CARDIAC FUNCTION:

- 4 chambers
  - L & R atria
  - L & R ventricles
- 4 valves
  - 2 AV valve (b/w atria & ventricles)
    a) Left: mitral valve (bicuspid)
    b) Right: tricuspid valve
  - 2 SL valves (vessels exiting heart)
    a) Left: aortic valve
    b) Right: pulmonary valve

CARDIAC CONDUCTION:

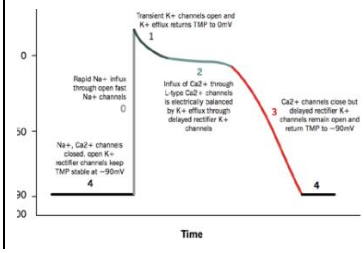
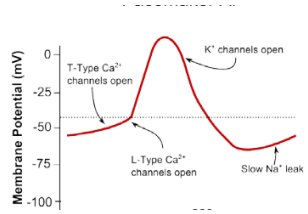
- Automaticity: spontaneous depolarization of some specialized cells
- Myogenic: muscle contraction is self-generated by myocyes
- Pace-making: controlled/regulated by cardiac conduction system
  1. SA node: pacemaker in right atrium
     - Initiates electrical impulse (normal rhythmic)
  2. AV node: right atrium
     - Delay of atrial impulse to ventricle
     - Results in delayed stimulation of ventricular tissues
  3. AV bundle: bundle of His
     - Conduction from atria to ventricle
  4. Purkinje fibers:
     - Conduction to both ventricles simultaneously

CARDIAC CYCLE:

- All events from one heart beat to next
- Frequency = heart rate
- Involves transit of blood through: *heart chambers* and into *pulmonary artery & aorta*

1. Isovolumic ventricular contrraction	• Ventricle myocyte contraction • All valves closed • NO FILLING
2. Ventricular ejection	• Full ventricular contraction • SL valves open • Blood pumps to body/lungs
3. Isovolumic ventricular relaxation	• Ventricles relax • All valves closed • Atria begin filling
4. Early diastole	• 80% passive ventricle filling • Ventricle relaxed • AV valves open (SL closed)
5. Atrial systole	• Atrial contraction • AV stays open (SL closed) • 20% more filling of ventricles = ATRIAL KICK

CARDIAC ACTION POTENTIAL: CELLS

Non-pacemaker cells	Pacemaker cells
NO automaticity	Automaticity
Excitability	Excitability
Atrial and ventricular myocytes	SA, AV nodes, Bundle of His, Purkinje Fibers
Na+ driven (phase 0)	Ca2+ driven (phase 0)
Phase 0 to 4	No phase 1 or 2
	

PHASES OF CARDIAC ACTION POTENTIAL:

Phase 0 – rapid depolarization	• Influx of Na through voltage gated Na channel (Nav 1.5) ○ In SA/AV node – calcium not sodium (Cav. 1.2) • Influx of +ve charge depolarizes cell	
Phase 1 – acute repolarization "notch"	• Inactivation of Nav channel • Efflux K through voltage gated K channels (Kv4.2/4.3)	
Phase 2 – plateau phase	• Influx Ca2+ through voltage gated calcium channel (Cav1.2) • Efflux K through voltage gated potassium channel (KvLQT1) • Sustained muscle contraction	
Phase 3 – rapid repolarization	• Inactivation Cav channels • Amplified K efflux (KvLQT1, **hERG**, Kir) • Net positive efflux repolarizes cells	
Phase 4 – resting membrane potential	• Only some K+ channels types are open and keep resting membrane potential at -90 mV • Cells are ready to refire	
Pacemaker cells: membrane slowly depolarizing through funny channels	• Hyperpolarization-induced cAMP gated channels (HCN) = non-selective channels (contribute to slow Na+ leak) • Reach threshold → refire cardiac AP • Sympathetic stimulation increases cAMP → activation of dunny channels and increased steepness of phase 4 slope → increased firing rate	
Refractory period: time frame in which normal cardiac AP cannot re-excite	Absolute refractory period: phase 0 to early 3 • Impossible due to inactivation of ion channels • Require hyperpolarization to reset channel conformation	Relative refractory period: phase early 3 – early 4 • Can initiate but requires stronger than normal stimulus

CARDIAC ARRHYTHMIAS:

DEFINITION:

- Abnormal rhythms in heart
- Dysfunction impacting rate or regularity of heart function
- Caused by alteration in cardiac conduction sequence (impulse generation or propagation)

SYMPTOMS:

- Palpitations/fluttering feeling in chest
- Shortness of breath
- Fatigue
- Dizziness, light-headed, syncope/fainting

MECHANISMS OF ARRHYTHMIA:

- Dysfunction in impulse formation or propagation/conduction results in arrhythmia
  - Automaticity in heart is spontaneous depolarization of cells
    - Primary: pacemaker cells in SA node
    - Alternate locations: AV node, bundle of His, Purkinje fibers

