ECG Interpretation Course

Small group discussion cases

Answers

Chapter 1: Hypertrophy / BBB

Case 1: Sinus rhythm at 90, LVH with strain. S in V1 plus R in V5 is about 48 mm and the P wave suggests biatrial abnormality. This man turned out to be HIV positive.

Case 2: HR 78, PR: .26, QRS: .18, Axis: -60, QT: .46, QTc: .51. First degree AV block, RBBB. LAD, LAFB. Possible trifascicular block. The near syncope could have been due to high degree AV block leading to asystole. The patient should be monitored to try to prove the cause of near syncope.

Case 3 : HR: 64, PR: .24, QRS: 0.20, Axis: -30, QT: 0.48, QTc: 0.5. 1 degree AV Block. Left atrial abnormality. LBBB with wide QRS. He is at risk of CHF, heart block, VT, VF, and atrial fibrillation.

Case 4: Atrial fibrillation with a controlled ventricular response (rate 60- 100 bpm).QRS- 0.10 sec. Prominent voltage consistent with left ventricular hypertrophy with strain pattern. Probable digoxin effect.

Case 5: ST (rate 104 bpm) PR: .14, QRS: .08, Axis: +120, QRc: .44. Left atrial abnormality (enlargement). Probable right ventricular hypertrophy. RAD. A cardio-embolic stroke is possible. This patient had moderately severe mitral stenosis.

Case 6: ST (104bpm) PR: .18, QRS: 08, Axis: +165. SR RAD Right ventricular hypertrophy

Case 7: Sinus tachycardia. RVH with strain (RAD, tall R in V1, V2, T inversion V1, V2). LVH (biventricular enlargement) (deep S in V3). Tall R and deep S V2-V4 usually suggests biventricular enlargement.

Case 8: Sinus tachycardia with rate 115 bpm. PR- 0.16, QRS- 0.10, QT- 0.30 sec, axis- + 60. Tall and wide P waves suggest bi-atrial enlargement. Prominent R waves V1-3 may represent septal enlargement but other causes are possible. Non- specific ST-T changes in the anterolateral leads.

Case 9: Sinus rhythm with rate of 85 bpm. Tall P waves, Axis +95. Delayed R wave progression V1-5. C/W COPD

Case 10: Sinus tachycardia at 105 bpm. PR- 0.20 sec, QRS- 0.14 sec, QT- 0.40 sec (QTc- 0.53 sec.) LBBB.

Case 11 Sinus tachycardia with 2:1 AV conduction with atrial rate of 132 and ventricular rate of 66 bpm. , QRS- 0.12 sec, QT -0.50 sec, Axis + 120 degrees.
RBBB and left posterior fascicular block. (With the previous ECG suggests alternating bundle branch block and pacing would be needed with the symptoms indicated.)

Chapter 2: Ischemia / Infarction

Case 1: Sinus rhythm. Acute anteroseptal current of injury.

Case 2: Sinus rhythm. T wave inversion in the anterolateral leads suggesting reperfusion of the previously occluded artery.

Case 3: 15 lead ECG- Acute inferior, right ventricular infarction. HR: 48, PR .16, QRS .10, Axis: +70 ST is elevated in inferior leads and in lead V4r.

Case 4 : ECG Sinus tachycardia at 100 bpm, PR: .22, QRS: .11, Axis: -15. Diffuse ST segment depression c/w subendocardial ischemia or Non ST elevation myocardial infarction. Coronary angiography might show 3 vessel disease or left main stem stenosis. Management should consider revascularization if feasible.

Case 4b: Sinus bradycardia at 48 bpm with near resolution of ST, and T changes except in leads I and aVL suggesting resolution of ischemia

Case 5 : Sinus bradycardia at 56 bpm. ST elevation in leads I, II, III, aVF, V4,5,6. Possible inferior and lateral wall current of injury, but the patient had "inflammation" on cardiac MRI and coronary arteries were normal. The patient had normal wall motion and remained pain free on ASA and colchicine.

Case 5b: Sinus bradycardia at 56 bpm with improvement in diffuse ST and T wave changes.

Case 6: Sinus rhythm at 80 bpm. PR: .16 QRS: .12, Axis: -60. Extensive recent anteroseptal myocardial infarction and likely apical. With extensive Q waves improvement of function in the infarct zone is not likely.

Case 7: Sinus at 70 bpm There is less T wave inversion (evolution) in the infarct zone.

Case 8: Sinus rhythm. Recent inferoposterolateral myocardial infarction. Axis in 4^{th} quadrant. This person had occlusion of the left circumflex artery.

