How to interpret the paediatric 12-lead ECG

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ABSTRACT

ECG interpretation is a core skill for any healthcare practitioner that looks after children. The article aims to educate the reader in basic interpretation of paediatric ECG in a succinct, interactive, organised manner in a way that it can be easily referenced and applied in everyday clinical practice. We include clinical examples as well as age and sex-related reference ranges for QT intervals, P-wave duration, Q-wave amplitude, QRS complex duration, R-wave and S-wave amplitude, R/S ratio and PR intervals.

INTRODUCTION

In an era that paediatric echocardiography has become readily available, the ECG may occasionally be considered unnecessary. This could not be more wrong. The ECG remains an irreplaceable diagnostic tool that is more likely to reveal heart rhythm abnormalities and a diagnosis than an echocardiogram, especially in the absence of other positive clinical examination findings (such as murmurs or hypertension). Subtle changes in the ECG can sometimes precede findings detected in echocardiography. In certain presentations such as syncope, palpitations and chest pain, the ECG can be the single most important investigation and should be performed without haste, especially in the acute setting. Confident interpretation of the ECG is a key skill for all physicians who look after children. While the subject is broad and an exhaustive discussion of ECG analysis is beyond the scope of the article, we have designed this paper with a view to educate the reader in basic interpretation of paediatric ECG in a succinct, interactive, organised manner in a way that it can be easily referenced and applied in everyday clinical practice.

BACKGROUND

The paediatric 12-lead ECG is a simple, reproducible and non-invasive test that mirrors the changes that occur in cardiac physiology with growth and age. The most important physical principle in interpreting any ECG is that myocardial depolarisation towards an ECG electrode produces a positive deflection (above the isoelectric line), while depolarisation away from the electrode produces a negative deflection. The greater the depolarisation (eg, due to a bigger myocardial bulk) results in a bigger deflection as the voltage is higher.

In fetal life, pulmonary vascular resistance (PVR) is high as lungs do not contribute to gas exchange and systemic vascular resistance (SVR) is low due to placental circulation. For this reason, there is right ventricular (RV) dominance in the newborn period, which with age gradually changes to left ventricular (LV) dominance (PVR drops, SVR rises). An adult pattern is usually achieved around 3 years of age, although inverted T waves

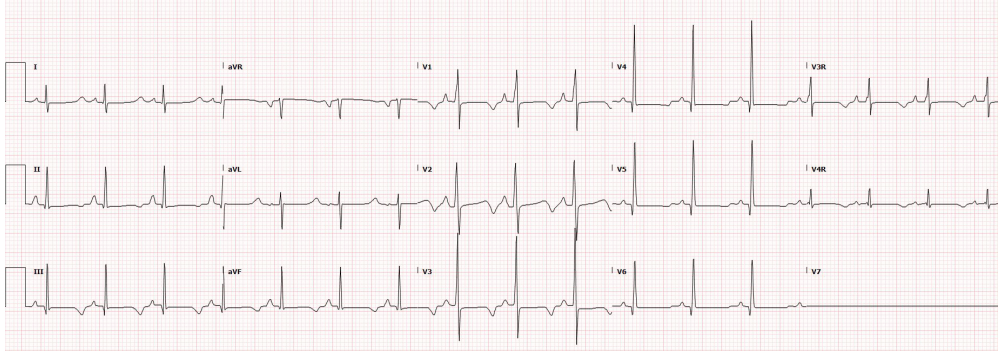

Figure 1 ECG of case 1. See box 3 for full interpretation.

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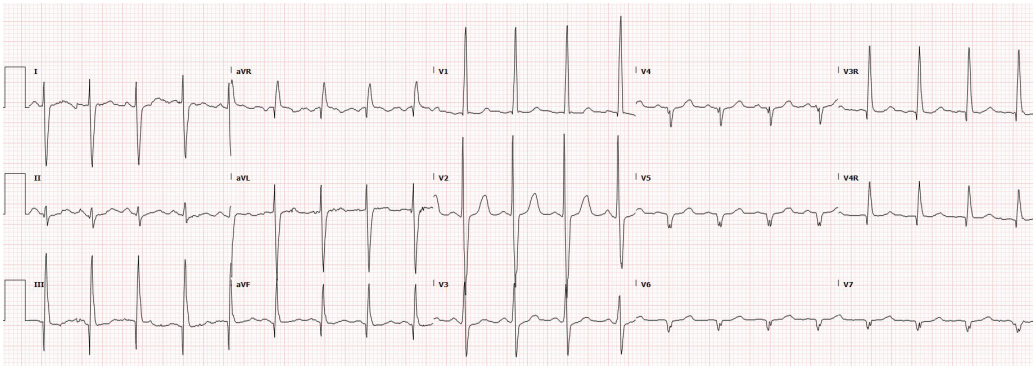

Figure 2 ECG of case 2. See box 4 for full interpretation.

