REVIEW ARTICLE

A Practical Guide to Paediatric ECG Interpretation for General Practitioners: From Age-Related Normality to Recognition of Pathology

Bello Abdullahi Ibrahim[1], Surajudeen Oyeleke Bello[2]

Abstract

The electrocardiogram (ECG) is a vital diagnostic tool in children presenting with syncope, chest pain, palpitations, and suspected heart conditions. However, paediatric ECG interpretation presents unique challenges due to significant age-related physiological changes affecting heart rate, axis, intervals, and wave morphology; hence, what is normal for a neonate may be abnormal for an adolescent. General practitioners (GPs) are often the first point of contact for children with these presentations, yet many report limited confidence in interpreting paediatric ECGs. Recent evidence from a 2025 study demonstrated a mean accuracy of 76.6% among clinicians, with notable misdiagnosis rates in emergency and resource-limited settings.

This article presents a practical, evidence-informed framework for paediatric ECG interpretation, developed through synthesis of peer-reviewed literature, published normative data, and established cardiology guidelines. We provide an overview of developmental physiology, a structured five-step approach to ECG analysis, and discussions of common arrhythmias and red flags requiring urgent cardiology referral. Reference tables offer age-adjusted normal parameters, and a summary algorithm supports clinical decision-making. Technical considerations, including lead placement, motion artefact, and the limitations of automated interpretations, are addressed to reduce misdiagnosis. The goal is to bolster GPs' confidence in distinguishing benign from dangerous conditions, enhance patient safety, and optimise the utilisation of specialist care.

Keywords: Paediatric electrocardiography, ECG interpretation, age-related changes, arrhythmias, medical education, general practice

Introduction

The electrocardiogram (ECG) is one of the most frequently performed non-invasive cardiovascular investigations in paediatric practice. Indications include evaluation of syncope, chest pain, palpitations, suspected arrhythmias, drug ingestion, electrolyte disturbances, and initial assessment of suspected congenital or acquired heart disease. [1,2] General practitioners (GPs) are often the first point of contact for children with these presentations, yet many lack formal training in paediatric ECG interpretation.

Despite the widespread availability of ECG machines, paediatric ECG interpretation remains a source of anxiety for many medical officers and general practitioners. This unease is understandable: the paediatric ECG is not simply a smaller version of the adult tracing. It is a dynamic record that evolves with the physiological changes of the growing heart, and findings that would be pathological in an adult may be entirely normal in a child. [3,4]

The clinical significance of this challenge is underscored by recent evidence. A 2025 study evaluating ECG interpretation skills among paediatric trainees and faculty reported a mean accuracy score of only 76.6%, with major over- or under-diagnosis (errors with potential for patient harm) occurring in 12.3% of interpretations.[5] These findings align with

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earlier work demonstrating that misinterpretation of paediatric ECGs in emergency department settings can lead to diagnostic delays and inappropriate clinical decisions.[6]

Despite the availability of numerous educational resources on paediatric ECG interpretation, many are either comprehensive textbooks that are impractical for rapid clinical reference or online materials that lack a systematic approach and age-specific normative data. Consequently, non-specialist frontline clinicians have limited access to concise, clinically oriented guidance that integrates developmental physiology, age-adjusted normal values, a structured interpretation algorithm, and clear referral criteria. This review aims to address this gap by providing an evidence-informed, stepwise approach to paediatric ECG interpretation for general practitioners and other frontline clinicians. It outlines the developmental basis of age-related ECG changes, presents practical reference tables of normal values, discusses common normal variants and clinically important abnormalities, highlights red flags requiring specialist referral, and reviews technical factors that influence ECG quality and interpretation.

Methodology

Evidence synthesis

This article is an evidence-informed narrative review developed to provide a practical clinical guide for frontline clinicians. Although not conducted as a formal systematic review, the literature search and evidence synthesis followed a structured and transparent approach consistent with recommendations for high-quality narrative reviews.

Literature search

Electronic searches were performed in PubMed, Google Scholar (for supplementary citation tracking), and the Cochrane Database of Systematic Reviews for publications from January 2000 to December 2025. Search terms included paediatric ECG, paediatric electrocardiogram, ECG interpretation, normal ECG values in children, age-related ECG changes, paediatric arrhythmias, long QT syndrome in children, and ECG referral criteria. Reference lists of key publications and relevant guidelines were also manually screened to identify additional eligible sources.