Case 9: Sinus rhythm at 75 bpm. Frequent pvc's with a couplet and a triplet. The second beat of the triplet is likely a fusion beat. There is prominent ST elevation leads V1-4 with prominent T waves V5, 6 and minimal reciprocal ST depression in lead aVL- C/W an acute anteroseptal MI.

Case 9b Idioventricular rhythm at 100 bpm, with isolated pvc's, (from a different ventricular origin, than the pvc's on the previous ECG)
ST segment elevation is present in leads V1-6, and II, III, aVF

Case 10: Acute inferior (+RV MI)-not shown. Sinus tachycardia at 108 with CHB, junctional escape at 50 bpm. Narrow QRS suggests the block is above the bundle of His and if hemodynamically stable, pacing is not required. This heart block most often will resolve.

Case:11: Marked ST elevation in leads V2-6, I and aVL. This patient has coronary artery spasm involving the left anterior descending artery, proven in the heart catheterization lab. Management includes coronary vasodilators.

Case 12: Heart rate average 160 BPM. PR - , QRS.09 seconds, Axis +45º. ST segment elevation leads 2-3 AVF with reciprocal ST depression leads 1 and AVL. Rapid, irregularly irregular heart rhythm. Interpretation: Atrial fibrillation with a rapid ventricular response. Acute inferior wall ST elevation MI.

Case 13: HR 68, PR .16, QRS .09, Axis +40. Sinus rhythm, with a single PVC. 1-2 mm ST elevation in 1 and aVL and reciprocal ST depression and T inversion in inferior leads. C/W acute high lateral MI

Premature Beats & Tachyarrhythmias

Case 1: AD

History: 77-yr-old female admitted with dizzy spell, nausea, shaking. (Diagnosed as torsades & given $MgSO_4$).

ECG: Artefact. True QRS complexes are visible through the tracings. Underlying rhythm is irregular – ?SR with PACs, ?AF.

Case 2: GJ

History: 96-yr-old male with history of CLL. Presented to ER with nausea / vomiting / diarrhoea.

ECG: Vent rate 69 BPM, PR interval 142 ms, QRS duration 82 ms, QT/QTc 388/415 ms, P-R-T axes * 43 64; "Wandering atrial pacemaker" then sinus bradycardia. Reduced R wave progression V1-3.

Case 3: RJ

History: 64-yr-old male. Attended ER with musculoskeletal chest pain.

ECG: Vent rate 86 BPM, PR interval 134 ms, QRS duration 86 ms, QT/QTc 384/459 ms, P-R-T axes 46 85 37; SR with frequent PACs, one conducted with RBBB (Ashman-type aberrancy).

Case 4: TB

History: 54-yr-old male. Athlete - trains for Iron-man competitions. VF arrest on bicycle. Normal coronaries on cath. Unknown FHx (adopted).

ECG: TB1 Strips show SR with bigeminal PACs – non-conducted on 1st 4 beats, contributing to the bradycardia (also sinus brady as well – time from PAC to next sinus P wave would give rate of ~50 bpm). Then one PAC conducted normally (compare this T wave for clear evidence of the PACs elsewhere), then one conducted with LBBB. NB also different P wave in front of 5th QRS complex – also an ectopic atrial beat.

ECG: TB02 SR, then wide-complex tachycardia. Independent P waves, seen best in Lead 1. 6th beat of the WCT is a fusion beat – preceding P wave is visible. VT. [ICD was implanted].

Case 5: PM

History: 67-yr-old male with no prior cardiac history. Presented with acute retrosternal CP.

ECG: PM01 Vent rate 79 BPM, PR interval * ms, QRS duration 129 ms, QT/QTc 392/449 ms, P-R-T axes * -70 101; Wide-complex rhythm with independent P waves, capture & fusion beats – accelerated idioventricular rhythm. Likely inferoposterior LV source based on morphology. Long PR interval on the captured / fused beats. Capture beats show inferior ST elevation.

ECG: PM02 Vent rate 60 BPM, PR interval 220 ms, QRS duration 108 ms, QT/QTc 408/408 ms, P-R-T axes 56 68 92; ECG in SR showing 1st degree heart block & acute inferoposterior MI – matches the territory of the idioventricular rhythm well. [Cath showed occluded mid-RAD → stented].

Case 6: AJ

History: 18-yr-old female. Three episodes of palps, lasting up to 40 min, triggered by intense cardiovascular exercise.