Table 1 Existing reference ranges for respiratory rate and heart rate

Age range (years)	Heart rate (beats/min)				
	APLS[7]/PHPLS[8]	PALS[9]	EPLS[10]	PHTLS[11]	ATLS[12]
Neonate	110–160	85–205*	85–205*	120–160†	<160
0–1	110–160	100–190*	100–180*	80–140†	<160
1–2	100–150	100–190	100–180	80–130	<150
2–3	95–140	60–140	60–140	80–120	<150
3–5	95–140	60–140	60–140	80–120	<140
5–6	80–120	60–140	60–140	80–120	<140
6–10	80–120	60–140	60–140	(60–80)–100	<120
10–12	80–120	60–100	60–100	(60–80)–100	<120
12–13	60–100	60–100	60–100	(60–80)–100	<100
13–18	60–100	60–100	60–100	60–100‡	<100

Adapted from Fleming *et al*.[3]

PALS and EPLS provide multiple ranges—ranges for awake children are tabulated.

*PALS and EPLS provide separate ranges for infants up to 3 months, and for those between 3 months and 2 years of age.

†PHTLS provides separate ranges for infants up to 6 weeks, and for those between 7 weeks and 1 year of age.

‡PHTLS does not provide ranges for adolescents over 16 years of age.

APLS, Advanced Paediatric Life Support; ATLS, Advanced Trauma Life Support; EPLS, European Paediatric Life Support; PHPLS, Pre-Hospital Paediatric Life Support; PHTLS, Pre-Hospital Trauma Life Support.

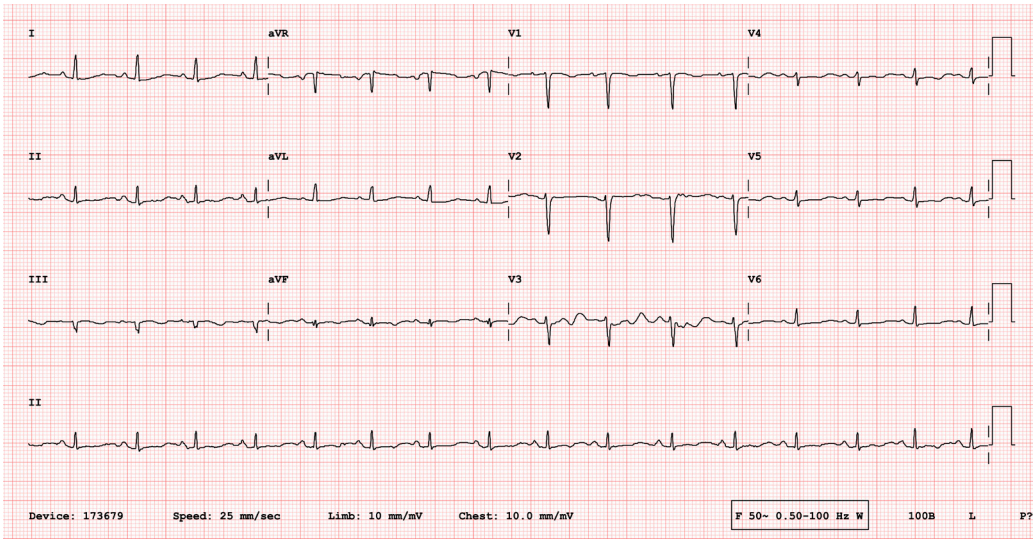

Figure 3 ECG of case 3. See box 5 for full interpretation.

