Cardiac Cycle:
Electrical and Mechanical Events
CARDIAC CYCLE

• The cardiac events that occur from the beginning of one heart beat to the beginning of the next.

• Events: Electrical
  Mechanical

• Electrical and mechanical events occur in a co-ordinated manner to generate effective contractions

• Duration – 0.8 s
ELECTRICAL EVENTS

- The rhythmical activity of the heart is controlled by electrical impulses
- A specialised conduction system generates and propagates impulses
- Cardiac muscle as a functional syncitium enables rapid and uniform passage of the impulses

Ensures: all parts of the ventricle contract simultaneously.

atrial contraction → 1/6th sec before ventricular contraction
CONDUCTION SYSTEM
SAN

ANT I/N TRACT OF BACHMAN

MIDDLE I/N TRACT OF WENCKEBACH

POST I/N TRACT OF THOREL

AVN

BUNDLE OF HIS

RBB

LBB

LAF

LPF

PURKINJE SYSTEM

Myocardium
SINOATRIAL NODE

- Junction of SVC with right atrium
- Specialised neurocardiac tissue; almost no contractile muscle filament
- Normal pacemaker
- Connects directly with the surrounding atrial muscle fibres so that action potential generated spreads immediately into the atrium.
- Properties of **Self-excitation** and **Rhythmicity**
What causes the self excitation?

- Membrane in natural state permeable to Na and Ca ions
- RMP in SA nodal fibre is less negative: -60 to -70 mv
- This voltage closes fast Na channels but slow Na - Ca channels are open
- The ions flow inward → membrane permeability further increased → Threshold reached → Action potential
- Half way through the action potential, slow Na-Ca channels begin to close. K channels open for a prolonged period.
- Inside of the membrane becomes more negative = HYPERPOLARIZATION
- This persists for 0.5s after the Action potential ends.
- The K conductance weans off → RMP reached → Na–Ca opening overbalances K closure → threshold for Action potential → Action potential.
SINO ATRIAL NODE

Self–excitation & AP → Recovery of AP

↑

Resting Memb Potential ← Hyperpolarisation
Conduction through the other parts

- From SAN impulses through the 3 bands (1m/s) and atria (0.3m/s)
- Delay in the AVN – of 0.09 s. Due to reduced gap junction number. Allows time for the ventricles to fill completely before they contract
- Rapid transmission through Purkinje fibres (4m/s) due to many gap junctions
- Transmission in ventricular muscle- 0.3 – 0.5m/s
Phase 0: rapid depolarization – opening of fast Na channels
Phase 1: rapid repolarisation – closure of Na channels
Phase 2: Plateau – slow prolonged opening of Ca channels
Phase 3: final repolarisation – closure of Ca channels
Phase 4: RMP (-85 to -90 mv) – opening of K channels
REFRACTORY PERIOD:

• Refractory to re-stimulation during Action potential

• Absolute refractory period = 0.25 – 0.3 s. Phase 0 to 2 and half of phase 3

• Relative refractory period = phase 4

• Refractory period of the atria is shorter than that of ventricle
Variation in length of action potential and associated phenomena with cardiac rate.¹

<table>
<thead>
<tr>
<th>Duration, each cardiac cycle</th>
<th>Heart Rate 75/min</th>
<th>Heart Rate 200/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of systole</td>
<td>0.27</td>
<td>0.16</td>
</tr>
<tr>
<td>Duration of action potential</td>
<td>0.25</td>
<td>0.15</td>
</tr>
<tr>
<td>Duration of absolute refractory period</td>
<td>0.20</td>
<td>0.13</td>
</tr>
<tr>
<td>Duration of relative refractory period</td>
<td>0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>Duration of diastole</td>
<td>0.53</td>
<td>0.14</td>
</tr>
</tbody>
</table>

¹ All values are in seconds.
Effect of autonomic nervous system on conduction
Effect of parasympathetic system

