

ECG interpretation

Mechanics of tracing:

- Small box = 1 x 1 mm & Large box = 5 x 5 mm
- Paper speed (horizontal boxes)
 - Standard speed = 25 mm/sec
 - Small block will be $1 \div 25 = 0.04$ sec
- Voltage calibration (vertical boxes)
 - Standard = 10 mm/mV (2 big boxes)
 - Half standard = 5 mm/mV (1 big box)
 - May have 10/5: standard for chest leads, half for precordial leads
- Note the calibration!!

1. HEART RATE

a. Measure in beats per minute

- 60/RR interval in seconds or
- 300/number of big boxes between consecutive QRS complexes or
- 1500/number of little boxes between consecutive QRS complexes

- b. Atrial and ventricular heart rates** may be different due to complete heart block or atrial flutter – so calculate separately (Parks p47)

Newborn	145	90-180
6 months	145	105-185
1 yr	132	105-170
4 yrs	108	72-135
14 yrs	85	60-120

c. Causes of sinus tachycardia

Any condition causing/requiring increased cardiac output

Infection	Anxiety
Fever	Pain
Hypervolemia	Carditis
Anaemia	Cardiac failure

d. Causes of supraventricular tachycardia

Metabolic	Drugs
Hypo/hyperkalaemia	Digoxin
Hypocalcaemia	Cocaine
Hypomagnesaemia	Caffeine
Hypoxia	Antiarrhythmics
Acidosis	Procainamide
Thyrotoxicosis	Amiodarone
Myocardial abnormalities	Channelopathies
Myocarditis	Long QT syndrome

Cardiomyopathy	Brugada syndrome
ARVD	
Muscular dystrophy	
Post-op scars	
Neoplasm	

e. Sinus tachycardia vs. supraventricular tachycardia

SINUS	SVT
HR <200 (exceptions)	HR >200 (usually)
Normal P waves	P waves abnormal (if discernible)
HR variation in response to stimulation	No beat to beat variation
Slows gradually in response to treatment	Sudden onset and termination

2. RHYTHM

- Sinus rhythm
- Subsidiary pacemaker
- Tachyarrhythmia
- Bradyarrhythmia
- Atrioventricular block

For normal sinus rhythm

- P wave before every QRS
- QRS following every P wave
- Normal P axis (0 to +90)
 - For P axis to be between 0 to + 90 degrees: P waves must be upright in leads I & aVF or at least not inverted in these leads
 - Normal PR interval is not required

3. QRS axis

Hexa axial reference system

- Bipolar limb leads
 - I, II, III
- Augmented unipolar leads
 - aVR, aVL, aVF

Horizontal reference system

- Precordial leads
 - V1-V7
 - Right sided leads (e.g. V4R)

Axis determination

- Calculate amplitude vector as follows
- Add net R-S in lead I, R-S in aVF
- Plot in mm on the grid (lead I horizontal, lead aVF vertical)
- Draw vector from origin to net amplitude
- Angle of vector = cardiac axis

Normal QRS Axis (Parks)

1 wk to 1 month	+110	+30 to +180
1-3 months	+70	+10 to +125
3 m to 3 yrs	+60	+10 to +110
> 3yr	+60	+20 to +120
Adult	+50	-30 to +105

Right axis deviation

- Axis >100 degrees significant in adults (see above table for normal reference range in children according to age)
- Suggestive of RVH

Causes:

- RVH
- TAPVR
- ASD secundum type (rsR pattern in V1 and aVR)
- RBBB
- Mitral stenosis
- TOF

Left axis deviation

- Axis < - 5 degrees
- Q waves in leads I & aVL
- Conduction abnormality
- Associated with atrioventricular septal defect
- No correlation with LVH
- Occurs in 5% of normal population

Causes

- Normal variant
- AV septal defect (including primum ASD)
- Perimembranous inlet VSD
- Tricuspid atresia
- Single ventricle
- Double outlet right ventricle
- Noonan syndrome
- Left anterior hemiblock after MI

Superior QRS / NW axis

S wave greater than R wave in aVF and lead I

It may occur with

- Left anterior hemiblock (in the range of -30 to -90) seen in
 - a. ECD – in asplenic syndrome
 - b. Tricuspid atresia
- RBBB

4. T axis

- Mean T axis is +45 with the mean between 0 to +90 degrees
 - T waves must be upright in leads I and aVF.
 - T waves can be flat but not inverted
- T axis outside the normal quadrant suggest conditions with myocardial dysfunction
 - Severe ventricular hypertrophy with strain
 - Ventricular conduction disturbances
 - Ischaemic or metabolic myocardial dysfunction

5. PR INTERVAL

- Onset of atrial contraction to onset of ventricular contraction (measures cumulative time of depolarization through atria, AV node, His-Purkinje system)
- Varies between leads
- Increases with age
- Decreases with heart rate

Normal PR interval: 0.07 to 0.18 (Parks p49)

