

MacPeds

Pediatric ECG Survival Guide

Second Edition

2019



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This guide is dedicated to my fellow MacPeds residents!

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Approach of ECG Interpretation:

Various approaches of ECG interpretation exist; listed below is a common one.

Approach:

1. Identification information: Name, age, date, indication of the ECG.
2. Calibration and paper speed.
3. Heart rate.
4. Rhythm.
5. Cardiac axis.
6. Intervals.
7. Wave amplitude.
8. Morphology.
9. Repolarization phase (ST segment & T wave).

Normal ECG variations in pediatrics:

ECGs of the normal pediatric population are different from those of normal adults. Many differences are due to the right ventricular dominance in infants, and the evolution to adult dynamics.

Listed below are the features that you may encounter in pediatric ECGs in comparison to adult ECGs:

- Faster heart rate.
- Sinus arrhythmia.
- Rightward QRS axis (up to 3 months and again in adolescence).
- T wave inversions in the right precordial leads (RPLs).
- Dominant R wave in V1.
- RSR' pattern in V1.
- Shorter PR interval and QRS duration.
- Slightly longer QTc.

Chest electrode positions:

V1: 4th intercostal space at right sternal border.

V2: 4th intercostal space at left sternal border.

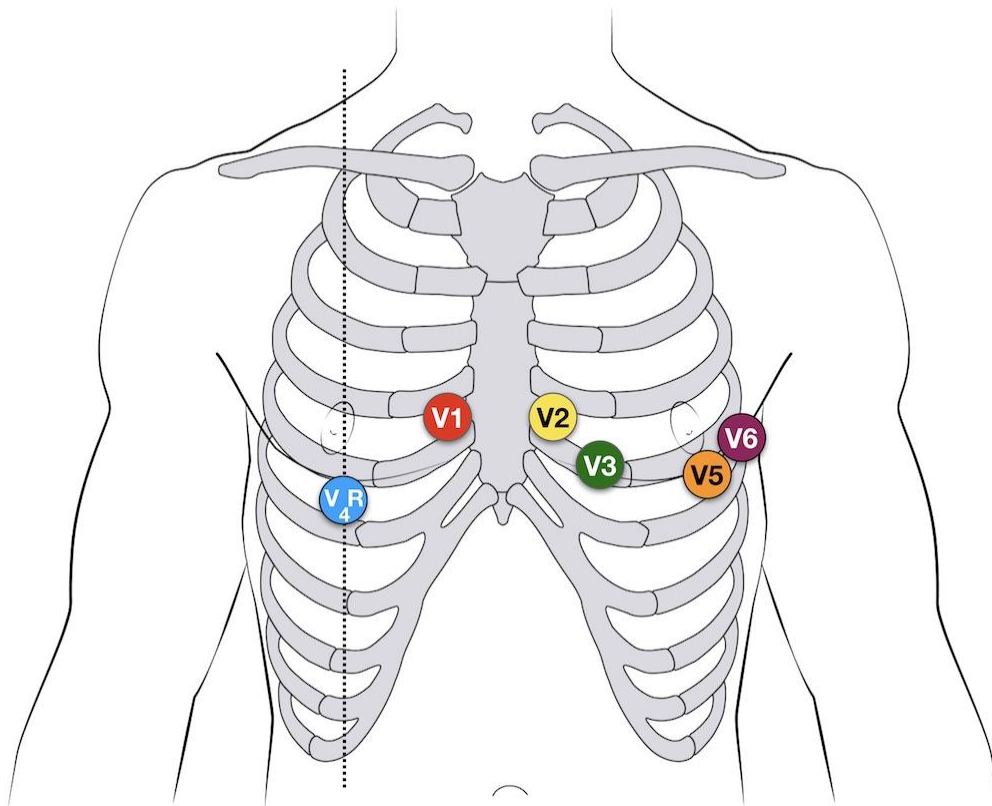
V3: Midway between V2 and V4.

V4: 5th intercostal space at midclavicular line.

V5: Anterior axillary line at the level of V4.

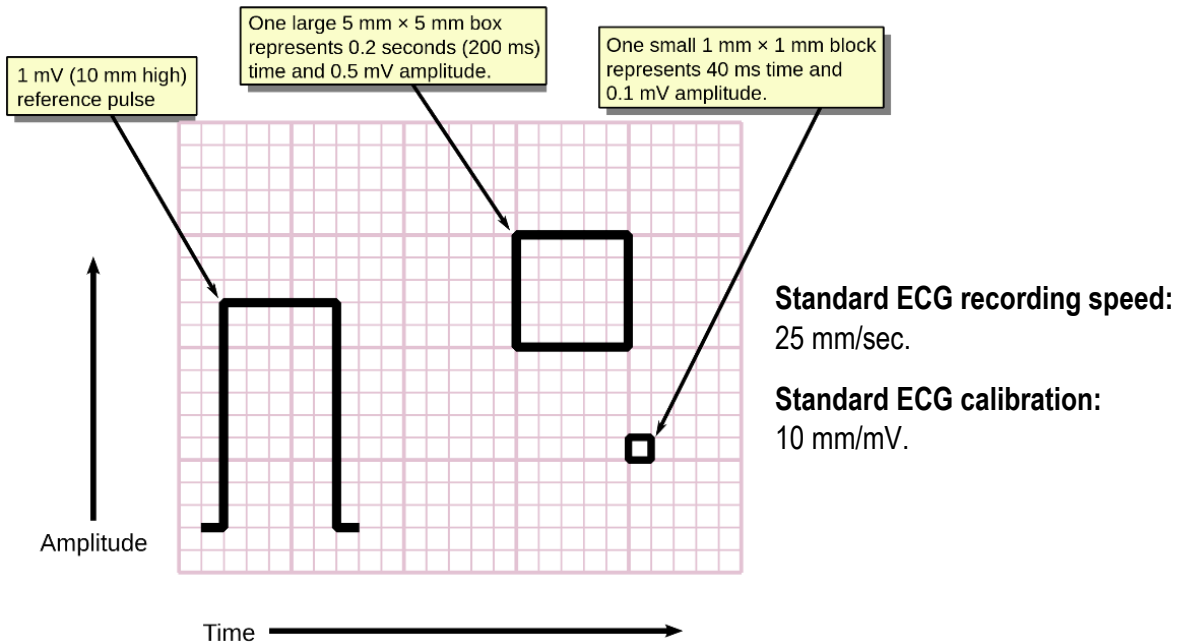
V6: Mid -axillary line at the level of V5.

V4R: Same position of V4 but on the right side.