Table 2 P-wave duration limits of normal

Age		0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
P-wave duration (ms), median (2nd, 98th centile)	♂	78 (64, 85)	79 (65, 98)	81 (64, 103)	80 (66, 96)	80 (63, 113)	87 (67, 102)	92 (73, 108)	98 (78, 117)	100 (82, 118)
	♀	79 (69, 106)	78 (62, 105)	78 (63, 106)	80 (64, 107)	83 (62, 104)	84 (66, 101)	89 (71, 107)	94 (74, 114)	98 (78, 122)

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

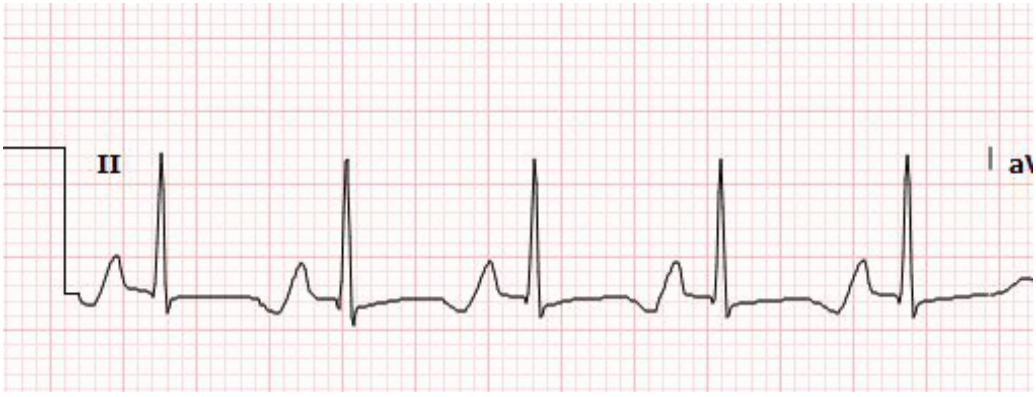

Figure 4 ECG showing p-pulmonale (p-wave >2mm in lead II).

characteristically remain until later years. As children grow, their heart rate slows down and their chest wall becomes thicker. Therefore, normal values for rate, axis, voltages and cardiac intervals vary with age.[1–3]

CLINICAL CASES

Case 1

A 9-year-old boy presents to the local emergency department following a syncopal episode in the middle of a football game. The parents report that he appeared pale and remained unresponsive for 1 min. He recalls that he had some chest discomfort but does not remember the rest of the event. A 12-lead ECG was obtained (figure 1).

Case 2

A 4-month-old infant who was born prematurely at 28 weeks with a previously patent arterial duct was treated for chronic lung disease with low flow oxygen. He had a routine ECG (figure 2) followed by an echocardiogram. Echocardiogram revealed stenosis of one pulmonary vein.

Case 3

A 15-year-old girl presents with a 2-day history of acute chest pain worse on exertion. She was normally fit and healthy with excellent exercise tolerance. She was tachypnoeic (32/min) and tachycardic (110/min) with bilateral crackles on chest auscultation. She was referred to acute paediatric services by her general practitioner (GP) with the clinical impression of lower respiratory tract infection. In the hospital, she was started on macrolides for atypical pneumonia and kept as inpatient in view of her observations. She made no clinical improvement 48 hours later; hence, a chest X-ray was performed. This showed cardiomegaly and signs of pulmonary oedema. Her 12-lead ECG is shown in figure 3.

ECG INTERPRETATION

For the interpretation of an ECG, it is important to be methodical and systematic.

Before any interpretation

It is vital to know the patient's age, medical history, medication, any electrolyte abnormalities and the clinical indication for the ECG. Check that the ECG is recorded in standard speed (25 mm/s) and voltage (1 mV=10 mm).

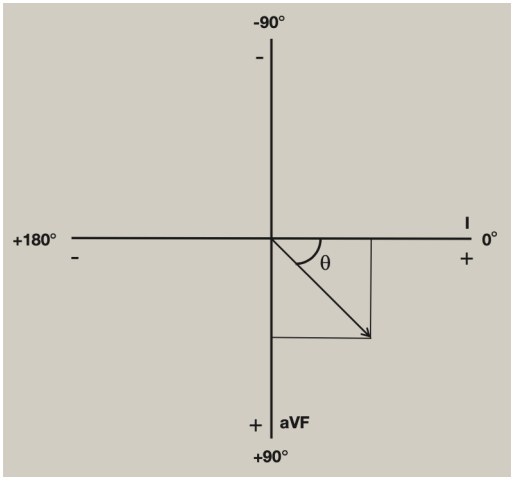

Figure 5 QRS axis calculation using leads I and aVF. aVF, augmented Vector Foot.