ECG: Vent rate 230 BPM, PR interval * ms, QRS duration 66 ms, QT/QTc 190/371 ms, P-R-T axes * 81 -78; Narrow-complex tachycardia, with widespread ST changes. QRS alternans. Probable P waves visible in the ST segments / T waves (best seen in Lead III) – negative in inferior leads, positive in V1, negative in V6 – makes most likely diagnosis AV reentrant tachycardia with left-sided accessory pathway. [Terminated with Valsalva manoeuvre. No preexcitation on baseline ECG].

Case 7: SM

History: 38-y-o male with episodes of palpitation, breathlessness & dizziness.

ECG: SM1 Vent rate 269 BPM, PR interval * ms, QRS duration 190 ms, QT/QTc 186/393 ms, P-R-T axes * 179 0; Wide-complex tachycardia. No visible atrial activity. Rapid initial forces & typical RBBB pattern – suggests possible SVT with aberrant conduction.

ECG: SM2 Vent rate 130 BPM, PR interval * ms, QRS duration 80 ms, QT/QTc 256/376 ms, P-R-T axes * 46 11 Wide-complex tachy, then narrow-complex tachy, then ventricular couplet, then slower irregular rhythm. When slows, flutter waves are visible (typical flutter). Flutter cycle length matches the rate on the prior ECG – i.e. confirms that this was 1:1 AV conduction. The ventricular couplet most likely causes period of AV nodal delay, allowing flutter waves to be seen.

Case 8: DL

History: 60-yr-old male with severe dilated cardiomyopathy. Conscious & talking during this ECG(!). Leads are appropriately placed and no electrical interference is present.

ECG: VF. [LVAD in place & history of recurrent VT / VF.] Measurements: irrelevant!

Case 9: AM

History: 39-yr-old female. Episodes of rapid palpitation. Nausea, vomiting. Elevated white cell count & pleural effusions.

ECG: Vent rate 158 BPM, PR interval 112 ms, QRS duration 68 ms, QT/QTc 240/389 ms, P-R-T axes 62 98 -10; SR, then narrow-complex tachycardia. Independent P waves are visible. QRS complexes are identical to conducted sinus beats. Junctional tachycardia.
[Should check electrolytes, serum magnesium & digoxin level (in case of accidental or deliberate overdosage)].

Case 10: JW

History: 75-yr-old male with prior anterior MI 1998. Poor LV function with extensive scar. Admitted with chest discomfort.

ECG: Vent rate 129 BPM, PR interval 41 ms, QRS duration 192 ms, QT/QTc 396/580 ms, P-R-T axes * 98 -36; Wide-complex rhythm with discernible independent P waves (best seen in Lead II rhythm strip). Atypical RBBB QRS morphology, with positive concordance across chest leads. VT.
[For amio & ICD].

Case 11: VT

History: 23-yr-old male. Recurrent palpitation.

ECG: VT1 Vent rate 264 BPM, PR interval * ms, QRS duration 140 ms, QT/QTc 138/289 ms, P-R-T axes * 182 0; Wide-complex irregular tachycardia, with slurred QRS upstroke & some variation in QRS morphology. No visible atrial activity. Preexcited AF. Shortest RR interval <200 ms – increased risk of VF / sudden death.

ECG: VT2 Vent rate 98 BPM, PR interval 146 ms, QRS duration 114 ms, QT/QTc 352/449 ms, P-R-T axes 71 82 69; ECG after DC cardioversion. SR. Normal PR interval, but subtle preexcitation – seen best in leads V3 & 4. WPW syndrome. Pathway most likely far from sinus node (i.e. left lateral) hence PR not short & most ventricular activation is over the normal conduction system. [Ablation of the pathway was performed].

Case 12: DB

History: 37-yr-old male with cardiac surgery one year previously for an ASD and partially anomalous pulmonary venous drainage. Presented with palpitation and exertional syncope.

ECG: Vent rate 152 BPM, PR interval 118 ms, QRS duration 154 ms, QT/QTc 324/515 ms, P-R-T axes * 82 -63; Narrow-complex regular tachycardia, with rate suspicious for atrial flutter with 2:1 AV conduction. Lead II demonstrates flutter waves. This could be typical atrial flutter, although he also may have potential circuits for scar-related macroreentry due to his surgery.

The ECG also suggests RVH.

[Syncope was likely related to 1:1 conduction of his flutter. Periods of 1:1 conduction were indeed seen on a Holter].

Chapter 4 - Heart Block & Pacemakers

Case 1: LV

History: 81-y-o male, presented with acute CP.
Angio showed 80% prox & 100% mid RCA lesions → PPCI with bare metal stent. CHB resolved with reperfusion.