Ref: <https://litfl.com/paediatric-ecg-interpretation-ecg-library/>

Calibration and paper speed:



Ref: <http://a-fib.com/treatments-for-atrial-fibrillation/diagnostic-tests/the-ekg-signal/>

Heart rate:

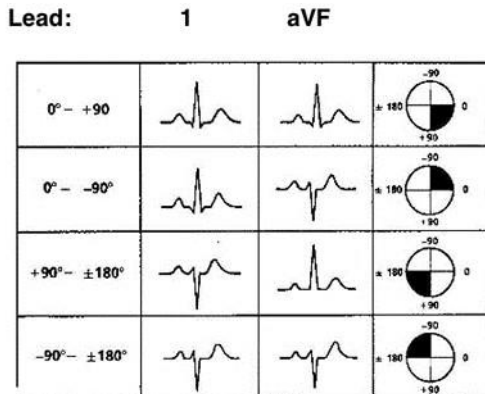
Calculation:

- **Regular rhythm:**
 - 300/number of large squares between 2 consecutive R waves.
 - 1500/number of small squares between 2 consecutive R waves.
- **Irregular rhythm:**
 - Multiply the number of QRS complexes on the rhythm strip by 6.

Age	HR
Newborn ≤ 1 wk	120-160
Newborn ≤ 1 mo	120-160
Infant ≤ 1 yr	110-140
Toddler 1-3 yr	90-130
Preschool 3-5 yr	90-120
Child 6-12 yr	80-110
Adolescent > 12 yr	70-100
Adult > 18 yr	60-100

Ref: MacPeds Survival Guide, 2018

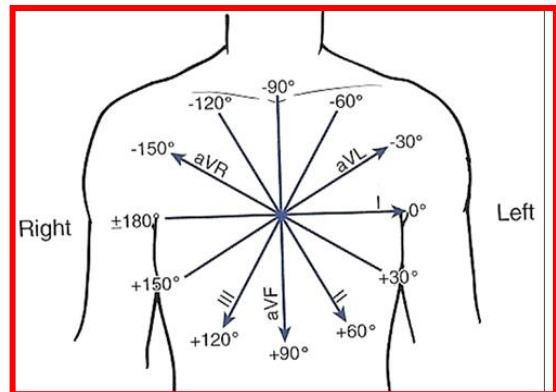
Cardiac (QRS) axis:



1. Use **lead I** and **aVF** to locate a quadrant.
2. Find a lead with an **equiphaseic QRS complex** (height of R equal to depth of S).
3. The QRS axis will be **perpendicular** to this lead in the **previously determined quadrant**.

Ref: Park's, the pediatric cardiology handbook, 5th edition.

- Full-term newborn: $+135^\circ$ (range $+58^\circ$ to $+168^\circ$).
- 1 week – 1 month: $+110^\circ$ (range $+65^\circ$ to $+159^\circ$).
- 1 month – 3 months: $+75^\circ$ (range $+31^\circ$ to $+115^\circ$).
- 3 months – 6 months: $+60^\circ$ (range $+7^\circ$ to $+105^\circ$).
- 6 months – 1 year: $+54^\circ$ (range $+7^\circ$ to $+98^\circ$).
- 1 year – 3 years: $+55^\circ$ (range $+8^\circ$ to $+100^\circ$).
- 3 years – 5 years: $+55^\circ$ (range $+7^\circ$ to $+104^\circ$).
- 5 years – 8 years: $+66^\circ$ (range $+10^\circ$ to $+140^\circ$).
- 8 years – 12 years: $+61^\circ$ (range $+9^\circ$ to $+115^\circ$).
- 12 years – 16 years: $+58^\circ$ (range $+11^\circ$ to $+133^\circ$).



Ref: Davignon et al, 1980.

A quick and fairly accurate method to determine the QRS axis for older children:

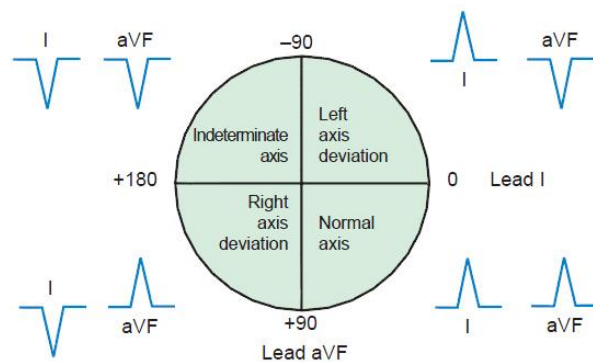
Look at **Lead I** and **aVF**:

- If both (+) → normal axis.
- If lead I (+) and aVF (-) → LAD.
- If lead I (-) and aVF (+) → RAD.
- If both (-) → extreme axis deviation.

Causes of QRS axis deviations:

RAD causes: RBBB, RVH.

LAD causes: AVSD, tricuspid atresia, LBBB, left anterior hemiblock, WPW (type b), LVH, ccTGA.



Ref: <https://soperedi.files.wordpress.com/2013/08/a0129-1.jpg>

In a patient with **Down syndrome** having LAD, suspect **AVSD** (ECHO is needed!).

Rhythm:

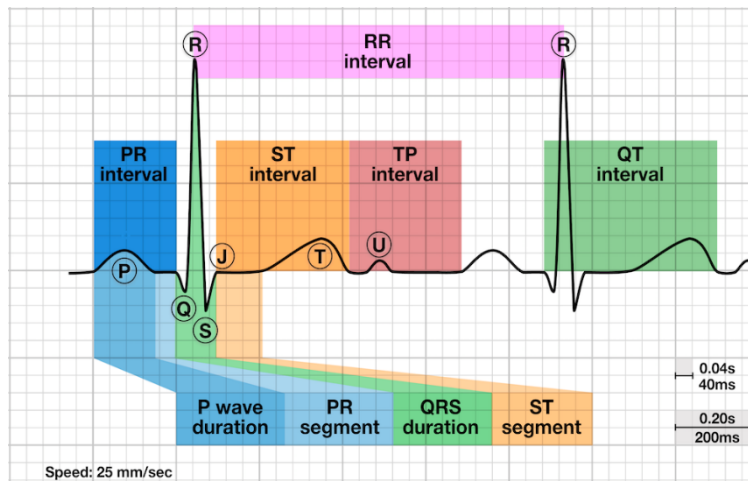
Look at the rhythm strip to examine the rhythm (often lead II or V1).

Regular: Constant RR interval.

Sinus (the 3 criteria should be met):

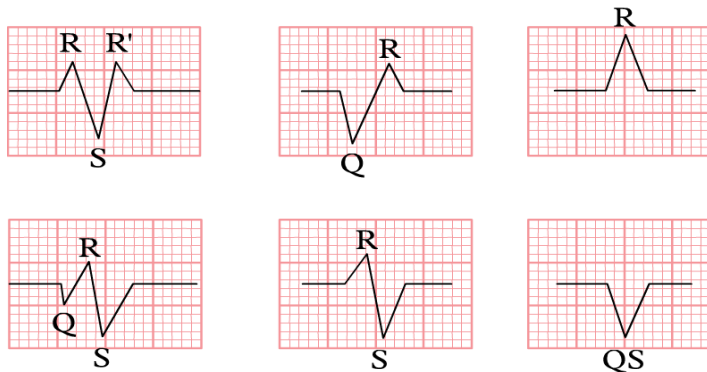
1. P wave preceding each QRS complex (i.e., QRS complex after every P wave).
2. Constant PR interval.
3. Normal P wave axis – refer to “P wave axis” section in page 8.

