Routine Interpretation

a. Rate & Rhythm
b. P-wave
c. PR interval
d. QRS interval
e. QRS complex & mean axis
f. ST segment
g. T wave
h. U wave
i. QT interval

Normal ECG

a. ***sinus rhythm*** - rate 60-100 bpm
   - 2 types of sinus arrhythmia,
   i. rate ***increases*** with ***inspiration***
   ii. no relationship to respiration

b. ***P wave***
   i. ***duration*** is argued - accept ≤ 0.11s
      - if bifid, < 0.04 s apart
      - if bifid, > 0.04 s suggests ***LA hypertrophy***
   ii. upright in - I, II, aVF, $V_{4\text{-}6}$
   iii. inverted in - aVR
   iv. ***amplitude*** < 3 mm in any lead
      > 3 mm in inferior leads suggests ***RA hypertrophy***

c. ***T_P wave***
   - atrial repolarisation, usually hidden in the QRS complex
   - broad, low voltage, usually opposite polarity to P wave, cf. the T wave
   - may be visualised in CHB

d. ***PR interval***
   - beginning of P wave to start of QRS
   - range 0.12-0.2 s, use the longest interval present
   - decreases with increasing HR
   - causes for a ***short*** PR interval,
   i. normal variant
   ii. ectopic atrial rhythms
   iii. WPW or LGL syndrome

e. ***PR segment*** - end of the P wave to the start of the QRS
- usually isoelectric
- may be elevated in atrial infarction or acute pericarditis

f. ***QRS complex - duration*** > 0.12 s is abnormal

i. ***ectopic ventricular mechanism*** - PVC's
- ventricular escape beats
- VT
- idioventricular rhythm
- accelerated idioventricular rhythm
- ventricular parasystole
- paced ventricular rhythm

ii. ***slowed ventricular conduction*** - intraventricular conduction block
- aberrant ventricular conduction

iii. ***accelerated conduction to one ventricle*** - WPW syndrome

g. ***QRS complex - amplitude*** * variable due to sensitivity etc. (see LVH)
- < 5 mm average in I, II, III abnormal
- < 10 mm average in precordial leads abnormal

h. ***Q wave***
- small, narrow Q in I, aVL, aVF, and V_{4-6} is normal
- > 0.03 s is suggestive
- effects of respiration, especially in inferior leads

i. ***QRS complex - axis***
- normally -30° to +90°, not unanimous
- transitional zone in precordial leads, normally V_{3-4}
- relative prominance of component waves
- normal progression of inferior leads

j. ***QRS complex - intrinsicoid deflection***
- onset of QRS to R wave peak normally < 0.02s in V_1
  < 0.04s in V_4
- prolongation implies a delay in conduction,

i. dilatation

ii. hypertrophy

iii. conduction disease

k. ***ST segment***
- from the J-point (take-off from the QRS) to the onset of the T wave
- range in limb leads -0.5 to + 1 mm
- range in precordial leads -0.5 to + 2 mm

l. ***T wave***

- usually upright in - I, II, V_{4-6} (ie. lateral chest leads)
  - V_{2-3} variable, V_3 in young males
- usually inverted in - aVR
- variable in others

m. ***QT duration***

- from the onset of the QRS to the end of the T wave
- in normal SR, usually < ½ the preceeding RR interval
- prolongation of the QT = ***delayed repolarisation***

i. congenital syndromes
- Jervell-Lange-Nielsen (auto-R, 1:100 deaf)
- Romano-Ward (auto-D, not deaf)
- familial VT

ii. electrolyte disturbances
- hypokalaemia (?)
- hypocalcaemia
- hypomagnesaemia

iii. drugs
- class Ia and Ic antiarrhythmics - quinidine, procainamide, disopyramide
- class III antiarrhythmics - amiodarone, sotalol
- psychotropic agents - phenothiazines, TCA's
- local anaesthetics - bupivacaine

iv. CNS disease
- SAH, ICH
- cryptococcal meningitis (?)

v. myocardial ischaemia / cardiomyopathy

vi. arrhythmias
- post-tachycardia syndrome
- cardiac arrest of any aetiology
- chronic idioventricular rhythms (inc. pacing)

- reduced QT interval
  - digoxin
  - hypercalcaemia
  - hyperkalaemia

n. ***U wave***

- genesis is uncertain
- often best seen in V_3, same potarity as the T wave
- influenced by many variables, especially increased in ***hypokalaemia***
- inverted in - LV overload
  - anterior wall ischaemia (often with absence of other signs)

Acute Myocardial Infarction

a. ***hyperacute***
   - i. increased ventricular activation time **qR > 0.04s**
   - ii. ST elevation - upsloping or concave-up
     ~ 80% of AMI
     - maximal at 2-4 hours
   - iii. T-wave tall and wide
   - iv. ST depression & T inversion ~ 10-25%

b. ***evolution***
   - i. ST elevation - convex-up
     plus - pathological **Q-wave > 2 mm, > 0.04s, > 25% R-wave**
     - onset at 1-3 hours, maximal at 12 hours
   - ii. T-wave flattening (early) or inversion (after 12-24 hours)

c. ***resolution***
   - i. Q-wave > 2-4mm, > 0.04s, may disappear
   - ii. ST-segment - isoelectric in 2-4 weeks
     * persistent elevation → ***aneurysm***
   - iii. T-wave - normal in 1-6 months
     - inversion may persist