ECG: Sinus rate 100, vent rate 44 BPM, PR interval *ms, QRS duration 122 ms, QT/QTc 478/408 ms, P-R-T axes * 69 107; complete heart block, junctional escape rhythm, acute inferoposterior MI.

Case 2: PT

History: 76-y-o female, at time of hip replacement.

ECG: SR, 4:3 & 3:2 Wenckebach, vent rate 49 BPM, PR interval 186 ms, QRS duration 98 ms, QT/QTc 490/442 ms, P-R-T axes 38 -8 -21. 1st conducted beats of each cycle narrow (likely due to longer recovery time for left bundle), others with LBBB & leftward axis. LVH, possible old anteroseptal MI (no evidence of this on echo).

Case 3: DC

History: 13-y-o boy with congenital heart block (maternal SLE with anti-Ro/SS-A antibodies). NB - junctional escape rate increased to peak of 125 bpm on treadmill – PPM not required at this time.

ECG: SR, with some ventriculophasic sinus arrhythmia, CHB, junctional escape. Vent rate 50 BPM, PR interval * ms, QRS duration 112 ms, QT/QTc 412/375 ms, P-R-T axes * 32 53. Large precordial voltages are normal for this age.

Case 4: FK

History: 81-y-o man admitted with dizziness, presyncope, chest pain. Moderate diffuse CAD on angio with ectatic vessels.

ECG: FK1 SR, Mobitz type 1 block, then junctional escape beat with retrograde P wave which resets the sinus node (slight delay to next P wave). Vent rate 49 BPM, PR interval * ms, QRS duration 92 ms, QT/QTc 450/406 ms, P-R-T axes 51 65 129. Non-specific anterior T wave inversion.

ECG: FK2 Dual chamber paced rhythm. QRS morphology suggests RV apical pacing site. Short paced AV delay (around 160 ms) – could be lengthened to minimize ventricular pacing & preserve battery life. Vent rate 60 BPM, PR interval 102 ms, QRS duration 150 ms, QT/QTc 468/468 ms, P-R-T axes 13 -78 93.

ECG: FK3 Dual chamber paced rhythm with longer AV delay (260 ms) showing pseudofusion. (AV delay could be lengthened even further).

Case 5: LM

History: 62-y-o male. Asymptomatic. Very fit – runs & cycles 50-60 km / week. Holter showed very frequent PACs and runs of atrial ectopy, intermittent marked first degree heart block, Mobitz type 1 and type 2 AV block (mostly nocturnal), junctional beats and ventricular ectopy. No indication for PPM – reasonable to follow with serial Holters & reassess if becomes symptomatic.

ECG: SR with marked 1st degree heart block (PR 600 ms); vent rate 56 BPM, PR interval 600 ms, QRS duration 92 ms, QT/QTc 438/422 ms, P-R-T axes * 41 35.

Case 6: GN

History: 64-y-o diabetic smoker, with CAD & CHF. Exercise intolerance & episodes of presyncope. Required pacing - LV EF was measured to determine whether ICD required - was 50%, therefore pacemaker alone was implanted.

ECG: SR, with 2:1 AV conduction ("high-grade AV block") and ventriculophasic sinus arrhythmia, vent rate 35 BPM, PR interval 314 ms, QRS duration 144 ms, QT/QTc 574/438 ms, P-R-T axes 83 266 104. Conducted beats have RBBB & likely LAFB (bifascicular block).

Case 7: RF

History: 33-y-o male with bicuspid aortic valve with severe AS & AR, s/p complete aortic root replacement, ascending aorta replacement & hemi-arch replacement. ECG performed day 1 post-op in cardiac ICU. Conduction did not recover & PPM was placed.

ECG: Vent rate 80 BPM, PR interval * ms, QRS duration 182 ms, QT/QTc 450/519 ms, P-R-T axes * 0 -80. VVI pacing at start of tracing with complete heart block (note independent P waves), pacing then turned off – P waves with no conduction & no escape rhythm.

Case 8: BM

History: 86-y-o male with reduced exercise capacity.

ECG: Vent rate 65 BPM, PR interval * ms, QRS duration 160 ms, QT/QTc 462/480 ms, P-R-T axes * -80 88. VVI pacing with sinus rhythm in the atrium and complete heart block.

Case 9: JB

History: 66-y-o male admitted with NSTEMI. Intermittent CHB noted on monitoring. VDD pacemaker implanted.