Waves, segments and intervals:



Ref: <https://litfl-wpengine.netdna-ssl.com/wp-content/uploads/2018/10/ECG-waves-segments-and-intervals-LITFL-ECG-library-3.png>

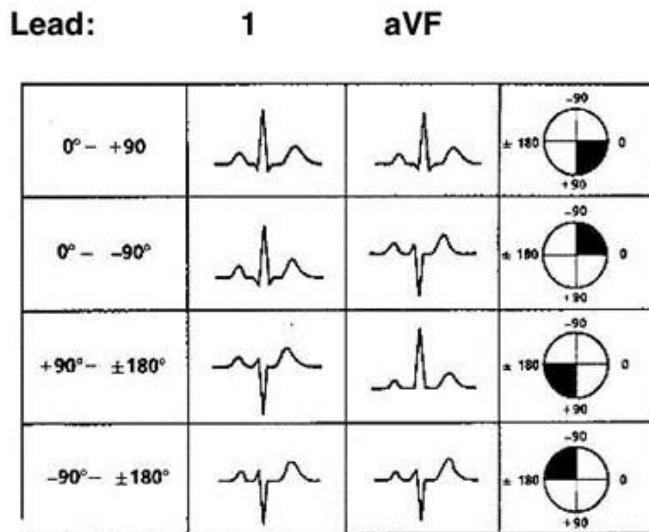
Denominations of the QRS complex:



QRS shapes - ECGPEDIA.ORG

P wave (duration of atrial depolarization):

P wave axis:



Ref: Park's, the pediatric cardiology handbook, 5th edition.

1. Look at **lead I** and **aVF**.
2. If the P wave is **upright** in lead I AND aVF, then the P wave axis is within the normal range (0° to $+90^\circ$).
3. If the P wave is **NOT upright** in lead I or aVF, then the P wave axis is outside the normal range (0° to $+90^\circ$).

NORMAL P WAVE AXIS: Within the 0° to $+90^\circ$ quadrant.

ABNORMAL P WAVE AXIS: Outside 0° to $+90^\circ$ quadrant.

Note: Normal P wave is positive in lead II and negative in aVR. P wave that is negative in lead II and positive in aVR (retrograde P wave) indicates an ectopic focus below the level of the SA node.

Left atrial enlargement (P Mitrale):

- **In Lead II** (both points should be met):
 - o Bifid P wave with > 40 msec between the two peaks.
 - o Total P wave duration > 80 msec in infants and > 100 msec in children.
- **In lead V1** (both points should be met):
 - o Biphasic P wave with terminal negative portion > 40 msec duration.
 - o Total P wave duration > 80 msec in infants and > 100 msec in children.

Right atrial enlargement (P Pumonale):

- **In Lead II:** P wave > 3 mm.
- **In lead V1:** P wave > 1.5 mm.

PR interval (reflects the transit time though the AV node):

PR Interval with Age and Rate (and Upper Limits of Normal)

Rate (bpm)	0-1 month	1-6 month	6-12 month	1-3 year	3-8 year	8-12 year	12-16 year	Adult
<60						160 (180)	160 (190)	170 (210)
60-79					150 (170)	150 (170)	150 (180)	160 (210)
80-99	100 (120)				140 (160)	150 (160)	150 (170)	150 (200)
100-119	100 (120)			(150)	130 (160)	140 (150)	150 (160)	150 (190)
120-139	100 (110)	110 (140)	110 (140)	120 (140)	130 (150)	140 (150)		150 (180)
140-159	90 (110)	100 (130)	110 (130)	110 (140)	120 (140)			(170)
160-179	100 (110)	100 (120)	100 (120)	100 (140)				
>180	90	90 (110)	100 (110)					

Ref: From Park MK, Guntheroth WG: How to Read Pediatric ECGs, ed 4, Philadelphia, 2006, Mosby.

Lower limit of PR interval by age

Younger than 12 months	0.075 second
1 to 3 years	0.080 second
3 to 5 years	0.085 second
5 to 12 years	0.090 second
12 to 16 years	0.095 second
Adults	0.120 second

Ref: Park's, the pediatric cardiology handbook, 5th edition.

Causes of prolonged PR interval: Refer to the “degrees of AV block” section below.

Causes of short PR interval:

1. WPW syndrome – refer to the “WPW syndrome” section in page 22.
2. Junctional rhythm (due to a retrograde P wave) – refer to the “P wave” section in page 8.
3. Left atrial enlargement (due to a widened P wave) – refer to the “P wave” section in page 8.

Causes of variable PR interval:

1. Wandering atrial pacemaker.
2. Typical Wenckebach – refer to the “degrees of AV block” section below.

Degrees of AV block:

1st degree AV block: Fixed prolongation of the PR interval.

Causes:

- Can occur in otherwise healthy children and young adults, particularly athletes.
- Certain CHD’s, specifically AVSD, ASD and Ebstein’s anomaly.
- Myocarditis – refer to the “myocarditis” section in page 19.
- Rheumatic fever – prolonged PR interval is a minor criterion among revised Jones criteria.
- Digitalis effect, beta blocker and calcium channel blocker toxicity – refer to the “Digitalis effect” section in page 24.
- Hyperkalemia – refer to the “hyperkalemia” section in page 22.
- Cardiac surgery.

2nd degree AV block:

Mobitz type 1:

- **Typical Wenckebach:** Progressive PR interval prolongation culminating in a non-conducted P wave.
- **Atypical Wenckebach:** No progressive PR interval prolongation, but instead the PR interval of the first conducted P wave is shorter compared to the last conducted P wave.

Mobitz type 2:

- Intermittent non-conducted P waves without progressive prolongation of the PR interval.

3rd (complete) AV block:

Independent atrial and ventricular activities; the P waves and QRS complexes are not associated (i.e. AV dissociation). AV dissociation may be very subtle when the atrial and ventricular rates are similar (isorhythmic AV dissociation).

Q Wave:

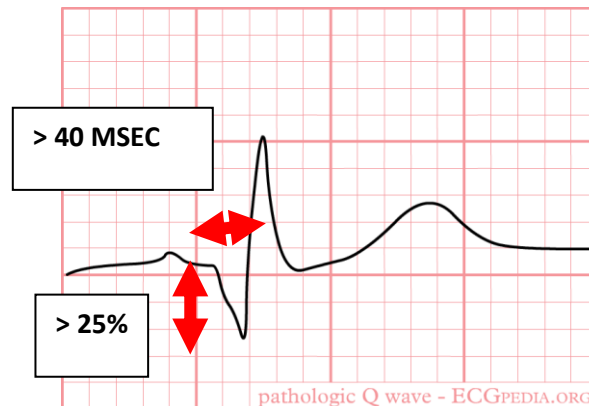
Normal Q Wave Voltages According to Age and Lead: Mean (and Upper Limit) in mm

Lead	0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adult
III	2 (5)	3 (6)	3 (6)	3 (6)	1.5 (4)	1 (3)	1 (3)	0.5 (4)
aVF	2 (4)	2 (5)	2 (6)	1.5 (5)	1 (5)	1 (3)	1 (3)	0.5 (2)
V5	1.5 (5)	1.5 (4)	2 (5)	2 (6)	2 (6)	2 (4.5)	1 (4)	0.5 (3.5)
V6	1.5 (4)	1.5 (4)	2 (5)	2 (4.5)	1.5 (4.5)	1.5 (4)	1 (2.5)	0.5 (3)

Modified from Guntheroth WG: Pediatric Electrocardiography. Philadelphia, WB Saunders, 1965.
*Voltages measured in millimeters, when 1 mV = 10 mm paper.