Eligibility criteria

Eligible publications included peer-reviewed original research, systematic reviews, clinical practice guidelines, consensus statements, and authoritative textbook chapters addressing paediatric ECG interpretation, normal age-specific values, cardiac conduction abnormalities, or arrhythmia recognition in children (0–18 years). Non-English publications, conference abstracts, editorials, and adult-only studies were excluded. Case reports were included only when illustrating clinically important diagnostic findings.

Evidence synthesis

Evidence was synthesized narratively, with emphasis on studies providing age-specific normative ECG data, diagnostic accuracy, and clinically relevant interpretation strategies. Greater weight was given to large population-based studies, internationally recognised reference datasets (including those of Davignon *et al*. and Rijnbeek *et al*.),[12][13] and current professional society guidelines. Where discrepancies existed between sources, recommendations were based on the best available evidence, prioritising contemporary guidelines and consistently validated reference values. Recommendations for referral criteria were cross-referenced with published cardiology society guidelines.

Limitations

As a narrative review, this article did not include a formal risk-of-bias assessment or quantitative meta-analysis. Consequently, recommendations should be interpreted alongside clinical judgement, institutional protocols, and specialist consultation where appropriate.

The Developmental Physiology of the Paediatric ECG

Understanding why the paediatric ECG changes with age requires an appreciation of the dramatic circulatory transition at birth and subsequent cardiac maturation.

The Foetal and Neonatal Circulation. In utero, the placenta serves as the organ of gas exchange. The pulmonary vasculature is constricted, resulting in high pulmonary vascular resistance, while the systemic circulation offers relatively low resistance. The right ventricle pumps against a high-pressure system and becomes the dominant chamber, hypertrophying to exceed the left ventricle at birth.[4,7] This right ventricular dominance explains why neonatal ECGs differ markedly from adult tracings.

Postnatal Transition. With the first breaths after delivery, the lungs expand, pulmonary vascular resistance falls dramatically, and systemic vascular resistance increases as the umbilical cord is clamped. The foramen ovale and ductus arteriosus close, establishing adult-type circulation. Over subsequent weeks and months, the left ventricle gradually assumes its role as the systemic pump, while right ventricular mass regresses relatively as the left ventricle hypertrophies in response to increasing systemic demands. [8] This ventricular remodelling is reflected in the gradual leftward shift of the QRS axis and the progression of precordial R-waves throughout childhood. [9] Adult-type R-wave progression typically emerges by 3–4 years of age, though individual variation is considerable.

Electrophysiological Correlates. The smaller physical dimensions of the infant heart mean electrical impulses travel shorter distances, resulting in shorter durations of all ECG intervals. The PR interval, QRS duration, and QT interval all increase progressively with age as the heart enlarges and conduction pathways elongate. [10] Similarly, the resting heart rate is highest in the neonatal period, when cardiac output is heart rate-dependent due to the relatively noncompliant immature ventricle, and gradually declines to adult values by adolescence. [4]

Special Considerations for Preterm Infants. Infants born prematurely may exhibit ECG patterns more typical of their corrected gestational age than chronological age. Normative data in this population are limited, and cardiology input is advisable when significant abnormalities are suspected in ex-preterm infants. [2]

These developmental changes mean that a single set of "normal" values cannot apply to all children. Age-specific normative data are essential, and the clinician's first step in interpretation must always be to note the child's age.

Limitations of ECG as a Diagnostic Tool

Clinicians must be aware that the ECG has important

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limitations in paediatric practice:

1. A normal ECG does not exclude structural heart disease. A child with a significant ventricular septal defect, aortic stenosis, or even hypertrophic cardiomyopathy may have a normal or near-normal ECG. Clinical decision-making must always integrate history and physical examination.
2. Poor sensitivity for certain conditions. The ECG has low sensitivity for detecting atrial septal defects, mild ventricular hypertrophy, and early cardiomyopathy.[14] A normal ECG should not provide false reassurance when clinical suspicion is moderate or high.
3. Age-related overlap. Normal and abnormal values overlap considerably at age extremes (neonates and adolescents), making borderline findings challenging to interpret without serial tracings or echocardiography.
4. Technical limitations. Paediatric ECGs are more susceptible to artefact from crying, movement, and lead misplacement than adult tracings. Poor-quality recordings may simulate pathology.
5. Machine interpretation is unreliable. Automated ECG reports frequently misclassify normal paediatric variants as abnormal and may miss significant abnormalities, particularly prolonged QTc intervals and subtle pre-excitation patterns.