- ***Diagnostic Criteria***

1. ST elevation > 1.0 mm → 2 adjacent limb leads V_4-V_6
2. ST elevation > 2.0 mm → V_1-V_3

- ***Posterior AMI***

a. V_1 & V_2 - tall R wave
   - tall & wide T-wave
   - ST depression

b. ± inferior changes of AMI

c. absence of other cause for ↑ V_1R

- ***RV Infarction***

NB: ST elevation > 1mm in any of **V_{4-6}R**
~ 90% specific in the presence of inferior AMI

■ ***AMI & LBBB***

- data from the GUSTO I trial
- factors independently predictive of AMI with LBBB,

1.	ST elevation ***concordant*** with QRS	> 1 mm	5 pts
2.	ST depression in V_{1-2-3}	> 1 mm	3 pts
3.	ST elevation discordant with QRS	> 5 mm	2 pts

- Sgarbossa *et al* NEJM 1996 used point score ≥ 3 pts for treatment →
  a. sensitivity ~ 40%
  b. specificity ~ 96%

AMI - Location by Q Waves

Location	Leads	Pseudo-Infarct Patterns
Anteroseptal	V_{1-2}	• WPW, RVH, early repolarisation • ? LVH, LBBB, CAL (R-Tompson)
Anterolateral	I, aVL, V_{4-6}	• LVH, LBBB, HOCM, VSD
Extensive Anterior	I, aVL, V_{1-6}	
High Anterolateral	I, aVL	
Apical	V_{2-4}	
Inferior	II, III, aVF	• WPW type B, HOCM, ?PTE
Inferoposterior	II, III, aVF V_1 - tall R wave	• WPW type A, HOCM
Posterolateral	V_1 - tall R wave V_{4-6}	• child, athlete, RBBB • WPW type A, HOCM, dextrocardia • Duchenne muscular dystrophy
Other causes of Pseudo-infarction		• incorrect lead placement • pericarditis • Prinzmetal angina

Normal Q-Wave		Pathological Q-Wave	
R-wave height	< 25%	R-wave height	> 25%
Height	< 2 mm	Height	> 2 mm
Duration	< 0.04 s	Duration	> 0.04 s
Appearance		Appearance	
• Normal	V_{5-6}, aVR	• Cardiac	AMI, etc
• RAD, vertical heart	II, III, aVF	• Extracardiac	PTE, CAL, etc.
• LAD, horizontal heart	I, aVL	* see over	

- ***Causes of Q-Waves***

1. infarction
2. ischaemia without infarction
3. ventricular hypertrophy - left or right
4. abnormal conduction
   - hemiblocks
   - pre-excitation
   - LBBB (complete or incomplete)
5. cardiomyopathy
   - hypertrophic
   - idiopathic
6. myocardial disease
   - myocarditis
   - amyloidosis
   - infiltration with tumour
   - sarcoidosis
7. extracardiac causes
   - PTE
   - CAL
   - pneumonia
   - pancreatitis

- ***Q Waves in Lead III***

1. normal variant
2. old inferior AMI
   > 0.04s
   + Q's in II, aVF
   + small R
3. pulmonary embolism
4. left posterior hemiblock
5. nodal rhythm

Myocardial Ischaemia

a. ***acute ischaemia***
   i. none ~ 50%
   ii. ST depression - horizontal or sagging
   iii. ST elevation - Prinzmetal's angina
   iv. LBBB
   v. ventricular ectopics ± VT, VF
   vi. AV block

b. ***Prinzmetal's variant angina***
   i. upsloping ST segment elevation > 2mm
   ii. tall wide T-wave
   iii. increased ventricular activation time - qR > 0.04s

c. other less common features
   i. tall R and deep S wave
   ii. transient LAHB
   iii. transient AV block
   iv. U wave inversion

Hypertrophy

NB: problems of poor ***sensitivity*** and ***specificity***

- ***LV Hypertrophy***

1.	SV_1 or V_2 + RV_5 or V_6	> 35 mm	(40)
2.	R or S in any limb lead	> 20 mm	
3.	RV_5 or RV_6	> 25 mm	(27)
4.	R + S in any V lead	> 45 mm	

- numbers in brackets from LIGW
- additional features stated,

1.	deepest R in V_{1-2-3}	> 13 mm	?? S
2.	R in aVL	> 13 mm	
3.	R in aVF	> 20 mm	

- ***RV Hypertrophy***

1. reversal of precordial pattern
   - V_1R > V_1s - age < 5yrs, posterior AMI
   - V_1R > V_2R - anterior AMI
   - V_1R > 0.9 mV - posterior AMI, WPW, dextrocardia
2. QRS interval within normal limits
3. late intrinsicoid deflection in V_{1-2}
4. right axis deviation - qR or rSR' in V_1
5. strain pattern in RV or leads with dominant R wave