Pathological Q waves:

- Q WAVES IN THE RIGHT PRECORDIAL LEADS:
 - o **Causes:**
 - May be normal in neonates.
 - Severe RVH – refer to the “ventricular enlargement” section in page 20.
 - Single ventricle.
 - ccTGA (also called L-TGA).
- DURATION > 40 MSEC.
- AMPLITUDE MORE THAN 25% OF THE FOLLOWING R WAVE.



- o **Causes of wide and deep Q waves:**
 - Myocardial infarction.
 - Myocardial fibrosis.
 - Deep Q waves in the left precordial leads may be seen with LVH (volume-overload type) – refer to the “ventricular enlargement” section in page 20.
- ABSENT Q WAVES IN THE LEFT PRECORDIAL LEADS:
 - o **Causes:**
 - ccTGA (also called L-TGA).
 - LBBB.

Note: In ccTGA, the Q waves are absent in the left precordial leads and present in the right precordial lead due to ventricular inversion.

QRS complex duration (duration of ventricular depolarization):

QRS Duration According to Age: Mean (and Upper Limits of Normal) (In msec)

	0-1 month	1-6 month	6-12 month	1-3 year	3-8 year	8-12 year	12-16 year	Adult
QRS (ms)	50 (70)	50 (70)	50 (70)	60 (70)	70 (80)	70 (90)	70 (100)	80 (100)

Ref: Derived from percentile charts in Davignon A, et al. Normal ECG Standards for Children and Infants. Ped cardio 1:123-131, 1979/80.

Common causes of wide QRS complex:

- Bundle branch block (RBBB, LBBB)*:

o RBBB:

- Terminal slurring of the QRS complex resulting in an **rsR' complex** (small R, small S, large R) in the right precordial leads (V1, V2, V4R) and a **slurred S wave** in the left-sided leads (I, V5, V6) – refer to page 7 for denominations of the QRS complex.

- **Complete RBBB:** QRS duration > 100 msec in children 4 to 16 years of age, and > 90 msec in children less than 4 years of age.
 - o Repolarization may be consequentially abnormal (i.e., negative T waves).
 - o Often associated with RAD and large QRS amplitudes.
 - o Causes: Post-intracardiac surgery (e.g., repair of VSD and TOF), post-cardiac catheterization, ASD, Ebstein anomaly.
- **Incomplete RBBB:** QRS duration 90-100 msec in children 4 and 16 years of age, and 86-90 msec in children younger than 4 years of age.
 - o Causes: Found in about 1% of normal children.

o LBBB:

- A **single R complex** in the left-sided leads (I, V5, V6) and a **QS wave** in the right precordial leads (V1, V2, V4R) – refer to page 7 for denominations of the QRS complex.
 - Repolarization may be consequentially abnormal (i.e., negative T waves).
 - Often associated with LAD and large QRS amplitudes.
 - Causes: Anatomic abnormalities of the conduction system (e.g., Lenegre disease), LVH, post-cardiac surgery.

- WPW syndrome*: Refer to the “WPW syndrome” section in page 22.

- Ventricular rhythm (e.g., premature ventricular contraction, ventricular tachycardia or fibrillation).

- Slight prolongation may be seen with ventricular hypertrophy.

- Intraventricular block:

- o Defined as prolongation of the QRS complex that does not fit the pattern of RBBB or LBBB.
- o Causes:
 - Hyperkalemia – refer to the hyperkalemia section.
 - Tricyclic antidepressant poisoning.

* Diagnosis of ventricular hypertrophy cannot be made in the setting of BBB or pre-excitation.

QRS complex amplitude:

R and S Voltages: Mean (and Upper Limits of Normal) According to the Lead and Age

Voltage	Lead	0-1mo	1-6mo	6-12mo	1-3yr	3-8yr	8-12yr	12-16yr	Young
R	I	4 (8)	7 (13)	8 (16)	8 (16)	7 (15)	7 (15)	6 (13)	6 (13)
	II	6 (14)	13 (24)	13 (27)	13 (23)	13 (22)	14 (24)	14 (24)	9 (25)
	III	8 (16)	9 (20)	9 (20)	9 (20)	9 (20)	9 (24)	9 (24)	6 (22)
	aVR	3 (7)	3 (6)	3 (6)	2 (6)	2 (5)	2 (4)	2 (4)	1 (4)
	aVL	2 (7)	4 (8)	5 (10)	5 (10)	3 (10)	3 (10)	3 (12)	3 (9)
	aVF	7 (14)	10 (20)	10 (16)	8 (20)	10 (20)	10 (20)	11 (21)	5 (23)
	v4R	6 (12)	5 (10)	4 (8)	4 (8)	3 (8)	3 (7)	3 (7)	
	V1	15 (25)	11 (20)	10 (20)	9 (18)	7 (18)	6 (16)	5 (16)	3 (14)
	V2	21 (30)	21 (30)	19 (28)	16 (25)	13 (28)	10 (22)	9 (19)	6 (21)
	V5	12 (30)	17 (30)	18 (30)	19 (36)	21 (36)	22 (36)	18 (33)	12 (33)
V6	6 (21)	10 (20)	13 (20)	12 (24)	14 (24)	14 (24)	14 (22)	10 (21)	
s	I	5 (10)	4 (9)	4 (9)	3 (8)	2 (8)	2 (8)	2 (8)	1 (6)
	v4R	4 (9)	4 (12)	5 (12)	5 (12)	5 (14)	6 (20)	6 (20)	
	V1	10 (20)	7 (18)	8 (16)	13 (27)	14 (30)	16 (26)	15 (24)	10 (23)
	V2	20 (35)	16 (30)	17 (30)	21 (34)	23 (38)	23 (38)	23 (48)	14 (36)
	V5	9 (30)	9 (26)	8 (20)	6 (16)	5 (14)	5 (17)	5 (16)	
	V6	4 (12)	2 (7)	2 (6)	2 (6)	1 (5)	1 (4)	1 (5)	1 (13)

Ref: From Park MK, Guntheroth WG: How to Read Pediatric ECGs, ed 4, Philadelphia, 2006, Mosby.

Causes of large QRS voltages: Ventricular hypertrophy, BBB, WPW syndrome.

Causes of low QRS voltages: Pericarditis, myocarditis, hypothyroidism, normal neonates, obesity.

