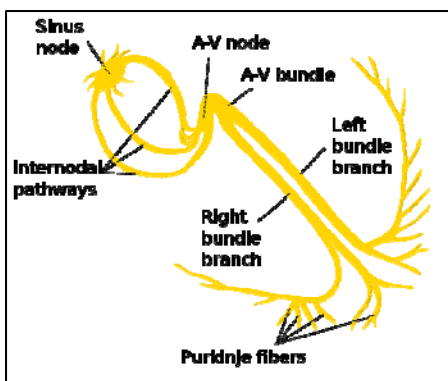
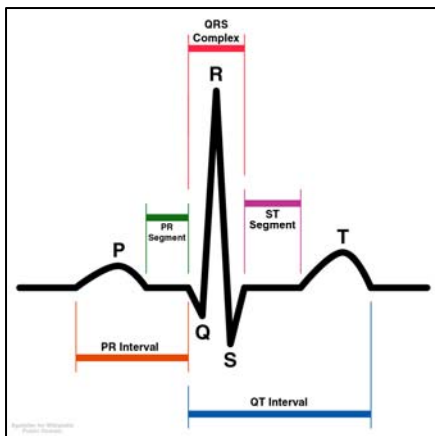


Overview-Arrhythmias are caused by disturbances of the electrical conduction system of the heart. They can result in either bradycardia or tachycardia, and be anywhere from asymptomatic to lethal. This section will rapidly review normal conduction, and the electrocardiogram (ECG), with that as a basis we will move onto a discussion in outline form of bradycardias and tachyarrhythmias. The management of stable and unstable rhythms is in accordance with the current 2010 American Heart Association guidelines.



Normal Conduction: The sinoatrial (SA) node discharges, and depolarization spreads through the atrial muscle. A delay occurs as the charge passes through atrial ventricular (AV) node, made up of specialized atrial tissue. The charge then passes through the single pathway A-V bundle (Bundle of His) in the ventricle before it divides into the right and left bundle branches.

ECG: The normal ECG consists of a P wave a QRS wave and a T wave. All ECG



machines run at standard rate and print on standard paper. Each large 5 mm square is 0.2 s, so there are 5 large squares to one second. Each small square is 0.04s (40 ms). The R-R interval is the time between beats, and can be used to calculate the HR from the rhythm strip; if there is one beat every 5 squares the HR is 60 bpm, if there is a beat every square it is 300

bpm. The PR interval is the time it takes for the excitation to spread from the SA node, through the atrial muscle and AV node, down the AV bundle and into the ventricular

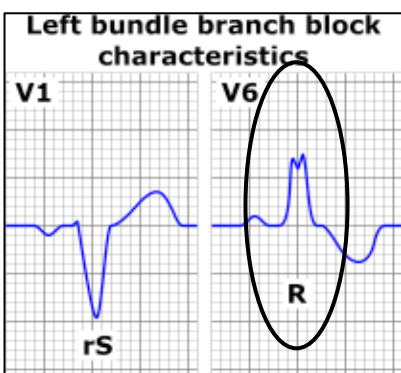
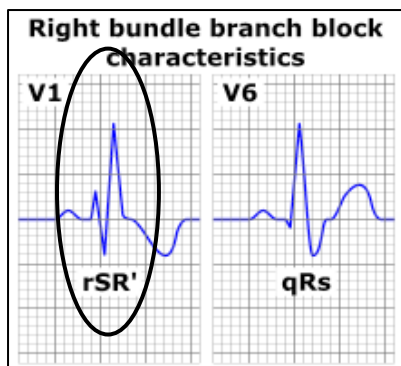
muscle. It is measured from the beginning of the P-wave to the beginning of the QRS wave. The normal PR interval is 120-200 ms or 3-5 small squares on the ECG strip. The normal QRS is <120 ms (3 squares). The ST segment is measured from the end of the S segment to the beginning of the P. The P-wave is atrial de-polarization and the QRS ventricular. T wave is re-polarization of the ventricular conduction pathway. ST elevation can result from a failure of ischemic tissue to re-polarize.

BRADYCARDIA

- Sinus Bradycardia: Arises from the SA node, normal PR. HR <60/min, not generally symptomatic unless <50/min. It can be normal or a result of the vasovagal reflex.
- AV Nodal Block: Can be caused by hyperkalemia, hypermagnesemia, digoxin and b-blockers.
 - First-Degree: Prolonged PR > 0.2 s (one large box on ECG), all P-waves are conducted, and the PR interval is fixed. Usually asymptomatic, no treatment or further work-up required.
 - Second-Degree Type 1 (Wenckebach): The P waves occur at regular intervals but there is, progressive prolongation of the PR interval with each eventual missed beat (P-wave with no QRS). It is a stable rhythm, once causal medication stopped resolves. Second-Degree Type 2: Sudden non-conducted P-wave without change in the PR interval. A sudden dropped QRS is observed. It is inherently unstable with P:QRS ratios of 3 or 4:1, and almost always an infra AV nodal conduction disturbance. It can be caused by an anterior wall MI, and can progress into third degree block

- Third-Degree: Complete dissociation of the P and QRS waves. The P-waves march through at regular intervals. The QRS waves can be narrow or complex. It can result from inferior and anterior wall MIs, and often requires permanent pacemaker placement.
- Treatment
 - Stable: Observation, identify and treat the cause, permanent pacemaker
 - Unstable: Identify and treat the cause, 1) Atropine (0.5 mg bolus, repeat every 3-5 minutes) 2) Transcutaneous pacing 3) Dopamine (2-10 mcg/kg/min) or Epinephrine (2-10 mcg/min) infusions 4) Transvenous pacing if available.