- LIGW states on of the following 2 patterns,

1. RAD (or S_1, S_2, S_3 syndrome)
   incomplete RBBB - QRS < 0.12s
   clockwise rotation
2. Rs in V_1
   ST depression & T-wave inversion V_{1-2-3}
   deep S in V_{5-6}

Features of Bundle Branch Blocks		
	Left BBB	**Right BBB**
I	monophasic R, no Q, or, wide notched rR' waves	wide S
V_1	qS or rS	late intrinsicoid deflection M-shaped QRS (rSR' variant) sometimes wide R or qR
V_6	monophasic R, no Q, or, wide notched rR' waves late intrinsicoid deflection	wide S, early intrinsicoid deflection
Causes:		
	• always pathological • ischaemic heart disease • hypertensive heart disease • cardiomyopathy	• normal ~ 2% • tachycardia • acute PTE, RV strain, RVH • ischaemic heart disease • myocarditis or cardiomyopathy • CAL • ASD
General Features		
• prolonged QRS > 0.12s • rSR or qR pattern in appropriate chest leads * I, V_1, V_6 • qRS or rS pattern in the "reciprocal" chest leads • secondary ST segment changes • T wave inversion • axis deviation ***is not*** a necessary criterea		

- ***Causes of rSR' Variants in V_{1-2} QRS < 0.12S***

1. normal in ~ 5% of young people
2. frequently associated with ***pes cavum***, or straight back deformities
3. incomplete RBBB
4. RV hypertrophy
5. acute cor pulmonale
6. RV diastolic overload
7. WPW syndrome
8. Duchenne muscular dystrophy

- ***Causes of Dominant R Waves in V_{1-2}***

a. occasionally a normal variant
b. children < 5 years
c. RV hypertrophy
d. RBBB
e. true posterior or lateral infarction
f. WPW syndrome - type A
g. LV diastolic overload
h. HOCM
i. Duchenne muscular dystrophy

HemiBlocks

Left Anterior Hemi-Block	Left Posterior Hemi-Block
Left axis deviation < -60°	Right axis deviation > +120°
small Q in I, aVL small R in II, III, aVF	small R in I, aVL small Q in II, III, aVF
late intrinsicoid deflection > 0.045s in **aVL**	late intrinsicoid deflection > 0.045s in **aVF**
↑ QRS voltage in limb leads	↑ QRS voltage in limb leads
normal QRS duration	normal QRS duration
	no evidence of RVH (exclusion)
Conditions Mimicked	
anterior AMI lateral AMI LVH	anterior AMI
Conditions Masked	
anterior AMI inferior AMI LVH RBBB	anterior AMI
Causes	
ischaemic heart disease cardiomyopathy anterolateral AMI ostium primum ASD	***rare*** RBBB + ***LAHB*** → CHB ~ 10% RBBB + ***LPHB*** → CHB **~ 100%**

Axis Deviation

Left Axis Deviation		Right Axis Deviation	
normal	~ 2% of population	normal	> 2% of population
LBBB		RBBB	
LAHB		LPHB	
WPW	* type B = RV anomalous pathway	WPW	* type A = LV anomalous pathway
AMI	* inferior	AMI	*anterolateral
hyperkalaemia		acute PTE	
ASD	*ostium primum	dextrocardia	

- ***Left Axis Deviation***

1. normal variant ~ 2% of population
2. LBBB
3. LAHB
4. WPW syndrome - RV path, ***type B***
5. inferior AMI
6. hyperkalaemia
7. ASD - ostium primum defect

- ***RAD***

1. normal variant ? >2% of population
2. RBBB
3. LPHB
4. WPW syndrome - LV path, ***type A***
5. anterolateral AMI
6. pulmonary emblous
7. dextrocardia

Aberrant Conduction

NB: three types,

i. fascicular refractoriness
ii. anomalous supraventricular activation - WPW, LGL
iii. paradoxical critical rate

- ***Features of Aberration***

1. triphasic contours - rsR' in V_1
   - qRs in V_6 ~ 90% specific for aberration
2. preceeding atrial activity
3. initial deflection identical with conducted beat if RBBB
4. second-in-the-row anomalous beat
   i. ***Ashman's phenomenon***
      - the refractory period is related to the previous cycle length
      - $\therefore$ an early beat following a long cycle is more likely to be aberrantly conducted $\rightarrow$ right bundle still refractory
      - however, be aware of,

   ii. ***rule of bigemini***
      - an ectopic beat is likely to occur following a pause
      - however, this is likely to occur at lower heart rates than Ashman's phenomenon
5. alternating BBB patterns separated by a single normally conducted beat

