Premature Atrial complexes. (PAC)

Causes: ① ischemia ② excess adrenergic ③ Alcohol ④ tobacco ⑤ electrolyte imbalance ⑥ hypoxia ⑦ infection

- This early beat arises within the atria, firing on its own.
- On E CG, look for early P waves that differ in morphology from the normal sinus P wave (because these P waves originate within the atria and not the sinus node).
- may cause palpitation or give rise PSVT
- asymptomatic (usually), don't need treatment Monitor for increased frequency. If symptomatic (e.g., palpitations), β-blockers may be helpful.

PVC

This early beat fires on its own from a focus in the ventricle and then spreads to the other ventricle

occur without structural heart dz

causes: ① hypoxia ② electrolytes imb. ③ medication ④ structural heart dz ⑤ caffeine ⑥ stimulants

Since conduction is not through normal conduction pathways, but rather through ventricular muscle, it is slower than normal, causing a wide QRS

Wide, bizarre QRS complexes followed by a compensatory pause are seen;a P wave is not usually seen because it is "buried" within the wide QRS complex.

PVCs appear in more than 50% of men who undergo 24-hour Holter monitoring.
6. Most patients are asymptomatic. Some patients may have palpitations and dizziness related to PVCs. If symptomatic, β-blockers may be used.
7. Presence of PVCs in patients with normal hearts is associated with increased mortality.
8. If a patient is found to have frequent PVCs, workup forund erlying structural heart disease should be initiated which may require specific treatment.
9. Patients with frequent,repetitive PVCs and underlying heart disease are at increased risk for sudden death due to cardiac arrhythmia (especially VFib). Order an electrophysiologic study because patients may benefit from an ICD or ablation.

Aldosterone antagonists (spironolactone, eplerenone)
Indicated for HFrEF with EF <35%
Improve all-cause mortality, CV mortality, and hospitalizations
Can cause hyperkalemia, hence BMP should be monitored (do not start if creatinine >2.5 in men or 2 in women)

A.fib:-

No effective contraction → ↓ diastolic filling

around the Pul. v.

Multiple foci in the atria fire continuously in a chaotic pattern ← irr rapid vent rate, atria quiver continuously

A.fib + underlying heart dz → ↑ risk thromboembolism and hemodynamic compromise

Atrial rate is over 400 bpm,but most impulses are blocked at the AV node so ventricular rate ranges between 75 and 175.

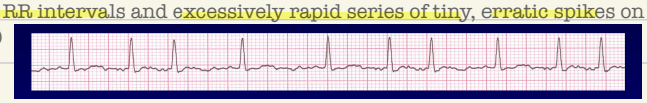
left atrial appendage → ↑ stasis → Embolize → ↑ thrombosis
- MCA stroke
- mesenteric A. → Acute mesentric ischemia
- Renal A. → Renal ischemia → AKI

① heart dz (CAD, MS, HTN, mitral stenosis)
② Pulmonary dz (PE, COPD, Lung Cancer)
③ Hx of cardiac surgery
④ Hyper thyroidism
⑤ Systemic illness (sepsis, malignancy)
⑥ stress (Post-operative, pain)
⑦ Pericarditis
⑧ Hyperadrenergic state, Pheochromocytoma, Cocaine, methamphetamine use
⑨ Excess alcohol use
⑩ Sedentary lifestyle / excess exercise

Clinical feature → ① fatigue and exertional dyspnea
② Palpitation, dizziness, angina, syncope
③ irregularly irr pulse
④ blood stasis (secondary to ineffective contraction) leads to formation of intramural thrombi (often in the left atrial appendage), which can embolize to the brain, causing ischemic stroke

Dx:- ECG findings: Irregularly irregular rhythm (irregular RR intervals and excessively rapid series of tiny, erratic spikes on ECG with a wavy baseline and no identifiable P waves)

(Fibrillatory wave)



- BMP
  - Serum electrolytes (Na+, K+, Mg2+, and Ca2+): to identify electrolyte imbalances
- TSH, fT4: to screen for hyperthyroidism