Therefore, the ECG should be viewed as one component of clinical assessment, not a standalone diagnostic test. Over-reliance on ECG findings without appropriate clinical correlation can lead to both under-diagnosis and over-investigation.

Technical Considerations Before Interpretation

Before interpreting any paediatric ECG, the clinician must ensure the tracing is technically adequate. Errors in lead placement, motion artefact, and crying can all produce abnormalities that mimic pathology. [11]

Lead Placement. Precordial lead misplacement is common and can dramatically alter R-wave progression. Key points:

· V1 and V2 should be placed in the 4th intercostal space, right and left of the sternum.
· V3 is midway between V2 and V4.
· V4 should be placed in the 5th intercostal space at the midclavicular line (not simply "under the nipple," as nipple position varies).
· Misplacement of V1 and V2 superiorly can produce an rSr' pattern mimicking right ventricular conduction delay. [11]

Motion Artefact. Young children rarely lie still during ECG recording. Crying, limb movement, and respiratory variation can produce baseline wander, muscle tremor artefact, and apparent arrhythmias. If the tracing is significantly degraded, repeating the ECG with adequate distraction, swaddling, or after feeding an infant is preferable to interpreting a poor-quality tracing.

Effects of Crying and Tachypnoea. Crying increases heart rate and can produce transient ST-segment changes due to tachycardia and altered ventricular repolarisation. Sinus tachycardia from crying should not be mistaken for supraventricular tachycardia; the gradual onset and offset, along with rate variability during crying, typically distinguish it. [11]

Half-Standardisation Recognition. Some paediatric ECGs are recorded at half-standardisation (5 mm/mV instead of 10 mm/mV) when the voltages are very high. This should be noted on the tracing (e.g., "5 mm/mV" or "half gain"). When half standardisation is used, voltage measurements must be doubled for interpretation. Conversely, double standardisation (20 mm/mV) may be used for low-voltage tracings, thereby halving the required voltage measurements. Always check the calibration marker at the beginning of the tracing before measuring voltages.

When to Repeat. If the tracing shows significant artefact, if lead placement is uncertain, or if findings are discordant with the clinical picture, repeating the ECG is always appropriate. A suboptimal tracing interpreted with false confidence is more dangerous than delaying interpretation for a technically adequate recording.

A Stepwise Approach to Paediatric ECG Interpretation

A systematic, reproducible approach minimises the risk of overlooking abnormalities. The following five-step framework is adapted from established paediatric cardiology guidelines and is suitable for routine clinical use. [2,4,12] Always begin by confirming that the ECG belongs to the correct patient and checking the recording speed (standard 25 mm/s) and voltage calibration (standard 10 mm/mV). A one-page summary algorithm is provided in Figure 1.

Step 1: Rate – Is It Appropriate for Age?

