CORE CONCEPTS: ECG 1

Basic Electrophysiology and Sinus Rhythms

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CARDIAC CELLS IN MYOCARDIUM (two distinct types):

1. **Contractile Cells*:** Also called "mechanical" or "working" cells in the myocardium (middle layer) of the heart. Contain contractile filaments that cause the cell to contract when electrically stimulated.

   ***NOTE:** *Can myocardial contractile cells generate an electrical signal?* NO.*

2. **Conducting Cells:** Also called "electrical" or "automatic" cells in the heart. Pacemaker cells have ability to spontaneously form electrical impulses (action potentials).

CARDIAC CELL PROPERTIES:

Term	*Definition*	*Key Points*
Automaticity	Ability of a **pacemaker cell** to CREATE an electrical impulse without being stimulated	• Not all cardiac cells have automaticity. • Influenced by electrolytes, especially calcium and potassium.
Excitability	Ability of cardiac cells to RESPOND to an external source of stimulus	• Has the cell repolarized? • Also referred to as "irritability"
Conductivity	Ability to receive and TRANSMIT (pass along) to adjoining cell.	• All cardiac cells have conductivity. • Can be changed by factors like meds and autonomic response.
Contractility	Ability of a myocardial muscle cell to mechanically respond (shorten in order to cause contraction)	• Myocardium: this thick middle layer of heart contains muscle fibers for contraction

INTRINSIC RATES OF CELLS WITH AUTOMATICITY:

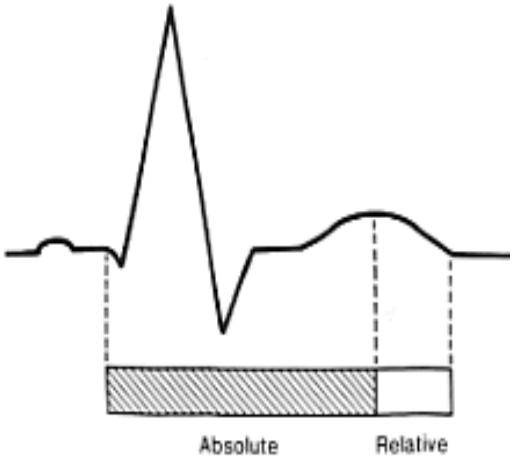
Source	*Associated with…*	*Intrinsic Rate (beats per min.)*
Sinus or Sinoatrial (SA) Node	***Sinus Rhythms***	**60-100 bpm**
AV Junction	***Junctional Rhythms***	**40-60 bpm**
Purkinje Fibers	***Ventricular Rhythms***	**20-40 bpm**

POLARIZATION STATES:

Polarization: "Resting & ready" state of conducting cell – inside cell is NEGATIVELY charged.

Depolarization: Stimulated state of conducting cell – inside cell becomes POSITIVELY charged.

- Depolarization typically results in contraction of the cardiac muscle
- **NOTE:** After depolarization, *"repolarization" occurs – movement of charged particles across cell membrane to restore NEGATIVE charge inside the cell*



REFRACTORY PERIOD: Time cell needs to recover (repolarize) before it can again respond to a new stimulus (depolarize).

- ***Absolute*** **Refractory Period:** the period of time in which a cell absolutely cannot respond again to another stimulus (fire an impulse or contract).

- ***Relative*** **Refractory Period (RRP):** the period of time where some cells are repolarized, but not all cells are ready for another impulse. If a stronger than normal impulse comes along, cells may respond.

Why is this important?

- An electrical impulse that comes through during RRP can cause a tachyarrythmia (fast abnormal rhythm) to begin.
- Risk for this happening increases with prolonged QT intervals.
- That's why RNs monitor QT intervals if patient is taking meds known to cause QT prolongation.
- This concept is key to understanding **synchronized cardioversion** *(see next section: ECG 2, p. 9)*

Image: http://nursegeorge.com/ecg2.html

SINUS RHYTHMS TO KNOW FOR EXAM:

	Sinus BRADYCARDIA (SB)	Sinus Rhythm	Sinus TACHYCARDIA (ST)
Rhythm	regular	regular	regular
Heart Rate	**less than 60**	**60 to 100**	**greater than 100**
P wave	present & normal	present & normal	present & normal
PR interval	"NORMAL" *0.12 to 0.20 sec*	"NORMAL" *0.12 to 0.20 sec*	"NORMAL" *0.12 to 0.20 sec*
QRS duration	"NARROW" *0.11 sec or less (unless abnormally conducted)*	"NARROW" *0.11 sec or less (unless abnormally conducted)*	"NARROW" *0.11 sec or less (unless abnormally conducted)*

*Note: Be aware of **sinus arrhythmia**. However, it will not be on exam since it "usually does not require treatment"*

Carefully review: **SINUS BRADYCARDIA** and **SINUS TACHYCARDIA**
"at a glance" on next 2 pages…

SINUS BRADYCARDIA (SB) at a glance

HOW DO I RECOGNIZE IT?

Rhythm	regular
Heart Rate	**less than 60**
P wave	present & normal
PR interval	"NORMAL" *0.12 to 0.20 sec*
QRS duration	"NARROW" *0.11 sec or less (unless abnormally conducted)*

WHAT CAUSES IT?