R/S ratio:

R/S Ratio According to Age: Mean, Upper and Lower Limits of Normal

Lead		0-1 mo	1-6 mo	6 mo-1 yr	1-3 yr	3-8 yr	8-12 yr	12-16 yr	Adults
V1	LLN	0.5	0.3	0.3	0.5	0.1	0.15	0.1	0.0
	Mean	1.5	1.5	1.2	0.8	0.65	0.5	0.3	0.3
	ULN	19	S = 0	6	2	2	1	1	1
V2	LLN	0.3	0.3	0.3	0.3	0.05	0.1	0.1	0.1
	Mean	1	1.2	1	0.8	0.5	0.5	0.5	0.2
	ULN	3	4	4	1.5	1.5	1.2	1.2	2.5
V6	LLN	0.1	1.5	2	3	2.5	4	2.5	2.5
	Mean	2	4	6	20	20	20	10	9
	ULN	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0	S = 0

LLN, lower limits of normal; ULN, upper limits of normal.

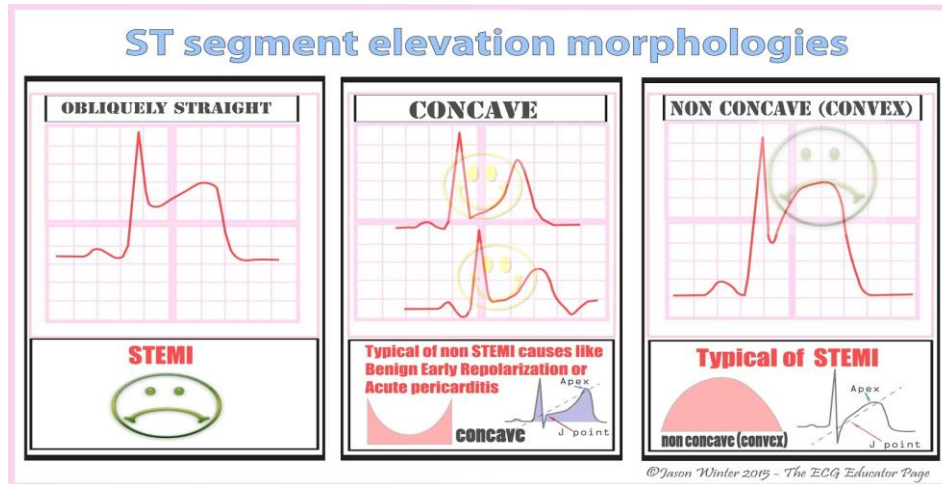
From Guntheroth WB: Pediatric Electrocardiography. Philadelphia, WB Saunders, 1965.

ST segment:

Pathological changes:

- Limb lead ST depression or elevation > 1 mm.
- Precordial lead ST depression or elevation > 2 mm.

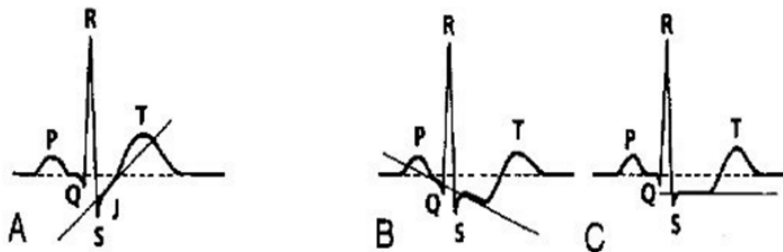
ST segment elevation:



Causes of ST segment elevation in children:

Pericarditis, early repolarization, STEMI, Prinzmetal angina, aortic dissection, myocarditis, Brugada syndrome.

ST segment depression:



Ref: Park's, the pediatric cardiology handbook, 5th edition.

A: Upsloping ST depression → Normal variant.
B: Downsloping ST depression → usually abnormal.
C: Horizontal ST depression → usually abnormal.

Causes of ST segment depression in children:

Subendocardial ischemia, ventricular hypertrophy with strain, digitalis effect, hypokalemia, STEMI (reciprocal change).

T waves:

Natural course of T waves:

- ✓ **During the 1st week of life**, the T waves in V1 may be positive, in V2-V4 are usually negative, in V5 are variable but usually positive, and in V6 are always positive.
- ✓ **After the 1st week of life**, the T waves become inverted in V1-4.
 - They will start becoming positive at 7 years of age, starting with V4, then V3, then V2 and lastly V1. This process does not skip leads, so if there are negative T waves in-between positive ones or vice-versa, this is abnormal. Most commonly, this is due to lead misplacement or chest electrodes touching each other.
 - The T waves should be positive in V5 and V6.

Key point: Upright T waves in V1 in children > 7 days to < 7 years are suggestive of RVH (pressure overload such as in pulmonary stenosis or TOF).

Causes of T wave inversion:

- Normal variant:
 - T waves usually become positive in V1 when the child becomes 7 years and older.
 - However, inverted T waves in V1 can persist into adolescence and early adulthood.
 - T wave inversion in leads V1-V4 in black/African athletes is normal.
- Ventricular hypertrophy with strain (RVH or LVH).
- BBB (right or left).
- Hypokalemia.
- WPW syndrome.
- Myocardial ischemia.
- Digitalis effect.
- Brugada syndrome.

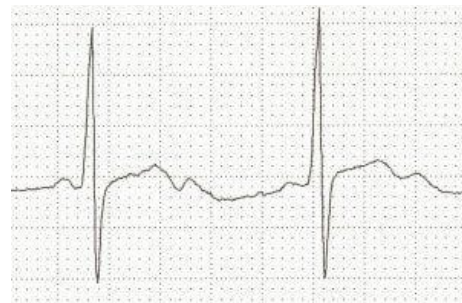
Causes of tall T waves:

- Normal variant (early repolarization).
- Hyperkalemia (early sign).
- Myocardial infarction (early sign).
- Raised intracranial pressure (e.g., CVD).

Causes of 'camel hump' (2-peaked) T waves:

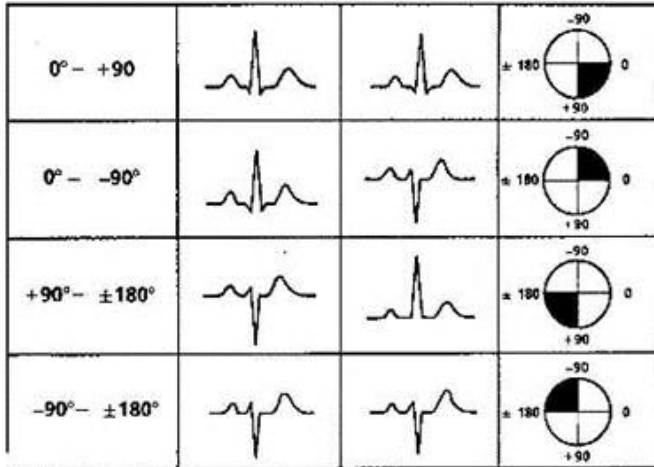
- Prominent U wave fused to the end of the T wave.
- Hidden P wave embedded in the T wave (as in sinus tachycardia or AV block).

'Camel hump' T wave



T wave axis:

Lead: 1 aVF



1. Use **lead I** and **aVF** to locate a quadrant.
2. Find a lead with an equiphase T wave (or a **flat T wave**).
3. The T wave axis will be **perpendicular** to this lead in the **previously determined quadrant**.

NORMAL T WAVE AXIS:

Within the 0° to $+90^\circ$ quadrant (when the T wave is upright in lead I AND aVF).