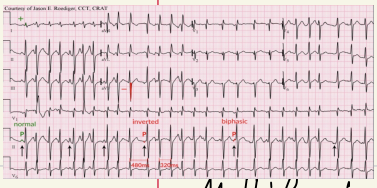
- Atrial fibrillation is a supraventricular arrhythmia.
- The exact mechanisms of Afib are not well understood. Suggested mechanisms include:
  - atrial hypertrophy and/or dilatation
  - Atrial ischemia
  - Inflammation
  1. Afib is triggered by one or both of the following
     - Bursts of electrical activity from automatic foci near the pulmonary veins or in diseased, fibrotic atrial tissue
     - Pre-excitation of the atria as a result of aberrant pathways (e.g., WPW syndrome)
  2. Afib is sustained by re-entry rhythms and/or rapid focal ectopic firing
     - Re-entry rhythms are more likely to occur with enlarged atria, diseased heart tissue, and/or aberrant pathways (e.g., WPW syndrome).
  3. Atrial remodeling
- Effects of Afib
  - The atria contract rapidly but ineffectively and in an uncoordinated fashion → stasis of blood within the atria → risk of thromboembolism and stroke
  - Irregular activation of the ventricles by conduction through the AV node → tachycardia

Atrial flutter:-

Pathophysiology
a. One irritable automaticity focus in the atria fires at about 250 to 350 bpm (typically very close to 300 bpm), giving rise to regular atrial contractions.
b. Atrial rate between 250 and 350, around 300 bpm. The long refractory periodin the AV node allows only one out of every two or three flutter waves to conduct to the ventricles.

Causes
a. Heart disease: Heart failure(most common association),rheumatic heart disease, CAD
b. COPD, other hypoxic pulmonary disease
c. Atrial septal defect(ASD)
d. Very similar risk factors to AFib

Diagnosis
1. ECG provides a saw-tooth baseline,with a QRS complex appearing after every second or third "tooth" (P wave). Saw-tooth flutter waves are best seen in the inferior leads (II, III, aVF)



Multifocal atrial tachycardia

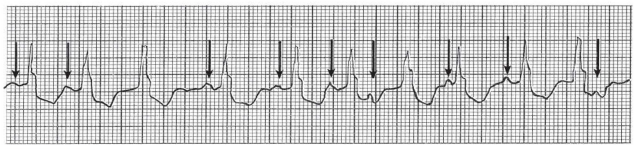


FIGURE 1.11 **ECG of multifocal atrial tachycardia with different P-wave configurations, varying PR intervals, and an irregular rate. Visible ectopic P waves are indicated by *arrows*.**

- Usually occurs in patients with severe pulmonary disease (e.g., COPD)
- ECG findings: Variable P-wave morphology and variable PR and RR intervals. At least three different P-wave morphologies are required to make an accurate diagnosis
- Can also be diagnosed by use of vagal maneuvers or adenosine to show AV block without disrupting the atrial tachycardia
- Treatment directed at the underlying disease, improving oxygenation and ventilation (strong association between MAT and lung disease). If left ventricular function is preserved, acceptable treatments include CCBs, β-blockers, digoxin, amiodarone, IV flecainide, and IV propafenone. If LV function is not preserved, use digoxin, diltiazem, or amiodarone. Electrical cardioversion is ineffective and should not be used.

Clinical features
- Palpitations
- Chest pain or discomfort
- Dyspnea
- Dizziness or presyncope
- Syncope
- Diaphoresis
- Symptom onset and resolution are typically abrupt, in contrast to sinus tachycardia (in which onset and resolution are more gradual).

Paroxysmal supraventricular tachycardia: pathophysiology

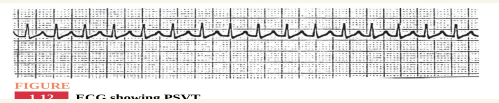


FIGURE 1.12 ECG showing PSVT

AV nodal reentrant tachycardia

Two pathways (one fast and the other slow) within the AV node, so the reentrant circuit is within the AV node
Most common cause of supraventricular tachyarrhythmia (SVT)
Initiated or terminated by PACs
ECG: Narrow QRS complexes with no discernible P waves (P waves are buried within the QRS complex). This is because the circuit is short and conduction is rapid, so impulses exit to activate atria and ventricles simultaneously. Can sometimes have retrograde P waves, or P waves which occur after QRS complex

In accordance with the electrical conducting system of the heart (SA node, atria, AV node, bundle of His, bundle branches, Purkinje fibers), tachycardias can be quickly separated into two categories based on the width of the QRS complex. Narrow QRS complexes suggest that the arrhythmia originates at or above the level of the AV node. Wide QRS complexes suggest that the arrhythmia originates outside of the normal conducting system or there is a supraventricular arrhythmia with coexisting abnormality in the His-Purkinje system.