<1 y	0.075 – 0.14
1-3 y	0.080 – 0.15
3-5 y	0.085 – 0.18
5-12 y	0.09 – 0.18
12-15 y	0.095-0.18
Adult	0.12 – 0.18

Prolongation of PR interval

1. First degree AV block
2. Drugs: Digitalis, Quinidine toxicity
3. Atrial surgery (scar tissue)
4. Acute rheumatic fever (minor Jones criteria)
5. Kawasaki disease
6. Myocarditis
7. Hyperkalaemia
8. Ebstein's anomaly
9. Normal heart with vagal stimulation

Short PR interval

1. Wolff-Parkinson-White syndrome
2. Glycogen storage disease type IIa (Pompe's disease)
3. Fabry's disease
4. GM1 gangliosidosis
5. Friedrich's ataxia
6. Duchenne Muscular Dystrophy
7. Lown-Ganong-Levine syndrome
8. Pheochromocytoma

6. QRS DURATION

- Normal QRS duration: 0.05-0.085 sec

0 m – 3 yr	0.05-0.075
>3 yr	0.6-0.75
Adults	0.085

- Beginning of Q wave to end of S wave
- Use a lead where a Q wave is visible
- Normal = 0.04 – 0.08 (may be up to 0.09 in adolescents)
- >0.12 = bundle branch block
- 0.10 – 0.12: evaluate morphology
- QRS duration prolonged in ventricular conduction disturbances

1. RBBB 2. LBBB 3. Pre excitation (WPW) 4. Myocardial fibrosis 5. Ventricular arrhythmias 6. Implanted ventricular pacemaker	7. Metabolic or ischaemic myocardial dysfunction 8. Intraventricular block <ul style="list-style-type: none"> • Hyperkalaemia • Toxicity from Quinidine • Procainamide
---	--

- RSR' morphology seen in right precordial leads: V1, rV3
- Commonly occurs in 7% of kids
- R and R' both small and short duration
- S wave larger than R and R'
- R' is less than 10 mm (15 mm in infants)
- Abnormal RSR' may reflect:
 - RBBB
 - RVH (volume overload type) – RSR in V1
 - ASD (primum and secundum type) - RSR in V1

7. QT INTERVAL – calculated as corrected QT (QTc)

- Onset of ventricular depolarization (Q wave) to end of ventricular repolarization (T wave)
- Do not include U waves
- Varies inversely with heart rate
- Best leads: II, V5, V6

Bazett's formula: $QT_c = QT / \sqrt{RR}$

- Normal 0.38 – 0.47
- May be as high as 0.45 sec in adolescents/adult females
- May be as high as 0.49 sec in newborns (to 6 months)

Long QT interval

<ol style="list-style-type: none">1. Long QT (congenital) syndrome2. Hypocalcaemia3. Myocarditis4. Hypertrophic & dilated cardiomyopathy5. Head injury6. Severe malnutrition7. Jervall-Lang-Nielson8. Romano-Ward	Drugs <ol style="list-style-type: none">1. Ia and III antiarrhythmics2. Phenothiazines3. TCA4. Antibiotics<ul style="list-style-type: none">○ Erythromycin○ Amantadine○ Co-trimoxazole○ Ampicillin
--	---

Short QT interval

- Digitalis
- Hypercalcaemia
- Hyperthermia
- Short QT syndrome – familial cause of sudden death with QTc <0.30 sec

8. CHAMBER HYPERTROPHY

Right atrial hypertrophy

Lead II – tall P wave >2.5 mm = P Pulmonale

Causes:

1. Bronchitis or chronic obstructive pulmonary disease (COPD)
2. Tricuspid stenosis
3. Mitral stenosis
4. Pulmonary embolism
5. Tricuspid regurgitation
6. Mitral regurgitation

Left atrial hypertrophy

- Lead II – widened and often notched P wave of duration:
 - >0.08 sec in infants – P mitrale
 - >0.1 sec in children
- In V1, P wave is diphasic with prolonged negative segment

Causes:

1. Obesity
2. High BP
3. Mitral stenosis
4. PDA (moderate to large shunt)
5. VSD (moderate to large shunt)

Bilateral atrial hypertrophy

- Combination of increased amplitude and duration of P wave
- Causes:
 - D-TGA with VSD or PS
 - L-TGA

Right ventricular hypertrophy

- RAD for patient's age
- Pure R waves >12 mm in V1 (Parks >10 mm)
- R wave with an S wave > 20 mm in V1 (> 29 in <1 month)
- Upright T wave in V1
- Q wave in V1 & V4R

Causes

1. Pulmonary stenosis/severe peripheral PA stenosis (William's, Rubella)
2. Mitral stenosis
3. TOF
4. TAPVR

Left ventricular hypertrophy

- Left axis deviation for patient's age (not always)
- R wave in V6 >30 (>20 1st yr)
- R wave in V6 + S wave in V1 >50 mm
- Deep/wide Q waves >5 mm in V5, V6
- Tall symmetric T waves = "LV diastolic overload"