ABNORMAL T WAVE AXIS:

Outside the 0° to $+90^\circ$ quadrant (when the T wave is inverted in lead I or aVF).

Causes of abnormal T wave axis:

- Ventricular hypertrophy with strain, BBB.

QRS-T angle:

Angle formed by the QRS axis and T wave axis.

NORMAL QRS-T angle: $< 60^\circ$ (except in neonates when it can be $> 60^\circ$ degrees).

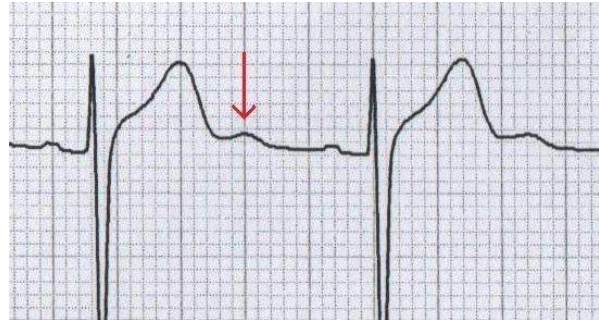
ABNORMAL T WAVE AXIS: $> 60^\circ$ ($> 90^\circ$ is certainly abnormal).

Causes of abnormal QRS-T angle:

- Ventricular hypertrophy with strain, BBB, ventricular arrhythmias, myocardial dysfunction of a metabolic or ischemic dysfunction.

U wave:

- An additional positive deflection **after the T wave**.



- U wave is best seen in leads V2 and V3.
- The U wave size is inversely proportional to the heart rate (the less the heart rate, the larger the U wave).
- If the U wave is $> 50\%$ of the amplitude of the T wave, include it in the QTc interval measurement.

Causes of U wave:

- Normal variant.
- Hypokalemia.
- Bradycardia.

Features of normal U wave:

- The voltage is normally $< 25\%$ of the T wave voltage.
- In the same direction of the T wave.
- More visible with slower heart rates.

Drugs associated with prominent U waves:

- Digoxin.
- Phenothiazines (thioridazine).
- Class Ia antiarrhythmics (quinidine, procainamide).
- Class III antiarrhythmics (sotalol, amiodarone).

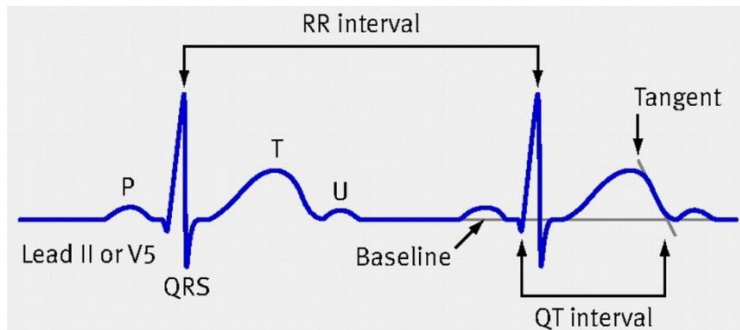
QT interval (time taken for both ventricular depolarization and repolarization):

QT interval varies with heart rate.

Bazett's formula is used to correct the QT for HR:

$$QTc = \frac{QT}{\sqrt{R-R}}$$

The R-R interval is the duration between the R waves **prior** to the measured QT interval.



Lower limit of the normal QTc range: 370 msec.

Upper limit of the normal QTc range:

- Prior to 15 years: 450 msec in boys and 460 msec in girls.
- After 15 years: 440 msec in boys and 450 msec in girls.

However, a definitely prolonged QTc interval is considered beyond 480 msec. A QTc interval that is within the upper normal limit and 480 msec is generally considered borderline.

A useful rule of thumb: Normal QT is less than half the preceding RR interval.

A normal QTc interval does not exclude the presence of a long QT syndrome. Up to 40% of people with the genetic mutation have a QTc interval within the normal limits.

JT interval:

Useful when the QT interval is prolonged secondary to a prolonged QRS complex.

Measured from the **J point** (junction between S wave and ST segment) to the end of T wave.

$$JTc = \frac{JT}{\sqrt{R-R}}$$

Prolonged JTc has the same significance as the prolonged QTc interval.

Normal JTc (mean +/- SD): 0.32 +/- 0.02 seconds.

Characteristic ECG patterns for particular conditions:

Pericarditis:

- Stage 1 – widespread ST elevation and PR depression with reciprocal (opposite) changes in aVR (occurs during the first two weeks).
- Stage 2 – Generalized T wave flattening (1 to 3 weeks).
- Stage 3 – flattened T waves become inverted (3 to several weeks).
- Stage 4 – ECG returns to normal (several weeks onwards).

Less than 50% of patients progress through all four classical stages and evolution of changes may not follow this typical pattern.



Pericardial effusion may produce QRS voltages ≤ 5 mm in all limb leads.

Myocarditis:

- AV conduction disturbances, ranging from PR prolongation to complete AV dissociation.
- Low QRS voltages (5 mm or less in all limb leads).
- Decreased T wave amplitude, negative or flat T waves.
- QT prolongation.
- Tachyarrhythmias including SVT and VT.
- 'Pseudo-infarction' pattern with deep Q waves and poor R wave progression in precordial leads.
- ST segment elevation that may vary in magnitude with the symptoms.

Ventricular enlargement:

Right ventricular enlargement (RVH):

- **Axis:**
 - RAD for the patient's age.
- **Voltages:**
 - Tall R waves (greater than limits for age) in the right precordial leads (V1, V2, V4R).
 - rSR' (small R, tall S, tall R') with R' > 15 mm before the age of 1 year or > 10 mm after that age in the right precordial leads (provided that there is no RBBB).
 - Deep S waves (greater than limits for age) in the left-sided leads (I, V5, V6).
- **R/S ratio:**
 - R/S ratio in V1 and V2 > the upper limits of normal for age.
- **T waves:**
 - Upright T waves in V1 in children > 7 days to < 7 years (provided that T waves are upright in the left precordial leads). This is enough to diagnose RVH.
- **Q waves:**
 - qR (small Q, tall R) or qRs (small Q, tall R, small S) pattern in V1.

Left ventricular enlargement (LVH):

- **Axis:**
 - LAD for the patient's age.
- **Voltages:**
 - Tall R waves (greater than limits for age) in the left-sided leads (I, V5, V6).
 - Deep S waves (greater than limits for age) in the right precordial leads (V1, V2, V4R).
- **R/S ratio:**
 - R/S ratio in V1 and V2 < the lower limits of normal for age.
- **T waves:**
 - Inverted T waves in lead I & aVL AND left precordial leads (LV strain pattern).
- **Q waves:**
 - Deep Q waves in V5 and V6 (volume-overload type).

Biventricular enlargement:

- Positive voltage criteria for RVH and LVH (with normal QRS duration).
- Positive voltage criteria for RVH or LVH and normal voltages for the other ventricle.
- Large equiphasic QRS complexes in two or more limb leads and in mid-precordial leads (V2-V5) – called "**Katz-Wachtel phenomenon**".