Table 3 Q-wave amplitude upper limits of normal

Age			0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
Q-wave (mm), 98th centile	II	♂	2.5	3.0	3.5	5.0	4.5	2.5	3.0	2.5	2.0
		♀	2.5	3.0	4.0	4.5	5.0	2.5	2.5	2.0	2.0
	III	♂	2.5	5.0	7.0	8.0	7.5	4.5	3.5	3.0	3.0
		♀	3.5	5.0	6.5	8.0	7.5	4.0	4.0	2.5	2.0
	aVF	♂	2.5	3.5	4.0	6.0	5.5	3.5	2.5	2.5	2.0
		♀	2.5	3.5	4.5	5.0	5.5	3.1	3.0	2.1	2.0
	V6	♂	2.0	3.0	3.5	6.0	5.5	4.0	4.0	4.0	4.5
		♀	1.5	3.5	4.0	4.0	5.0	4.0	4.1	3.5	2.5

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

aVF, augmented Vector Foot.

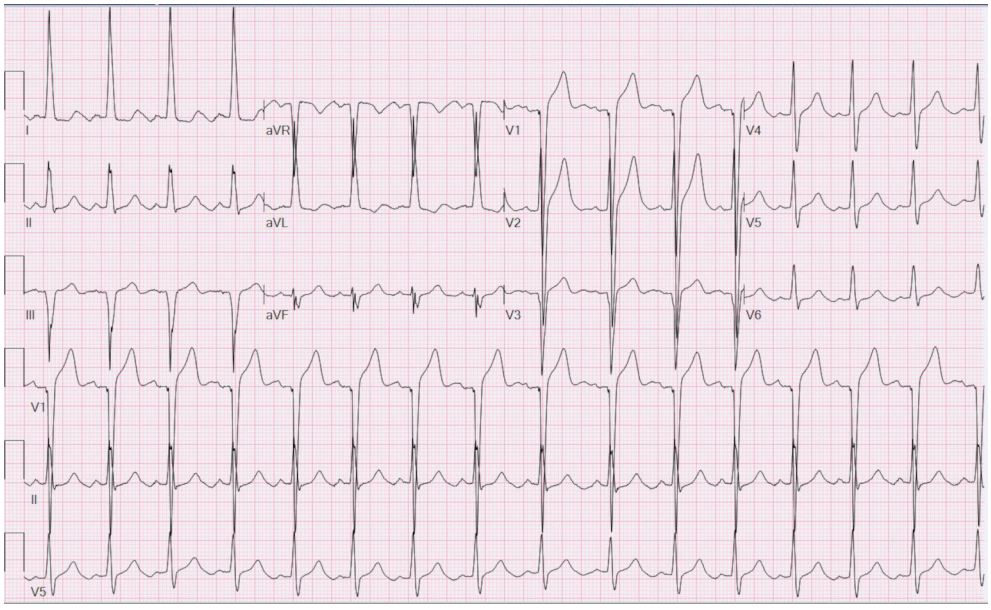

Figure 6 ECG showing deep Q-waves in leads V1-3 in a child with congenitally corrected transposition of great arteries. aVL, augmented Vector Left; aVF: augmented Vector Foot; aVR, augmented Vector Right.

Suggested systematic approach

1. Rate.
2. Rhythm.
3. P-waves.
4. QRS axis.
5. QRS complexes.
6. T-waves and ST segments.
7. Intervals (PR, QTc).

Rate

If ECG paper is recorded in conventional speed (25 mm/s), 1500 mm is covered in 1 min. Therefore, heart rate is 1500/R-R interval in mm (number of small squares). Bradycardia or tachycardia is defined as a heart rate lower or higher, respectively, than the range of normal for age[3] (table 1).

Table 4 QRS complex duration limits of normal

Age		0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
QRS duration (ms), median (2nd, 98th centile)	♂	67 (50, 85)	64 (52, 77)	66 (54, 85)	69 (52, 86)	71 (54, 88)	75 (58, 92)	80 (63, 98)	85 (67, 103)	91 (78, 111)
	♀	67 (54, 79)	63 (48, 77)	64 (50, 78)	64 (52, 80)	68 (54, 85)	71 (58, 88)	77 (59, 95)	82 (66, 99)	87 (72, 106)

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

Table 5 R-wave and S-wave amplitude upper limits of normal

Age			0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
R-wave (mm), 98th centile	V1	♂	20.5	21	22	21.5	21	18	15	11	12
		♀	22	20	20	19	19	14	12	11	11
	V6	♂	18	22	27	28	29	31	30	32	30.5
		♀	16	27	28	27	26	29	32.5	30	25
S-wave (mm), 98th centile	V1	♂	14	16	20	19	23	21	23	25	24
		♀	15	16	16	19	22	21	25	26	20.5
	V6	♂	8	11	12.5	12	9	9	9	8	8.5
		♀	10	8	10	7	9	6	8	7.5	6

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

Rhythm

Normal sinus rhythm originates from the sinoatrial node and is defined as follows:

- P-wave preceding every QRS complex.
- Uniform PR interval.
- Normal p-wave axis (0–90^0) manifested as positive p-wave in I and augmented Vector Foot (aVF).