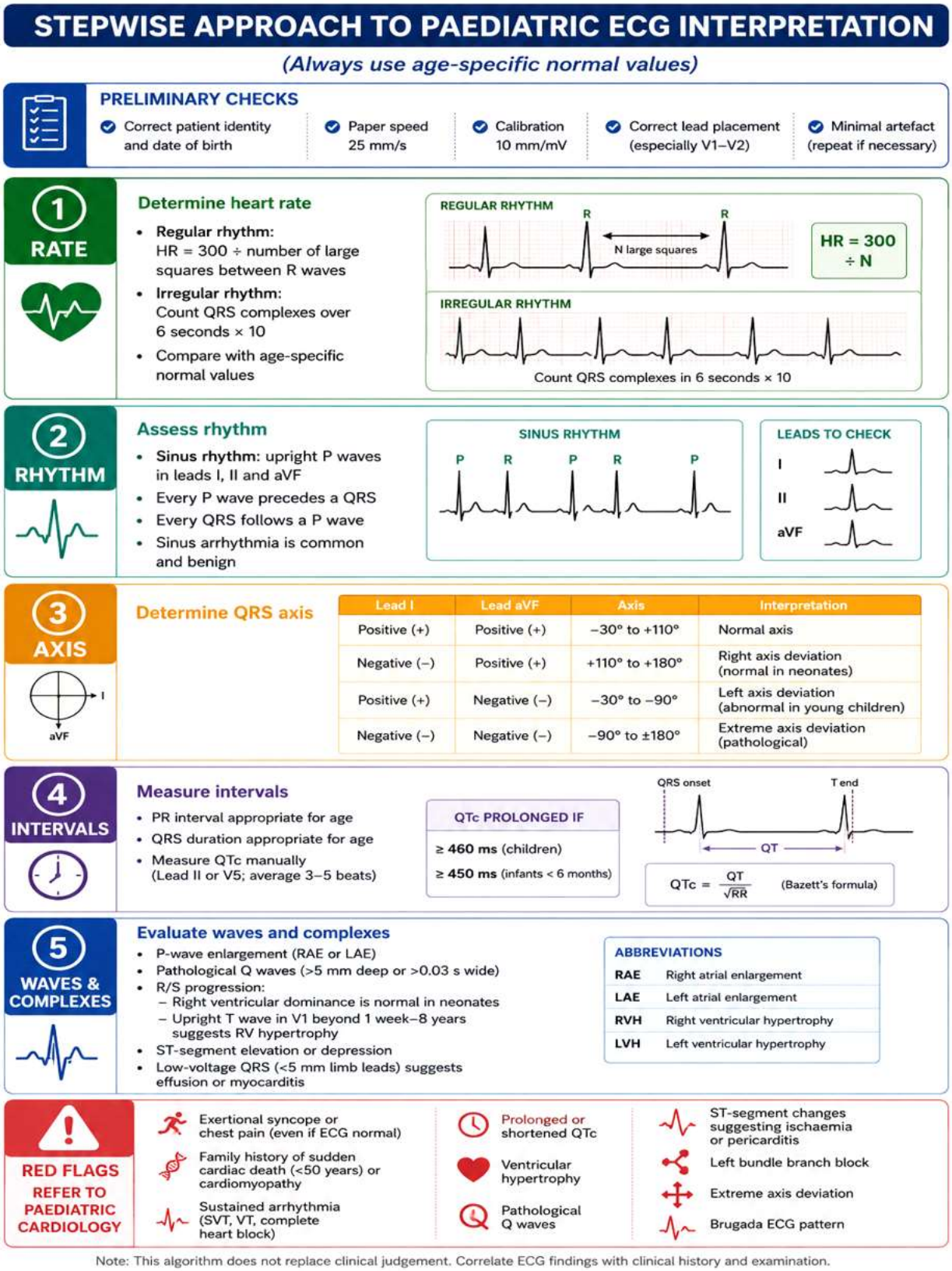
Heart rate varies inversely with age. Tachycardia or bradycardia must be assessed against age-specific reference ranges (Table 1). An approximate rate can be calculated by dividing 300 by the number of large squares (0.2 s) between consecutive R waves (for regular rhythms). For irregular rhythms, count the number of complexes over a 6-second interval (30 large squares at 25 mm/s) and multiply by 10 to obtain the rate in bpm.

Table 1: Age-Specific Normal Heart Rates and Upper Limits of Intervals

Age	Heart Rate (bpm)	QRS Axis (°)	PR Interval (ms) (upper limit)	QRS Duration (ms) (upper limit)	QTc Upper Limit (ms)†
Preterm infant*	120–180	60–180	120	70	460
Neonate (0–30 days)	90–180	30–180	120	70	450–460‡
Infant (1–12 months)	110–160	5–125	140	75	450
1–5 years	100–150	5–110	170	75	440
6–12 years	80–120	20–120	190	85	440
12+ years	60–100	–30–105	210	100	440 (males) / 460 (females)

Compiled from references [2,4,12,13]; normative data for preterm infants are limited—values shown are approximations. †All QTc values require **manual measurement**; automated readings are unreliable; ‡Some sources use 460 ms for all children under 6 months; clinical correlation advised.

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Note: Manual interpretation is recommended for all paediatric ECGs as machine-generated reports may be unreliable in children. Always verify the heart rate, QTc interval, and the presence of delta waves/pre-excitation [Developed by the authors based on published evidence]

Figure 1: Paediatric ECG Interpretation Algorithm

Step 2: Rhythm – Is It Sinus?

Sinus rhythm is present when: (1) P waves are upright in leads I, II, and aVF (indicating a normal P-wave axis of 0° to +90°); (2) a QRS complex follows every P wave; and (3) every QRS complex is preceded by a P wave. [2] The rhythm should be roughly regular, although **sinus arrhythmia**, cyclic variation in the R-R interval with respiration is an extremely common and entirely benign finding throughout childhood (see Figure 2). [15]

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Step 3: Axis – Is the QRS Axis Normal for Age?
The net vector of ventricular depolarisation determines the mean QRS axis in the frontal plane. The normal axis shifts leftward with age (Table 1). In neonates, right axis deviation (up to +180°) is normal; by adolescence, the axis should fall within the adult range (–30° to +105°).