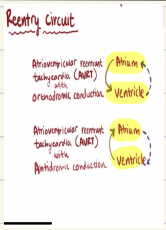
Orthodromic AV reentrant tachycardia

An accessory pathway between the atria and ventricles that conducts retrogradely
Called a "concealed bypass tract," and is a common cause of SVTs
Initiated or terminated by PACs or PVCs
ECG: Narrow QRS complexes with P waves which may or may not be discernible, depending on the rate. This is because the accessory pathway is at some distance from the AV node (reentrant circuit is longer), and there is a difference in the timing of activation of the atria and ventricles

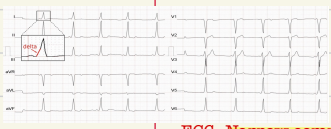
Wolff-Parkinson-White (WPW) syndrome

An accessory conduction pathway from atria to ventricles through the bundle of Kent causes premature ventricular excitation because it lacks the delay seen in the AV node. → paroxysmal tachycardia

Orthodromic reciprocating tachycardia (orthodromic AVRT)
The impulse travels through the AV node (anterograde limb) and depolarizes the ventricles. Then it travels back through the accessory pathway (the retrograde limb) and depolarizes the atria again, creating a reentry loop.
No delta waves because conduction occurs retrograde over the accessory pathway.



Supraventricular tachycardias (AFib or atrial flutter)
Usually, AV node only allows certain impulses to get to ventricles. With an accessory pathway, all or most of the impulses may pass to the ventricles. A fast ventricular rate may occur and cause hemodynamic compromise.



ECG: Narrow complex tachycardia ,a short PR interval (usually<100ms),and a delta wave (upward deflection seen before the QRS complex)

Vent. tach.

Always suspect VT in a patient with a wide (>0.12 second) QRS tachycardia.

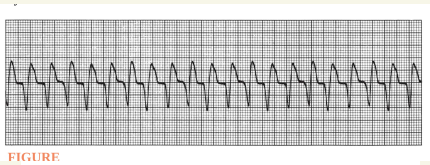
Etiology
1. CAD with perior MI (M/C)
2. Active ischemia, hypotension
3. Cardiomyopathies
4. Vent. scar tissue
5. congenital defect
6. Long QT syn. (torsade de pointes)
7. Drugs toxicity

sustained
1. >30 sec
2. symp.
3. marked hemodynamic compromised / development of myocardial ischemia
4. life threatening
5. Progress to V.fib if untreated

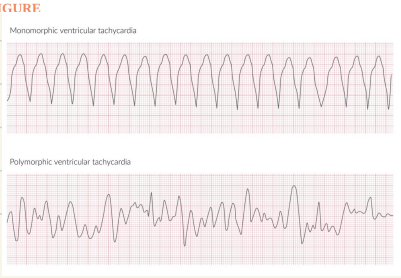
non-sustained
Brief, self-limited runs of VT
Usually asymptomatic
When CAD and LV dysfunction are present, it is an independent risk factor for sudden death. Therefore, patients with nonsustained VT should be thoroughly evaluated for underlying CAD and LV dysfunction.

It is important to distinguish monomorphic VT from SVT with aberrant conduction. Wide complex tachycardia in adults with a history of structural heart disease is much more likely to be due to VT than SVT with aberrancy.

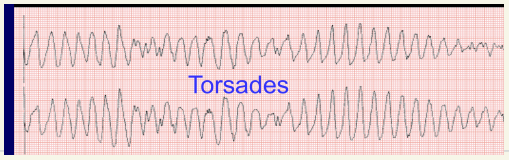
Sx: (1) Palpitations, dyspnea, lightheadedness, angina, impaired consciousness (syncope)
2. May present with sudden cardiac death
3. Signs of cardiogenic shock may be present
4. May be asymptomatic if rate is slow
5. Cannon A waves in the neck
6. S1 that varies intensity



FIGURE



Dx:
1. ECG: Wide and bizarre QRS complexes.
2. QRS complexes may be mono-or polymorphic.
a. In monomorphic VT, all QRS complexes are identical.
b. In polymorphic VT,the QRSc omplexes are different.
3. Unlike PSVT, VT does not respond to vagal maneuvers or adenosine.