In coronary sinus/low atrial rhythm (normal variant), there is a p-wave before every QRS, but p-waves are inverted in inferior leads (II, III, aVF). In situs inversus, the p-wave may be inverted in lead I. Tachycardias with abnormal p-wave axis can suggest tachyarrhythmias such as atrial tachycardias.

P-waves

P-waves are generated by atrial depolarisation. The right atrium (RA) depolarises first, this translates to the early portion of the p-wave being generated by the RA and the late portion by the left atrium.

- **P-wave duration:** Increases slightly with age. P-wave duration longer than the upper limit of normal *(*see table 2*)* can indicate left atrial enlargement (eg, left to right shunts, mitral stenosis).
- **P-wave amplitude:** Remains unchanged with age. Voltage higher than 2.5 mm in lead II suggests right atrial hypertrophy. This is known as p-pulmonale and can be seen in conditions like Ebstein's anomaly and pulmonary hypertension (figure 4).

QRS axis

We can determine the frontal QRS axis by using leads I and aVF which give a visual representation of the direction of depolarisation at right angles to each other (figure 5). The overall deflection of the RS wave is calculated and plotted. Once the coordinates are joined, we create an angle (theta) with the zero axis. Theta is the mean frontal plane of the QRS axis. The QRS axis changes with age: from +125° at birth (range +30° to +180°) to a mean value of +50 by the age of 3 (adult range −10° to +110°).

Superior axis deviation (−90° to 180°) is an abnormal finding and can be seen in children with atrioventricular septal defects, tricuspid atresia, large ventricular septal defects or pacing.

QRS complexes

- **Q-wave:** The q-wave represents septal depolarisation and is normally present in inferior leads (II, III, aVF), anterolateral leads (I, aVL) and almost always present in V_5 and V_6. Generally, the q-wave should not be larger than 25% of the associated R-wave amplitude in any lead. Table 3 describes the 98th centiles for q-wave amplitudes for age and gender.

Abnormally high q-wave voltages can be seen in ventricular hypertrophy, cardiomyopathies and

Box 1 Criteria for left ventricular hypertrophy (LVH)

Left ventricular hypertrophy

- R-wave above 98th centile for age on V6 and S-wave above 98th centile for age on V1 (table 5)
- Inverted T-waves in II, III, aVF, V4–6 → Strain patter
  - Highly suggestive LVH (not always present)
- Additional markers (not always present)
  - Tall R-waves in aVF
  - Left axis deviation
  - Deep Q-waves in V4–6

Box 2 Criteria suggesting right ventricular hypertrophy (RVH)

Right ventricular hypertrophy

Any of the four criteria suggests RVH

- R-wave above 98th centile for age on V1 (table 5)
- S-wave above 98th centile for age on V6 (table 5)
- R/S ratio above 98th centile on V1 or below 2nd centile on V6 (table 6)
- Upright T-waves in V1 (*ages: 1 week–10 years)

Supportive findings

- Neonatal type R-wave progression in precordial leads in children/adolescents
- Right axis deviation (not to be used in isolation, especially in infants/young children)

myocardial infarction. Deep q-waves in lead aVL may signify anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA). Presence of q-waves in leads V_{1-3} may be consistent with the rare condition congenitally corrected transposition of great arteries (figure 6). In single functional ventricles (eg, hypoplastic left heart syndrome), there may be no q-waves in the left precordial leads.

- **QRS duration:** QRS duration increases with age (table 4). QRS prolongation can be due to various pathologies including conduction disturbances caused by electrolyte abnormalities, drugs, bundle branch blocks and Wolff–Parkinson–White (WPW) syndrome.
- **RS amplitude and progression:** Table 5 shows the upper limits of normal for R-waves and S- waves in leads V_1 and V_6. Amplitudes higher than the above limits may indicate ventricular hypertrophy.[4] Boxes 1 and 2 describe the criteria for left and right ventricular hypertrophy, respectively. It is important to note that the diagnosis of left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH) (figure 2 and box 2) should not be based on voltage criteria alone. Low voltages (limb lead deflections of less than 5 mm) can occur with pericardial effusions, chronic constrictive pericarditis and hypothyroidism, but may be normal in the newborn.