- ***Harrison***

NB: $\rightarrow$ Ventricular origin more likely with,

1. QRS ***duration*** > 0.14s
2. morphology ***not typical*** of RBBB or LBBB
3. AV ***dissociation*** or variable retrograde conduction
4. ***superior axis*** $\rightarrow$ NW quadrant
5. QRS ***concordance*** in the precordial leads (ie. all +'ve or all -'ve)

- ***Distinguishing VT from SVT with Aberration***

1. ***AV dissociation*** * useful
   - absence is not helpful
   - ~ 50% of VT have retrograde VA conduction
   - use clinical information, cannon waves, variable S_1, plus ECG
   - not infallable, junctional tachycardia with block & AV dissociation occurs (rarely)
2. ***fusion beats*** * useful but rare
   - usually at slower rates
   - although rarer, aberrantly conducted junctional beats can also fuse with sinus beats
3. ***capture beats***
   - occur early in the cycle & are also rare and at slower rates
   - need for more than one lead
4. ***QRS morphology***
   i. **V_1**
      - rS or Q wave
        + a slick downstroke to an early intrisicoid deflection (≤ 60 msec)
        → 90% specific for LBBB aberrancy
      - rsR' → 90% specific for RBBB aberrancy
      - LV ectopics usually produce positive deflection
        → monophasic R or qR 90% specific for ventricular origin

   ii. **V_6**
      - LV ectopics ~ 70% rS
      - may occur in RBBB + LAHB
      - QS more diagnostic but less common

   iii. ***concordance***
      - positive: - LV ectopy DD_X: WPW type A
      - negative: - RV ectopy DD_X: LBBB & late transition

   iv. ***frontal plane axis***
      - in north-west quadrant DD_X: complex CHD, multiple MI's

   v. ***QRS duration*** > 0.14s

   vi. ***RVT*** - specific case
   = RAD, LBBB in V_6 plus broad, small rV_1
5. ***clinical features*** - including age

NB: irregularity of arrhythmia ***incorrect***, but frequently quoted
most paroxysmal arrhythmias are regular, VT or SVT

Broad Complex Tachyarrhythmias		
	Ventricular Tachycardia	Supraventricular Tachycardia
H_X	• elderly • chest pain, dyspnoea • past/recent AMI	• history WPW
E_X	• cannon waves • variable S_1 intensity • pulmonary oedema	• reduction in ventricular rate with carotid sinus massage
BP	• ***hypotension*** with beat/beat variability	• hypotension less common
HR	• < 170	• > 170 bpm
QRS	• > 140 msec	• < 140 msec
ECG	• capture/fusion beats rare • ***AV dissociation*** / VA conduction • ***concordance*** across chest leads • usually regular, but - VT + capture beats - AF + accessory pathway • ***extreme LAD*** = VT, may have RAD	• normal axis or RAD • capture/fusion beats diagnostic
RBBB - Like Pattern		
V_1: **V_6:** Axis:	• monophasic/biphasic **Rs** • initial deflection = SR • tallest = 1st • S wave > R wave **rS** • often < -30°	• triphasic **rsR'** • different to SR • tallest = second • S < R **Rs** • usually > -30°
LBBB - Like Pattern		
V_1: **V_6:** V_{2-6}: Axis:	• R wave > 40 msec • QS or QR • negative deflection > V_1 • LAD or RAD	• rS or Q with sharp downstroke • triphasic • smaller • normal, RAD rare
Dr Vohra, "if in doubt treat as VT"		

ECG Changes with CNS Disease

1. bradycardia
2. T wave - flattening or inversion
3. ST segment - depression
4. U waves
5. prolonged QT_C
6. ventricular ectopics

NB: tumour, trauma, SAH, post-operatively, infection
acute onset, may last for up to 2 weeks

Afterload & Preload

a. ***increased afterload*** (systolic overload)
   i. tall ± inverted T waves
   ii. ST depression
   iii. "strain" pattern

b. ***increased preload*** (diastolic overload)
   i. tall, peaked T waves
   ii. deep Q waves
   iii. ST elevation * left ventricle
   iv. RBBB * right ventricle

Apparent "Bigeminy"

1. ventricular ectopics - parasystole
   - frequent ectopics
2. atrial or nodal ectopics
3. 3:2 block
   i. AV block - type I & type II
   ii. SA block - type I & type II
   iii. atrial tachycardia or flutter, with alternating conduction (eg. 2:1 and 4:1)
4. nonconducting atrial trigeminy
5. concealed AV extrasystoles every 3rd beat
6. reciprocal beating

Atrial Bradyarrhythmias

- ***Atrial Ectopics***

    1. different P wave morphology
    2. PR interval usually greater than normal
    3. normal QRS ***or*** rate dependent RBBB
    4. compensatory pause

- ***SA Wenckebach***

    1. P-P (and R-R) interval shortens
    2. then absent sinus beat

- ***SA Block***

    1. P-P interval is in multiples of basic P-P interval
    2. results in dropped beats
    3. significant if > ***3s pause***

    NB: ***sinus pause*** = SA block for > 2x P-P interval

- ***Sinus Arrest***

    Def'n: P-P interval is greater than basic P-P interval, but not a simple multiple

