Concepts:

- Fluid flow
- APs in contractile & autorhythmic cells
- Cardiac cycle (elec. & mech. events)
- HR regulation
- Stroke volume & cardiac output

Running Problem: Heart Attack

Developed by John Gallagher, MS, DVM
Modulation of Heart Rate by ANS

- ANS can alter permeability of autorhythmic cells to different ions

- **NE/E (i.e. sympathetic stimulation):** $\uparrow$ flow through $I_f$ and Ca$^{2+}$ channels
  - Rate AND force of contraction go up

- **Ach (parasympathetic):** $\uparrow$ flow through K$^+$ channels $\downarrow$ flow through Ca$^{2+}$ channels
  - Membranes become hyperpolarized

Fig 14-16
The Heart as a Pump  (p477)

- Communication starts in autorhythmic cells in the SA node (the **Pacemaker**)
- Move from events in single cell to events in whole heart
- **Cardiac cycle**
  1. electrical events
  2. mechanical events
- Electrical conduction in heart coordinates contraction
THE CONDUCTING SYSTEM OF THE HEART

1. SA node depolarizes.
2. Electrical activity goes rapidly to AV node via internodal pathways.
3. Depolarization spreads more slowly across atria. Conduction slows through AV node.
4. Depolarization moves rapidly through ventricular conducting system to the apex of the heart.
5. Depolarization wave spreads upward from the apex.
Electrocardiogram ECG (EKG)
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- Surface electrodes record electrical activity deep within body - How possible?
- Reflects electrical activity of whole heart not of single cell!
- EC fluid = “salt solution” (NaCl) ⇒ good conductor of electricity to skin surface
- Signal very weak by time it gets to skin
  - ventricular AP = ? mV
  - ECG signal amplitude = 1mV
- **EKG tracing** = Σ of all electrical potentials generated by all cells of heart at any given moment

Fig 14-22
Depolarization = signal for contraction

Since:

Segments of EKG reflect mechanical heart events
Components of EKG

- **Waves** (P, QRS, T)
- **Segments** (PR, ST)
- **Intervals** (wave-segment combos: PR, QT)

Mechanical events lag slightly behind electrical events.
Einthoven’s Triangle and the 3 Limb Leads:

Fig 14-19
Info provided by EKG:

1. HR
2. Rhythm
3. Relationships of EKG components
   - each P wave followed by QRS complex?
   - PR segment constant in length? etc. etc.
For the Expert:

Find subtle changes in shape or duration of various waves or segments.

Indicates for example:

- Change in conduction velocity
- Enlargement of heart
- Tissue damage due to ischemia (infarct!)
Injury to AV bundle can increase duration of QRS complex (takes longer for impulse to spread throughout ventricular walls).
Heart Sounds (HS)

- **1st HS**: during early ventricular contraction \(\Rightarrow\) AV valves close
- **2nd HS**: during early ventricular relaxation \(\Rightarrow\) semilunar valves close
Gallops, Clicks and Murmurs
(clinical focus, p 486)

Turbulent blood flow produces heart murmurs upon auscultation
Cardiac Cycle: some definitions

- **Systole** (time during which cardiac muscle contracts)
  - atrial
  - ventricular

- **Diastole** (time during which cardiac muscle relaxes)
  - atrial
  - ventricular

- **EDV** = End diastolic volume
- **ESV** = End systolic volume
- **SV** = Stroke Volume—that which is pumped in one stroke

Heart at rest: atrial & ventricular diastole

\[
SV = EDV - ESV
\]

70mL = 135 mL - 65 mL
Cardiac Output (CO) – a Measure of Cardiac Performance

**CO = HR x SV**

- **HR controlled by ANS** (p 475)
  - parasympathetic influence?
  - sympathetic influence?
  - without ANS, SA node fires 90-100x/min

- *What happens with ANS when resting HR goes up (e.g. during exercise)??*
CO = \text{HR} \times \text{SV}

Force of contraction

Length of muscle fibers (Starling curve/law) due to venous return, influenced by skeletal muscle pump and respiratory pump

Sympathetic activity (and adrenaline)
venous constriction by sympathetic NS and Increased Ca^{2+} availability
Frank-Starling Law (p 490)

- SV $\alpha$ EDV
  - i.e., the heart pumps all the blood sent to it via venous return
- Therefore, Venous Return = SV
- **Preload** = the amount of load, or stretch of the myocardium before diastole
- **Afterload** = Arterial resistance and EDV combined
- **Ejection Fraction** = % of EDV that is actually ejected; e.g., 70 ml/135ml x 100 = 52% at rest
Myocardial Infarction

The End