BUNDLE BRANCH BLOCK: Right and left-bundle branch block can be a sign of ischemic disease. In addition if the patient is tachycardic they can be confused with more malignant rhythms, and ischemia.



- Right Bundle Branch Block (RBBB): No RB conduction-RV depolarizes from the left causing the r wave before the QRS (rSR) in V1, and a small Q-wave in V6. The RV depolarizes after the left, causing R' in V1 and deep S in V6.
- Left Bundle Branch Block (LBBB): No LB conduction, depolarizes from the right; causing a very small Q wave in V1, and an R wave in V6. Further depolarization causes an S-wave in V1 and a second R wave in V6.

TACCYCARDIA: Is defined as a HR > 100 bpm and is characterized as wide complex or narrow complex QRS, regular or irregular, and stable or unstable. A narrow complex QRS is < 120 ms. Rates < 150 bpm do not usually cause instability.

SUPRAVENTRICULAR: Narrow-complex, however can be wide if LBBB/RBBB

- Sinus Tachycardia (Narrow QRS, regular): A physiologic response to an external stimuli (pain, fever etc), usually not a source of instability in the short term.
- Re-entry Tachycardia, nodal tachycardia (Narrow QRS, regular) Younger, healthy patients rate 120-250 bpm, twice as common in women, rapid onset, often relieved by Valsava, can be wide complex if impulse enters non-specialized ventricular myocardium and depolarizes slowly.
- Atrial Fibrillation (Irregular, no P-waves, HR variable) and Flutter (Saw-tooth P-waves, usually 150 bpm and regular). Associated with cardiovascular pathologies, including CAD and HTN, affects 1-2% of the population, can be associated with slow conduction or AV block, often unstable.
- Multifocal Atrial Tachycardia (MAT) (P waves present, irregular)
 - Rapid and irregular, P wave morphology variable, rarely causes instability and reverses with electrolyte correction, usually stable
- Treatment: Correction of electrolyte abnormalities, removal of inciting causes
 - Stable: Atrial Fibrillation/Flutter: B-blocker, amiodarone, digoxin (in HF patients), calcium channel blockers, burst pacing in pts with pacemaker.
 - Amiodarone: Effective in converting both fibrillation and flutter, controls HR, low Torsades risk, not a negative inotropic agent, safe in critical illness

- Unstable: Synchronized Cardioversion
 - Irregular QRS (atrial fibrillation/flutter): 120-200 J
 - Narrow regular: 50-100 J, can consider adenosine 1st
 - Adenosine: Terminates AV nodal re-entry tachycardia, will *not* terminate atrial fibrillation/flutter but can slow the rate to make the diagnosis. 6 mg IV given rapidly (1-3 sec), 2nd dose 12 mg IV.

VENTRICULAR ARRHYTHMIAS: Wide complex >120 ms, morphology of QRS is different than in prior ECGs, abnormal T waves, usually regular rate 120-170. It is caused by underlying ischemic disease, hypothermia and electrolyte abnormalities.

- Ventricular Tachycardia (VT): 3 or more beats, prolonged if > 30 seconds
 - Monomorphic: QRS waves have similar morphologies
 - Consider supra-ventricular tachycardia with LBBB/RBBB (look at prior ECG)
 - If A-V dissociation VT
 - Polymorphic (normal QT): QRS waves have different morphologies can degenerate into ventricular fibrillation.
 - Polymorphic (Torsades de points): Evolves from prolonged QT interval, often caused by medications that prolong the QT. Rapid VT that vary in amplitude and polarity, or twisting around the baseline. Initiated when a beat falls prematurely on a T-wave.
 - Treatment: Oxygen, remove inciting events, electrolyte correction, IV access 12 lead ECG.

- Stable: Monomorphic ONLY consider adenosine. Polymorphic anti-arrhythmic infusion:
 - Procainimide
 - 20-50 mg/min until suppressed, follow by infusion
 - Stop if hypotension, or QRS increase by > 50%
 - Amiodarone
 - 150 bolus over 5 mins, then drip, repeat as needed if VT recurs
 - Sotalol
 - 100 mg (1.5mg/kg) over 5 minutes
 - Avoid if prolonged QT
- Unstable: Synchronized cardioversion, ACLS protocol if loss of vital signs.
- Ventricular Fibrillation (VF): No waveform, may be coarse or fine
 - Treatment: ACLS protocol
- ACLS protocol for Cardiac Arrest with VT/VF: Call for help, give O₂, attach monitor
 - Shock-Biphasic at maximum dose (120-200 J), 2 full minutes of CPR, IV access, check rhythm, Shock, 2 full minutes of CPR, Epinephrine, advanced airway, capnography, check rhythm, Shock, 2 full minutes of CPR, Amiodarone