- ***Wandering Atrial Pacemaker***

    1. P waves of different morphology
    2. PR interval shorter than normal

Atrial Tachyarrhythmias

- ***Differential Diagnosis of Atrial Fibrillation***

1. multiple atrial ectopics
2. atrial flutter
3. MAT

- ***Atrial Flutter***

1. aetiology
   - i. ischaemia, infarction
   - ii. myocarditis, cardiomyiopathy
   - iii. drugs - inotropes, β agonists
     - rarely digoxin toxicity
   - iv. rheumatic heart disease
   - v. thyrotoxicosis
   - vi. ASD
2. symptoms
   - i. sudden onset palpitations, dyspnoea, light-headed
   - ii. underlying heart disease
3. signs
   - i. regular tachycardia ~ 150 / min
   - ii. hypotension
   - iii. JVP apparent loss of 'a' wave, often with LVF
   - iv. may present as progressive cardiac failure
   - v. decreased rate with carotid sinus massage but ***not reversion***
4. ECG
   - i. atrial rate 250-350/min - flutter waves in II
   - ii. variable AV block - ventricular rate 150-100/min
   - iii. decreased ventricular rate with carotid sunus massage
5. management
   - i. stable haemodynamic state
     - slows ventricular rate - IV digoxin, Verapamil, β-blocker
     - revert to SR - Flecainide, Procainamide, Amiodarone
   - ii. unstable haemodynamics - cardioversion, IV digoxin
   - iii. refractory - Amiodarone followed by cardioversion
     - overdrive atrial pacing

- ***Multifocal Atrial Tachycardia***

1. rate > 100 bpm
2. P waves of at least 3 different morphologies, not of SA node
3. irregular PP, PR, and RR intervals
4. ? rapid form of wandering atrial pacemaker

- ***Paroxysmal SVT***

1. aetiology
   i. WPW
   ii. ischaemia
   iii. myocarditis
   iv. alcohol
   v. emotional upset
   vi. idiopathic
2. ECG
   i. regular rapid rate ~ 140-250 / min
   ii. abnormal P waves but fixed relation to QRS
   iii. may have rate-dependant RBBB → ***aberrancy***

Atrial Fibrillation

- ***Chronic***

1. ischaemic heart disease* **comonest causes*
2. mitral stenosis*
3. hypertensive heart disease
4. cardiomyopathy

- ***Paroxysmal***

1. thyrotoxicosis*
2. WPW
3. pericarditis
4. pulmonary embolus
5. AMI
6. hypoxia
7. viral myocarditis
8. myocardial contusion
9. drugs
   - alcohol, inotropes, β agonists, theophylline, caffeine
   - rarely digoxin toxicity
10. others
    - idiopathic
    - chronic pericarditis
    - ASD, cor pulmonal, acute right heart strain
    - post-cardiothoracic surgery
    - MVP, atrial myxoma

"Sinus" Tachycardia & Anaesthesia

1. inadequate depth of anaesthesia, pain
2. hypovolaemia, hypotension
3. hypoxia, hypercarbia, acidosis
4. hyperthermia
5. sepsis
6. drug induced
   i. sympathomimetic, parasympatholytic, reflex
   ii. idiosyncratic, anaphylactic
   iii. toxic *digitalis → PAT & 2:1 block
7. malignant hyperpyrexia
8. thyroid storm
9. congestive cardiac failure
10. acute pulmonary thromboembolism
11. "apparent" sinus tachycardia
    i. atrial flutter with 2:1 block
    ii. paroxysmal atrial tachycardia
    iii. AV reentry tachycardia - SVT ± aberration

NB: degree of haemodynamic compromise,
impaired coronary perfusion and 2° ischaemia,
differentiation when rate ~ 150 bpm → vagal manoeuvres, edrophonium

- ***Management***

1. treat underlying cause
2. vagal manoeuvres
3. IV verapamil
4. β-blockade
5. metaraminol
6. edrophonium
7. digitalisation
8. overdrive pacing
9. DC cardioversion

AtrioVentricular Block

1. **1st Degree** = prolonged PR interval (> 0.22 s)
2. **2nd Degree**
   - i. **Mobitz I** - progressive lengthening ***PR interval***
     - progressive shortening of the ***RR interval***
     - culminating in a dropped beat
     = ***Wenckebach***
     - caused by disease at the AV node (high)
     - rarely causes significant bradycardia, or progresses to higher degree block
   - ii. **Mobitz II** = fixed PR & RR intervals (long) with regular dropped beats
     - caused by disease below the AV node, ie. the bundle of His
     - may be associated with anterior AMI
     - ventricular rates can be quite slow, with dyspnoea, syncope & fatigue
     - frequently progresses to a higher level of block & ***requires pacing***
     - pacing ***does not*** alter 60-70% mortality rate ∝ native disease
3. **3rd Degree** = complete AV dissociation
   - ventricular 'escape' rhythm should be regular
   - beware SR bradycardia with junctional escape & occasional capture beats

