drop. Ventricular volume (white) is at a minimum and is ready to be filled again with blood.

ECG:

Heart sounds:
The second heart sound (S2, "dup") occurs when the semilunar (aortic and pulmonary) valves close. S2 is normally split because the aortic valve closes slightly earlier than the pulmonary valve.
RAPID VENTRICULAR FILLING

Heart:
Once the AV valves open, blood that has accumulated in the atria flows rapidly into the ventricles.

Pressures & Volume:
Ventricular volume increases rapidly as blood flows from the atria into the ventricles.

ECG:
Heart sounds:
A third heart sound (S3) is usually abnormal and is due to rapid passive ventricular filling.
It occurs in dilated congestive heart failure, myocardial infarction, or mitral incompetence.
REDUCED VENTRICULAR FILLING (DIASTASIS)

Heart:

Pressures & Volume:
Ventricular volume (white) increases more slowly now. The ventricles continue to fill with blood until they are nearly full.

ECG:

Heart sounds: