Q1 What is the likely diagnosis?

a. SVT with VT
b. Bidirectional VT
c. SVT with aberrancy
d. Ventricular bigeminy

**The correct answer is “b” – Bidirectional VT.**

The 12 lead ECG show a classic bi-directional tachycardia, best identified in the limb leads. It is a wide QRS tachycardia with 2 distinct morphologies alternating with each other; the QRS axis is either $-90^\circ$ or $+120^\circ$. Lead V1 remains positive.

The origin of this VT is from the left fascicular system and is seen in digitalis toxicity. Catecholaminergic VT and aconite poisoning can also produce such patterns.
Q2. 14 yr boy, h/o palpitations & CHF

The correct diagnosis is
a. Atrial tachycardia
b. Sinus tachycardia
c. Atrial flutter
d. a + b

The correct answer is “a” - Atrial tachycardia.
The 12 lead ECG shows two different tachycardias. In the beginning, the ventricular rate is 86 bpm and the underlying atrial rhythm shows two P’ waves for every QRS complex, suggestive of atrial tachycardia. The atrial tachycardia rate is 172 bpm and there is 2:1 AV conduction. This is well seen in the inferior leads and V4, where every alternate P’ is superimposed on the T wave. One P’ wave precedes the QRS complex with a prolonged PR interval and the next non-conducted P wave falls within the T wave. Soon after the 5th QRS complex, an earlier P’ terminates the atrial tachycardia. The subsequent rhythm shows 1:1 AV conduction with P wave morphology generally consistent with sinus node origin. However, the P is negative in lead I. Since the rate is 110 bpm, this is another atrial tachycardia. This patient had clinically presented with heart failure, most likely because of tachycardiomyopathy.
Q.3. 38 yrs-old, recurrent palpitations

This ECG shows
a. SVT with aberrancy
b. VT with VA conduction
c. VT with VA dissociation
d. None of the above

The correct answer is “b” - VT with VA conduction
This 12 lead ECG shows a regular, wide QRS tachycardia. There is no definite BBB patternand thus, an SVT with aberrancy is ruled out. Moreover, the LBBB-like QRS morphology (inlead V1) with right axis deviation as seen here, is a combination that is pathognomonic for VT. Also, there are fewer P waves than QRS complexes. This is a VT with retrograde, inverted P waves interspersed between the QRS complexes. There is definite P-QRS relationship, wherein a Pwave follows two consecutive QRS complexes with an increasing RP interval followed bythe third QRS with no P wave conducted retrogradely. Thus, there is a VA conduction with VA Wenckebach phenomenon.
4. 49 yr old man. Recurrent chest discomfort, only on exertion. ECG at rest.

This ECG is suggestive of:
- a. Coronary ischemia
- b. Hypertrophic cardiomyopathy
- c. Raised ICT
- d. Don’t know

The correct answer is “b” - Hypertrophic cardiomyopathy

The 12-lead ECG shows deep, ‘dagger-shaped’ T wave inversions in leads V4- V6, I, aVL, II, III and aVF. Coronary ischemia is unlikely in view of the T wave inversions present even at rest. Also, there is evidence of left ventricular hypertrophy by voltage criteria. The history of exertional chest pain does not support raised ICT. LVH with widespread T wave inversions at rest suggest hypertrophic cardiomyopathy and these patients often complain of exertional chest pain in view of increased myocardial oxygen demand during exercise.
Q5. Asymptomatic 42 year old sportsman. Normal echo

The Holter shows:

a. Atrial flutter
b. NSVT
c. Atrial flutter with intermittent bundle branch block
d. Both a + b

The correct answer is “d” - Both a + b

The four Holter strips show atrial flutter throughout being conducted at variable ventricular rate. The bottom two strips show two episodes of ill-sustained wide QRS tachycardia. The rate of this tachycardia is faster, there is no definite bundle branch block pattern and the tachycardia has two different morphologies. Thus, this patient has both atrial flutter and NSVT.
Q6. 60 year old man. Old anterior MI 6 months ago. Coronary Angio then had shown an ectasia of the LAD artery. This time he came with recent chest pain with collapse

This ECG shows:

a. SVT with aberrancy
b. Ventricular tachycardia
c. Atrial fibrillation with fast ventricular rate
d. None of the above

The correct answer is ‘b’ – Ventricular tachycardia

The ECG shows a regular wide QRS tachycardia. The QRS complexes are monomorphic. Unless proved otherwise, a wide QRS tachycardia in subjects with a history of MI must be considered a ventricular tachycardia. Moreover, one can even discern AV dissociation in lead V1.

Monomorphic VT in coronary artery disease is due to an underlying scar of an old MI.
The same patient was given a DC shock, but went on to develop a VT storm. Shown below are some of the monitor strips during these episodes, which required repeated DC shocks.

Does this patient have acute ischemia?

a. Yes  
b. No  
c. Can’t say  
d. Don’t know

The correct answer is ‘a’ – Yes!
The ECG strips show a wide QRS tachycardia. The R-R intervals are irregular (measure carefully) and the QRS pattern is changing, often from complex to complex. Hence, we are seeing polymorphic VT. In coronary artery disease patients, polymorphic VT is a defining arrhythmia of acute ischemia.

The VT storm was refractory to anti-arrhythmics, electrolyte optimization, sedation and nitrates. Hence he was taken up for emergent angiography. The angiogram showed a thrombus in the ectatic segment of the LAD artery. Angioplasty with a covered stent immediately restored sinus rhythm, as seen below.

The ECG shows QS complexes in leads V1-V3, consistent with an old anteroseptal MI. There is gross ST elevation in leads V4-V6, due to the superimposed recent MI. The patient subsequently stabilized. The monomorphic scar VT recurred as expected. An ICD was implanted.
A 22-year-old man. Frequent rapid palpitations since 1 month; echo showed moderate biventricular dysfunction.

This ECG shows:

a. Atrial flutter
b. Ventricular tachycardia
c. Junctional tachycardia
d. Atrial tachycardia

The correct answer is ‘c’ — Junctional tachycardia

There is a regular narrow QRS tachycardia. Only in lead V2 there is mild prolongation of the QRS complex. Hence a myocardial VT is ruled out. Lead II (especially the long lead) shows tall, “nipped” T waves intermittently—these are due to superimposed P waves. Hence there is clearly AV dissociation. The only “supraventricular” tachycardia that can show AV dissociation is junctional ectopic tachycardia. This arrhythmia is more common in children who have recently undergone cardiac surgery. However, it can also be seen in myocarditis, which was the diagnosis in this patient.

The likely diagnosis:
- a. Acute coronary syndrome
- b. Intracerebral bleed
- c. Hypokalemia
- d. None of the above

The correct answer is ‘c’ – Hypokalemia

The ECG shows an obvious QT prolongation (QT & QTc 560 ms), with bifid T waves in leads V3-V6. Acute ischemia can produce QT prolongation. However, absence of both ST depressions/elevations and angina make this diagnosis untenable.

Raised intracranial tension can produce marked QT prolongation. However, this is associated with deep, symmetrical T inversion. Also, such patients remain in altered sensorium. Many diuretics can produce hypokalemia. Though indapamide is considered safer in this regard, in this patient it proved to be the culprit. The serum potassium was only 1.8 mEq/L. After withdrawing the drug and replacing potassium, his ECG normalized.
Q10. A 22 yr old man; asymptomatic; nationally competitive tennis player

This ECG shows:

a. Mobitz type 1 AV block (Wenckebach)
b. Mobitz type 2 AV block
c. Intermittent 2:1 AV block
d. SA exit block

The correct answer is ‘a’ – Mobitz type 1 AV block (Wenckebach)
There is clearly progressive PR prolongation followed by a blocked P wave. The sinus rate is 60/min. The Holter did not show any higher grades of AV block. Repeatedly, AV Wenckebach was seen. Asymptomatic AV Wenckebach in this man was the consequence of extreme fitness resulting in a high vagal tone.
Q11. A 53 yr old lady with effort dyspnea. At start of stress test.....

After 3 minutes of exercise. She felt tired. The profile suggests: 

This ECG shows:
- a. Ischemia
- b. AV nodal block
- c. Infranodal block
- d. None of the above

The correct answer is ‘c’ – Infranodal block

Careful observation of ECG during exercise shows that there is deformation of the T wave which has become pointed in lead II, suggesting the presence of 2:1 AV block. This is confirmed by the pointed T wave lying exactly between the mid-point of the 2 surrounding P waves. The atrial rate has increased with exercise to 150/min.

Ischemia is unlikely as there are no ST segment changes. The T wave changes have occurred because of superimposed P waves. In any case, ischemia does not produce isolated AV block on exercise.

Any AV block which comes or worsens on exercise, has to be ‘Infranodal block’ as AV nodal conduction improves with exercise. This patient therefore needs a pacemaker.
Q12. A 45 year old man with severe chest pain

This ECG suggests:
- Proximal LAD occlusion
- RCA occlusion
- Left main critical stenosis
- Acute Pericarditis

The correct answer is ‘d’ – Acute pericarditis

Except leads aVR and V1, all leads show ST elevation. Also, there is ST depression in lead aVR. Close scrutiny shows the ST elevation to be “concave upward”, best seen in lead V5; however, this is only a supportive criterion for the diagnosis. Acute pericarditis causes inflammation of the epicardium resulting in repolarization abnormalities of both atria and ventricles. In the ventricles, it results in ST elevation which is seen in most of the leads. In the atria, repolarization occurs during the PR segment; the current of injury thus causes PR segment elevation in aVR, since this lead ‘faces’ the atria. In several other leads there may be PR segment depression, a hint of which is seen in lead II. In acute pericarditis, PR and ST segment change directions. In conclusion, aVR may show PR elevation with ST segment depression whereas other leads show PR depression with ST elevation.
Q13. Uncontrolled HT. Pacemaker implanted for bradycardia a few years ago. Patient now comes with ill-health and dyspnea.

Likely cause for symptoms:
a. Ischemia
b. Renal failure
c. Pacemaker malfunction
d. Can’t say

The correct answer is ‘b’ - Renal failure
The QRS complexes are all paced. They are extremely wide, measuring 280 ms. The P waves bear no relation to the QRS complexes. Hence, we are talking of single chamber ventricular pacing (VVI). The P waves is distinctly abnormal as seen in lead C1 (V1). This suggests a biatrial abnormality. The left atrial abnormality could be because of ventricular dysfunction and enlargement because of long-standing uncontrolled hypertension.

The T waves are extremely tall and peaked. The height measures almost 18 mm in C3 (V3) and the peaked nature is best seen in lead C2 (V2). Ventricular pacing does give wide QRS complexes. However, these are typically in the range of 140-180 ms, depending on the site of pacing and the size of the heart. A QRS complex of 280 ms is distinctly unusual for ventricular pacing. Secondary T wave change opposite to the QRS complex is expected with ventricular pacing. However, the peaked nature seen here is distinctly abnormal. Also, the amplitude is too high for secondary T wave changes. This ECG picture along with the clinical scenario suggests that the patient is having a metabolic problem. Hyperkalemia is the most important cause for this T wave abnormality. If one then evaluates this retrospectively, then the QRS width suggesting myocardial delay is also a feature of sever hyperkalemia.

After correction of hyperkalemia
There is marked reduction in the QRS widths to about 120 ms. The T waves also become much lower measuring only 8 mm in lead V3. The P wave abnormality is still seen, suggesting residual ventricular abnormality.

Q14. A 42 yrs-old smoker with uneasiness & chest discomfort...

**The ECG shows:**

a. AV Wenckebach  
b. Atrial bigeminy  
c. SA block  
d. Sinus node dysfunction

**The correct answer is ‘b’ – Atrial bigeminy**

The long lead II clearly shows paired QRS complexes, which then would be described as a bigeminal rhythm. A clear P wave is seen before the first of these pairs of QRS complexes. The T wave shows alternating amplitude and width. The first T wave of the bigeminal pair is broader and taller. This clearly suggests a P wave sitting on a T wave. If one looks at lead V1(C1), one can clearly make out these premature ectopic P waves (arrow). Since the P wave is very premature, it finds the AV conduction partly refractory and conducts with a prolonged PR interval, best seen in V1.

Therefore, it is clear that there are alternating unifocal atrial ectopics producing an atrial bigeminypattern.
Q15. 29 yr old man with recurrent rapid palpitations, sometimes with near-syncope

Figure 1

As the episodes continues....... 

Figure 2

What are these arrhythmias?

a. Monomorphic VT and A Fib
b. Polymorphic VT and A Fib
c. AVRT and A Fib
d. LQTS arrhythmias

The correct answer is ‘c’ – AVRT and A Fib
The first ECG shows initially a rapid regular wide QRS tachycardia @ 200 bpm. It shows atypical LBBB pattern. The latter portion of the trace shows an irregular wide QRS tachycardia with a different morphology. There is a delta wave which is seen in V2-V4. This is diagnostic of atrial fibrillation with pre-excitation (WPW). Therefore, this is WPW syndrome with orthodromic AVRT (with LBBB), which degenerates into A Fib. WPW syndrome patients have a high incidence of A Fib, the commonest mechanism being degeneration of AVRT, as in this slide.

Figure 2 shows atrial fibrillation; there is an abrupt cessation of preexcitation with a sudden drop in ventricular rate. The A fib now continues with a normal QRS complex. This was recorded during radiofrequency ablation of the accessory pathway, which eliminated the preexcitation.