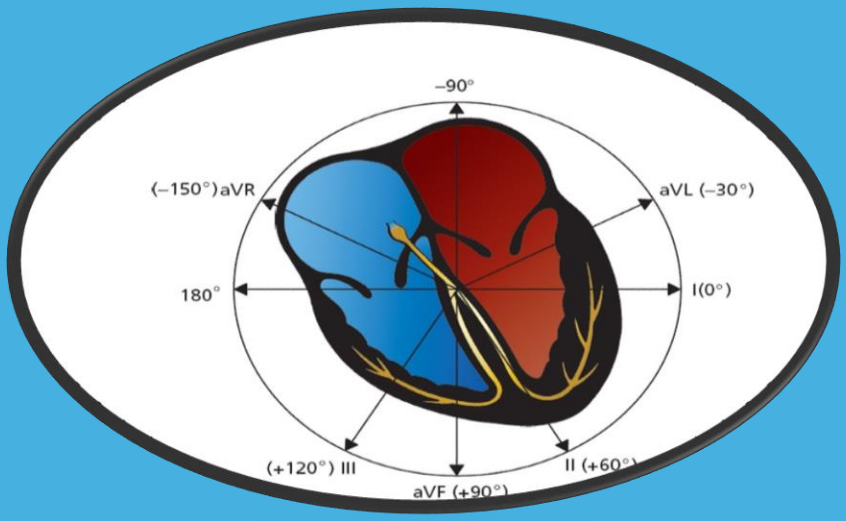
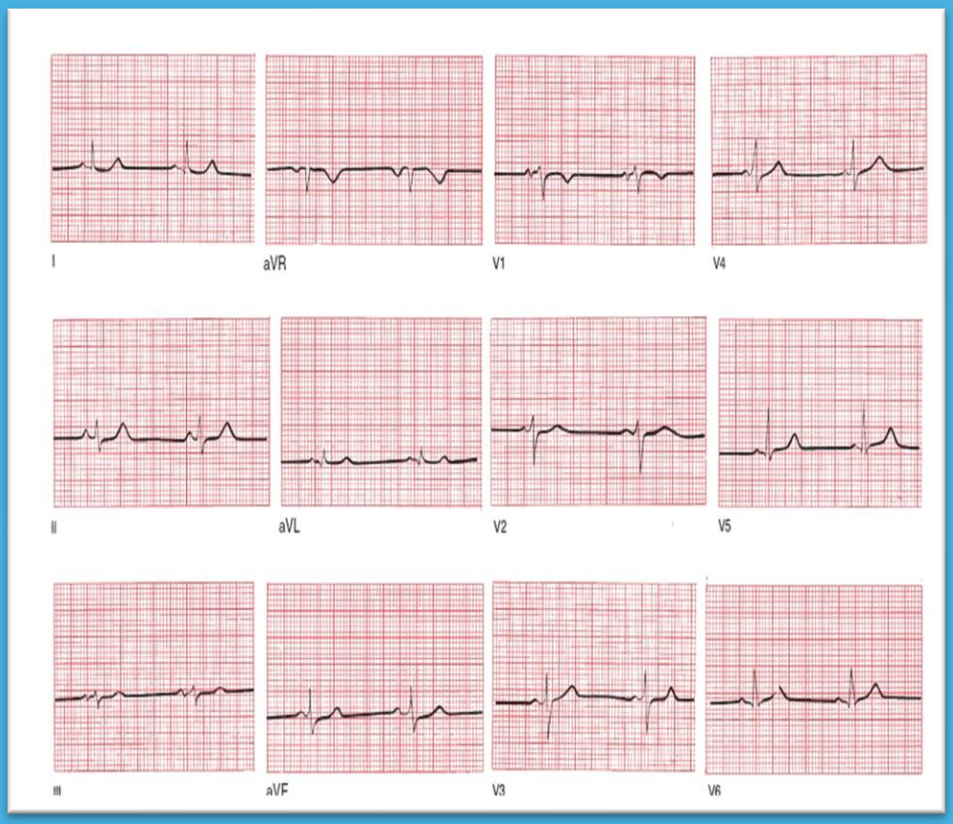
ECG CHANGES





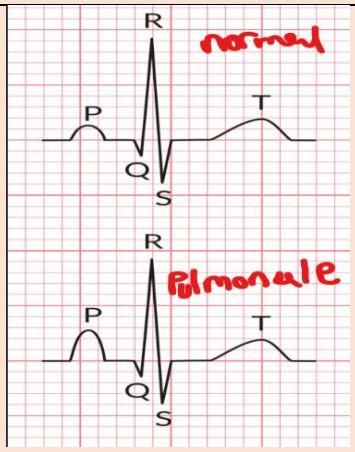
DONE by:
Noor Abu Hantash

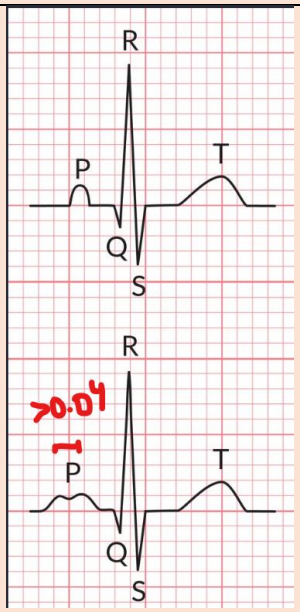
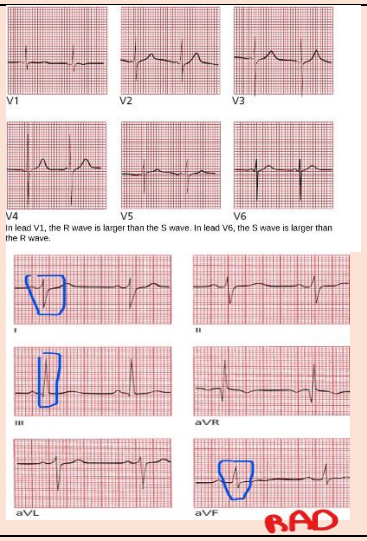
-Get familiar with the ECG in cover page, it is not just for design:>

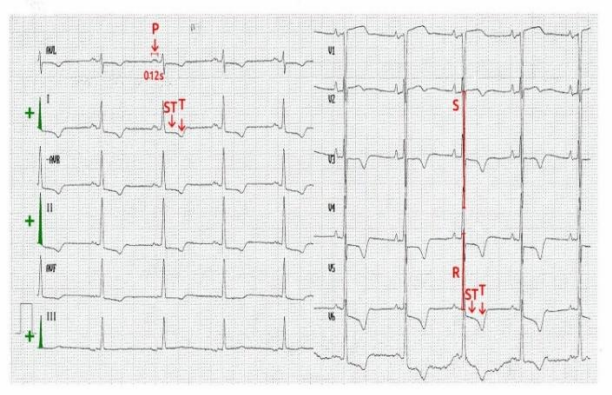
-normal ECG value from chatgpt

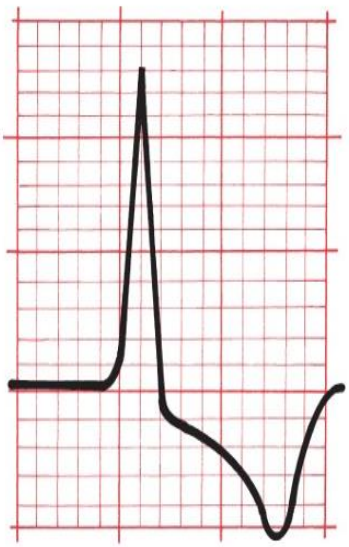
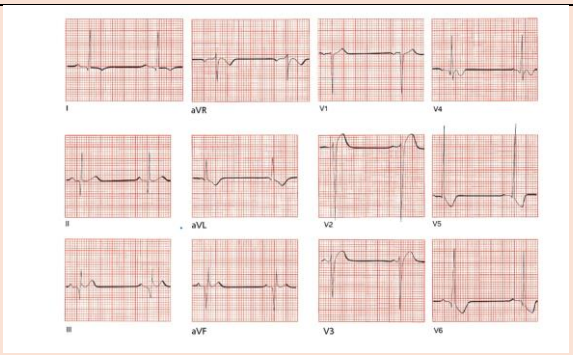
ECG Component	Duration (Seconds)	Amplitude (Voltage)
P Wave	0.08 - 0.10	≤ 2.5 mm (0.25 mV)
PR Interval	0.12 - 0.20	—
QRS Complex	0.06 - 0.10	5 - 30 mm (0.5 - 3.0 mV)
QT Interval	0.35 - 0.44 (varies with HR)	—
T Wave	0.10 - 0.25	≤ 10 mm (1.0 mV) in precordial leads
ST Segment	0.08 - 0.12	Isoelectric (± 0.1 mV deviation)

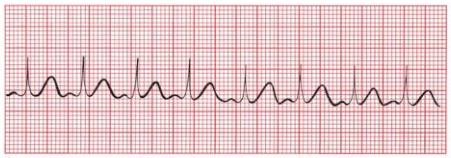
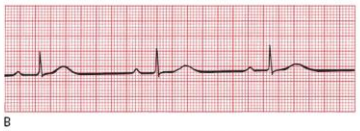
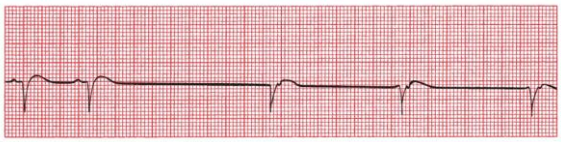
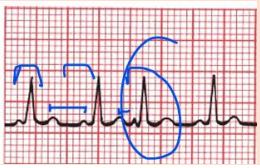
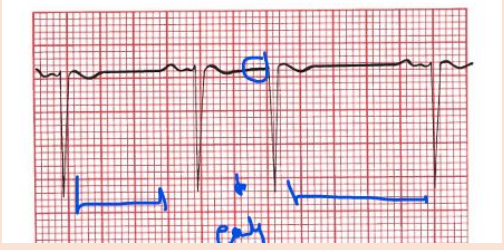
-now let's go through our big table, it is collected from only ekg book you will never need(mainly) , medstudy, amboss, FA, and Google, it will be helpful for revision or preparation for diving into ecg :

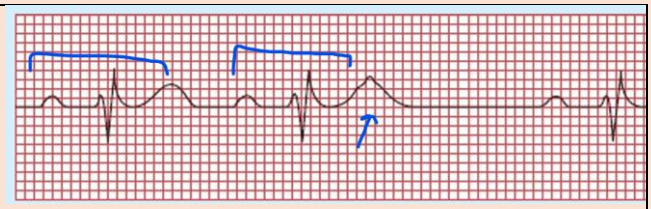
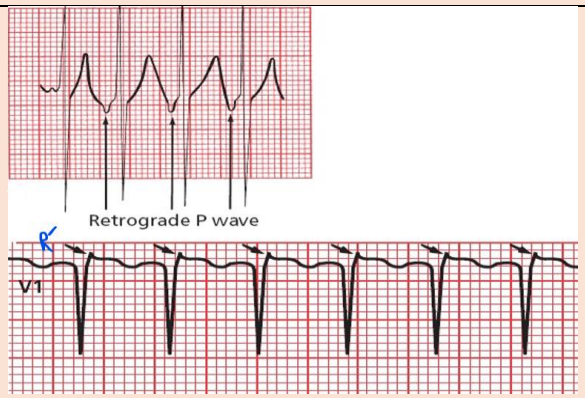
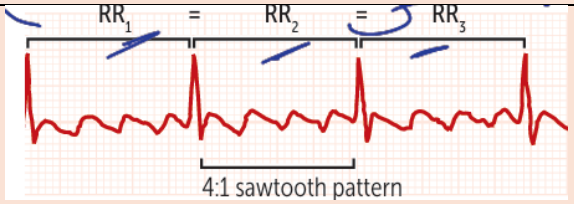
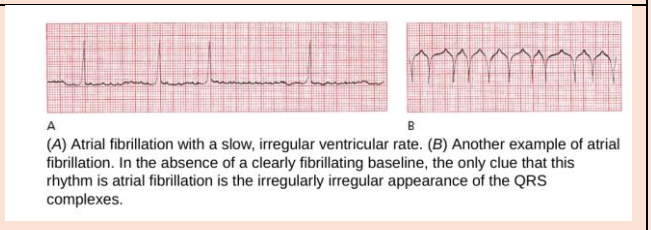
disorders	ECG findings	ECG illustration
RT Atrial enlargement	-p pulmonale, increased p wave amplitude exceeding 2.5 mm (0.25 mvolt) on inferior leads -no change in duration - possible rt axis deviation -best seen on lead 2, v1 -since v1 is biphasic, the 1st curve of p wave will seem larger in amplitude	

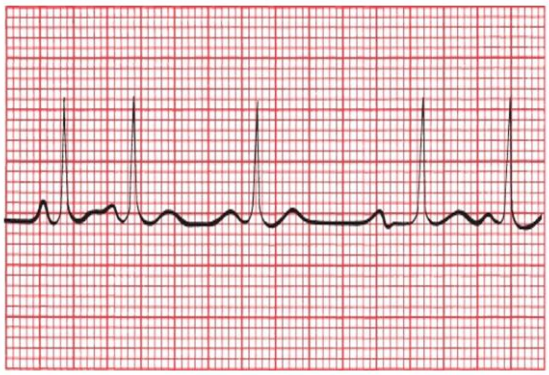
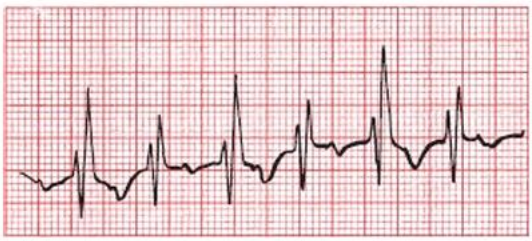
Lt atrial enlargement	-P mitrale -The amplitude of the terminal(negative) component of the P wave may be increased and must descend at least 1 mm below the isoelectric line in lead V1. -The duration is increased, and the terminal (negative) portion of the P wave must be at least 1 small block (0.04 second)in width. -No significant axis deviation is seen because the left atrium is normally Electrically dominant.	
Rt ventricular hypertrophy	-RAD, exceeding 100˚+ -The R wave is larger than the S wave in V1, whereas the S wave is larger than the R wave in V6.(disruption of R progression	

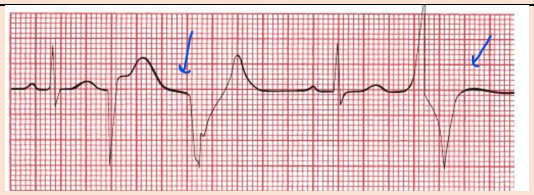
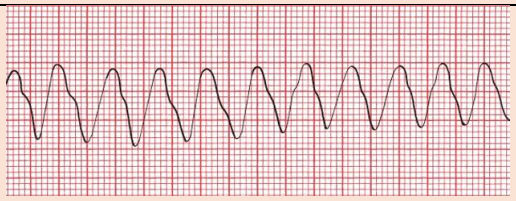
Lt ventricular hypertrophy	**-There should be increased R-wave amplitude in leads overlying the left ventricle and increased S-wave amplitude in leads overlying the right ventricle** **-**The most useful criteria are the following: 1. The R wave in V5 or V6 plus the S wave in V1 or V2 exceeds 35 mm. 2. The R wave in aVL is 11 mm. 3. The R wave in aVL plus the S wave in V3 exceeds 20 in women and 28 in men. 4. Left axis deviation exceeding −15° is also often present -if both ventricles are hypertrophied, there will be mix feature, with lt ventricular features predominance	 12-lead ECG (paper speed: 25 mm/s) – Heart rate: ~55/min – Regular sinus rhythm – Normal cardiac axis: positive (+) QRS complex polarity in leads I, II, and III – Broad and bifid P waves (P): referred to as "P mitrale" and suggestive of left atrial enlargement – S_{V2} (S) + R_{V5} (R) >3.5 mV: meets Sokolow-Lyon criteria for left ventricular hypertrophy – Left ventricular strain pattern: ST depression (ST) with T-wave inversion (T) in left-sided leads I and V4–V6 Positive Sokolow-Lyon criteria and left ventricular strain pattern are characteristic of left ventricular hypertrophy.

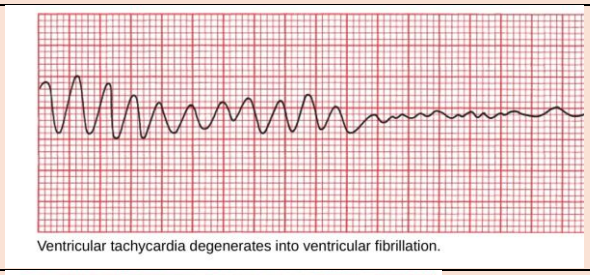
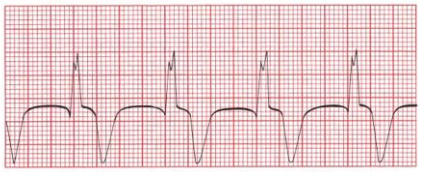
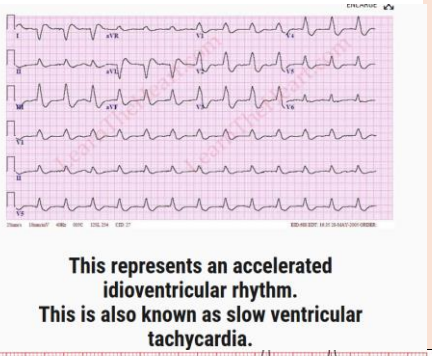
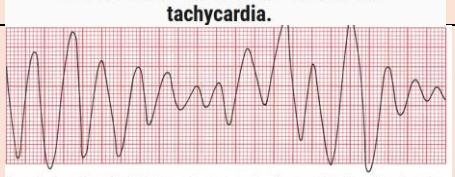
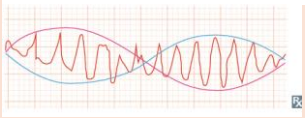
Secondary Repolarization Abnormalities of Ventricular Hypertrophy	asymmetric, T-wave inversion and downsloping ST-segment depression -right ventricular repolarization abnormalities will be seen in leads V1 and V2, and left ventricular repolarization abnormalities will be most evident in leads I, aVL, V5, and V6. - Left ventricular secondary repolarization abnormalities are far more common than right ventricular abnormalities.	 Note how the depressed ST segment and the inverted T wave appear to blend together to form a single asymmetric wave. The downward slope is gradual; the upward slope is abrupt.
HTN	Normal in most cases -echo done to check LVH	
HOCM, IHSS (idiopathic hypertrophic subaortic stenosis)	1. Ventricular hypertrophy 2. Repolarization abnormalities in those leads with the tallest R waves 3. Narrow, deep Q waves, of uncertain etiology, most often in the inferior and lateral leads	

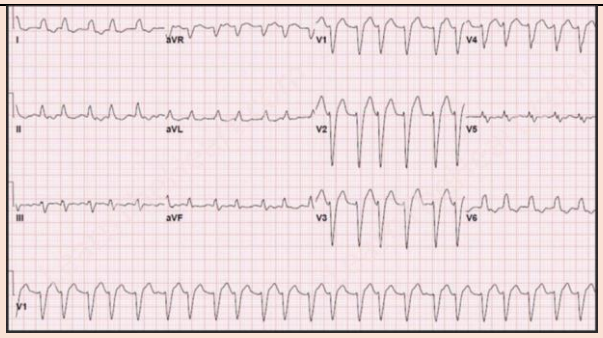
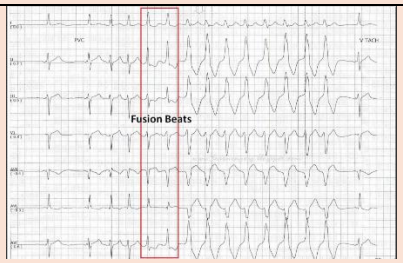
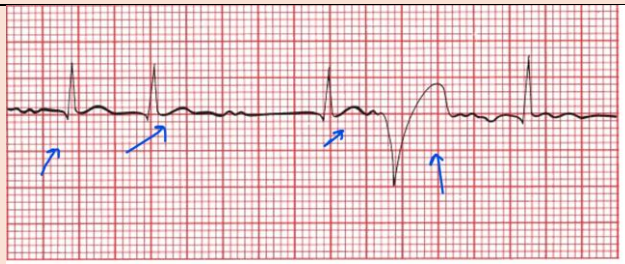
Sinus arrythmias	300divided by number of large boxes with Prescence of p wave ->300 sinus tachy <60 sinus brady	 A  B (*A*) Sinus tachycardia. Each beat is separated by two and one-half large squares for a rate of 120 beats per minute. (*B*) Sinus bradycardia. More than seven large squares separate each beat, and the rate is 40 to 45 beats per minute.
Junctional escape -av node is the pacemaker(40-60) in sinus arrest	Most often, there is no P wave at all. Occasionally, however, a retrograde P wave may be seen -may appear before, with(will be obscured), after QRS	 Junctional escape. The first two beats are normal sinus beats with a normal P wave preceding each QRS complex. There is then a long pause followed by a series of three junctional escape beats occurring at a rate of 40 to 45 beats per minute. Retrograde P waves can be seen buried in the early portion of the T waves (can you
Sinus arrest or sinus exit block	There is a failure of the sinus mechanism to deliver its current into the surrounding tissue (no depolarization in sa—arrest, no delivery—exit block)	
Atrial premature beats(PACs): P wave is present but abnormal contour and timing Junctional premature beat: P wave is absent	Like junctional escape beat, but it presents early in the beat rather than late after cessation	 

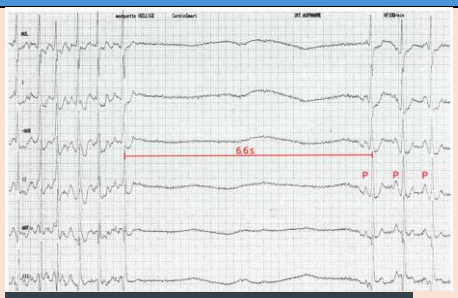
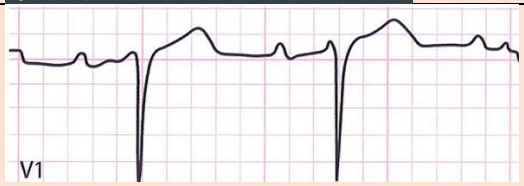
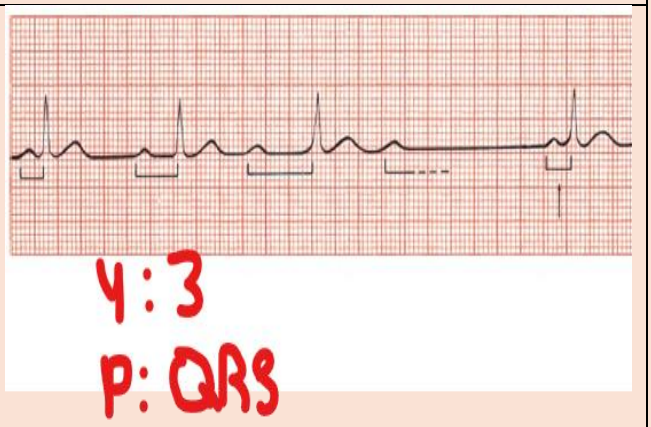
blocked atrial premature contraction	an atrial premature beat may occur sufficiently early that the AV node will not have recovered (i.e., repolarized) from the previous conducted beat and will therefore be unable to conduct the atrial premature beat into the ventricles	
AVNRT(prev: Paroxysmal SVT)	-regular rhythm -narrow QRS -150-250bpm -Retrograde P waves may sometimes be seen in leads II or III -lead V1→pseudo-R'	
Atrial flutter	-regular -P waves appear at a rate of 250 to 350 bpm -leads II and III→saw-toothed pattern. -2:1, 3:1, 4:1 AV blocks -clockwise(+ve flutter deflection in inf lead), counterclockwise(-ve def)	
Atrial fib	-irregularly irregular 120-180bpm -No true P waves can be seen. Instead, the baseline appears	 (*A*) Atrial fibrillation with a slow, irregular ventricular rate. (*B*) Another example of atrial fibrillation. In the absence of a clearly fibrillating baseline, the only clue that this rhythm is atrial fibrillation is the irregularly irregular appearance of the QRS complexes.

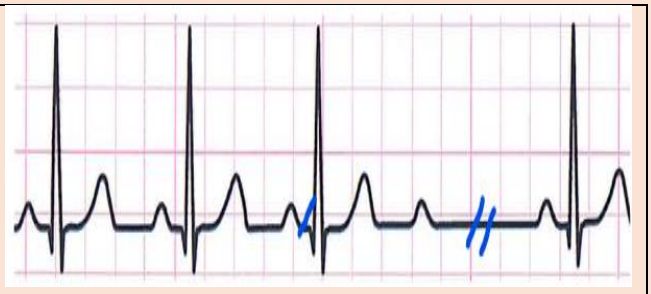
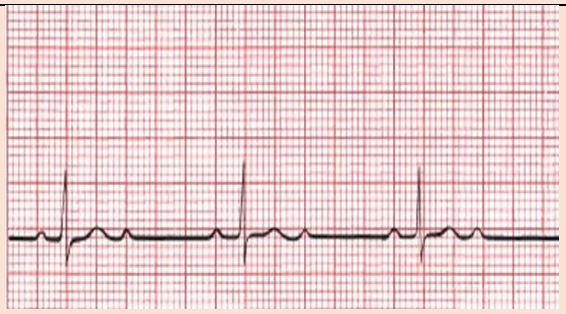
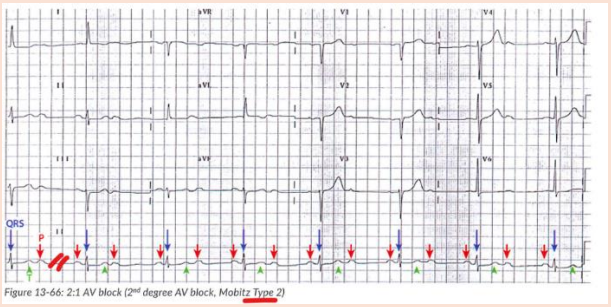
	flat or undulates slightly	
Multifocal Atrial Tachy(MAT)	-irregularly irregular -100-200bpm -Sometimes, the rate is less than 100 bpm→wandering atrial pacemaker.(not tachy) -The P waves, originating from multiple sites in the atria, will vary in shape, and the interval between the different P waves and the QRS complexes may vary as well. - In order to make the diagnosis of MAT, you need to identify at least three different P-wave morphologies	 Multifocal atrial tachycardia. Note that (1) the P waves vary dramatically in shape, (2) the PR intervals vary, and (3) the ventricular rate is irregular.
Paroxysmal Atrial Tachycardia (PAT)	-regular rhythm with a rate of 100 to 200 bpm -hard to differentiate it from AVNRT **-if you see a warm-up or cool-down period on the EKG, the rhythm is likely to be PAT. In addition, carotid massage can be very helpful: Carotid massage**	

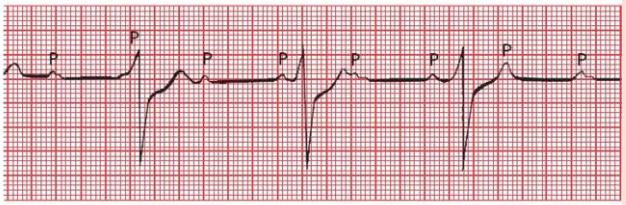
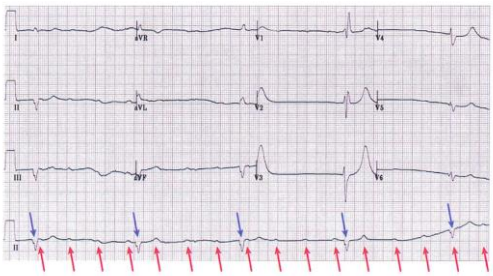
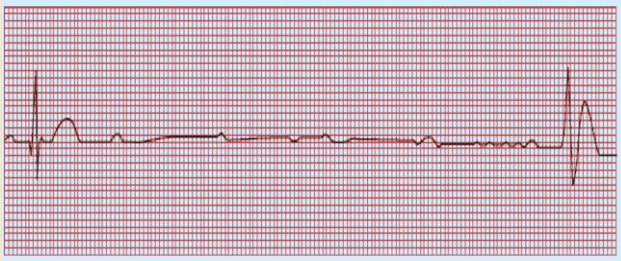
	will slow or terminate AVNRT, whereas it has virtually no effect on PAT	
Premature ventricular contraction (PVCs)	-Wide and bizarre QRS with at least 0.12s duration -A retrograde P wave may sometimes be seen, but it is more common to see no P wave at all. -A PVC is usually followed by a prolonged compensatory pause before the next beat appears. Less commonly, interpolated PVCs. -bigeminy 1:1, or Trigeminy 2(sinus beat):1(pvc) 3:1quadrigeminy -3 and more consecutive pvcs→NSVT (<30 s) if >30 s: sustained VTs=vts -R on T phenomenon -Multiform PVCs	
VTs	-run of three or more consecutive PVCs >30s -usually extreme axis deviation -120-200bpm -uniform (monomorphic), or polymorphic	

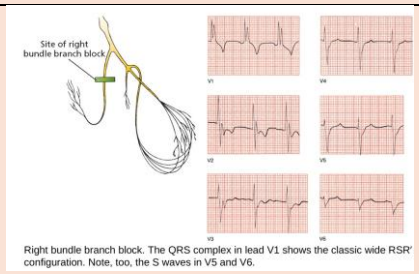
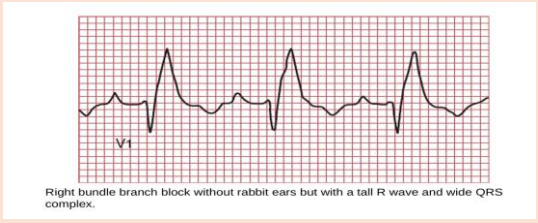
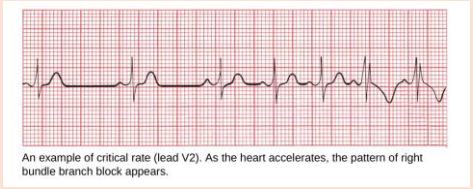
	(torsade de pointes) -superwide QRS (>160msec) -more QRSs than Ps opposite to AV blocks	
Ventricular fib	-coarse or fine -no true QRS -preterminal	 Ventricular tachycardia degenerates into ventricular fibrillation.
Accelerated Idioventricular Rhythm	-regular rhythm occurring at 50 to 100 bpm -< 50 bpm idioventricular without accelerated	 Accelerated idioventricular rhythm. There are no P waves, the QRS complexes are wide, and the rate is about 75 beats per minute.  **This represents an accelerated idioventricular rhythm. This is also known as slow ventricular tachycardia.**
Torsade de pointes	-ventricular tachycardia in patients with prolonged QT intervals -PVC falling during the elongated T wave can initiate torsade de pointes	 Torsade de pointes. The QRS complexes seem to spin around the baseline, changing their axis and amplitude. 

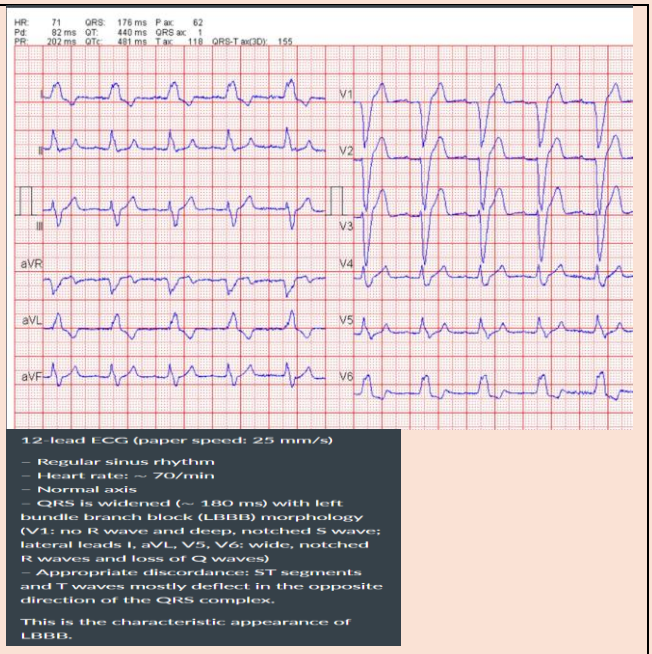
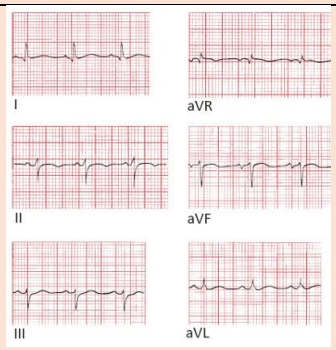
SVT with aberrancy	-Results in a wide QRS complex, as the depolarization of the ventricle happens more slowly (from myocyte to myocyte) rather than through rapidly conducting Purkinje fibers -SVT with aberrant conduction can have a very similar appearance to ventricular tachycardia.	
Fusion beat (capture beat) =normal beat, indicates ventricular origion	-may be seen in VT not AVNRT -supraventricular impulse and ectopic ventricular depolarization coincide at the ventricle, resulting in a complex with elements of both a regular QRS complex and a premature ventricular complex	
Ashman phenomenon	-wide QRS complex due to a physiologic, aberrantly conducted supraventricular beat in a patient with otherwise narrow QRS complexes. -Seen in supraventricular tachyarrhythmias,	 The Ashman phenomenon. The fourth beat looks like a PVC, but it could also be an aberrantly conducted supraventricular beat. Note the underlying atrial fibrillation, the short interval before the second beat, and the long interval before the third beat—all in all, a perfect substrate for the Ashman phenomenon.

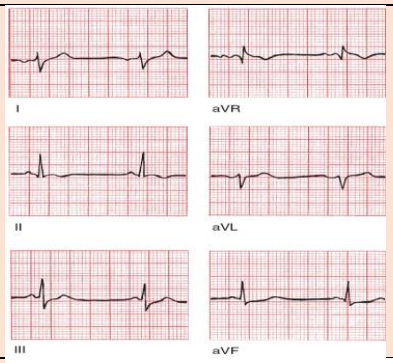
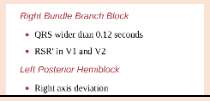
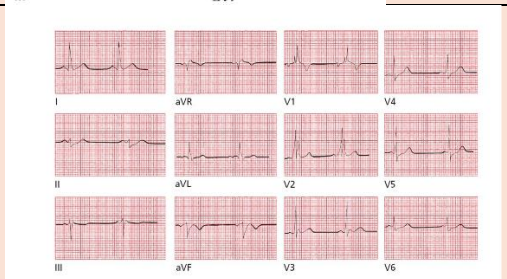
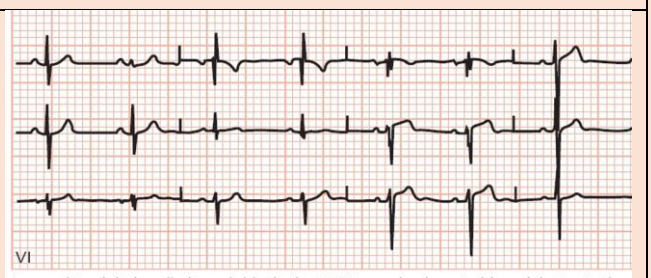
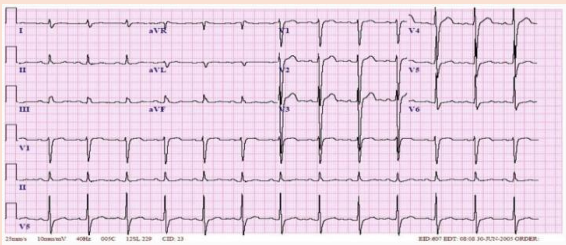
	especially atrial fibrillation similar morphological appearance to a premature ventricular complex, the mechanism is different	
sick sinus syndrome (bradytachycardia syndrome) =SA dysFx	bradyarrhythmias (e.g., sinus bradycardia, sinoatrial pauses >4s, blocks, and arrest) and/or tachyarrhythmias (e.g., Afib, SVT)	 The initial rhythm is atrial fibrillation (absent P waves and irregular RR intervals), at a rate of ~ 110/min. There is then a pause of ~ 6.6 s, followed by restoration of normal sinus rhythm at a rate of ~ 75/min (P waves present).
1st degree AV block (delay not block)	-PR interval>0.2 s -Every QRS complex is preceded by a single P wave	
Mobitz type I second-degree AV block (called Wenckebach block)	-progressive lengthening of the PR interval with each beat and then suddenly a P wave that is not followed by a QRS complex (a "dropped beat"). After this dropped beat, during which no QRS complex appears, the sequence repeats itself, over and over, and often with impressive regularity	

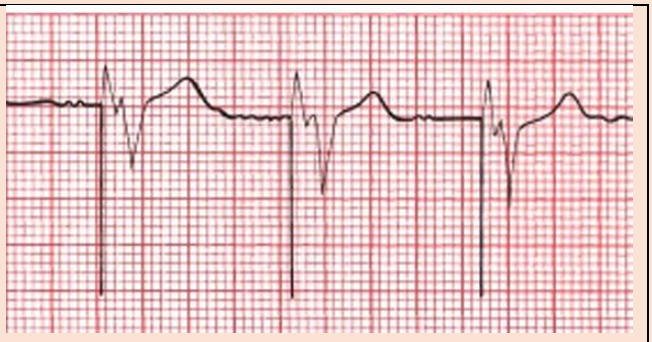
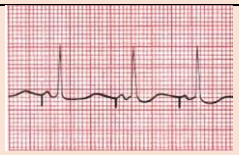
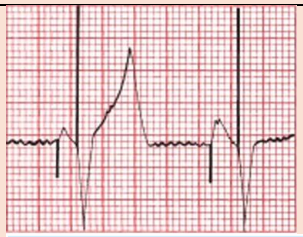
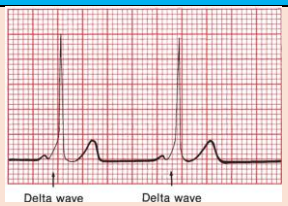
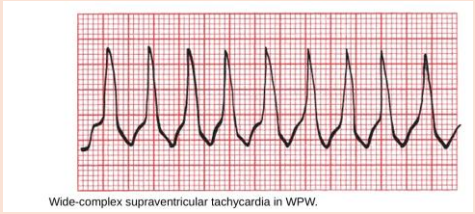
Mobitz type II second-degree AV block	-All-or-nothing conduction, in which QRS complexes are periodically dropped without prolongation of the PR interval -The ratio of conducted beats to nonconducted beats is rarely constant	
2:1 AV block	-impossible to tell whether it is due to Wenckebach block or Mobitz type II block. The distinction between these two types of second-degree heart block depends on whether or not there is progressive PR lengthening, but with a 2:1 ratio in which every other QRS complex is dropped, it is impossible to make this determination. -because you cannot see either the progressive lengthening of PR interval (Mobitz 1) or the fixed PR interval (Mobitz 2) until QRS drops. One way to begin to distinguish between them is to look at the QRS	  Figure 13-66: 2:1 AV block (2nd degree AV block, Mobitz Type 2)

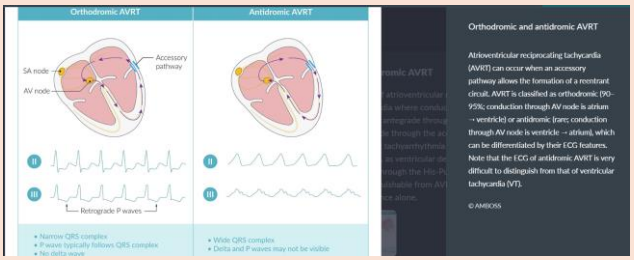
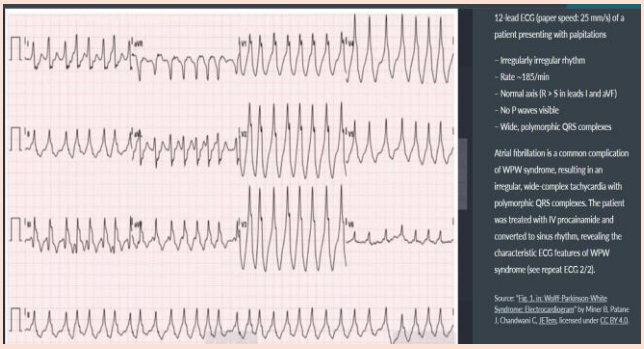
	; if narrow, then Mobitz 1 is more likely; if wide, then Mobitz 2 is more likely, but it is just confirmed Holter monitor, clinically and EPS	
3rd degree AV block	-complete heart block -P waves marching across the rhythm strip at their usual rate (60 to 100 waves per minute) but bearing no relationship to the QRS complexes that appear at a much slower escape rate -the location of escape determines the width of QRS -irregular A fib→regular with 3rd AV block(bad sign) -ventricular rate is slower than the sinus or atrial rate.	 Third-degree AV block. The P waves appear at regular intervals, as do the QRS complexes, but they have nothing to do with one another. The QRS complexes are wide, implying a ventricular origin.  igure 13-64: 3rd degree AV block (a.k.a. complete heart block) with junctional escape rhythm
Stokes–Adams attacks	-AV blocks! -third-degree heart block, there may be a delay (or even complete absence) in the appearance of a ventricular escape rhythm. The EKG will then show sinus beats (P waves) activating the atria with no	 This patient was in normal sinus rhythm (see the first complex) when he suddenly went into complete heart block. There is a long pause during which you can see nothing but P waves; no escape beats can be seen for several seconds. Finally, the first ventricular escape beat saves the day, but during the long pause, the patient experienced a Stokes–Adams attack.

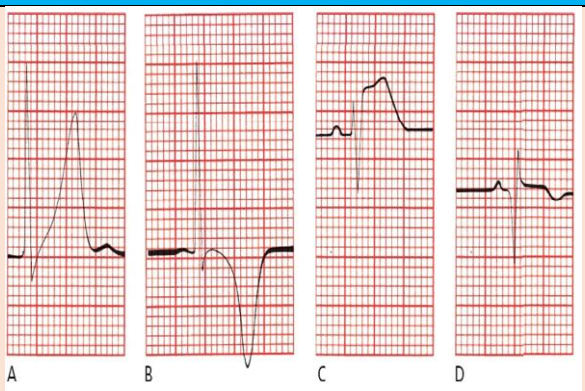
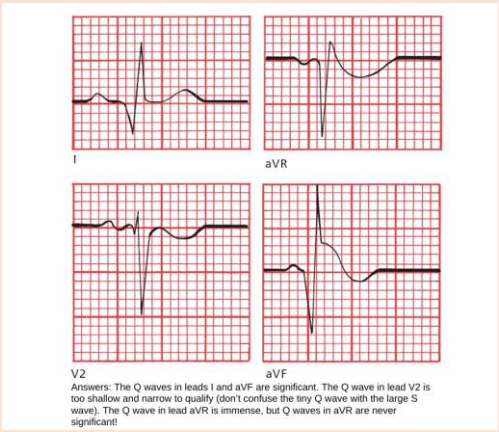
	ventricular activity at all for two or more beats before either normal AV conduction resumes or a ventricular escape rhythm finally appears. When there are 4 or more seconds without ventricular activity, the patient usually experiences a near or complete faint.	
RBBB	-**Bundle branch block is diagnosed by looking at the width and configuration of the QRS complexes.** -QRS complex widened to greater than 0.12 seconds -RSR' (rabbit ears) or a tall R wave in V1 and V2 with ST-segment depression and T-wave inversion - Reciprocal changes in V5, V6, I, and aVL(late deep S wave)	 Right bundle branch block. The QRS complex in lead V1 shows the classic wide RSR' configuration. Note, too, the S waves in V5 and V6.  Right bundle branch block without rabbit ears but with a tall R wave and wide QRS complex.  An example of critical rate (lead V2). As the heart accelerates, the pattern of right bundle branch block appears.

LBBB	-left axis deviation may be present -QRS complex widened to greater than 0.12 seconds. - Broad or notched R wave with prolonged upstroke in leads V5, V6, I, and aVL, with ST-segment depression and T-wave inversion. - Reciprocal changes in V1 and V2(deep S) Note: if bundle branch block is present. Specifically, right bundle branch block precludes the diagnosis of right ventricular hypertrophy, and left bundle branch block precludes the diagnosis of left ventricular hypertrophy. In addition, the diagnosis of a myocardial infarction can be extremely difficult in the presence of left bundle branch block	
Hemiblocks anterior lt fascicle block LAFB	-LAD(-30- -90): Lead1 +ve, avf -ve, lead2 -ve, after excluding other causes -normal QRS duration, no T, ST changes -more common	

Post lt fascicle block **LPFB**	-RAD(+90- 180) after exclusion -normal QRS duration, no T, ST changes	
bifascicular block (combination of right bundle branch block with either left anterior or left posterior hemiblock)	-combination of features of both hemiblock and right bundle branch block  	 This is an example of right bundle branch block combined with left anterior hemiblock.
Underachieved blocks	**-nonspecific intraventricular conduction delay (IVCD**) :occurs when there is QRS widening greater than 0.10 seconds without the other criteria for either bundle branch block or bifascicular block. **–An incomplete bundle branch block** occurs when the EKG tracing shows a left or right bundle branch appearance (e.g., rabbit ears in V1 in right bundle branch block), but the QRS duration is between 0.10 and 0.12 seconds	 Incomplete right bundle branch block; the QRS complex is not widened, but note the classic rabbit ears configuration in V1.  **ECG Findings:** 1. Normal Sinus Rhythm 2. Non-specific Interventricular Conduction Delay (IVCD)

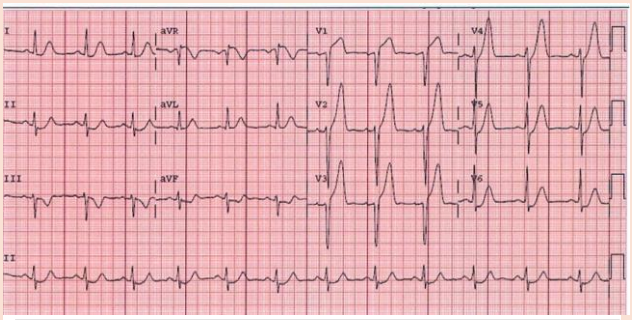
Ventricular pacemaker	-The ensuing QRS complex will be wide and bizarre, just like a PVC. -Because the electrodes are located in the right ventricle, the right ventricle will contract first and then the left ventricle. This generates a pattern identical to left bundle branch block, with delayed left ventricular activation. - A retrograde P wave may or may not be seen.	
Atrial pacemaker	spike followed by a P wave, a normal PR interval, and a normal QRS complex	
(Dual)sequential pacemaker	two spikes will be seen, one preceding a P wave and one preceding a wide, bizarre QRS complex	 In some patients, pacemaker spikes can be difficult to see on a standard EKG because their amplitude may be less than 1 mV. If you are examining an EKG from a patient unknown to you that demonstrates wide QRS complexes and left axis deviation, you must always suspect the presence of a pacemaker even if the tiny pacemaker spikes cannot be seen. Obviously, examination of the patient or—if the patient is lucid—a simple question or two will reveal the presence or absence of an electrical pacemaker.
WPW	-PR interval less than 0.12 seconds -QRS complex is widened to more than 0.1 second by the presence of what is called a delta wave -classic ecg+symptoms= WPW syndrome -no delta wave—not PWP—either due to small bypass	 Delta wave Delta wave  Wide-complex supraventricular tachycardia in WPW.

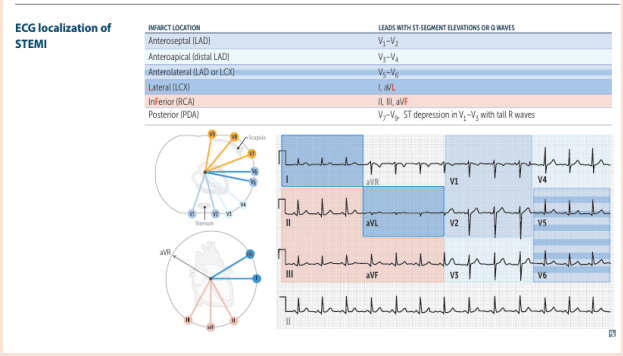
	pathway within or very close to the AV node, or an AV node that conducts more rapidly than normal. -Patients with a short PR interval without delta waves and who have had at least one tachyarrhythmia are said to have Lown–Ganong Levine syndrome -The two tachyarrhythmias most often seen in WPW are a supraventricular tachycardia and atrial fibrillation -supraventricular mechanism: AV(atrioventricular) reciprocating tachycardia (AVRT): *orthodromic tachycardia: tachycardia activates the ventricles in an antegrade manner through the AV node, generating a narrow QRS complex *antidromic tachycardia: Reciprocating tachycardias that activate the ventricles through the accessory pathway, generating a wide QRS complex -atrial fib: *very rapid and rarely can lead to ventricular fib *300bpm!	 

	-cant determine axis	
STEMI	- T-wave peaking (hyperacute T wave, its length equals or exceeds the QRS) followed by T-wave inversion few hours later(A and B). *T inversion is reversile unless true infarction happens, it persists for months to years * One helpful diagnostic feature is that the T waves of MI are inverted symmetrically, whereas in most other circumstances they are asymmetric, with a gentle downslope and rapid upslope *think about pseudonormalizatio n and persistent juvenile T-wave pattern, isolated inverted T in lead III, and normal inverted T in aVR -ST-segment elevation compared to isoelectric TP segment (C), it turns normal within hours. Persistent ST-segment elevation often indicates the formation of a ventricular aneurysm, a	 

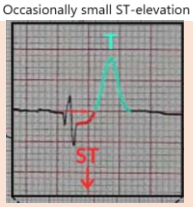
weakening and bulging out of the ventricular wall.
*make sure it isn't J point elevation

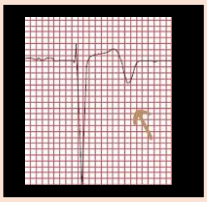
- The appearance of new Q waves (D)
*The Q wave must be greater than 0.04 seconds in duration. &. The depth of the Q wave must be at least 25% the height of the R wave in the same QRS complex.
*in 2 contagious leads
*isolated deep Q is normal in lead III
*Q waves in aVR are not significant.
-reciprocal changes in distant leads
-the amplitude of the R wave should normally exceed that of the S wave by lead V4. This pattern may vanish with anterior infarction, and the result is called poor R wave progression. One simple criterion for the diagnosis of poor R-wave progression is if the R wave in lead V3 is not larger than 3 mV(not specific)
-T waves types in LAD occlusion: in table beside
-the presence of an R wave of greater amplitude than the corresponding S wave in lead V1 is



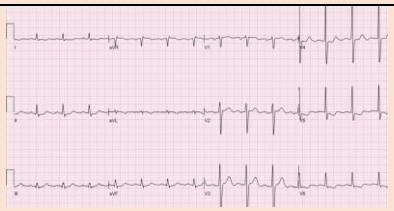
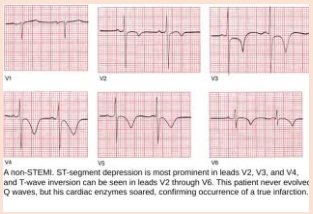
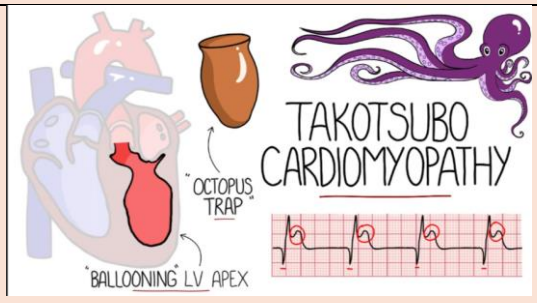


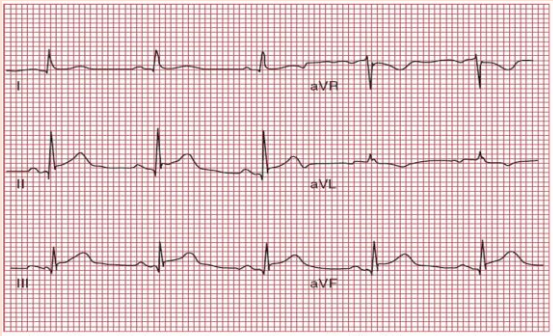
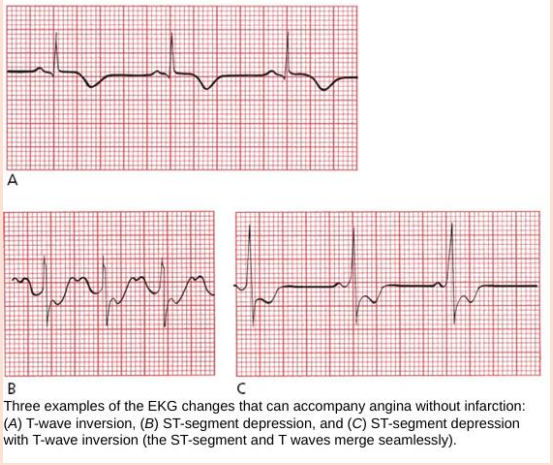
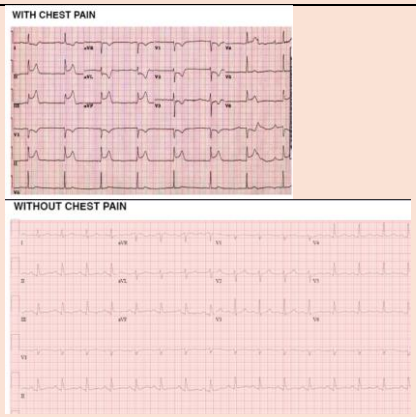
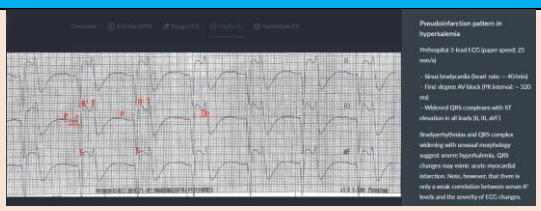
Feature	de Winter Syndrome	Wellens Syndrome
T-wave pattern	Prominent, symmetrical, **hyperacute T-waves** in precordial leads with a characteristic **upsloping ST-segment depression** in V1-V6.	Deeply **inverted T-waves** or **biphasic T-waves**, especially in V2-V3.
ST-segment	**Upsloping ST-segment depression** in precordial leads, often with no ST-elevation. Occasionally small ST-elevation in aVR.	ST-segment is typically **isoelectric** or shows minimal elevation.

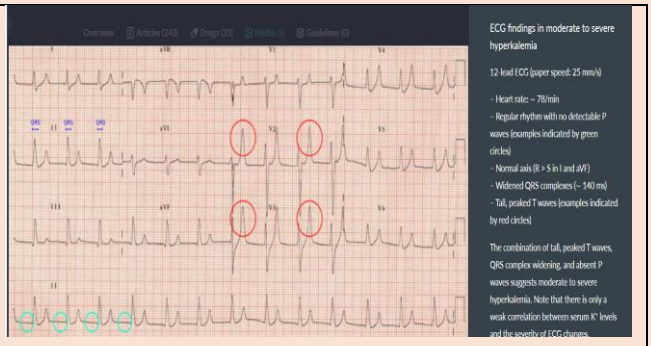
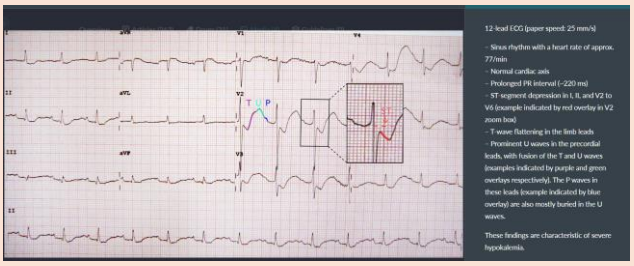
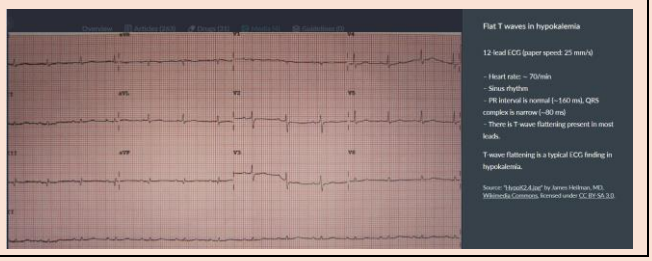


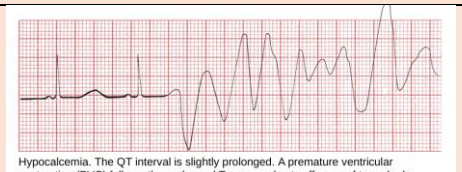
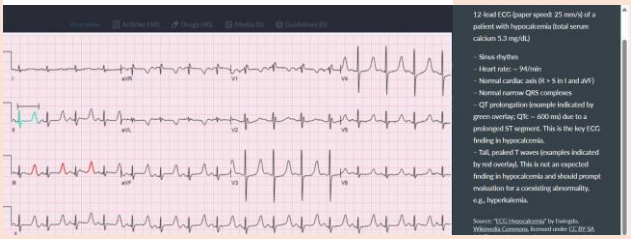
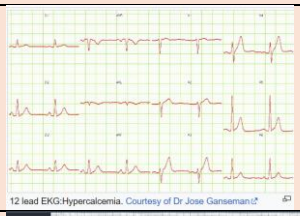
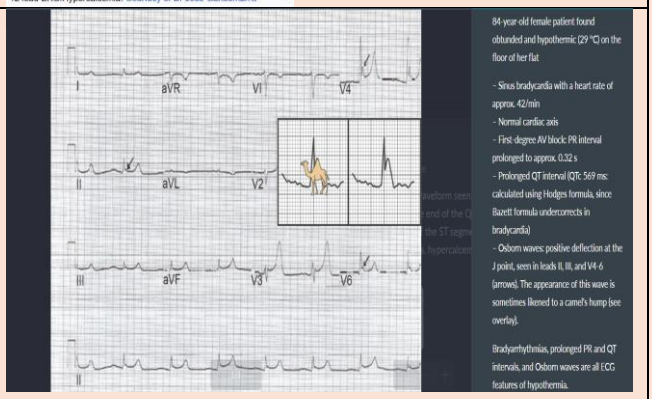
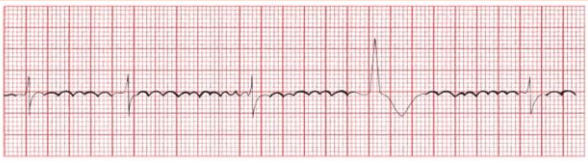


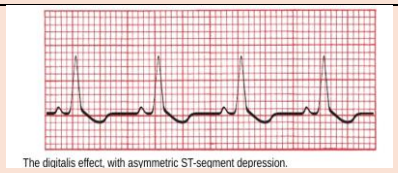
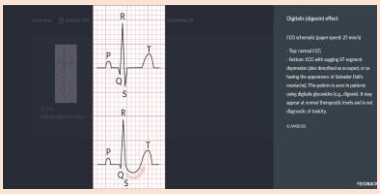
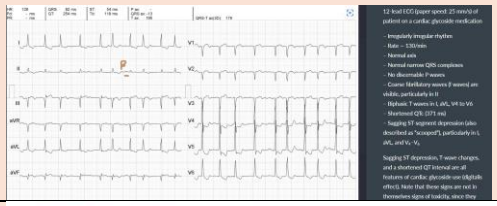
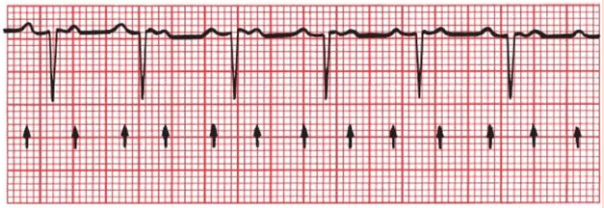
In patients with WPW, the delta waves are often negative in the inferior leads (II, III, and aVF). This pattern is therefore often referred to as a pseudoinfarct pattern because the delta waves may resemble Q waves. The short PR interval is the one remaining clue that can distinguish WPW from an infarction on the EKG

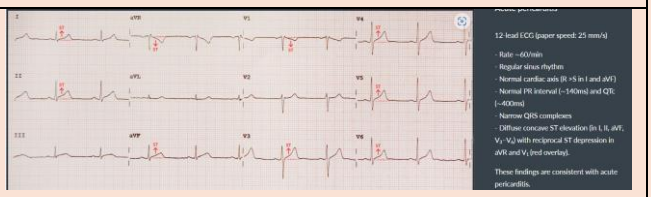
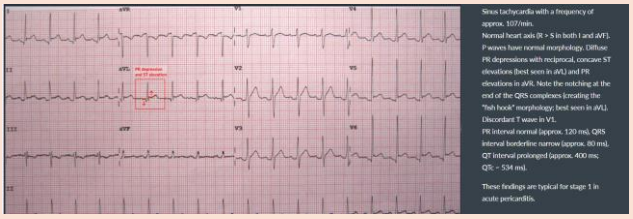
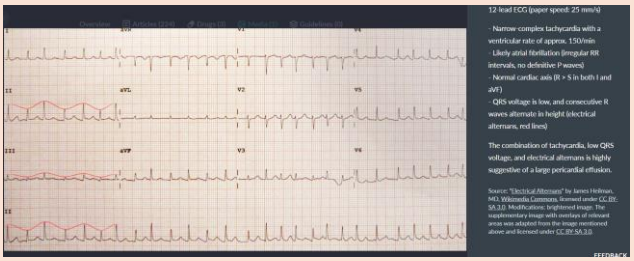
	highly suggestive of a posterior infarction, no RAD differentiating it from RVH -lt ventricular infarction more common than rt -rt is almost accompanied by inferior infarction(most rightward anterior lead, V1. If there is also ST elevation in lead V2, it will be of smaller magnitude than that in V1, and often V2 will show ST depression. In the limbs leads, the ST elevation in lead III is greater than that in lead II, or place ther electrodes on rt chest wall)	in a patient with left bundle branch block the presence of ST segment elevation of at least 1 mm in any lead with a predominant R wave or ST-segment depression of at least 1 mm in leads V1–V3 if deep S waves are present is strongly suggestive of an evolving infarction
NSTEMIs	-T-wave inversion and ST-segment depression -more common than STEMIs -lower initial mortality rate but a higher risk for further infarction and mortality than STEMIs	 
Takotsubo Cardiomyopathy (apical ballooning syndrome, broken heart syndrome)	-ST elevations (most common finding), typically in the precordial leads -ST depressions are uncommon (< 10% of cases). -Diffuse T-wave inversions -Prolonged QT interval	

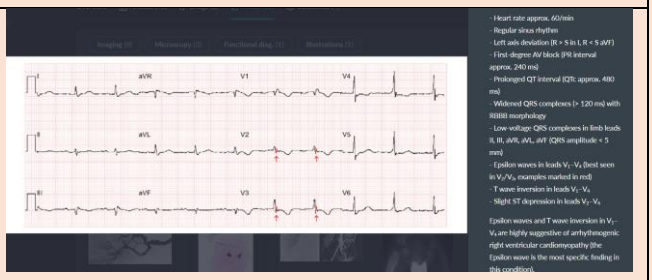
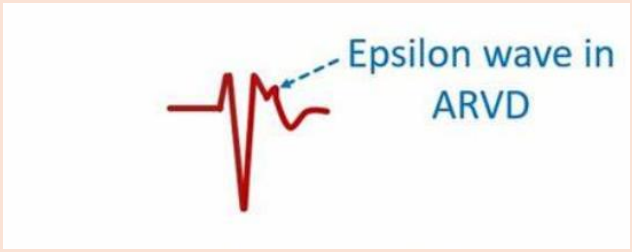
	-There are no electrocardiographic criteria that can reliably distinguish takotsubo cardiomyopathy from a STEMI caused by coronary artery occlusion. The distinction is made in the cath lab; patients with takotsubo cardiomyopathy will not show the occluded coronary arteries seen with a STEMI.	 Limb leads in a patient with takotsubo cardiomyopathy. The ST-segment elevation looks for all the world like a typical inferior wall STEMI.
Angina without infarction	-During an attack of angina, the EKGs of patients with both stable and unstable angina may demonstrate T-wave inversion and often ST-segment depression. In between attacks, the EKG is usually normal.(measure cardiac enzymes to exclude infarction)	 Three examples of the EKG changes that can accompany angina without infarction: (*A*) T-wave inversion, (*B*) ST-segment depression, and (*C*) ST-segment depression with T-wave inversion (the ST-segment and T waves merge seamlessly).
Prinzmetal angina	-ST elevation, the contours often will not have the rounded, domed appearance of true infarction, and it will return quickly to baseline when the patient is given antianginal medication (e.g., nitroglycerin)	
hyperkalemia	-The great imitator. Evolution of (1) peaked T waves, (2) PR prolongation and P-wave flattening, and (3)	

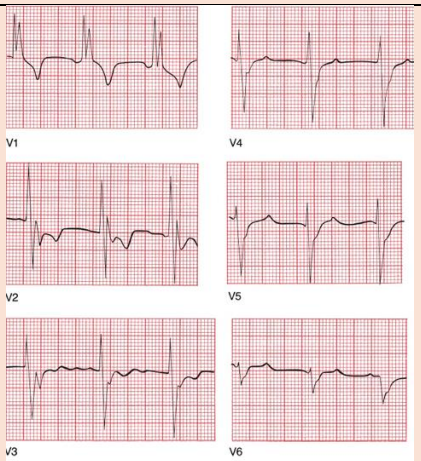
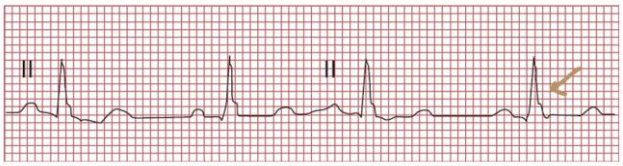
	QRS widening. Ultimately, the QRS complexes and T waves merge to form a sine wave -the presence of a rightward axis (a negative QRS complex in lead I, a positive QRS in aVF) may be an important clue that the wide QRS complexes are the result of hyperkalemia -Conduction blocks—high-degree AV blocks and bundle branch blocks—can also appear as the serum potassium rises. Asystole or ventricular fibrillation may eventually develop	
Hypokalemia	particular order: - ST-segment depression - Flattening of the T wave with prolongation of the QT interval(may cause supraventricular and ventricular tachyarrhythmias) -Appearance of a U wave -Rarely, severe hypokalemia can cause ST-segment elevation *don't forget to exclude MI!	 

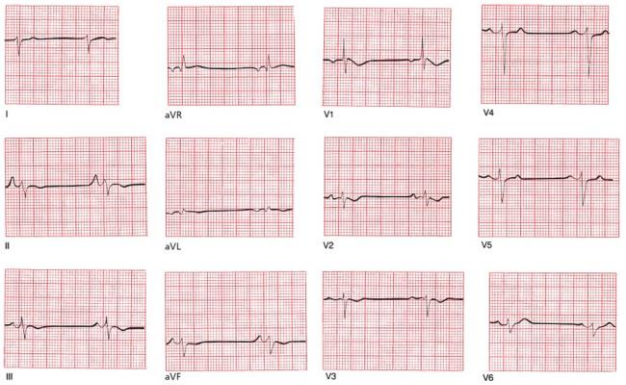
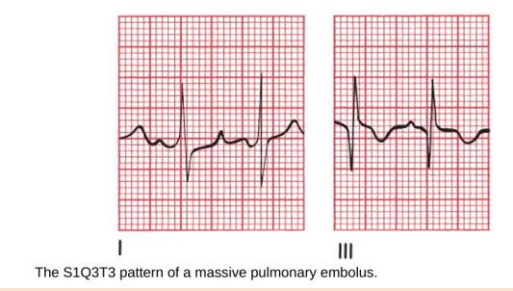
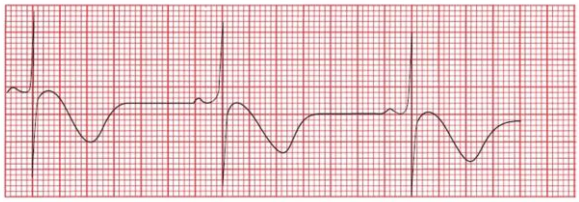
Hypocalcemia, hypomagnesemia, hypokalemia	-prolonged QT→torsade de poites? Drugs prolong QT: -antiarrhythmic agents (e.g., sotalol, quinidine, procainamide, disopyramide, amiodarone, dofetilide, and dronedarone) - Antibiotics: macrolides (*e.g.*, erythromycin, clarithromycin, azithromycin) and fluoroquinolones (*e.g.*, levofloxacin and ciprofloxacin) Antifungals (*e.g.*, ketoconazole) Nonsedating antihistamines (*e.g.*, astemizole, terfenadine) Psychotropic drugs: antipsychotics (*e.g.*, haloperidol, phenothiazines), tricyclic antidepressants (*e.g.*, amitriptyline), selective serotonin reuptake inhibitors (*e.g.*, citalopram, fluoxetine), and methadone Plus some gastrointestinal medications, antineoplastic agents, and diuretics (the last by causing hypokalemia or hypomagnesemia)	 Hypocalcemia. The QT interval is slightly prolonged. A premature ventricular contraction (PVC) falls on the prolonged T wave and sets off a run of torsade de pointes.  12-lead ECG (paper speed: 25 mm/s) of a patient with hypocalcemia (total serum calcium 5.3 mg/dL) - Sinus rhythm - Heart rate: ~ 94/min - Normal cardiac axis (II > 5 in I and aVF) - Normal narrow QRS complexes - QT prolongation (example indicated by green overlay; QTc ~ 600 ms) due to a prolonged ST segment. This is the key ECG finding in hypocalcemia. - Tall, peaked T waves (examples indicated by red overlay). This is not an expected finding in hypocalcemia and should prompt evaluation for a coexisting abnormality, e.g., hyperkalemia. Source: "ECG: Hypocalcemia" by Ewingdo, Wikimedia Commons, licensed under CC BY-SA $$QTc = \frac{QT}{\sqrt{RR}}$$ The QTc should not exceed 500 ms during therapy with any medication that can prolong the QT interval (550 ms if there is an underlying bundle branch block); adhering to this rule will reduce the risk for ventricular arrhythmias. This simple formula for determining the QTc is most accurate at heart rates between 50 and 120 beats per minute; at the extremes of heart rate, its usefulness is limited.
hypercalcemia	Shortens QT interval <360s	 12 lead EKG:Hypercalcemia. Courtesy of Dr Jose Gansemans
Hypothermia	1-Everything slows down. Sinus bradycardia is common, and all the segments and intervals—PR, QRS, QT, etc.—become prolonged. 2. A distinctive and virtually diagnostic type of ST-segment elevation may be seen. It consists of an abrupt ascent right at the J point and then an equally sudden plunge back to baseline. The resultant configuration is called a J wave or Osborn wave. J waves will disappear as the	 84-year-old female patient found obtunded and hypothermic (29 °C) on the floor of her flat - Sinus bradycardia with a heart rate of approx. 42/min - Normal cardiac axis - First-degree AV block: PR interval prolonged to approx. 0.32 s - Prolonged QT interval (QTc 569 ms; calculated using Hodges formula, since Bazett formula undercorrects in bradycardia) - Osborn waves: positive deflection at the J point, seen in leads II, III, and V4-6 (arrows). The appearance of this wave is sometimes likened to a camel's hump (see overlay). Bradyarrhythmias, prolonged PR and QT intervals, and Osborn waves are all ECG features of hypothermia.  A muscle tremor artifact resembles atrial flutter.

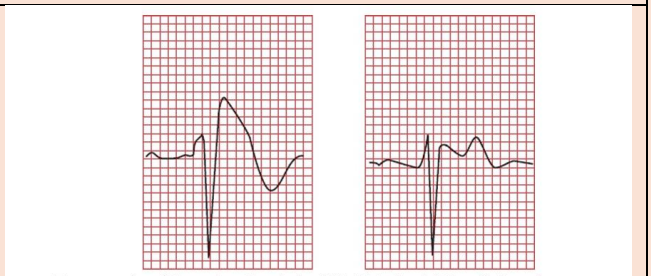
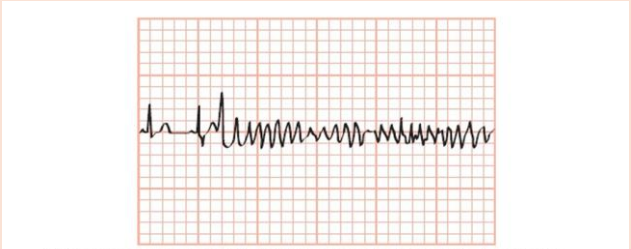
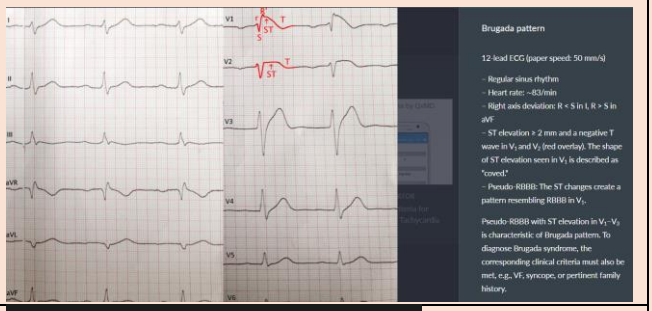
	patient is rewarmed. 3-Various arrhythmias may appear, including sinus bradycardia, a slow junctional rhythm and slow atrial fibrillation. 4. A muscle tremor artifact due to shivering may complicate the tracing. A similar artifact may be seen in patients with Parkinson disease. The tremor of Parkinson disease can be easily mistaken for atrial flutter, since both tend to cycle at about 5 Hz, or 300 times per minute.	
Digitalis:EKG Changes Associated With Therapeutic Blood Levels	ST-segment and T-wave changes in leads with tall R waves;ST segment depression with flattening or inversion of the T wave. The depressed ST segments have a very gradual downslope, emerging almost imperceptibly from the preceding R wave	 The digitalis effect, with asymmetric ST-segment depression.  
Digitalis: toxic blood level	-SA suppression -tachyarrhythmias - conduction blocks; -Paroxysmal atrial tachycardia (PAT) and PVCs are the	 PAT with 2:1 block. The *arrows* point to each P wave.

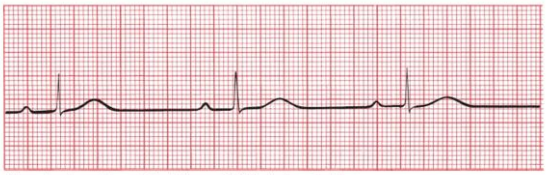
	most common, junctional rhythms are fairly common, and atrial flutter and fibrillation are the least common -PAT with block(2:1 mc) is most characteristic.	
pericarditis	-Stage 1: **diffuse** ST elevations, reciprocal ST depression in aVR and V1, PR segment depression Stage 2: ST segment normalizes in ~ 1 week. Stage 3: inverted T waves Stage 4: ECG returns to normal baseline (as prior to onset of pericarditis) after weeks to months -no Q wave according to the book	 
Pericardial effusion, and some tamponade	-low voltage in all leads -the criteria: The most sensitive are either (1) the sum of the total QRS voltage in leads I, II, and III is less than 15 mV or (2) the sum of the total QRS voltage in leads V1, V2, and V3 is less than 30 mV. More specific criteria are (1) the QRS voltage in all limb leads is less than 5 mV or (2) the QRS voltage in all precordial leads is less than 10 mV.	A pericardial effusion is not the only cause of low voltage. Anything that dampens the ability of the surface electrodes to detect the electricity generated by the heart can be responsible, such as the expanded, air-filled lungs of chronic lung disease; a pneumothorax; a large pleural effusion; or the marked adiposity of someone who is very obese. Also, anything that reduces the heart's ability to generate normal voltage can be the culprit, for example, infiltrative diseases of the heart (such as amyloidosis), severe hypothyroidism, and end-stage cardiomyopathy caused by multiple infarctions. 

	-in large effusion—electrical alternans(electrical axis of the heart varies with each beat, This can affect not only the axis of the QRS complex but also that of the P and T waves. A varying axis is most easily recognized on the EKG by the varying amplitude of each waveform from beat to beat).	
ARVD(Arrhythmogenic right ventricular cardiomyopathy/ dysplasia)	-The most common feature on the EKG is T-wave inversion in leads V1 through V3 -Repolarization disturbances in the right precordial leads (V1-3) -Possibly epsilon wave (at the end of a widened QRS complex):Looks like the Greek letter epsilon: ε Highly specific for ARVC but only occurs in ~ ⅓ of patients -Increased QRS duration -Ventricular tachycardia -Ventricular extrasystoles	 

Myocarditis	-Most common are conduction blocks, especially bundle branch blocks and hemiblocks	 Right bundle branch block in a patient with active myocarditis following a viral infection.
Atrial Septal Defect	-The EKG may be normal. With enlargement of the right atrium and right ventricle, however, you can see first-degree AV block, atrial tachyarrhythmias, incomplete right bundle branch block, and, with the more common secundum ASD, right axis deviation (you may see left axis deviation with a primum ASD) -crochetage pattern:most characteristic finding,small notch in the QRS complexes in the inferior leads. It can occur early or late in the QRS complex. Interestingly, the size of the notch is proportional to the size of the ASD and the size of the shunt. Crochetage can also be seen in patients with a patent foramen	 The small notch in the terminal portion of these QRS complexes is the crochetage pattern of an atrial septal defect.

	ovale and sometimes in perfectly normal hearts	
COPD	-low voltage, right axis deviation, and poor R-wave progression in the precordial leads -COPD can lead to chronic cor pulmonale and right-sided congestive heart failure. The EKG may then show right atrial enlargement (P pulmonale) and right ventricular hypertrophy with repolarization abnormalities.	 Chronic obstructive pulmonary disease. Note the low voltage, extreme right axis deviation, right atrial enlargement (in lead II), and precordial criteria for right ventricular hypertrophy.
Acute PE	-Right ventricular hypertrophy with repolarization abnormalities, right bundle branch block, S1Q3 or S1Q3T3. Sinus tachycardia and atrial fibrillation are the most common arrhythmias -non massive PE→ normal ECG, or only sinus tachy	 The S1Q3T3 pattern of a massive pulmonary embolus. 
(CNS) catastrophes, such as a subarachnoid bleed or cerebral infarction	diffuse T-wave inversion and prominent U waves. The T waves are typically very deep and very wide, and their contour is usually symmetrical (unlike the asymmetrical	 V4 Deeply inverted, wide T waves in lead V4 in a patient with a central nervous system bleed.

	inverted T waves of secondary repolarization associated with ventricular hypertrophy). Sinus bradycardia also is commonly seen.	
Brugada syndrome (BRUGADA PATTERN+ SYMPTOMS)	-(1) a pattern resembling right bundle branch block with a slow, prolonged downslope of the R' component of the QRS complex, (2) T-wave inversion in leads V1 and/or V2, and (3) ST-segment elevation in leads V1, V2, and V3. The ST-segment elevation is often concave and descends into an inverted T wave, a pattern referred to as coving -The importance of Brugada pattern lies in its propensity to cause ventricular arrhythmias that can lead to sudden death. The most typical of these is a fast polymorphic ventricular tachycardia that looks just like torsade de pointes	 Two examples of Brugada pattern in lead V1. Note the right bundle branch appearance and the inverted T wave in V1. The ST-segment elevation can appear coved (first figure) or saddle-backed (second figure).  Polymorphous ventricular tachycardia with unusually narrow QRS complexes in a patient with Brugada syndrome.  Brugada pattern 12-lead ECG (paper speed: 50 mm/s) - Regular sinus rhythm - Heart rate: ~83/min - Right axis deviation: R < S in I, R > S in aVF - ST elevation > 2 mm and a negative T wave in V_1 and V_2 (red overlay). The shape of ST elevation seen in V_2 is described as "coved." - Pseudo-RBBB: The ST changes create a pattern resembling RBBB in V_2. Pseudo-RBBB with ST elevation in V_1–V_3 is characteristic of Brugada pattern. To diagnose Brugada syndrome, the corresponding clinical criteria must also be met, e.g., VF, syncope, or pertinent family history.
The Athlete's Heart	Nonpathologic findings can include sinus bradycardia, junctional rhythms and a wandering atrial pacemaker, nonspecific ST-segment and T-wave changes, left and right ventricular	**Wandering atrial pacemaker** An arrhythmia defined by the presence of multiple atrial pacemakers. Characterized by at least 3 different P wave morphologies. If tachycardia is also present (HR > 100), it is referred to as multifocal atrial tachycardia.

	hypertrophy, incomplete right bundle branch block, first-degree or Wenckebach AV block, and a notched QRS complex in lead V1 *Preparticipation Screening for Athletes* Findings that require further evaluation: • T-wave inversion beyond lead V2 in white athletes or beyond V4 in African American or Caribbean athletes • T-wave inversion in the lateral leads • ST-segment depression in any lead • Evidence of a congenital heart condition such as hypertrophic cardiomyopathy, long QT syndrome, Wolff–Parkinson–White syndrome, Brugada syndrome, and arrhythmogenic right ventricular cardiomyopathy	 Sinus bradycardia and first-degree AV block in a triathlete.

Done

Best of luck