- ***Causes of AV Block***

1.	congenital	- frequently have associated VSD's
2.	ischaemic heart disease	
3.	myocarditis / cardiomyopathy	
4.	drug induced	- digoxin, β-blockers, halothane, CEB's
5.	post cardiac surgery	
6.	sclerodegenerative	* HPIM, possibly the commonest cause of ***isolated*** CHB
	i. Le***v***'s disease	- also affects ***v***alves, skeleton
	ii. Lenegre's disease	- affects the conducting system only
7.	fibrosis	- longstanding aortic stenosis - chronic rheumatic carditis
8.	tumours	
9.	granulomatous disease	
10.	incidental	* some fit young adults have 1°HB or Wenckebach

- ***Main Determinants of A-V Conduction***

1. state of the AV junction and bundle branches
   i. physiological refractoriness
   ii. pathological refractoriness
2. autonomic nervous system influences
3. atrial rate
4. R-P relationships
   - refractory period is determined by the preceeding cycle length
5. ventricular rate
6. level of the ventricular pacemaker (concealed AV conduction may occur)

Associations of Second Degree AV Block

Characteristic	Mobitz Type I	Mobitz Type II
Clinical	usually acute inferior MI rheumatic fever digoxin toxicity propranolol	usually chronic anteroseptal MI Lenegre's disease Lev's disease cardiomyopathy
Anatomical	usually AV node rarely His bundle	always sub-nodal usually bundle branches
Electrophysiological	relative refractory period decremental conduction	no relative refractory period all-or-none conduction
Electrocardiograph	RP / PR ***reciprocity*** prolonged PR normal QRS duration	stable PR normal PR bundle branch block

- ***RP-PR Reciprocity***

• the AV node has a relatively ***short absolute*** and a ***long relative*** refractory period
• the deeper into the relative refractory period an impulse occurs, the longer it takes to get through the node
• ∴ the closer an atrial impulse to the prior ventricular beat, the more refractory will be the AV node, and the longer the PR interval to the next ventricular beat
• hence, the PR interval is ***inversely*** or ***reciprocally*** related to the preceding RP interval
• when the P-wave occurs very close to the prior QRS, the absolute refractory period of the AV node is reached → non-conducted PACs

Cor Pulmonale

1. RVH
2. RAD
3. RBBB
4. P pulmonale
5. rS pattern in lateral chest leads
6. T wave inversion in chest leads
7. sloping PR interval
8. low voltages

Dextrocardia

1. large R in V_1
2. inverted P, QRS, and T waves in lead I → ***mirror*** of leads I, aVL
3. rS complex in V leads

NB: swapped arm leads results in mirror of I & aVL, but V-leads normal

Digoxin Effect

1. down-sloping ST segment depression
2. T wave inversion, or decreased amplitude
3. U wave
4. prolonged PR interval
5. widening of QRS
6. shortening of QT_C
7. arrhythmias
   - SVT with AV block (PAT & 2:1)
   - 2° or 3° HB
   - VE's, bigeminy, VT

Early Beats - Causes

1. ***extrasystole***
   i. different P wave morphology - if atrial or nodal ectopic
   ii. different QRS morphology - if ventricular
   iii. regular coupling interval
   iv. compensatory pause
2. ***parasystole***
   i. protected focus
   → ECG complex when chamber is responsive, none when refractory
   ii. the inter-ectopic interval is usually an integer multiple of the ***shortest*** ectopic-ectopic interval
   iii. variable coupling interval ? variable compensatory pause
   iv. fusion beats
3. capture beats - including supranormal conduction during AV block
4. reciprocal beat
5. better (eg. 3:2) interrupting poorer (eg. 2:1) AV blockade
6. rhythm resumption following inapparent bigeminy

- ***Underlying Pathology***

1. normal variant
2. bradycardia
3. drugs * β-blockers, digoxin, diuretics
4. ischaemic heart disease
5. hypertensive heart disease
6. valvular heart disease
7. hypokalaemia

Causes of Bradycardia

1. sinus bradycardia
2. non-conducted atrial bigemini
3. SA block, 2nd & 3rd degree
4. AV block, 2nd & 3rd degree
   - which may be associated with a number of supraventricular rhythms

Causes of Pauses

1. non-conducted atrial extrasystoles
2. 2nd degree AV block
   i. type I - Wenckebach (1899)
   ii. type II - Wenckebach (1906), and Hay (1906)
3. 2nd degree SA block - type I & type II
4. concealed conduction
5. concealed AV extrasystoles

Causes of Bigemini

NB: extrasystolic, supraventricular, or ventricular

1. due to 3:2 block
   i. SA block - type I & type II
   ii. AV block - type I & type II
   - atrial tachycardia or flutter with alternating conduction (eg. 2:1 / 4:1)
2. non-conducting atrial trigeminy
3. concealed atrial extrasystoles every third beat
4. reciprocal beating