- Vagal stimulation causes release of Ach
- Two effects:
  - ↓ rate of rhythm of SAN – RMP becomes more negative
  - ↓ excitability of A-V junctional fibres
- Slowing of heart rate, complete block in transmission may occur
- Strong vagal stimulus → ventricles stop beating for 5-20s → purkinje fibres take over → ventricles contract at 15-40 beats per minute → VENTRICULAR ESCAPE
Effect of sympathetic system

- Sympathetic stimulation → Noradrenaline released

- Three effects:
  - ↑ rate of SAN discharge – RMP more positive
  - ↑ rate of conduction and overall excitability
  - ↑ the force of contraction – increases Ca permeability

- Heart rate may increase 3 times and strength of contraction may increase 2 times the normal
Mechanism by which AP causes myofibrils to contract

- AP over cardiac muscle membrane
  - Reaches interior through T Tubules
  - T tubule AP acts on longitudinal sarcoplasmic reticulum
  - Release of Ca ions into sarcoplasm
  - Ca ions catalyze sliding of actin-myosin filaments
How is this different from the skeletal muscle contraction?

- **T tubules** also pump Ca ions into the sarcoplasm

- Ca from sarcoplasmic reticulum + T tubules = effective contraction

- Sarcoplasmic reticulum in cardiac muscle – less well developed

- T tubule diameter and volume in cardiac muscle >> skeletal muscle

- Since T tubules are in contact with ECF in the interstitium, the ECF Ca concentration determines the strength of the contraction
MECHANICAL EVENTS

The heart contracts and relaxes alternately during every heart beat and this occurs in concordance with the electrical events. The mechanical events can be studied in various phases such as:
5. Isovolumic ventricular relaxation—As ventricles relax, pressure in ventricles falls, blood flows back into cups of semilunar valves and snaps them closed.

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

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

3. Isovolumic ventricular contraction—First phase of ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.
ATRIAL SYSTOLE

Atrial Depolarization

Atrial contraction

Atrial pressures rise

Blood flows across AV valves

- Ventricular filling: 80% - direct flow from SVC
  20% - atrial contraction.

ATRIA = PRIMER PUMPS.
JVP – ‘a’ wave

ECG – P wave precedes the atrial systole. PR segment – depolarization proceeds to the AVN. The brief pause allows complete ventricular filling

Heart sounds - $S_4$ – pathological. Vibration of the ventricular wall during atrial contraction. Heard in ‘stiff’ ventricle like in hypertrophy and in elderly. Also heard in massive pulmonary embolism, cor pulmonale, TR
ISOVOLUMETRIC CONTRACTION

Increase in ventricular pressure > atrial pressure →
AV valves close

After 0.02s, semilunar valves open

Period between AV valve closure and semilunar
valve opening → contraction occurs without
emptying

Tension develops without change in muscle length
- **JVP** – ‘c’ wave → due to the bulging of the Tricuspid valve into RA secondary to increased pressure in the ventricle.
  - ‘x’ descent
- **ECG** – Interval between QRS complex and T wave (QT interval)
- **Heart Sounds** – $S_1$: closure of the AV valves. Normally split as mitral valve closure precedes tricuspid valve closure.
EJECTION

- When LV pres > 80 mm Hg
  RV pres > 8 mm Hg,
  The semilunar valves open.

- Rapid Ejection – 70% emptying in first 1/3

- Slow Ejection – 30% in last 2/3

- The pressure in the ventricle keeps decreasing until it becomes lower than that of the great vessels
- **JVP** – no waves
- **ECG** – T wave
- **Heart sounds** – none
- **Aortic pressure** - Rapid rise in the pressure = 120 mm Hg
  
  Even at the end of systole pressure in the aorta is maintained at 90 mm Hg because of the elastic recoil
ISOVOULUMETRIC RELAXATION

- When ventricle pressure < arterial pressure → backflow of blood → forces semilunar valves to close.