- ↑ parasympathetic tone
  - too much of a drug that ↓ HR (parasympathomimetic or cholinergic)
  - vagal stimulation, such as bearing down for BM
- myocardial infarction (MI) *(inferior or posterior)*
- ↑ intracranial pressure (ICP)
- hypoxia
- ↑ K+
- ↓ K+
- hypothyroidism

Can SB be normal for some people? **YES.** Well-trained athletes and some people during sleep.

WHAT DO I DO ABOUT IT?

TREATMENT:

1. **atropine** (anticholinergic)
2. **dopamine or epinephrine** (sympathomimetic)
3. **pacemaker (temporary or permanent)**
   - *Note: if caused by too much drug, then hold next dose, discontinue, or ↓dosage*

Do we always treat SB? **NO.** Only if it is symptomatic = *"Pt is not tolerating rhythm…"*

SYMPTOMS of hemodynamic compromise (↓ CO) due to low heart rate

- Acute changes in mental status (↓ LOC, syncope)
- Chest pain or discomfort
- Cold, clammy skin
- Fall in urine output (↓ renal perfusion)
- Hypotension, later turning to shock
- Respiratory issues:
  - Pulmonary congestion (crackles, rhonchi, dyspnea) related to heart failure
  - Shortness of breath (↓ O_2 sat, ↑ RR, ↑ work of breathing)

Note: It is NOT necessary to have EVERY symptom listed here before getting treatment.

SINUS TACHYCARDIA (ST) at a glance

HOW DO I RECOGNIZE IT?

Rhythm	regular
Heart Rate	**greater than 100**
P wave	present & normal
PR interval	"NORMAL" *0.12 to 0.20 sec*
QRS duration	"NARROW" *0.11 sec or less (unless abnormally conducted)*

WHAT CAUSES IT?

- ↑ sympathetic tone
  - exercise or physical exertion
  - too much of drug that ↑ HR (sympathomimetics including cocaine, meth, MDMA, caffeine, nicotine)
- pain
- anxiety
- fever
- hypoxia
- dehydration, hypovolemia
- shock
- myocardial infarction
- hyperthyroidism

WHAT DO I DO ABOUT IT?

TREATMENT:

1. **Treat underlying cause…**
   - reduce pain, anxiety, or fever
   - fluid replacement for dehydration, hypovolemia
   - supplemental oxygen for hypoxia (even intubation if necessary)
   - decrease or stop drugs causing ST
   - if MI, obtain 12-lead ECG & cardiac enzymes to confirm, then send to cardiac cath lab
   - in clinically stable patients, vagal maneuvers may be used to lower HR

2. **Medication options:**
   - *beta blockers* or *CCBs*
   - *adenosine* may be used for diagnosis or treatment of different kinds of tachycardias

WAVEFORM TERMS AND ILLUSTRATIONS:

Term	*Definition*	*Key Points*
Baseline	A straight line recorded when electrical activity is not detected	• Also called **isoelectric line**
Waveform	Also known as a *deflection:* Movement away from baseline in either a positive or negative direction	• **Positive deflection = UP** from baseline *(towards measurement lead)* • **Negative deflection = DOWN** from baseline *(away from measurement lead)*
Segment	A line between two different waveforms	• Know how to calculate intervals below • Know: **ST elevation** and **ST depression**
Complex	Several waveforms	• Know for exam: **QRS complex**
Interval	A waveform and a segment	• Know: **PR Interval** and **QT Internal**