Myocardial ischemia/infarction:

- **Infarction (death of cardiac tissue):**
 - o ST segment elevation in contiguous leads with reciprocal ST segment depression elsewhere.
- **Injury (prolonged oxygen deficiency, usually > 20 minutes):**
 - o Horizontal ST segment depression.
- **Ischemia (oxygen deficiency for a shorter period, usually < 20 minutes):**
 - o Flat/negative T waves with QRS-T axis angle > 90 degrees.

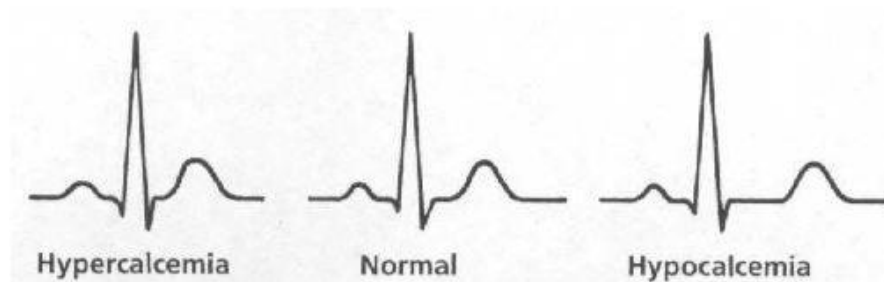
Correlation between ECG leads and coronary artery territory affected:

I Lateral Circumflex Artery	aVR	V1 Septal Left Anterior Descending Artery	V4 Anterior Right Coronary Artery
II Inferior Right Coronary Artery	aVL Lateral Circumflex Artery	V2 Septal Left Anterior Descending Artery	V5 Lateral Circumflex Artery
III Inferior Right Coronary Artery	AVF Inferior Right Coronary Artery	V3 Anterior Right Coronary Artery	V6 Lateral Circumflex Artery

Ref: WHO, 2011

Hypocalcaemia and hypercalcemia:

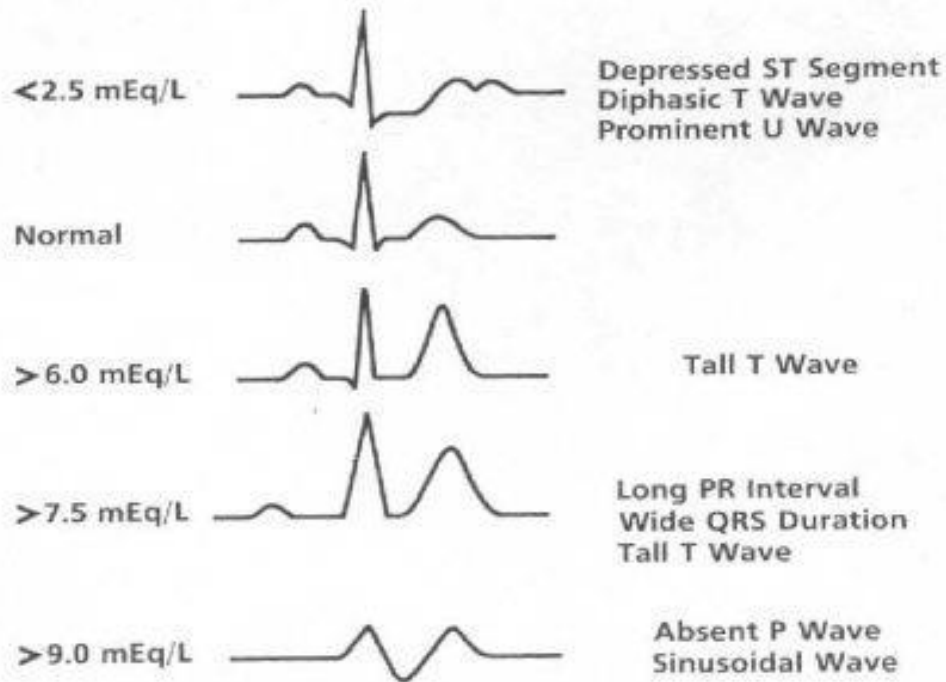
- Hypocalcaemia prolongs the ST segment with resulting **prolongation of the QTc**.
- Hypercalcaemia shortens the ST segment with resulting **shortening of the QTc**.



Ref: Park's, the pediatric cardiology handbook, 5th edition.

Hypokalemia and hyperkalemia:

SERUM K



Ref: Park's, the pediatric cardiology handbook, 5th edition.

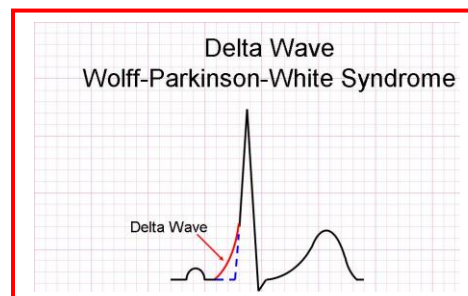
WPW syndrome:

Criteria:

1. Short PR interval (refer to [page 9](#) for normal limits of the PR interval).
2. Delta wave – slurring slow rise of initial portion of the QRS.
3. QRS prolongation (refer to [page 12](#) for normal limits of the QRS duration).

Repolarization may consequentially be abnormal (i.e., negative T waves).

Increases the risk of SVT.

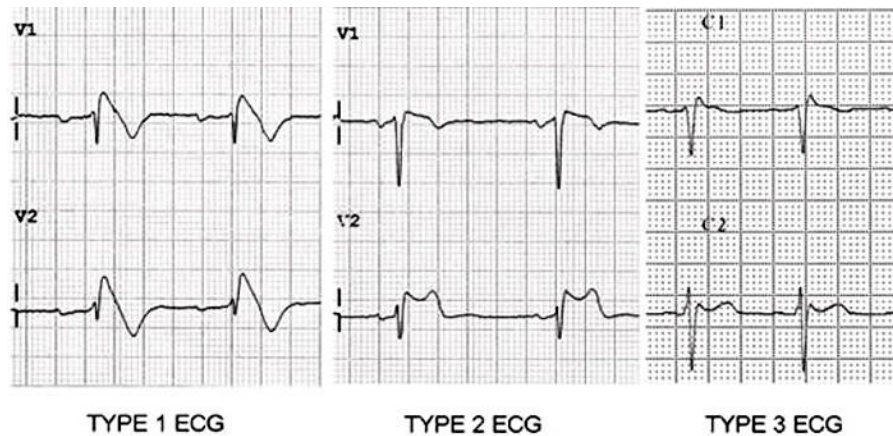


Brugada syndrome:

Type 1: Coved ST segment elevation > 2 mm in > 1 of V1-V3 followed by a negative T wave.

Type 2: Has > 2 mm of saddleback-shaped ST elevation.

Type 3: Can be the morphology of either type (1 or 2), but with < 2 mm of ST segment elevation.