Causes of Chaos

1. atrial fibrillation
2. atrial flutter or PAT with varying AV conduction
3. MATor wandering atrial pacemaker
4. multiple VEB's
5. parasystole
6. marked sinus arrhythmia
7. combinations of the above

Causes of Regular Rhythms at Normal Rates

a. sinus rhythm
b. accelerated AV nodal rhythm
c. accelerated idioventricular rhythm
d. atrial flutter with 4:1 block
e. sinus or supraventricular tachycardia with 2:1 block
f. ventricular tachycardia with 2:1 (exit) block

Re-entry versus Ectopic Tachycardia

- ***Re-Entry:***

a. acceleration is absent
b. initial P wave differs from subsequent P waves
c. premature stimulus does not reset, but may terminate the arrhythmia
d. prolongation of the first PR interval is usual

- ***Ectopic Automatic***

a. presence of warm-up, or ***acceleration***
b. all P waves, including the first, are the same
c. premature stimulus resets the tachycardia

AV Dissociation

Mechanism	Diagnosis
slowing of SA node	• sinus bradycardia
acceleration of subsidiary pacemaker	• accelerated idiojunctional rhythm • accelerated idioventricular rhythm • junctional or ventricular tachycardia
post-extrasystolic pause & escape	• atrial, junctional, or ventricular extrasystole
combinations of the above	

Antiarrhythmics

Vaughan Williams'				
Class		Electrophysiology		Examples
I.	Na^+-Channel Blockers			
	Ia.	↓ ↓↓ ↑	phase 0 conduction repolarisation	quinidine disopyramide procainamide
	Ib.	↔,↓ ↓ ↓	phase 0 conduction repolarisation	lignocaine phenytoin tocainide, mexiletine
	Ic.	↓↓↓ ↓↓↓ ±	phase 0 conduction repolarisation	flecanide
II.	β-blockers			
III.	Prolong Repolarisation	↑↑	repolarisation	amiodarone bretylium, sotalol
IV.	Calcium Entry Blockers			verapamil

Group Ia Effects

a.	increase most ECG intervals	* PR, QRS, QT, T wave
b.	QRS width proportional to dose	
c.	U wave and decreased T amplitude	~ same as for hypokalaemia
d.	in toxic doses	- all degrees of heart block - VT, torsade

NB: most effects are ***proportional*** to the dose

Hyperkalaemia

i. ↑ PR interval
ii. peaked T waves
iii. widening of the QRS
iv. ↓ R wave height
v. loss of ST segment
vi. loss of P wave
vii. loss of U wave
viii. occasionally left axis deviation

NB: decreased resting V_M & decreased v_c
can look like "bradycardia + 1°HB + RBBB"

Hypokalaemia

i. ST depression
ii. flattened or inverted T waves
iii. U waves * ***apparent*** increase in QT duration
- prominent U wave, flat T wave produces long "QU", which appears as QT
- QT actually ***not increased***
- looks like ***hypocalcaemia,*** which does increase QT but doesn't predispose to arrhythmias as does hypokalaemia

iv. prolonged PR
v. arrhythmias - SA block
- VE's, VT, torsade, VF

NB: increased resting V_M & increased v_c
decreased rate of repolarisation, digoxin toxicity, tachyarrhythmias

Hypothermia

i. shivering muscle tremor artifact
ii. sinus bradycardia
iii. J point elevation - commences < 33°C
- proportional to prolongation of QRS

iv. prolongation of PR & QT intervals
v. arrhythmias

< 34°C	AF
< 33°C	J point elevation
< 30°C	1°HB
< 28°C	VF
< 20°C	3°HB, asystole

Swapped Right & Left Arm Leads

NB: = lead I inverted and leads II & III swapped

1. inverted complexes in I
2. R wave in III > R wave in II
3. absence of tall R wave in V_1 * ie. not dextrocardia

Atrial Hypertrophy

- ***P Pulmonale***

1.	peaked P wave	- II, III, aVF > 2.5 mm
2.	cor pulmonale	
3.	PTE	
4.	RVF	
5.	TS	

- ***P Mitrale***

1.	wide P wave	> 0.12s
2.	bifid or notched P wave	> 0.04s → I, II, aVF, aVL
3.	biphasic P wave	- V_1 , with predominantly ***negative*** deflection
4.	IHD	
5.	MS	
6.	hypertensive heart disease	

Low Voltages

1. incorrect calibration
2. obesity
3. emphysema
4. pericardial effusion
5. hypothermia
6. myxoedema

P Wave Abnormalities

- ***Absent P Waves***

  i. SA block
  ii. AF
  iii. hyperkalaemia
  iv. nodal rhythm

- ***Inverted P Waves In Lead I***

  i. incorrect arm leads
  ii. dextrocardia
  iii. nodal rhythm
  iv. ectopic atrial rhythm - low atrial focus