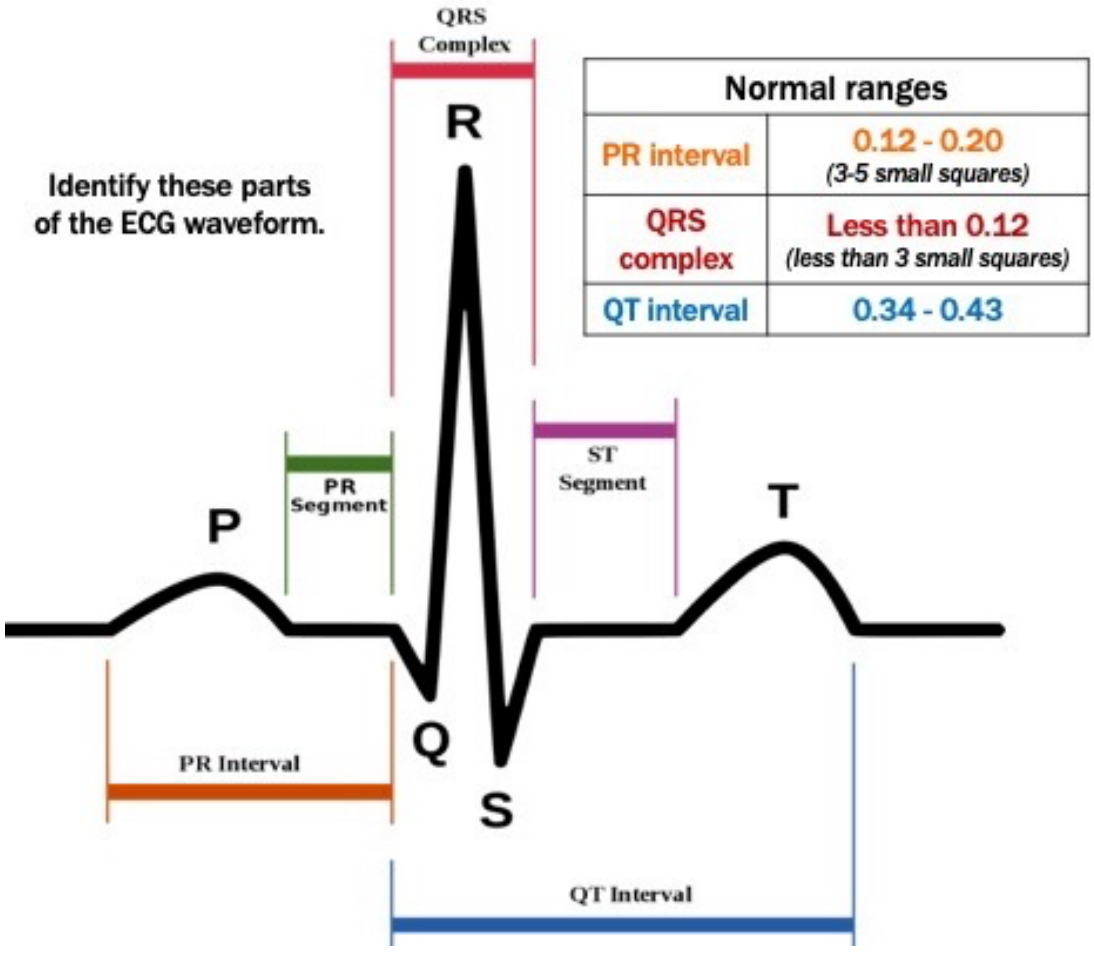


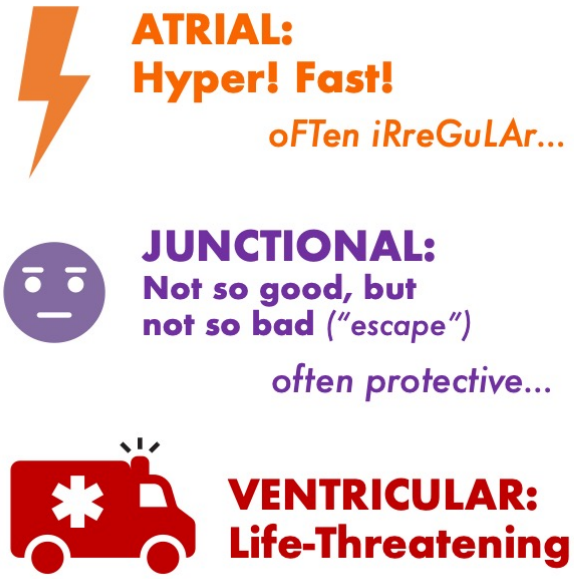
Image: https://commons.wikimedia.org/wiki/File:SinusRhythmLabels.svg