R/S progression: In adults and children over 3 years of age, there is smooth progression through the precordial leads, with a dominant S wave in V_1 and V_{4R}, comparable R and S voltages in V_2 and V_3 and dominant R waves in V_{4-6}. In the neonatal period, there may be a complete reversal of this progression, with a dominant R wave in the right precordial leads and a dominant S wave in V_5 and V_6. Typically, between these ages there is a dominant R wave both in leads V_1 and V_6.

R/S ratio

In normal infants, this ratio is large in the right precordial leads and small in the left precordial leads (table 6). This pattern is reversed in adults.

T-waves and ST segments

T-waves

These represent ventricular repolarisation and display important changes throughout childhood. In lead V_1, T-wave is upright at birth and becomes inverted by week 1 of life. It remains inverted up until the age of 10 years (although the exact age can vary). Upright T-waves in lead V_1 between first week of life and 10 years can suggest right ventricular hypertrophy (box 1).

High T-wave amplitude can be seen in conditions like hyperkalaemia, ventricular hypertrophy, myocardial infarction and cerebrovascular episodes. Flat T-waves, on the other hand, can be encountered in hypothyroidism, myocarditis, electrolyte disturbances (hypokalaemia, hypocalcaemia) and are also a normal finding in the newborn period.

ST segments

A shift (elevation/depression) of up to 1 mm may be normal in limb leads, whereas up to 2 mm is normal in left precordial leads attributed to early repolarisation of the heart, although the J-point remain not elevated in early repolarisation. In pericarditis, superficial epicardial involvement may cause ST segment elevation followed by abnormal T-wave inversion as healing progresses. Administration of digoxin is associated with sagging of the ST segment and abnormal inversion of the T-wave. Depression of the ST segment may also occur in any conditions that produce myocardial damage, for example, ALCAPA, glycogen storage disease affecting the heart, myocardial tumours and cardiomyopathies.

Intervals (PR and QTc)

PR interval

It is measured from the start of the P-wave to the beginning of the QRS complex and gradually increases with age. Table 7 shows the age-specific lower and upper limits of normal. Abnormally short PR interval can be seen in WPW syndrome and glycogen storage disease.

Long PR interval can be seen in first-degree heart block, myocarditis and rheumatic fever. It can also be encountered in electrolyte disturbances, drugs and hypothermia.

A variable PR interval can be produced by some forms of second-degree heart block (Wenkebach phenomenon) and complete heart block (figure 7).

QTc interval

The QT interval is measured from the onset of the QRS complex to the end of the T-wave. The interval varies with heart rate and is corrected for heart rate

Table 6 R/S ratio in leads V1, V6 limits of normal

Age			0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
R/S ratio: 2nd–98th centile	V1	♂	0.8–3.7	0.5–5.0	0.4–4.9	0.7–4.2	0.5–2.9	0.3–1.9	0.1–1.7	0.1–1.2	0.1–1.1
		♀	1.0–4.9	0.6–4.4	0.4–4.1	0.4–3.4	0.5–2.8	0.2–1.8	0.1–1.4	0.1–1.1	0.1–1.0
	V6	♂	1.0–3.7	0.8–8.3	0.4–S=0	1.1–S=0	0.8–S=0	1.9–S=0	1.8–S=0	1.7–S=0	2.0–S=0
		♀	1.0–3.7	1.7–8.7	1.1–S=0	1.8–S=0	0.5–S=0	2.7–S=0	1.7–S=0	2.0–S=0	1.3–S=0

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

Table 7 PR interval limits of normal

Age		0–1 month	1–3 months	3–6 months	6–12 months	1–3 years	3–5 years	5–8 years	8–12 years	12–16 years
PR interval (ms), median (2nd, 98th centile)	♂	99 (77, 120)	98 (85, 120)	106 (87, 134)	114 (82, 141)	118 (86, 151)	121 (98, 152)	129 (99, 160)	134 (105, 174)	139 (107, 178)
	♀	101 (91, 121)	99 (78, 133)	106 (84, 127)	109 (88, 133)	113 (78, 147)	123 (99, 153)	124 (92, 156)	129 (103, 163)	135 (106, 176)

Adapted from Rijnbeek *et al*.[1]

The normal limits of the group of 0–1 month should be used with caution, because the sample size of this group in Rijnbeek et al study was relatively small

variability by using Bazett's formula and expressed as QTc:

$QTc = QT/\sqrt{RR}$.