FORMATION DEFECTS: disturbance in impulse formation

	Enhanced automaticity	Triggered activity	
Cells involved	In pacemaker or non-pacemaker cells > Cells other than SA node (ECTOPIC FOCI) causes abnormal conduction of AP through rest of heart	Non-pacemaker cells	
Mechanism	1. Increase in phase 4 slope 2. Greater RMP – closer to threshold potential > Both lead to increased depolarization frequency (i.e. enhanced automaticity)	Depolarization during repolarization → transient triggered premature depolarization	
		Two types	
		Early afterdepolarization (EAD) • Spontaneous depolarizations during phase 2/3 • Influx of Na+ (different from 0 Na+ channels) or Ca2+ through ion channels • Block of K+ channels	Delayed afterdepolarization (DAD) • Spontaneous depolarizations during early phase 4 • Elevated diastolic Ca2+
Causes	• Increased phase 4 slope: o Catecholamines/caffeine o Increased sympathetic stimulation (stress, HF, cardiac overstretch as with increased end-diastolic pressure in cardiomyopathy/HF) • Less negative RMP o Anti-arrhythmic drugs (K+ channel blockers) o Ischemia o Altered potassium levels (HF)	• EAD: enhanced Ca2+ current or reduced K+ current o Same as in enhanced automaticity • DAD: ESPECIALLY HF, digitalis toxicity, catecholamines	

CONDUCTION DEFECTS: disturbance in impulse conduction

Re-entry	• Aka circus movement • Most common cause of arrhythmias
Mechanism	• Spread of an impulse through already stimulated tissue in a circular movement • Same initial impulse
Criteria for re-entry	• 2 functionally or anatomically distinct potential pathways that join proximally and distally around non-conducting tissue to form a closed circuit of conduction (ex// around infarct/scar tissue) o One of the pathways must have a refractory period that is substantially longer than the refractory period of the other pathway o The pathway with the shorter refractory period must conduct electrical impulses more slowly than the other pathway
Causes	• Ischemia vs. infarction (ischemia enhances conduction while infarction hinders conduction) • Hypokalemia, acidosis • HF, cardiomyopathies, cardiac overstretch • Stress/catecholamine/caffeine/sympathetic activation
Wolf-Parkinson-White (WPW) syndrome 1/1000 people	• Anatomical defect in conduction system • Accessory AV connection (BUNDLE OF KENT) - Type A (left) - Type B (right) o Fast response tissue (AV node is normally slow response tissue) • Excitation through accessory pathway → bypass gatekeeper AV node • Re-entrant supraventricular tachycardia

CLASSIFICATION:

BY RATE:

- Bradyarrhythmia: < 60 bpm
- Tachyarrhythmia: > 100 bpm

BY ORIGIN:

Supraventricular: origin above bundle of His	Ventricular: origin below bundle of His
• **Sinus bradycardia**: SA node origin < 60 bpm • **Sinus tachycardia**: SA node origin > 100 bpm • **WPW syndrome:** re-entrant tachycardia • Paroxysmal supraventricular tachycardia: 100-200 bpm at/above AV node • Premature atrial contraction: atrial (non-SA) premature beat • **Atrial flutter:** abnormal heart rhythm in atria (can cause supraventricular tachycardia) • **Atrial fibrillation:** disruption of normal SA impulse	• **Premature ventricular contractions:** premature beat originating below AV node • **Ventricular tachycardia**: rapid heart beat originating in ventricle • **Ventricular fibrillation:** uncoordinated ventricular contractions

COMMON ARRHYTHMIAS:

	AFib	Atrial flutter	Premature ventricular contraction (PVC)	Ventricular tachycardia	Vfib
Intro	• Most common arrhythmia	• 2nd most common arrhythmia	• Common even in healthy people • Extra heart beat	• 3 or more PVCs in a row with fast heart rate (> 120 bpm) • Inefficient ventricular filling	• Severe fatal arrhythmia • Ventricle quivers (rapid, uncontrolled, inefficient contractions)
Leads to	• High risk to cause ischemic stroke (blood pools and clots in heart)			• Serious – life threatening leading to Vfib	No cardiac output results in cardiac collapse within minutes (heart not getting blood/nutrients)
Risk	• Age • Diabetes • Heart disease		• Age, male • Stress, catecholamines • CAD, MI, myocarditis	• Metabolic abnormalities • MI • Drug toxicity	

Cause	• Multiple re-entry loops
	• Extremely rapid atrial activity (400-600 bpm)
	• Highly disorganized ventricular contraction (120-180 bpm)
	  ○ AV node acts as brake for some but not all signals
	  ○ Sower refractory period
		• Single ectopic dominant re-entry loop
		• Extremely rapid atrial activity (270 – 330 bpm)
		• Not transmitted to ventricle
		  ○ AV node acts as brake
		  ○ Regular input to ventricle
			• Abnormal origin in ventricle
			  ○ Ventricle depolarizes and contracts early
			  ○ May be due to re-entry, triggered activity or enhanced automaticity
			• Refractory pause
			  ○ Missed beat following PVC
			  ○ Ventricles are refractory when SA signal reaches them
				• Ventricular ectopic foci	• Enhanced automaticity
					• Trigerred activity
					Re-entry

ECG	• No P wave – ectopic origin in atria • Irregular R-R interval	• MANY merged P wave – saw tooth pattern • Regular RR-interval but fast	• Premature QRS with no P wave • Irregular QRS shape (sometimes inverted) • Skipped beat (refractory pause R-R is 2x)	• Abnormal location / absence P wave (hidden) • Monomorphic – beats look the same • Polymorphic – beats are irregular from beat to beat (**Torsade de pointes)**	Irregular LARGE QRS complexes
Symptoms	• Irregular fast heart beat • Heart palpitations (thumping in chest) • SOB increases with exertion • Fatigue, dizziness		• Can be asymptomatic • Skipped heart beat, palpitations • Stronger heart beat	• Heart palpitations • breathlessness	• Unconsciousness • Sudden cardiac death