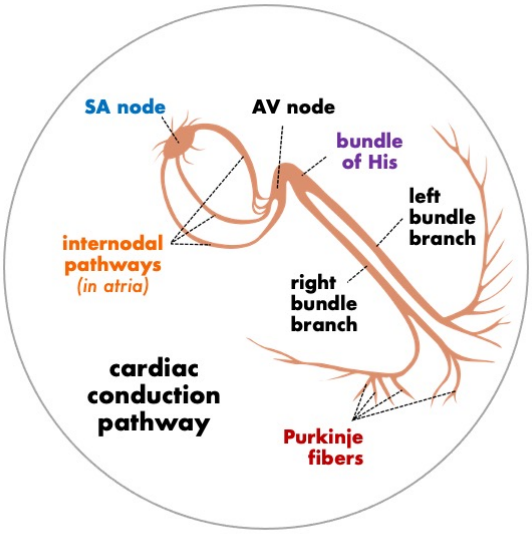
CORE CONCEPTS: ECG 2

Atrial Rhythms

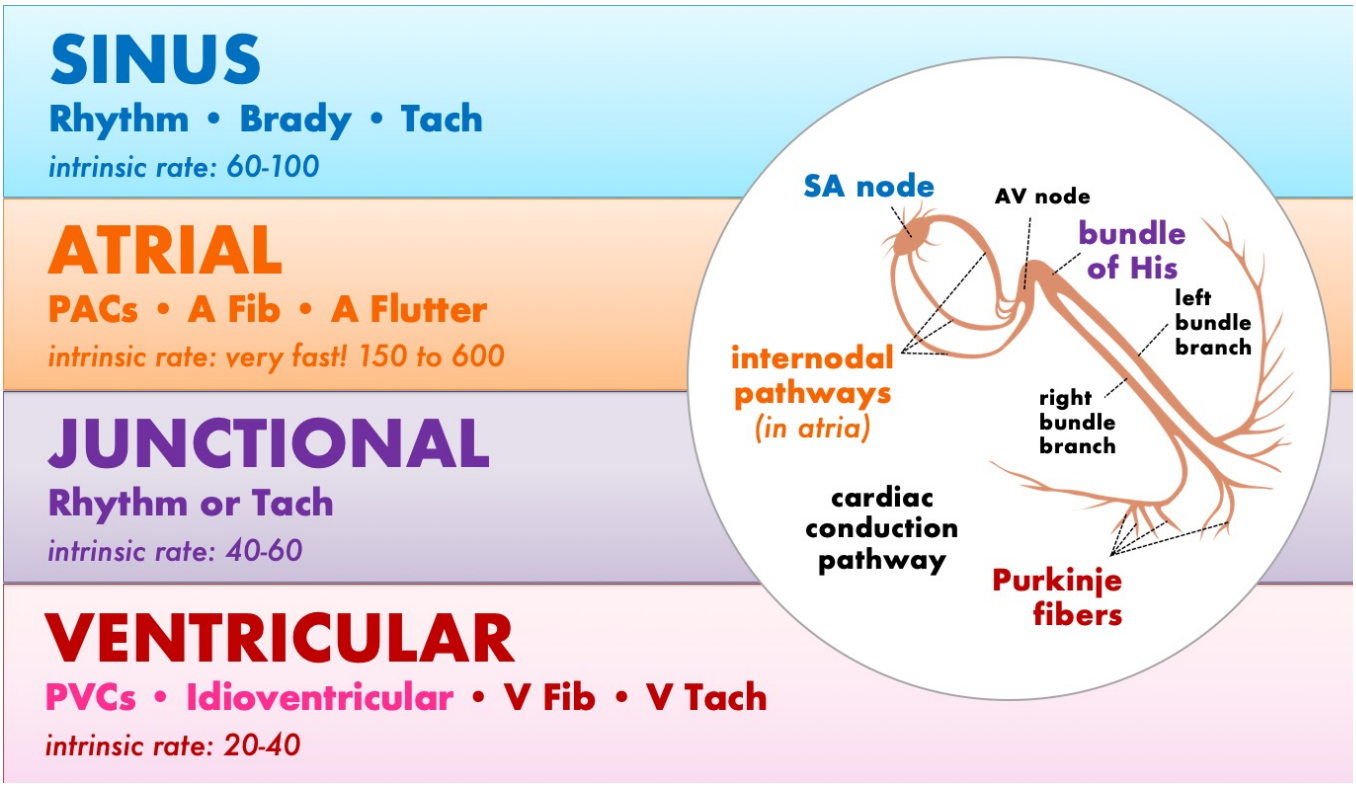
Junctional Rhythms

Ventricular Rhythms





Cardiac conduction pathway: adapted from
https://commons.wikimedia.org/wiki/File:Electrical_conduction_system_of_the_heart_ca.svg#/media/File:Electrical_conduction_system_of_the_heart.svg



ATRIAL KICK: Blood pushed into ventricles because of atrial contraction.

- Ventricles are 70% filled before atria contract. Atrial contraction during ventricular diastole forces additional blood (~10 to 30% of ventricular capacity) into ventricles: this is the atrial kick.
- Loss of atrial kick results in 20% to 30% drop in cardiac output. *Think about CO equation…*
    - **CARDIAC OUTPUT (CO) = HEART RATE (HR) x STROKE VOLUME (SV)**

"*FAST!*" ATRIAL RATES VS. VENTRICULAR RESPONSE RATES

- A healthy AV node protects the ventricles from extremely fast atrial rates.
    - AV node normally cannot conduct faster than 180 impulses per minute.
    - AV node designed to slow down and/or block impulses in this way.
- Ventricular Response is determined by how many electrical signals get through the AV node to cause ventricular contractions. This rate is either **SLOW, CONTROLLED, OR RAPID.**

RVR	Rapid Ventricular Response *(If there is RVR, the atrial rate is considered "uncontrolled")*	greater than 100 bpm
CVR	Controlled Ventricular Response *(Nurses see this often in hospital due to interventions)*	60-100 bpm
SVR	Slow Ventricular Response *(much less common – be aware it can occur, but won't be on exam)*	less than 60 bpm

ATRIAL RHYTHMS at a glance

HOW DO I RECOGNIZE IT?

	Atrial FLUTTER	Atrial FIBRILLATION
Atrial Rhythm	Regular	Irregular
Atrial Rate	"FAST!" can get as high as 300 bpm	"FAST!" can get as high as 600 bpm
Ventricular Rhythm	Regular or irregular: *varies depending on AV conduction of atrial beats.*	Irregular *(sometimes called "irregularly irregular")*
Ventricular Rate	Usually less than 180 bpm	Variable: RVR, CVR, or SVR *(see table below)*
P wave	**Saw-toothed "flutter" waves**	• **No identifiable P wave** • **Fibrillatory waves present *(isoelectric line = ERRATIC or WAVY)***
PR interval	"ABSENT" *(no P wave = no PR interval)*	"ABSENT" *(no P wave = no PR interval)*
QRS duration	"NARROW" *0.11 sec or less* *(unless abnormally conducted)*	"NARROW" *0.11 sec or less* *(unless abnormally conducted)*

WHAT CAUSES ATRIAL DYSRHYTHMIAS? *Underlying heart condition (CAD, hypertension, cardiomyopathy), toxic levels of certain medications, ETOH abuse, sleep apnea, etc.*

A FLUTTER: What Do I Do About It?