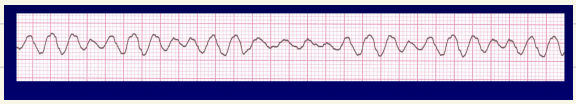


Torsades de pointes is a rapid polymorphic VT. It is a dangerous arrhythmia that often can lead to VFib.
It is associated with many factors that prolong the QT interval.
Risk factors for long QT include: hypokalemia, hypomagnesemia, hypocalcemia, drugs (antiemetics, antipsychotics, SSRIs, TCAs, macrolide and fluoroquinolone antibiotics, among others), and congenital long QT syndrome.
IV magnesium provides cardiac stabilization.
Electrical cardioversion for unstable patients. May require drugs to increase heart rate such as isoproterenol to shorten QT interval while underlying cause is addressed.
Address the underlying cause.

V.fib

Underlying cardiovascular disease
- Most common: coronary artery disease
- previous myocardial infarction, myocarditis, cardiomyopathy; congestive heart failure

Electrophysiologic disorders
- Wolff-Parkinson-White syndrome
- Long-QT syndrome → torsade de pointes



Sx
1. Chest pain
2. Palpitation
3. Shortness of breath
4. Dizziness

Can't measure BP

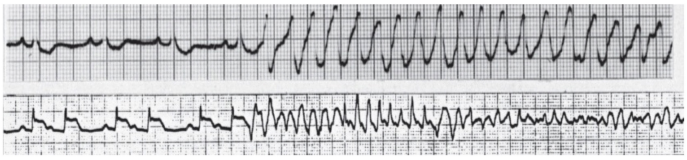
- Ultimately: loss of consciousness, death

Dx:

① Ventricular fibrillation
- Commonly preceded by ventricular tachycardia
- General appearance
  - Arrhythmic, fibrillatory baseline; > 300 bpm
  - indiscernible QRS complexes
  - No atrial P waves

Narrow complex tachycardias originate above ventricles. Wide complex tachycardias originate within ventricles and are more ominous because they are more likely to progress to VFib.

② Evaluation of underlying conditions
- ECG
- Laboratory
  - Cardiac enzymes
  - Electrolytes
  - TSH
  - Drug levels and toxicology screen
- Imaging
  - Coronary angiography
  - Echocardiography



FIGURE

Most common ECG abnormalities

Condition	Most relevant ECG findings	Most important clinical features
Myocardial infarction		

STEMI	• In early stages of ischemia
	  ○ Hyperacute T wave (peaked T wave) (without ST elevations)
	  ○ ST-segment elevations in
	  ■ Anteroseptal: V1–V2
	  ■ Anteroapical: V3–V4
	  ■ Anterolateral: V5–V6
	  ■ Lateral: I, aVL
	  ■ Inferior: II, III, aVF
	  ■ Posterior: V7–V9
	• Intermediate stage
	  ○ Absence of R wave
	  ○ T-wave inversion
	  ○ Pathological Q wave
		• Acute retrosternal chest pain
		  ○ Typically dull, squeezing
		  ○ Commonly radiates to left chest, arm, shoulder, neck, jaw, and/or epigastrium
		  ○ Precipitated by exertion or stress
		• Dyspnea (especially with exertion)
		• Nausea, vomiting
		• Diaphoresis, anxiety
		• Dizziness, lightheadedness, syncope
		• New heart murmur on auscultation (e.g., new S4)

Supraventricular tachycardia		
Atrioventricular nodal reentrant tachycardia	• Regular rhythm • Typically, narrow QRS complexes • Invisible P wave (it falls in or is "buried" in the QRS complex) • Heart rate typically 150–220/minute • ECG may be normal between episodes of tachycardia.	• Symptom onset and resolution are typically abrupt • Palpitations • Dyspnea • Dizziness or presyncope • Diaphoresis

