TOPIC 15: CARDIOVASCULAR SYSTEM: CARDIAC CYCLE & CONTROL

(All cd references refer to Interactive Physiology cd, Cardiovascular menu)

I. Mechanical Events in the Cardiac Cycle (Figs 13.18 to 13.21; cd cardiac cycle 5 to 17)
   A. Introduction
      1. Systole: Contraction and emptying of the chambers
      2. Diastole: Relaxation and filling of the chambers
      3. Atria and Ventricles go through separate cycles of systole and diastole
      4. The contraction status (being in systole or diastole) determines heart chamber pressure which determines whether valves are open or closed.
   B. TP interval: Ventricular diastole
      1. Atria and ventricles are in diastole (i.e., relaxed).
      2. Blood flows from veins into atria
      3. Ventricular Pressure < Aortic Pressure = Aortic Valve closed
      4. Atrial pressure > Ventricular Pressure = AV valve open so
      5. Blood flows from atria directly into ventricles.
   C. P wave and PQ interval: Late ventricular diastole
      1. Ventricular Pressure < Aortic Pressure = aortic valve closed
      2. SA node reaches threshold and fires.
      3. Atrial depolarization occurs.
      4. Atria contract = atrial systole
      5. Atrial Pressure > Ventricular Pressure = AV valves open.
   D. QR Interval: End of ventricular diastole
      1. Ventricular pressure < Aortic Pressure = Aortic Valve closed
      2. Atrial Pressure > Ventricular Pressure = AV valves open.
      4. Electrical impulse enters ventricles from the AV node.
      5. Ventricles begin to depolarize.
      6. R peak is end of ventricular diastole and start of ventricular systole.
   E. RS interval: Early ventricular systole
      1. Ventricles begin to contract.
      2. Atrial Pressure > Ventricle Pressure = AV valves close
      3. Atrial contraction and ventricular filling are completed.
      4. Ventricular pressure still not high enough to open aortic valve
      5. No blood entering or leaving ventricles. Blood volume in ventricles called end-diastolic volume (EDV) which = 135 ml/ventricle in a resting adult
      6. Atria repolarize.
   F. ST segment: Ventricular systole
      1. Ventricular Pressure > Atrial Pressure = AV Valve closed
      2. Ventricular Pressure > Aortic Pressure = Aortic Valve open
      3. Blood ejected into aorta from ventricles.
      4. Atria in diastole and filling with blood.
   G. Start of T wave: Late ventricular systole.
      1. Repolarization of the ventricles begins.
H. **T wave peak to end of T wave: Early ventricular diastole.**

1. Peak of T wave = end of systole and start of diastole.
2. Ventricles begin to relax.
3. Ventricular Pressure < Aortic Pressure = aortic valve closes.
4. No more blood can leave ventricles.
5. Ventricular Pressure > Atrial Pressure = AV valves closed = No blood can enter ventricles.
6. This remaining volume of blood is called the **End-Systolic Volume (ESV)**. Typically, about half of the end diastolic volume remains in the ventricles (about 65 ml/ventricle in a resting adult).
7. Atria in diastole and filling with blood.

I. **TP interval: Ventricular diastole (back where we started)**

1. Atria and ventricles are in diastole (i.e., relaxed).
2. Blood flows from veins into atria.
3. Ventricular Pressure < Aortic Pressure = Aortic Valve closed.
4. Atrial Pressure > Ventricular Pressure = AV valve open so blood flows from atria directly into ventricles.

J. **The stroke volume (SV) is equal to EDV-ESV = volume of blood pumped by one ventricle per heart beat.** This averages ~ 70 ml/beat when at rest.

K. Sounds (fig 13.23)

   a) first sound is turbulent rushing of blood as AV valves are closing
   b) second sound is turbulent rushing of blood as aortic and pulmonary valves are closing.

II. Cardiac Output and its Control (cd cardiac output 3 to 8)

A. **Cardiac output**

1. Cardiac Output (CO) is volume of blood pumped by each ventricle/minute (not volume of blood pumped by whole heart)
2. cardiac output (CO) = heart rate (HR) multiplied by stroke volume (SV)
3. **Control of cardiac output is accomplished by controlling heart rate and stroke volume!!!!!!**
4. At rest for average individual
   a) HR = 70 beats/min
   b) SV = 70 ml/beat (see above)
   c) CO = 70 beats/min x 70 ml/beat = 4900 ml/min
   d) Because total blood volume in a person is 5 to 5.5 liters, each ½ of the heart pumps nearly the whole blood volume each minute at rest

B. **Control of Heart Rate** (Fig 13.24)

1. Review: SA node sets baseline heart rate at ~ 70 beats per minute
2. Parasympathetic can modify baseline rate.
   a) Vagus nerve is primary parasympathetic nerve to heart, supplies the atrium, especially the SA and AV nodes, but has little/no effect on ventricles
   b) Decreases heart rate by:
      (1) decreasing rate of depolarization in SA node
      (2) increasing AV node delay
c) Mechanism: Na\(^+\) and Ca\(^{++}\) leaks into SA node cells decreased, K\(^+\) out increased = membrane potential more negative (Figs 13.25b & 13.26)

3. Sympathetic can modify baseline rate.
   a) Sympathetic cardiac nerves supply the atria, including the SA and AV nodes, and also the ventricles
   b) Increases heart rate by
      (1) increasing rate of depolarization in SA node
      (2) reducing AV node delay
      (3) speeding up spread of AP through Bundles of His and Purkinje fibers
   c) Mechanism: Binding of epi or norepi to Beta 1 receptors on pacemakes cells increases rate of Na\(^+\) and Ca\(^{++}\) leaks into cell (Figs 13.25a & 13.26) = membrane potential more positive.

C. Control of *Stroke Volume*
   1. Sympathetic Effects: norepi or epi binding Beta 1 receptors on contractile cells (Fig 13.27)
      a) increases contractile strength of ventricle cells by:
         (1) increasing Ca\(^{++}\) permeability of contractile cells
         (2) increasing Ca\(^{++}\) movement from the SR
         (3) increasing the rate of myosin ATPase activity
         (4) increasing rate of pumping Ca\(^{++}\) back into SR.
   2. Varying length of heart muscle fibers
      a) longer the fiber at start of contraction, the stronger the contraction
      b) more blood that is in chamber, the longer the fiber (i.e. they are stretched) so the stronger the contraction (Fig 13.29)
      c) **Bottom line:** when venous return of blood to heart is increased (by many factors to be discussed later) the blood volume in heart is increased, which increases fiber length, which increases strength of contraction, which allows pumping of larger volume. This is called the *Starling effect.*

D. Summary of Control of Cardiac Output
   1. Figs 13.30, 13.31 & 13.32

III. Nourishing the Heart Muscle
   A. Heart muscle does **not** extract nutrients from blood in chambers
      1. Endocardial lining prevents exchange
      2. Even if exchange could occur, heart walls too thick for effective exchange
   B. Coronary circulation
      1. Coronary arteries branch from aorta just beyond aortic valve
      2. Heart muscle receives most (70%) of nutrient blood supply during diastole because coronary arteries compressed during systole, and entries to these arteries partially blocked during systole