- *If RVR:* **control ventricular response**
  - **β blockers** –or– **CCBs** that directly affect heart: ***diltiazem, verapamil***
- *If RVR and signs of hemodynamic compromise:*
  synchronized cardioversion

RVR: Rapid Ventricular Response

Also: Radiofrequency **ablation** – *destroys the cells ("ectopic focus") causing dysrhythmia. Cardiac cath lab procedure.*

A FIB is the most common dysrhythmia.

A FIB (below) is different than A FLUTTER (previous page); however, treatment for both is similar.

A FIB: What Do I Do About it?

- Monitor ECG telemetry, VS, O_2 sat
  - ➢**H**ead of Bed: ↓ HOB, *if hypotensive*
  - ➢**O**xygen: administer supplemental O_2, *if saturation drops*
  - ➢**T**welve-lead ECG: obtain, *if new onset or change in condition*
- Consider causes *(underlying heart condition, ETOH abuse, sleep apnea, etc.)*

ASSESS: Is patient tolerating rhythm?

("tolerate" = no symptoms of hemodynamic compromise)

ISBARR provider, *if new onset or change in condition.*

Remember: 25% of patients with A Fib have no symptoms

A FIB: Is Patient Tolerating Rhythm?

☺ YES

- Control ventricular rate
  - **CCBs** (*diltiazem, verapamil*), **β blockers,** or **digoxin**
- Convert to SR with anti-dysrhythmics *(amiodarone)*
- Anticoagulation
- Ablation

NO ☹

- Control ventricular rate
  - **adenosine**
  - **CCBs** (*diltiazem, verapamil*), or **β blockers**
- *If meds not effective, then* **synchronized cardioversion***

*TEE *(transesophageal echocardiogram)* might be done first to assess for clots in atria.

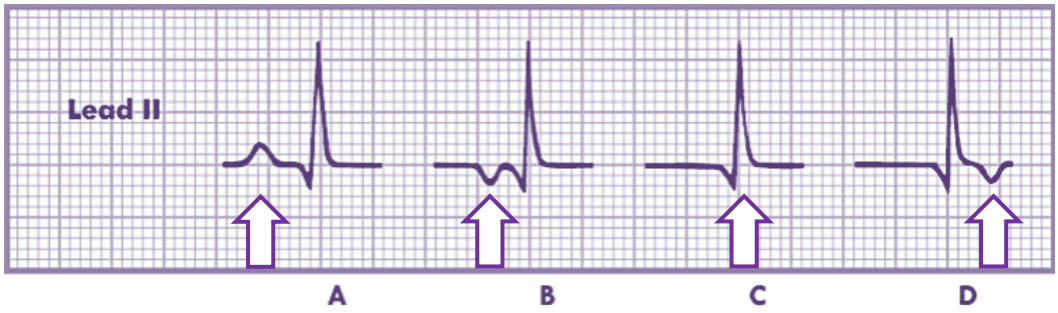
Note: treatment of A Fib and A Flutter are very similar.

JUNCTIONAL RHYTHMS:

- **AV** (atrioventricular) **Junction** consists of the **AV node** and the **Bundle of His**

- **The AV junction will assume responsibility for pacing the heart if…**
  - SA node doesn't fire
  - SA node generates a rate of impulses lower than intrinsic pacemaker rate of AV junction
  - SA node impulse is blocked: either (a) before leaving SA node or (b) before entering AV node

- ***Key Point:*** **Junctional rhythms primarily recognized by differences in P wave & PR interval.**
  - **P wave differences** - it will be **short** (<0.12 sec), **inverted** (not upright), or **absent**
    Why? Remember that with a junctional rhythm the impulse is starting at the AV junction and may be traveling in the opposite direction of a typical impulse to get to the atria.



Notice how the P-wave is occurring a little bit later in each example…

A: normal P-wave and PR interval

B: P-Wave inverted, *before* QRS *(note: this drawing has the PR interval a bit long at 0.12 sec)*

C: No visible P-wave, occurs *during* QRS *– atrial & ventricular depolarization occur at same time*

D: P-wave inverted, *after* QRS *(note: FYI, but this type of example will not be on exam)*

JUNCTIONAL RHYTHMS at a glance

HOW DO I RECOGNIZE IT?

	Junctional ESCAPE Rhythm	**ACCELERATED Junctional**	**Junctional TACHYCARDIA**
Rhythm	Regular	Regular	Regular
Rate	**40-60 bpm**	**61-100 bpm**	**greater than 100 bmp**
P wave	"NOT NORMAL" • Absent or inverted in lead II *Note: May occur before, during, or after QRS (see drawing on previous page)*		
PR interval	"SHORT or ABSENT" *less than 0.12 sec*		
QRS duration	"NARROW" *0.11 sec or less (unless abnormally conducted)*		

WHAT CAUSES IT?

- ↑ parasympathetic tone
- adverse effect of drugs *(digoxin, beta blockers, CCB, etc.)*
- hypoxia
- acute coronary syndrome *(such as MI)*
- and more…

WHAT DO I DO ABOUT IT?

TREATMENT:

- Obtain 12-lead EKG and ensure IV access.
- Assess for signs and symptoms of drug toxicity
    - Review drugs patient is taking
        - *prescribed:* digoxin, beta blockers, CCB
        - *other:* nicotine, amphetamine, caffeine
    - If toxicity is cause, then withhold med until symptoms subside.
- If drug toxicity is not present, then other treatments may include:
    - If heart rate 40-60: atropine
    - if HR greater than 60: a med for rate control may be used (beta blockers or CCB)
- *Also: patient may be scheduled for an **ablation** procedure in cath lab.*

JUNCTIONAL: What Do I Do About it?