Simplified Bedside Axis Determination: For clinicians less comfortable with vector calculations, a rapid assessment can be made:

- **Normal axis:** QRS predominantly positive in both lead I and lead aVF.
- **Right axis deviation (RAD):** QRS predominantly negative in lead I but positive in aVF.
- **Left axis deviation (LAD):** QRS predominantly positive in lead I but negative in aVF.
- **Extreme ("north-west") axis:** QRS negative in both lead I and aVF (range –90° to ±180°). This is always pathologic.

Left axis deviation (less than –30°) is abnormal in young children and may suggest conditions such as atrioventricular septal defect, tricuspid atresia, or left ventricular hypertrophy. [2,15] Extreme axis deviation warrants urgent echocardiography.

Step 4: Intervals – PR, QRS, and QTc
PR Interval. Measured from the onset of the P wave to the onset of the QRS complex (best in lead II), the PR interval reflects conduction time through the atria and AV node. It increases with age and decreases with faster heart rates. A **short PR interval** (below the **lower** limit for age) may be a normal variant but should prompt a search for delta waves suggestive of Wolff-Parkinson-White (WPW) syndrome. [12] A **prolonged PR interval** (above the upper limit for age) indicates first-degree AV block, which can be idiopathic, seen in myocarditis, or associated with congenital heart disease such as Ebstein anomaly or atrial septal defect. [2]

QRS Duration. Measured from the onset of the Q wave to the termination of the S wave (best in lead II or V5/6), QRS duration reflects ventricular depolarisation time and increases with age as ventricular mass increases (Table 1). Prolongation beyond age-specific norms suggests bundle branch block or ventricular conduction delay. **Right bundle branch block** (RSR' pattern in V1 with wide terminal S in V6) is common after cardiac surgery but can occur as an isolated finding. **Left bundle branch block** is rare in children and, if present, almost always indicates underlying structural heart disease. [2,16]

QT Interval and QTc. The QT interval varies inversely with heart rate, necessitating correction (QTc). The Bazett formula (QTc = QT / √RR) remains the most widely used method despite its limitations at extremes of heart rate, i.e., it tends to overcorrect at fast rates (>150 bpm) and undercorrect at slow rates. In infants with tachycardia, alternative formulae (Fridericia, Framingham) may be more accurate, but cardiology review is advisable if QTc is borderline. [17]

Critical Point: Automated QTc readings are unreliable and must not be used in isolation. Measurement should be performed manually in lead II or V5, averaging 3–5 beats to account for respiratory variation. [2] Prolonged QTc (≥460 ms in children, ≥450 ms in infants under 6 months) is a marker for potentially life-threatening ventricular arrhythmias and requires cardiology referral. [18] Causes include congenital long QT syndromes, electrolyte disturbances (hypokalaemia, hypocalcaemia, hypomagnesaemia), and medications. A QTc < 340 ms (short QT syndrome) is also associated with arrhythmia risk and should prompt specialist review. [15]

Step 5: Waves and Complexes – P, QRS, ST, T
P Waves. Normal P waves are ≤3 mm in amplitude and <0.10 s duration in children (<0.08 s in infants). [2] Tall, peaked P waves (P pulmonale) suggest right atrial enlargement; broad, notched P waves (P mitrale) suggest left atrial enlargement.
QRS Morphology and Voltage. The normal evolution of precordial R-wave progression reflects the changing balance of ventricular forces. In neonates and infants, dominant R waves in the right precordial leads (V1–V3) with deep S waves in the left precordial leads are normal, mirroring right ventricular dominance.[4] Adult-type R-wave progression (R > S in V5–V6, S > R in V1–V2) typically emerges by 3–4 years of age. An RSR' pattern in V1 without QRS prolongation (incomplete right bundle branch block pattern) is a common normal variant, present in up to 10% of healthy children. [16]

Simplified Screening for Ventricular Hypertrophy:

- **Suspect RVH if:** R wave in V1 > 98th percentile for age; upright T wave in V1 between 1 week and 8 years of age; pure R wave or qR pattern in V1. [12,16]
- **Suspect LVH if:** S in V1 + R in V6 (or V5) > age-specific 98th percentile; R wave in V6 > 25 mm in infants or >30 mm in older children; inverted T waves in V5–V6 (strain pattern). [12,16]

While detailed age-based 98th percentile criteria have been published (Davignon et al., 1979) [13], we recommend simplified voltage thresholds (e.g., RV6 > 2.5 mV or SV1 > 2.0 mV for LVH) during initial screening. These fixed cut-offs are derived from the upper limits of normal and offer a practical, bedside-friendly alternative without compromising diagnostic utility for the purposes of this review. Regardless of which ECG criteria you choose (percentile-based or simplified), it is essential to remember that echocardiography remains the gold standard for confirming ventricular hypertrophy, given the inherent low sensitivity of ECG

Q Waves. Small Q waves (≤5 mm depth, ≤0.03 s duration) are normal in the inferior (II, III, aVF) and left precordial (V5–V6) leads after the first few days of life. Pathological Q waves, i.e., deep (>5 mm) and/or wide (>0.03 s) suggest myocardial infarction or fibrosis and may be seen in anomalous left coronary artery from the pulmonary artery (ALCAPA) or hypertrophic cardiomyopathy. [2,15]

ST Segment and T Waves. The ST segment should be isoelectric. Minor ST elevation (≤1 mm in limb leads, ≤2 mm in precordial leads) can be normal, particularly in adolescents with early repolarisation.[15] Significant ST depression or elevation suggests ischaemia, pericarditis, or ventricular strain. Low-voltage QRS (<5 mm in limb leads) may indicate pericardial effusion or myocardial dysfunction and warrants echocardiography.

T-wave interpretation is highly age-dependent. At birth, T waves may be upright in all precordial leads. By **day 3–7 of**

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life, the T waves in V1–V3 normally invert, reflecting changing haemodynamics and ventricular regression. This **"juvenile T-wave pattern"** of inverted T waves in the right precordial leads can persist into adolescence (typically resolving by 12–14 years, though variable) and is a normal finding. [4,8] However, T waves should remain upright in the inferior and lateral leads (I, II, aVF, V5–V6) after the first week. **Persistently upright T waves in V1 between 1 week and approximately 8 years of age suggest right ventricular hypertrophy until proven otherwise.** [2,16] Some normal infants may retain upright T waves in right precordial leads beyond day 7 without pathology, but correlation with other RVH signs is essential.

Common Normal Variants in Children

Several findings that might trigger concern in an adult ECG are entirely benign in children. Recognising these variants prevents unnecessary investigation and anxiety.

- **Sinus Arrhythmia:** Phasic variation in heart rate with respiration is nearly universal in children and represents normal autonomic modulation. [15]
- **Juvenile T-Wave Pattern:** Inverted T waves in leads V1–V3 (and occasionally V4) are normal throughout childhood and may persist into early adulthood; they typically resolve by adolescence. [4,8]
- **Incomplete Right Bundle Branch Block:** An rSR' pattern in V1 with a normal QRS duration is a frequent finding (up to 10% of healthy children), attributed to delayed activation of the right ventricular outflow tract. Longitudinal studies confirm its benign nature in isolation. [16,19]
- **Sinus Bradycardia of the Athlete:** Well-conditioned adolescents may exhibit resting heart rates in the 40s–50s bpm, similar to adult athletes. [15]
- **Early Repolarisation:** Concave ST elevation with notching at the J point, most prominent in the lateral precordial leads, is a common normal variant in adolescents, particularly males. [15]
- **Neonatal Right Ventricular Dominance:** Dominant R waves in V1–V3 with deep S waves in V5–V6 are normal in the first months of life, gradually transitioning to the adult pattern by 3–4 years. [4]

Recognising Common Pathologies (With Example ECG Tracings)