- ***Dissociated P Waves & QRS Complexes***

  i. 3°HB
  ii. AV dissociation
  iii. ventricular parasystole
  iv. VT
  v. VE's

Pericarditis

1. stage I - concave ST elevation in most leads, except V_1 & aVR
2. stage II - ST return to baseline
   - PR prolongation
   * absent in many cases
3. stage III - widespread T wave inversion
   - similar appearance to myocarditis
4. stage IV - slow return to normal

NB: variants: - PR segment depression "apparent ST elevation"
- permenant T wave inversion
- ST elevation in only a few leads

- ***Chronic Pericarditis***

  1. low voltages
  2. low voltage T waves, isoelectric, or inverted

PR Interval

Def'n: 0.12-0.2 s

- ***Prolonged PR 1st Degree Heart Block***

1. IHD
2. cardiomyopathy, myocarditis
3. BBB
4. drugs - digoxin, β-blockers, Ca^{++} channel blockers, class Ia
5. hyperkalaemia
6. rheumatic fever
7. rarely a normal variant

- ***Short PR***

1. pre-excitation - WPW / LGL
2. nodal rhythm - inverted P wave in I
3. AV dissociation - apparent short PR

Pulmonary Embolism

1. normal ECG * ie. no change
2. sinus tachycardia - common finding
3. S_I, Q_{III}, T_{III} - rare finding, due to right axis shift
4. right axis deviation
5. RBBB - partial or complete
6. deep S wave in V_5 & V_6
7. T wave inversion in V_{1-3} - RV "strain"
8. tall P wave in II - P pulmonale

QT Interval

a. ***normal***
   i. best measured in aVL
   ii. roughly < ½ RR interval
   iii. $QT_C = QT / \sqrt{RR}$ < 0.44 s female
   < 0.40 s male

b. ***short QT***
   i. hypercalcaemia
   ii. digoxin

c. ***long QT***
   i. congenital LQTS
   ii. AMI
   iii. cardiomyopathy
   iv. myocarditis
   v. MVP
   vi. electrolyte disorders - $\downarrow Ca^{++}$, $\downarrow Mg^{++}$
   - $\downarrow K^{+}$ results in ***apparent*** $\uparrow$QT
   vii. drugs - antiarrhythmics (Ia, Ic, III)
   - tricyclics, phenothiazines
   - lithium
   - bupivacaine
   viii. hypothermia
   ix. CVA
   x. neck surgery ? sympathetic imbalance

Sick Sinus Syndrome

Def'n: ***symptomatic*** bradyarrhythmias,
sometimes interrupted by atrial tachyarrhythmias

1. SA block - SA block for exactly 2 x PP interval
2. sinus arrest - lack of sinus beat for > PP interval
   ± slower escape rhythm
3. sinus pause - SA block for > 2 x PP interval
4. brief episodes of AF, PAT, or atrial flutter
   • these often precede the bradyarrhythmia

ST Elevation

1. AMI
2. pericarditis * concave
3. aneurysm
4. early repolarisation ≤ 2 mm
5. coronary spasm * Prinzmetal's angina

ST Depression

1. myocardial ischaemia
2. digoxin effect
3. ventricular "strain"
4. hypokalaemia
5. non-specific

T Wave Changes

a. ***normal***
   i. > 2mm & < ½ R wave height
   ii. negative in V_1 and V_2

b. ***peaked T wave*** * > ½ R wave height
   i. hyperkalaemia
   ii. subendocardial infarction
   iii. acute ischaemia
   iv. posterior AMI - V_1 and V_2

c. ***inverted T waves***
   i. ischaemia
   ii. infarction
   iii. "strain"
   iv. digoxin effect
   v. hypokalaemia
   vi. pericarditis

U Waves

a. ***prominent U wave*** = > 1 mm, or > T wave height
   i. normal ~ 80%
      - usually in V_{2-3}
   ii. hypokalaemia
   iii. hypomagnesaemia
   iv. hyperthyroidism
   v. bradycardia
   vi. CNS disease
   vii. LVH
   viii. drugs - class I antiarrhythmics
      - tricyclics, phenothiazines
      - digoxin

b. ***inverted U wave***
   i. hypertensive heart disease
   ii. ischaemic heart disease
   iii. anterior ischaemia

Wolf-Parkinson-White Syndrome

a. short PR interval < 0.12s

b. delta wave - pre-excitation of the ventricle

c. tall R wave in V_1 - LV pathway, ***type A***

d. may simulate
   i. AMI
   ii. RVH
   iii. BBB's

e. normal ECG
   i. distal LV free wall anterograde pathway, or
   ii. rapid AV conduction, or
   iii. retrograde accessory pathway only

f. paroxysmal SVT
   i. antegrade AV node ~ 95%
      - orthodromic tachycardia, narrow QRS
   ii. retrograde AV ~ 5%
      - antidromic, wide complex tachycardia

g. AF with rapid ventricular rate > 220 bpm

Wolf-Parkinson-White Accessory Pathways			
	Bidirectional Path	Retrograde Path "concealed"	Multiple
LV free wall	42%	20%	10%
Septal	20%	9%	30%
RV free wall	6%	1.5%	
Undetermined	2%		