Monitor ECG telemetry, VS, O_2 saturation

- **H**ead of Bed: ↓ HOB, *if hypotensive*
- **O**xygen: administer supplemental O_2, *if saturation drops*
- **T**welve-lead ECG: obtain, *if new onset or change in condition*

Consider causes (↑ *parasympathetic tone, med overdose, rule out MI)*

ASSESS: Is patient tolerating rhythm?

("tolerate" = no symptoms of hemodynamic compromise)

ISBARR provider, *if new onset or change in condition.*

JUNCTIONAL: Is Patient Tolerating Rhythm?

☺ YES

- Treat underlying cause
  - *If overdose, hold that med:* **digoxin, β blockers,** *or* **CCBs**
  - *If possibly MI,* rule out by:
    - obtaining 12-lead ECG and cardiac enzymes
    - monitor for ST elevation

NO ☹

- *If HR too low,* **atropine** or **temporary pacing**
- *If MI,* prepare patient for **cardiac cath lab**
- *If junctional tach,* **adenosine** or **vagal maneuvers** may be used to slow rhythm temporarily to allow diagnosis

VENTRICULAR RHYTHMS TO KNOW FOR EXAM:

IDIOVENTRICULAR RHYTHM

Rhythm	Heart Rate	P wave	PR interval	QRS duration
Regular	20-40	"NONE" *(occasionally a P wave may appear after QRS)*	None *(no P wave = no PR interval)*	"WIDE & BIZARRE" *0.12 sec or longer*

VENTRICULAR TACHYCARDIA (VT or V Tach)

Rhythm	Heart Rate	P wave	PR interval	QRS duration
Usually regular *Note: Polymorphic VT may be irregular*	greater than 100	"NONE" *(if a P wave occurs, no relationship to QRS)*	None *(no P wave = no PR interval)*	"WIDE & BIZARRE" *0.12 sec or longer*

What Do I Do About V Tach?

See slide on next page.

VENTRICULAR FIBRILLATION (VFib)

Rhythm	Heart Rate	P wave	PR interval	QRS duration
Chaotic	None • No discernable waves or complexes to measure. • Heart muscle is just trembling instead of pumping.			

What Do I Do About V Fib?

V FIB = DEFIB (!!!)

and CPR

V TACH: What Do I Do? Call a code!

*Does pt have a **pulse**?* **NO**

- CPR
- **Defibrillation[1]**
- Follow ACLS algorithm

*Does pt have a **pulse**?* **YES**

- Stable but symptomatic
  - Oxygen, if indicated
  - IV access
  - Ventricular antidysrhythmics (lidocaine IV, etc.)
- Unstable patient
  - Oxygen, IV access, sedation
  - **Synchronized cardioversion[2]**

Remember…

[1]Defibrillation

- **Non-synchronized (random) electrical current (shock)** through chest wall to the heart by paddles or electrode pads.
- ***Higher dose** of electricity (joules)*
- ***Do not turn on SYNC***

[2]Synchronized Cardioversion

- **Synchronized (timed) electrical current (shock)** through chest wall to heart by paddles or electrode pads.
- ***Lower dose** of electricity (joules)*
- ***Must turn on SYNC***

Cardioversion must be synchronized with patient's innate rhythm. This prevents sending an electrical shock during the relative refractory period (RRP), which would cause a tachyarrhythmia (see previous section: ECG 1, p. 2).

Defibrillation is used when patient is pulseless and electrical activity is chaotic and disorganized (in other words, there is no innate rhythm with a T-wave to synchronize with or disrupt.)

CORE CONCEPTS: ECG 3

AV Blocks

Premature Complexes

Acronym	Definition	Key Points
AVB: Atrio-ventricular Block	A delay or interruption in impulse conduction from the atria to the ventricles. AV blocks are recognized by assessing the PR interval length and regularity.	*There are 4 types of AV blocks:* ○ ***1st degree AVB**** ○ *2nd degree AVB type I* ○ *2nd degree AVB type II* ○ ***3rd degree AVB (also called Complete Heart Block)**** **Only 1st and 3rd will be on exam.*
Premature Complexes:	A premature (occurring before the next anticipated beat) electrical complex that arises from a site outside the SA node.	*If premature <u>atrial</u> complex (PAC)*:* • *QRS is **normal*** *If premature <u>ventricular</u> complex (PVC)*:* • *QRS is **wide & bizarre*** **Only PAC and PVC will be on exam.*
Patterns of Premature Complexes	*Sometimes premature complexes can occur in patterns:* • **Couplet:** 2 premature complexes in a row • **Runs** *(also called "bursts"):* 3+ premature complexes in a row • **Bigeminy:** Every other beat is a premature complex • **Trigeminy:** Every third beat is a premature complex	

AV BLOCKS TO KNOW FOR THE EXAM:

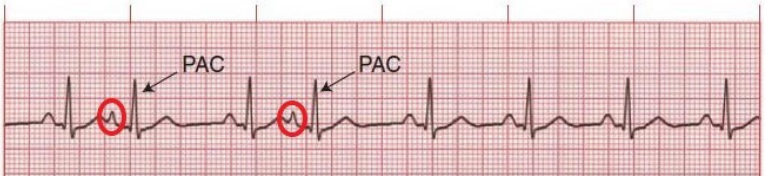
- Heart blocks are a **delay** or **complete block** of impulse conduction from atria to the ventricles.
- There are four in all, but we will focus on the following two…

	FIRST-DEGREE AV BLOCK	**THIRD-DEGREE AV BLOCK**
Definition	**Delay** in impulse conduction = prolonged PR interval *(greater than 0.20 sec)*	**Complete block** in impulse conduction = atria and ventricles *are* not communicating impulses to each other.
Rhythm	Regular	Two separate and unrelated rhythms: • Atrial: Regular • Ventricular: Regular
Heart Rate	Usually in normal range (60-100 bpm), but depends on underlying rhythm	• **Atrial rate: faster** than (and independent of) ventricular rate. • **Ventricular rate: slower,** determined by the origin of the escape pacemaker *(review intrinsic rates).*
P wave	**Normal** in size and shape with one P wave for every QRS.	Normal in size and shape. **More P waves than QRS complexes: a P wave will NOT precede every QRS.**
PR interval	"LONG" *greater than 0.20 sec* PR interval is constant and does not vary	"NONE" Atria and ventricles beat independently of each other
QRS duration	"NARROW" *0.11 sec or less (unless abnormally conducted)*	"NARROW or WIDE" *depending on where escape rhythm is coming from: the ventricle or AV junction.*

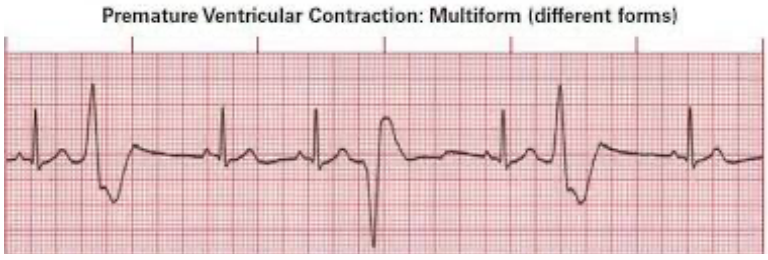
PREMATURE COMPLEXES TO KNOW FOR EXAM: *Compare and Contrast*

	Premature ATRIAL Complexes (PACs)	**Premature VENTRICULAR Complexes (PVCs)**
Rhythm	Early beats *cause irregularity* to underlying rhythm	Early beats *cause irregularity* to underlying rhythm
Heart Rate	Usually within normal range (60-100), but depends on underlying rhythm	Usually within normal range (60-100), but depends on underlying rhythm
P wave	• Premature • Positive deflection *(upright)* • 1 before each QRS • ***Often differ in shape from sinus P waves***	• Usually absent • May appear after the QRS complex
PR interval	"NORMAL" *0.12 to 0.20 sec* *May be prolonged depending on how early the premature beat comes in*	None
QRS duration	**"NARROW"** *0.11 sec or less* *(unless abnormally conducted)*	**"WIDE & BIZARRE"** *0.12 sec or longer*

Sinus Rhythm with **PACs**



Sinus Rhythm with **PVCs**



- *Q: Why do these PVCs have different forms/shapes?*
- *A: Because they are coming from different ectopic points in the ventricle wall.*

- PAC image: https://afibemory.wordpress.com/2017/09/15/premature-atrial-contractions-pacs-and-atrial-fibrillation/
- PVC image: http://jantungoke.blogspot.com/2012/12/premature-ventricular-contraction-pvc.html

"Why do ventricular complexes *(PVCs and ventricular rhythms)* have different forms/shapes?"

VENTRICULAR COMPLEXES: MORPHOLOGY AND UNIFORMITY

- These terms refer to the shape and amplitude (think "height") of the QRS complex.
- When the electrical impulse originates from the same place in the ventricle, it produces QRS complexes that have the same shape and amplitude.
- Electrical impulses originating from different foci (places) in the ventricle will display a different QRS shape and amplitude.

  - ***Monomorphic and Uniform***
    - PVCs with same shape and amplitude are referred to as **Uniform PVCs.**
    - Ventricular Tachycardia with QRS complexes that have same shape and amplitude are called **Monomorphic VT.**

  - ***Polymorphic and Multiform***
    - PVCs with a different shape and amplitude are referred to as **Multiform PVCs.**
    - Ventricular Tachycardia with QRS complexes that have a different shape and amplitude is called **Polymorphic VT.**

CORE CONCEPTS: ECG 4

Antidysrhythmic Meds

"Flashcards"

Because beta$_1$ receptors are coupled to calcium channels in the heart,

beta blockers
like propranolol

and

calcium channel blockers
like diltiazem and verapamil

have an identical effect on the heart.