- For 0.03-0.06 s, ventricle relaxes despite no change in its volume

- Meanwhile, atria fill up and atrial pressure gradually rises

- Pressures in ventricle keep falling till it is < atrial pressure
- **JVP** – ‘v’ wave – due to venous return to the atria from SVC and IVC
- **ECG** - no deflections
- **Heart sounds** – S₂: closure of the semilunar valves. Normally split because the aortic valve closes slightly earlier than the pulmonary valve
- **Aortic pressure curve** – INCISURA - when the aortic valve closes. Caused by a short period of backflow before the valve closes followed by sudden cessation of the backflow when the valve closes.
VENTRICULAR FILLING

• Begins with the opening of AV valves
• Rapid filling – first 1/3 of diastole
• Reduced filling (Diastasis) – middle 1/3 of diastole
• Atrial contraction – last 1/3 of diastole
• As the atrial pressures fall, the AV valves close and left ventricular volume is now maximum
  → EDV (120 ml in LV)
JVP – ‘y’ descent

ECG – no deflections

# VARIOUS PRESSURE VALUES

<table>
<thead>
<tr>
<th>CHAMBERS</th>
<th>NORMAL RANGE (mm of Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Atrium</td>
<td>2 – 6</td>
</tr>
<tr>
<td>Right Ventricle</td>
<td>15 – 25</td>
</tr>
<tr>
<td></td>
<td>2 – 8</td>
</tr>
<tr>
<td>Pulmonary Artery</td>
<td>15 – 25</td>
</tr>
<tr>
<td></td>
<td>8 – 15</td>
</tr>
<tr>
<td>Left Atrium</td>
<td>6 – 12</td>
</tr>
<tr>
<td>Left Ventricle</td>
<td>100 – 140</td>
</tr>
<tr>
<td></td>
<td>3 – 12</td>
</tr>
</tbody>
</table>
VENTRICULAR PRESSURE- VOLUME LOOP

a – Ventricular filling
b – isovol contraction
c – ejection
d – isovol relaxation

1 – Mitral valve closes
2 – Aortic valve opens
3 – Aortic valve closes
4 – Mitral valve opens
• The filling phase moves along the end-diastolic pressure-volume relationship (EDPVR)

• The slope of the EDPVR is the reciprocal of Ventricular Compliance

• The maximal pressure that can be developed by the ventricle at any given left ventricular volume → end-systolic pressure-volume relationship (ESPVR), which represents the inotropic state.
SYSTOLIC DYSFUNCTION

- Impaired ventricular contraction

↓ slope of ESPVR i.e. ↑ ESV

Compensatory rise in preload i.e. ↑ EDV

↓ SV

↓ EF

↓ Work

↑ EDP
FORCE- VELOCITY RELATIONSHIP

At any given preload and afterload, a loss of inotropy results in decrease in shortening velocity of the cardiac fibres.
DIASTOLIC DYSFUNCTION

Reduced venous return / compliance / relaxation (lusitropy)

↓ EDV

↓ SV

↓ or = EF

↓ Work

↑ EDP

Effects of left ventricular diastolic failure caused by decreased ventricular compliance (e.g., hypertrophy) on left ventricular pressure-volume loop. Heart rate, inotropy and systemic vascular resistance are unchanged.
MITRAL STENOSIS

Impaired LV filling

↓ EDV

↓ afterload ; ↓ ESV

↓ SV and CO
MITRAL REGURGITATION

- Afterload on LV ↓
- Outflow resistance is ↓
- EDV and EDP ↑
- ↑ SV
- ↓ EF
AORTIC STENOSIS

High outflow resistance; LV emptying impaired

↑ Peak systolic pressure; ↑ afterload

↓ SV

↑ ESV

↑ EDV
AORTIC REGURGITATION

No true isovolumetric relaxation

Blood from aorta to ventricle throughout diastole

↑ EDV

↑ SV (if no failure)

↑ ESV and ↓ SV in failure
SUMMARY

The conduction system and the atrial and ventricular muscle in the normal heart work in an extremely well co-ordinated manner to ensure correct opening and closure of the atrio-ventricular and semilunar valves and movement of the blood through the heart in an appropriate direction with minimal regurgitation.
REFERENCES