Causes

1. Aortic stenosis
2. Hypertension
3. PDA
4. VSD
5. Tricuspid atresia

Biventricular hypertrophy

In the absence of BBB and pre-excitation

- In V3, R > 30 mm with S wave > 30 mm
- In V4, R > 40 mm with S wave > 20 mm
- Large equiphasic QRS complexes in two or more of the limb leads and mid precordial (V2-V5) leads – "Katz-Wachtel" phenomenon

Causes:

1. Eisenmenger's syndrome
2. ECD complete
3. Persistent truncus arteriosus
4. VSD large shunt
5. PDA – large shunt (Parks p176)

9. CONDUCTION DEFECTS

Right bundle branch block

- Prolongation of QRS duration in the terminal portion ("terminal slurring")
- Delayed conduction through RBB prolongs depolarization of RV
- RAD

- Terminal slurring R' in aVR and V1, V2, V3r
- ST segment shift, T wave inversion (in adults)
- Intraventricular block – prolongation throughout the duration of QRS complex
- Pre-excitation with delta waves

Causes:

1. ASD/PAPVR
2. Right ventriculotomy
3. Ebstein's anomaly
4. Coarctation (<6 months)
5. Brugada syndrome
6. Myocardial disease
 - Cardiomyopathy
 - Myocarditis
7. Muscle diseases
 - Duchenne muscular dystrophy
 - Myotonic dystrophy

Brugada syndrome

- Sudden unexpected death syndrome
- Mutations in Na channels
- Abnormal ECG without abnormality in structural pattern of heart
- ECG shows RBBB with J point elevation and concave ST elevation
- Patient may present with blackout or palpitation
- There may be family history of sudden death
- CVS examination is usually normal

Left bundle branch block

- Prolonged and slurred portion of QRS complex leftward
- QRS looks like M in I & V6 and W in V1 & AVR
- Wide S waves in V1, V2
- Absent Q wave in V6 (may also be in lead I & V5)
- Prominent QS pattern in V1
- Tall R wave in V6
- ST depression & T wave inversion in V4, V6 is common

Wolff-Parkinson-White syndrome

- Pre-excitation: initial slurring of QRS
- Accessory conduction pathway
 - Premature polarization of part of the myocardium
 - Slow conduction delta wave
- Criteria
 - Short PR interval for age
 - Delta wave
 - Wide QRS for age

Pre-excitation syndromes

- Lown-Ganong-Levine syndrome
 - Short PR interval
 - Normal QRS duration
 - Fibers pass upper AV node, but conduct normally
- Mahaim fiber
 - Normal PR interval
 - Long QRS duration
 - Delta wave
 - Fiber bypasses His bundle, enters RV myocardium

Mechanism of WPW

- Recurrent episodes of abnormal rapid heart beats
- Extra muscle bundle with electrical pathway between atrial Kent bundle or ventricle (Mahaim fiber), results in rapid heart beat
- Most of Kent fibers conduct faster than AV node, so atrial depolarization transmitted to ventricles prematurely results in short PR interval
- Rapid conduction along the accessory pathway results in:
 - Short PR interval <0.12
 - A broad QRS complex 0.12 or more with delta wave and often abnormal T wave
- Pre-excitation refers to early depolarized muscle, this results in delta wave
- Congenital in nature
- May be functional at birth and became dormant
- Higher incidence in Ebstein's anomaly and mitral prolapse

10. ST CHANGES

Non pathological ST-segment shift

- Elevation or depression of up to 1 mm in the limb leads
- And up to 2 mm in the pre-cordial leads – is within normal limits

Pathologic ST segment shift / T wave changes

- Abnormal shifts of ST segment are often accompanied by T-wave inversion
- Downward slant followed by a diphasic or inverted T wave
- Horizontal elevation or depression sustained for more than 0.08 seconds

Causes:

1. LVH & RVH with strain
2. Digitalis effect
3. Post-operative state
4. Myocarditis
5. Myocardial infarction
6. Hypokalaemia or hyperkalaemia

ECG changes in myocardial infarction

- Wide Q waves (>0.035 sec with or without Q wave notching)
- ST segment elevation (>2 mm)
- Prolongation of QTc interval (>0.48 sec) with abnormal q waves

Causes of myocardial infarction in children

1. Atherosclerosis
2. Inflammatory disease of the myocardium
3. Lupus erythematosus
4. Syphilis
5. Polyarteritis nodosa
6. Hypertension
7. Diabetes mellitus
8. Endocardial fibroelastosis
9. Anomalous origin of left coronary artery from pulmonary artery
10. Kawasaki disease
11. Dilated cardiomyopathy
12. Coronary artery embolization resulting from:
 - Infective endocarditis or
 - Surgical procedures performed on the left side of the heart