Atrioventricular reciprocating tachycardia	• Regular rhythm
	• Heart rate 150–250/minute
	• Orthodromic AVRT
	  ○ Narrow QRS complex
	  ○ P wave typically follows QRS complex.
	• Antidromic AVRT
	  ○ Wide QRS complex
	  ○ Shortened PR interval	

Multifocal atrial tachycardia	• Heart rate 100–200/min • Irregularly irregular rhythm • P waves: ≥ 3 varying morphologies	
Paroxysmal atrial tachycardia	• Rhythm can be regular or irregular • Heart rate > 100 bpm • P wave with an unusual morphology (highly variable) before each normal QRS	

Wolff-Parkinson-White syndrome	• Regular rhythm
	• While in sinus rhythm, a preexcitation pattern may be present:
	  ○ Short PR interval
	  ○ ECG delta wave
	  ○ Widened QRS	

Ventricular tachycardia		

Torsades de pointes	• Regular rhythm
	• Heart rate typically ≥ 100/min
	• At least 3 consecutive wide QRS complexes (monomorphic VT or polymorphic VT)
	• Signs of AV dissociation
	  ○ Dissociated P waves
	  ○ Fusion complexes
	  ○ Capture beats
		• Often asymptomatic
		• Palpitations, syncope
		• Chest pain/pressure
		• Dyspnea, orthopnea
		• Dizziness
		• Hypotension
		• Cardiac arrest

Tachyarrhythmia		
Atrial fibrillation	• Irregularly irregular RR intervals • Commonly tachycardia (atrial rate > ventricular rate) • Indiscernible P waves • Typically narrow QRS complex (< 0.12 sec)	• Mostly asymptomatic • Less commonly, symptoms of arrhythmias (e.g., palpitations, dizziness, syncope)	• Tachycardia with an irregularly irregular pulse • Thromboembolic events (e.g., stroke/TIA)

Atrial flutter	• Heart rate: typically 75–150/minute (atrial rate ≥ ventricular rate)
	• The rhythm may be:
	  ○ Regularly irregular if atrial flutter occurs with a variable AV block occurring in a fixed pattern (2:1 or 4:1)
	  ○ Irregularly irregular with a variable block
	• Regular, narrow QRS complexes
	• Sawtooth appearance of P waves: identical flutter waves (F waves) that occur in sequence at a rate of ~ 300/minute
		• Tachycardia with a regular pulse

Ventricular fibrillation	• Commonly preceded by ventricular tachycardia (heart rate: > 300 bpm) • Arrhythmic, fibrillatory baseline • Indiscernible QRS complexes • Absent P waves	• Chest pain • Palpitation • Shortness of breath • Dizziness • Ultimately: loss of consciousness, death
AV block		
First degree	• Rate of SA node = heart rate • PR interval > 200 ms • No interruption in atrial to ventricular conduction	• Mostly asymptomatic, especially with first-degree and Mobitz type I blocks • Fatigue • Dizziness • Syncope
Second degree — Mobitz type I (Wenckebach)	• Mostly regular rhythm separated by short pauses, which may lead to bradycardia (regularly irregular rhythm) • Rate of SA node > heart rate • Progressive lengthening of the PR interval until a beat is dropped (a normal P wave is not followed by a QRS complex)	• Palpitations in the case of irregular rhythms (e.g., Mobitz I) • In third-degree: stokes-Adams attacks
Second degree — Mobitz type II	• Single or intermittent nonconducted P waves without QRS complexes • The PR interval remains constant. • The conduction of atrial impulses to the ventricles typically follows a regular pattern.	
Third degree	• Complete block with no conduction between the atria and ventricles • P waves and QRS complexes have their own regular rhythm but bear no relationship to each other (AV dissociation). • Sudden onset 3° AV block can result in ventricular asystole.	
Bundle branch block		
Left bundle branch block (LBBB)	• No R wave in lead V1 • Deep S waves (forming a characteristic W shape) • Wide, notched R waves in leads I, aVL, V5, V6 (forming a characteristic M shape)	• LBBB itself is asymptomatic. • Signs of the underlying condition (e.g., chest pain in MI)
Right bundle branch block (RBBB)	• An rsr′, rsR′, or rSR′ complex (forming a characteristic "rabbit ears" or M shape) in leads V1, V2 • Tall secondary R wave in lead V1 • Wide, slurred S wave in leads I, V5, V6	• RBBB itself is asymptomatic. • Signs of the underlying condition (e.g., cough in COPD)
Hereditary channelopathies		
Brugada syndrome	• Pseudo-RBBB with ST elevation in leads V1–V3	• Mostly asymptomatic • Syncope • Palpitation • Dizziness