Accurate calculation is very important as an abnormally long QTc can be associated with long QT syndrome and sudden cardiac death (figure 1). Normal QTc duration is up to 440 ms. Up to 450 ms in adolescent females and up to 460 ms in neonates can also be considered normal; however, values 440–460 ms are considered borderline and would warrant follow-up. Other reasons for abnormally long QT are drugs, myocarditis and hypocalcaemia.

A new cardiac entity that is associated with sudden death is the short QT syndrome. It is characterised by an abnormally short QT interval and should be suspected if QTc is shorter than 370 ms especially in the presence of symptoms or a positive family history of sudden cardiac death.[5]

CASE DISCUSSION

Case 1

Although syncope is very common presentation in the paediatric population and its causes are almost always benign, exertional syncope, on the other hand, is exceedingly rare. Loss of consciousness at the peak of exercise with minimal (chest pain, palpitations) or no prodromal symptoms should always be investigated for underlying cardiac disease. A 12-lead ECG should be performed in all children presenting with fainting.

As shown in box 3, systematic interpretation of the ECG shows an abnormally long QTc of 540 ms. In the absence of acquired causes for long QT (drugs, electrolyte abnormalities), this would be consistent with congenital long QT syndrome, especially in the presence of pathologic syncope. Diagnostic approach should include a comprehensive personal and family history, examination and referral to specialist paediatric cardiology services for inherited cardiac diseases, which typically include genetics. The patient should be advised to avoid exercise and drugs prolonging QT pending further investigations.

Case 2

In premature infants, persistent oxygen requirements post 36 weeks' corrected gestational age are usually attributed to chronic lung disease. And that is usually accurate. However, in our case, the ex-28-week gestation infant had a relatively unremarkable postnatal course. He did not require mechanical ventilation, was on continuous positive airway pressure support for 1 week and subsequently on low flow oxygen. His oxygen requirements persisted. He had a history of a patent arterial duct which was not haemodynamically significant and closed spontaneously. His 12-lead ECG is interpreted on box 4.

Upright T-waves in lead V_1 beyond the first week of life (even with otherwise normal R and S voltages in leads V_1 and V_6) should raise the suspicion of right ventricular hypertrophy. In our case, routine echocardiogram following the ECG showed evidence of a single pulmonary vein stenosis. He was referred to paediatric cardiology services. He subsequently

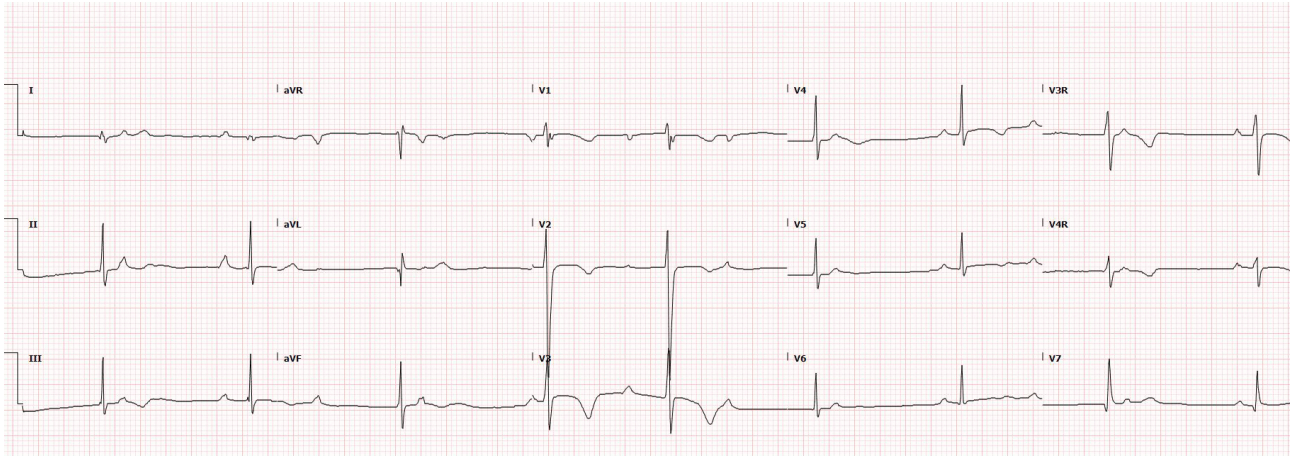

Figure 7 ECG showing complete A-V dissociation and complete heart block. aVL, augmented Vector Left; aVF: augmented Vector Foot; aVR, augmented Vector Right.