Ref: <https://lifeinthefastlane.com/ecg-library>

Long QT syndrome (causative medications):

Antiarrhythmic	Antimicrobial	Antidepressants	Antipsychotics	Others
Amiodarone	Levofloxacin	Amitriptyline	Haloperidol	Cisapride
Sotalol	Ciprofloxacin	Imipramine	Quetiapine	Domperidone
Quinidine	Clarithromycin	Desipramine	Ziprasidone	Ondansetron
Procainamide	Erythromycin	Fluoxetine	Droperidol	Sumatriptan
Dofetilide	Ketoconazole	Sertraline	Thioridazine	Methadone
Ibutilide	Itraconazole	Venlafaxine		Arsenic

Ref: up-to-date.

A more complete list of specific drugs that prolong QT interval is available at:

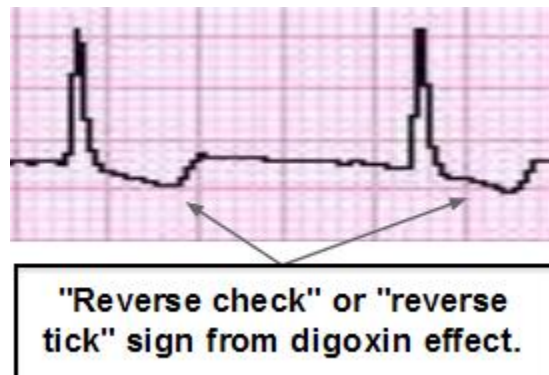
www.crediblemeds.org

Digoxin effect

These ECG features of digoxin effect are seen with therapeutic doses of digoxin.

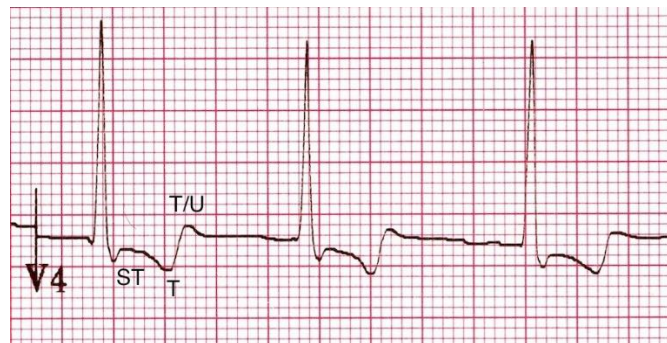
Characteristic findings:

1. Downsloping ST depression with a characteristic “reverse tick” appearance.



2. T wave changes:

- Flattened, inverted, or biphasic T waves.
 - The most common T wave abnormality is a **biphasic T wave** with an initial negative deflection and terminal positive deflection.
 - The initial negative deflection of the T wave is typically continuous with the depressed ST segment.
 - The terminal positive deflection of the T wave may be peaked or have a prominent U wave superimposed upon it.



3. Prolonged PR interval – due to increased vagal tone.
4. Shortened QTc.

Normal ECG findings in athletes:

1. Sinus bradycardia (≥ 30 bpm).
2. Sinus arrhythmia.
3. Ectopic atrial rhythm.
4. Junctional escape rhythm.
5. 1° AV block (PR interval > 200 msec).
6. Mobitz Type I (Wenckebach) 2° AV block.
7. Incomplete RBBB.
8. Isolated QRS voltage criteria for LVH:
 - **Except:** QRS voltage criteria for LVH occurring with any non-voltage criteria for LVH such:
 - i. Left atrial enlargement.
 - ii. Left axis deviation.
 - iii. ST segment depression.
 - iv. T-wave inversion.
 - v. Pathological Q waves.
9. Early repolarisation:
 - ST elevation, J-point elevation, J-waves or terminal QRS slurring.
10. Convex ('domed') ST segment elevation combined with T-wave inversion in leads V1–V4 in black/African athletes.

These common training-related ECG alterations are physiological adaptations to regular exercise, considered normal variants in athletes and do not require further evaluation in asymptomatic athletes.

Ref: Jonathan A Drezner, et al. Electrocardiographic interpretation in athletes: the '**Seattle Criteria**'. BMJ. 7th Dec. 2012.

Abnormal ECG findings in athletes (refined criteria):

Left atrial enlargement*:	Negative portion of the P wave in V1 ≥ 0.1 mV in depth and ≥ 40 msec in duration.
Right atrial enlargement*:	P wave amplitude ≥ 2.5 mm in II, III and aVF.
Left QRS axis deviation*:	-30° to -90° .
Right QRS axis deviation*:	$>115^\circ$.
RV hypertrophy*:	Sum of R wave in V1 and S wave in V5 or V6 ≥ 1.05 mV.
Corrected QT interval**:	>470 msec in women and >480 msec in men.
Complete LBBB*:	QRS duration ≥ 120 msec with predominantly negative QRS complex in V1 (QS or rS) and upright monomorphic R wave in lead I and V6.
Complete RBBB*:	RSR' pattern in anterior precordial leads with QRS ≥ 120 msec.
Interventricular conduction delay*:	Any QRS >120 msec in duration including RBBB and LBBB.
Pathological Q waves:	≥ 40 msec in duration or $\geq 25\%$ of the ensuing R wave.
Significant T wave inversion**:	>1 mm in depth in 2 or more of the leads V2-V6, II and aVF or I and aVL (excludes V1, III and aVR).
ST segment depression*:	≥ 0.5 mm in ≥ 2 leads.
Ventricular preexcitation**:	PR interval <120 msec with a delta wave.

* European Society of Cardiology ** Seattle Criteria

When to proceed with further cardiovascular evaluation:

As per the Refined Criteria, Athletes would not receive further cardiovascular evaluation when presenting with the following recognized training-related ECG changes in isolation:

1. Left atrial enlargement.
2. Right atrial enlargement.
3. Left axis deviation.
4. Right axis deviation.
5. Sokolow-Lyon voltage criteria for RVH or LVH.

However, importantly, the presence of two or more of the above ECG patterns would warrant secondary investigation.

Also, T wave inversion preceded by convex ST segment elevation in leads V1-V4 in asymptomatic black athletes do not require further evaluation.

Ref: Riding N. et al. Comparison of three current sets of electrocardiographic interpretation criteria for use in screening athletes. Heart 2015.

Abbreviations:

LVH (left ventricular hypertrophy); RVH (right ventricular hypertrophy); LBBB (left bundle branch block); RBBB (right bundle branch block); WPW (Wolf-Parkinson-White syndrome); QTc (corrected QT interval); PVC (premature ventricular contraction); AV (atrioventricular); bpm (beats per minute); msec or ms (milliseconds); sec (seconds); mo (month); yr (year); TOF (tetralogy of Fallot); HCM (hypertrophic cardiomyopathy); RPL (right precordial leads); LPL (left precordial leads); SVT (supraventricular tachycardia), ccTGA (congenitally corrected transposition of the great arteries), L-TGA (levo-transposition of the great arteries).

Acknowledgment:

We would like to express our gratitude to our **cardiologists** at McMaster Children's Hospital for their significant contribution to this guide.

References:

- Park's, the pediatric cardiology handbook, 5th edition
- <https://lifeinthefastlane.com/ecg-library>
- Pediatrics Review Core Curriculum, 7th edition, 2016-2017

Helpful ECG resources:

Please, visit the cardiology section of the MacPeds website (<https://macpeds.com/cardiology.html>) for important ECG resources, including review articles of common ECG arrhythmias.