CCBs: end in -DIPINE block calcium channels in **ARTERIES**	**arterial vasoDILATION**
CCBs: diltiazem and verapamil block calcium channels in **ARTERIES** –AND– block calcium channels in **HEART**	**arterial vasoDILATION** –AND– *Heart:* ↓ HR ↓ AV conduction ↓ contractility

BETA BLOCKERS: end in -LOL β_1 therapeutic benefit comes from blocking **beta$_1$ receptors in heart**	*Heart:* ↓ HR ↓ AV conduction ↓ contractility
DIGOXIN: • inhibits a key enzyme: *Na+,K+ ATPase* • narrow therapeutic range • K+ ions compete with digoxin to bond with this enzyme, so **abnormal K+ levels can cause digoxin to become toxic or subtherapeutic.** (see Lehne textbook, p. 523)	**positive inotrope** *(↑ contractility)*

atropine (ANTIcholinergic) blocks parasympathetic ("rest & digest") system	↑ HR ↑ AV conduction
adrenergic agonists cathecholamines (e____, n____, d____) activates sympathetic ("fight or flight") system	β_1 ↑ HR ↑ AV conduction ↑ contractility α_1 **vasoCONSTRICTION** • *veins* • *arterioles in intestines, mucous membranes, skin*

E.N.D. = epinephrine, norepinephrine, dopamine

amiodarone • **blocks K+ channels** • **delays repolarization** • highly effective but has serious toxicities *(lung damage, visual impairments)* • very long half-life (1-4 months) so when toxicity occurs, it's not quickly fixed by stopping med. • As a result, FDA has officially approved ("on-label") for ventricular dysrhythmias only. • However, commonly used "off-label" for atrial dysrhythmias in the hospital. (see Lehne textbook, p. 546)	↓ HR ↓ conduction of impulse through AV junction, bundle branches, Purkinje fibers ↓ contractility

CORE CONCEPTS: ECG 5

Nonpharmacological Interventions

Cardiac Catheterization Lab Interventions

Intervention	Definition	Lewis reading
Ablation	**Radiofrequency catheter ablation therapy** uses electrical energy to "burn" or ablate areas of the conduction system. An electrode-tipped ablation catheter "burns" ectopic sites or abnormal pathways in the atria, AV node, and ventricles. It is the nonpharmacologic treatment of choice for some atrial dysrhythmias	*p. 773*
Balloon Angioplasty	During cardiac catheterization, a catheter with a deflated balloon tip is inserted into the blocked coronary artery. The deflated balloon is positioned inside the blockage and inflated. This compresses the plaque against the artery wall, resulting in vessel dilation and a larger vessel diameter.	*p. 715*
Stent Placement	A balloon angioplasty can be used to place a permanent intracoronary stent to keep vessel open.	*p. 715 and Fig. 33.6*

PACEMAKERS

TEMPORARY (nurse has direct role in helping manage settings):

	INDICATIONS	KEY POINTS
Transcutaneous Pacemaker	★ **Emergently** restore adequate heart **rate** and **rhythm**	• Temporary • Electrical energy delivered by pads attached to skin • PAINFUL! • Use lowest possible voltage to get capture • Provide analgesia and/or sedation when possible
Transvenous Pacemaker	★ Emergently restore adequate heart **rate** and **rhythm** *-or-* ★ Maintained prophylactically after open heart surgery	• Temporary • Used in emergency departments or ICUs in emergency situations • **Can be a bridge to implantation of permanent pacemaker**

IMPLANTED:

Permanent Pacemaker (PPM)	★ Sinus node dysfunction = **bradycardia** ★ AV node dysfunction = **heart blocks** ★ **Heart failure**	• Permanent • Implanted totally within the body • Used to pace the heart when the normal conduction pathway is not functioning appropriately • Most pacemakers are ***demand*** pacemakers, meaning that they only fire a signal when the HR drops below a certain rate

Implantable Cardioverter-Defibrillator	★ Patients with history of **sudden cardiac arrest**
	★ Patients with spontaneous sustained VT
	★ Syncope with VT
	★ Cardiomyopathy = high risk for life-threatening dysrhythmias
		• Also capable of pacing if heart rate falls below programmed lower rate
		• ICD implantation and shocking can cause a variety of emotions for the patient including:
		  ○ *Fear of shock*
		  ○ *Fear of recurrent dysrhythmia*
		  ○ *Body image concerns*
		  ○ *Expectation of pain during shock*

Vagal Maneuvers to Slow Heart Rate

SOURCE: https://www.webmd.com/heart-disease/atrial-fibrillation/vagal-maneuvers-and-heart-rate

1. **Valsalva maneuver.** Hold your nose, close your mouth, and try to blow the air out. This creates pressure in your chest that may activate the vagus nerve. Sitting or squatting may help. Try it for 10 seconds.
    - *Note: Also can be stimulated by instructing patient to bear down as if having a bowel movement*
2. **Cough.** You need to cough hard to generate pressure in your chest and stimulate the vagus nerve. Children with tachycardia may not be able to cough hard enough to get a response from the vagus nerve.
3. **Hold your knees against your chest**: Do it for a minute. This may work best for babies and children.
4. **Cold water treatment.** You might hear this called the diving reflex. You may need to put a plastic bag of ice on your face for 15 seconds. Or you can immerse your face in icy cold water for several seconds. It might also work to step into a cold shower or a cold bath.
5. **Carotid sinus massage**: Only a physician should perform this one: Lie down and stick out your chin. The MD will put pressure on your carotid sinus, a bundle of nerves surrounding the carotid artery in your neck just below your jaw. You'll be monitored during the procedure.