ECG: Vent rate 99 BPM, PR interval 196 ms, QRS duration 148 ms, QT/QTc 394/505 ms, P-R-T axes 89 -68 114. Sinus rhythm, A-sensing, V-pacing. The ST changes cannot be interpreted in the presence of ventricular pacing.

Case 10: AC

History: 75-yr-old female with PPM. Complains of fatigue.

ECG: VVI pacing with failure to capture. Second QRS complex on the rhythm strip is not sensed or falls in the blanking period after the prior pacing spike. Underlying ECG shows possible junctional rhythm (or sinus bradycardia with PR prolongation and P waves masked by the pacing artefacts), leftward axis, and widespread T wave changes – possible "memory T-wave changes" secondary to long-term ventricular pacing.

Case 11:

History: Patient with history of dizzy spells & syncope. CSM performed.

ECG: Sinus rhythm, with sinus slowing, then sinus arrest – cardioinhibitory response. Pacemaker is indicated if recurrent syncope & no other cause identified.

Case 12: MT

History: 54-year-old female with diabetes & hypertension, presenting with recurrent unheralded syncope.

ECG: MT1 Vent rate 54 BPM, PR interval 224 ms, QRS duration 138 ms, QT/QTc 492/466 ms, P-R-T axes 23 -57 -8. Sinus bradycardia with 1st degree heart block, RBBB & left anterior fascicular block (bifascicular block).

ECG: MT2 The first strip appears to show sinus bradycardia, but based on the P-P interval on the second strip, this is sinus rhythm with 2:1 AV conduction ("high-grade" AV block). Complete heart block then occurs, without an escape rhythm. The sinus rate increases in the last strip. External pacing was employed, then implantation of a permanent system.

Chapter 5: Nonspecific ST changes

Case 1: 73 year old female with gastric hemorrhage: Sinus Rhythm at 90 with changes consistent with hypokalemia (K = 2.4 mmol//L)

Case 2: 40 year old male for routine physical. Early repolarization; normal variant.

Case 3: Sinus bradycardia at 40 bpm; PR 0.20, Generalized nonspecific ST-T changes, consistent with digitalis effect. Serum digoxin level (3.4 nmol/dL - toxic)

Case 4a: 83 year old male with an "unusual heart": Sinus rhythm. P waves inverted in I and aVL. Axis is in the right upper quadrant. Poor R wave progression across precordial leads consistent with dextrocardia.

Case 4b: Same patient as Case 4: Chest leads are across the right chest and limb leads are reversed - P wave is upright in leads I and aV, and there is now normal progression of the precordial voltage. Consistent with dextrocardia.

Case 5: History of chronic renal failure. QRS is very wide (about 300 msec.), P waves not readily identified consistent with sinoatrial block, and peaked T waves are all changes consistent with hyperkalemia (K^+ - 8.2)

Case 6: Same patient as Case 5: Post therapy: Sinus rhythm with minor T wave changes, and left axis deviation (QRS axis -60^0) (K^+ -5.6)

Case 7a: Arm leads are reversed – Differential of "p" inversion in leads I and aVL includes dextrocardia, and ectopic atrial rhythm.

Case 7b: ECG redone: Leads now positioned correctly. HR 46; PR .16, QRS 0.10 , QT .42, QTc- 0.37, Axis -15

Case 8: 21 year old female with syncope. Sinus rhythm with long QT interval (Romano Ward Syndrome - long QT with normal hearing) HR- 50 bpm, PR-0.14 sec., QRS-0.1sec., QT-0.68sec., QTc-0.56sec., Axis- +50°
These patients can have Torsade de pointes and syncope or sudden death.

Case 9a: Sinus bradycardia (42bpm) QT interval – 0.44 sec QTc- 0.37), elevated ST and T waves. Osborne waves (just after QRS complex) consistent with hypothermia. These changes should resolve with warming.

Case 9b: Sinus rhythm at 90 bpm, PR- 0.18, QRS- 0.10, QT- 0.38, QTc- 0.47, axis- 60^0
The J wave (Osborne wave) is no longer present.)

Case 10: Stab wound to the pericardium. Sinus rhythm with changes of pericarditis. There may be PR depression in leads V4, aVF, and I

Case 11: Unconscious in the ER. Sinus bradycardia. Marked T wave inversion with QT prolongation. Consistent with history of subarachnoid hemorrhage. Anterolateral ischemia or infarction is possible.

Case 12: Sinus rhythm at 70 bpm, PR- 0.18, QRS- 0.09 msec, QT- 0.42 sec, QTc- 0.45. Axis- $+130^0$ P pulmonale. Overall consistent with RVH and chronic lung disease.