Box 3 12-Lead ECG interpretation: case 1 (figure 1)

- Rate (1500/R-R interval in mm): 100 bpm
- Rhythm: normal sinus (upright p-wave I, aVF, p-wave before every QRS, uniform PR interval)
- P-wave: normal duration and amplitude for age
- QRS complexes
  - Axis 76° (normal)
  - No abnormal q-waves
  - QRS duration (within normal limits for age)
  - RV_1=8 mm, SV_1=7 mm (within normal limits for age)
  - RV_6=12.5 mm, SV_6=2 mm (within normal limits for age)
- T-waves
  - Appropriately inverted in V_1 (extending to V_2, V_3)
  - Abnormally inverted in lead III, aVF, V_4
- ST segments: no depression or elevation
- Intervals
  - PR interval: 140 ms (within normal limits for age)
  - **QTc interval using Bazett's: 540 ms (abnormally long)**

Summary: 12-lead ECG suggests marked QTc prolongation.

Box 5 12-Lead ECG interpretation: case 3 (figure 3)

- Rate (1500/R-R interval in mm): 96 bpm
- Rhythm: normal sinus (upright p-wave I, aVF, p-wave before every QRS, uniform PR interval)
- P-wave: normal duration and amplitude for age
- QRS complexes
  - Axis –2° (normal for age)
  - No abnormal q-waves
  - QRS duration (within normal limits for age)
  - **Low QRS voltages**
- T-waves
  - **Abnormal T wave inversion in anterior leads**
  - **Flattened T waves in lateral leads**
- ST segments: no depression or elevation
- Intervals:
  - PR interval: 140 ms (within normal limits for age)
- QTc interval using Bazett's: 425 ms (normal)

Summary: 12-lead ECGs show low QRS voltages with abnormal repolarisation (T-wave inversion in anterior leads and flattened T-waves in lateral leads).

developed second pulmonary vein stenosis and underwent pulmonary venous dilatation via cardiac catheterisation.

Case 3

This case demonstrates that cardiac failure may not always present with the classical signs described in textbooks. Especially in the first year of life, infants may present with symptoms resembling lower respiratory tract infections and can mislead even experienced paediatricians. Detailed history combined with thorough clinical examination is the key to raise the suspicion of an underlying cardiac defect.

The 15-year-old girl in our case had decreasing exercise tolerance over a period of 4 weeks. Two days prior to her presentation, she became increasingly tachypnoeic, with orthopnoea and chest pain with minimal activity. On clinical examination, she had gallop rhythm, hepatomegaly, evidence of pedal oedema and raised jugular venous pressure. She remained inappropriately tachycardic throughout her admission despite the absence of fever.

Her 12-lead ECG (figure 3) shows low voltage QRS complexes, T wave inversion in anterior leads and flattened T waves in lateral leads suggesting abnormal repolarisation (see box 5 for full interpretation). Echocardiogram showed LV dilatation with impaired LV function which would suggest dilated cardiomyopathy. She was transferred to a specialist paediatric cardiac unit where she was treated conservatively with anti-failure medications. She remains under regular follow-up.

Box 4 12-Lead ECG interpretation: case 2 (figure 2)

- Rate (1500/R-R interval in mm): 120 bpm
- Rhythm: normal sinus (upright p-wave I, aVF, p-wave before every QRS, uniform PR interval)
- P-wave: normal duration and amplitude for age
- QRS complexes
- Axis 147° (right axis deviation, can be normal for age)
  - No abnormal q-waves
  - QRS duration (within normal limits for age)
  - RV_1=21 mm, SV_1=0.5 mm (within normal limits for age)
  - RV_6=0 mm, SV_6=3 mm (within normal limits for age)
  - **R/S ratio V_1=42 (>98th centile for age)**
  - **R/S ratio V_6=0 (<2nd centile for age)**
- T-waves
  - **Abnormally upright in V_1**
- ST segments: no depression or elevation
- Intervals
  - PR interval: 120 ms (within normal limits for age)
  - QTc interval using Bazett's: 417 ms (normal)

Summary: 12-lead ECG suggests right ventricular hypertrophy.

CLINICAL BOTTOM LINE

There is not a single best way to interpret a 12-lead ECG; however, it is important that the clinician adopts an approach that is systematic and methodical to ensure that important abnormalities are not being missed.[5 6] It is worth reiterating that every ECG must be interpreted in the light of clinical information about the patient. The age-specific limits of normal are an essential tool for an accurate assessment and we recommend that are readily available to the interpreter.

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