Congenital long QT syndrome	• Long QT interval corrected for heart rate (QTc) interval
	  ○ Males: > 440 ms
	  ○ Females: > 460 ms
		• Mostly asymptomatic
		• Palpitations
		• Dizziness
		• Syncope
		• Cardiac arrest
		• Deafness in Jervell and Lange-Nielsen syndrome (not seen in Romano-Ward syndrome)

Unspecific changes		
Acute pericarditis	• Diffuse (saddle-shaped) ST-segment elevation • Diffuse PR-segment depressions • T-wave inversions	• Pleuritic chest pain • Low-grade intermittent fever, tachypnea, dyspnea, nonproductive cough • Pericardial friction rub
Cardiac tamponade	• Tachycardia • Low voltage QRS • Electrical alternans	• Beck triad (hypotension, muffled heart sounds, and distended neck veins) • Pulsus paradoxus • Pallor, cold sweats • Obstructive shock, cardiac arrest
Hypertrophic cardiomyopathy	• Signs of LVH • Nonspecific ST- and T-wave changes • Septal Q waves in inferior and lateral leads	• Frequently asymptomatic • Exertional dyspnea • Chest pain • Dizziness, lightheadedness, syncope • Palpitations • Sudden cardiac death (particularly during or after intense physical activity)
Restrictive cardiomyopathy	• Low voltage ECG (especially in amyloidosis) • LBBB and other conduction disorders	• Most common: dyspnea • Jugular venous distention • Peripheral edema, ascites • Hepatomegaly
Pulmonary embolism	• SIQIIITIII-pattern • New RBBB	• Dyspnea and tachypnea • Sudden pleuritic chest pain • Cough and hemoptysis • Possibly decreased breath sounds
Electrolyte imbalances — Hypokalemia	• Flattened T waves • ST depression • Presence of U waves	• Palpitations, irregular pulse, syncope • Muscle cramps, muscle weakness • Decreased deep tendon reflexes • Nausea, vomiting, constipation • Polyuria
Electrolyte imbalances — Hyperkalemia	• QRS complex widening • Peaked T waves • Widening and flattening of P wave	• Muscle weakness, paralysis, paresthesia • Decreased deep tendon reflexes • Nausea, vomiting, diarrhea
Electrolyte imbalances — Hypocalcemia	• Prolonged QT interval	• Tetany, spasms, and cramps (positive Chvostek sign, Trousseau sign) • Paresthesias • Seizure
Electrolyte imbalances — Hypercalcemia	• Shortened QT interval	• Nephrolithiasis, nephrocalcinosis • Bone pain, arthralgias, myalgias, fractures • Nausea and vomiting, constipation, anorexia • Peptic ulcer disease • Pancreatitis
Electrolyte imbalances — Hypomagnesemia	• Prolonged PR and QT intervals	• Anorexia, nausea, vomiting • Muscle weakness, muscle cramps/spasms • Tremor • Ataxia, nystagmus • Seizures
Atrial/ventricular enlargement		
Right atrial enlargement	• P pulmonale	• Signs of the underlying condition (e.g., cough in COPD)
Left atrial enlargement	• P mitrale	• Signs of the underlying condition (e.g., jugular venous distension in restrictive cardiomyopathy)
Left ventricular hypertrophy	• Sokolow-Lyon criteria: RV5 or RV6 + SV1 or SV2 ≥ 3.5 mV	• Signs of the underlying condition (e.g., syncope in aortic stenosis)
Right ventricular hypertrophy	• Right axis deviation • Dominant R wave in lead V1 (R wave > 0.6 mV or R/S > 1) • Sokolow-Lyon criteria: RV1 or R5 + SV5 or S6 ≥ 1.05 mV	• Signs of the underlying condition (e.g., cough in COPD)