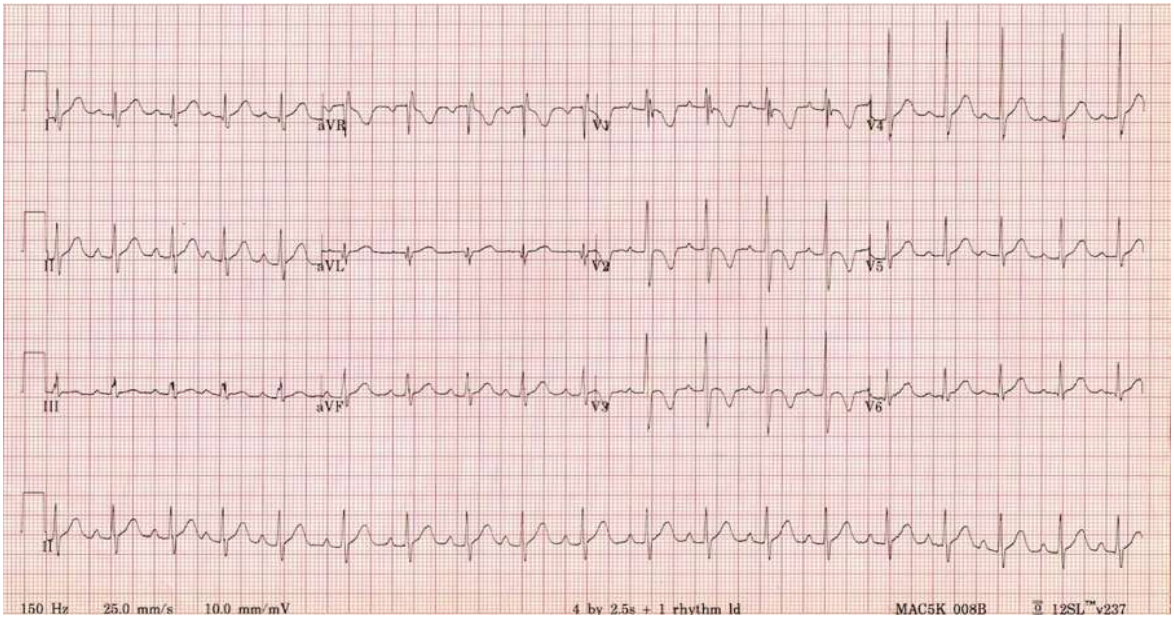


Figure 2: 12-lead ECG of an 8-year-old female illustrates normal paediatric ECGs(It also shows: Heart rate of 110 bpm (normal for age), Juvenile T-wave pattern (T wave inversion in V1-3), Dominant R waves in V1-3, RSR' pattern (partial RBBB morphology) in V1.[8] **Written informed consent was obtained from the patient's guardian for publication of this image.**

ECG recorded using a MAC 5K device with Marquette™ 12SL ECG Analysis Program v237, GE HealthCare.

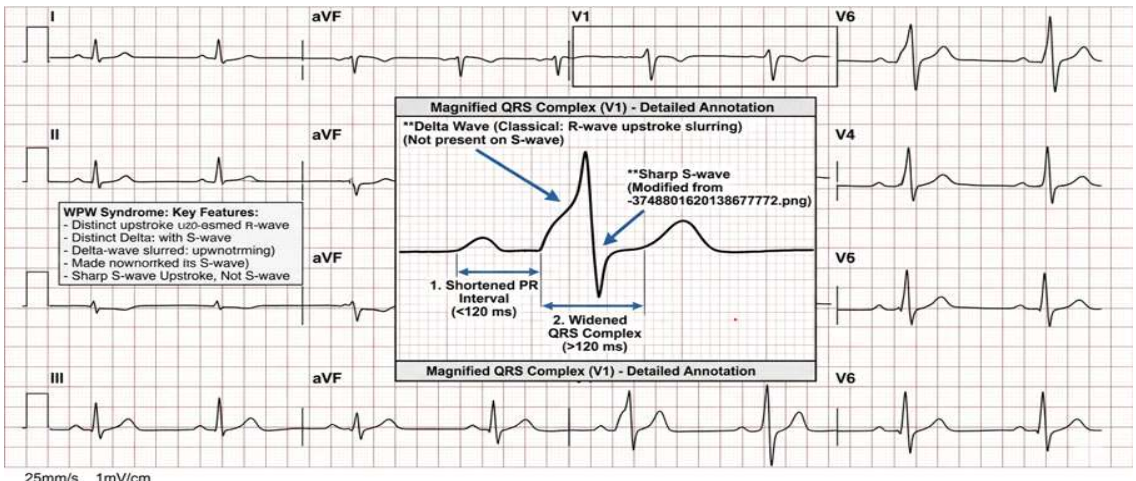


Figure 3a: shows the characteristic delta wave of Wolff-Parkinson-White (WPW) syndrome [Focus on slurred upstroke of the R]. The image is generated with CorelDRAW 13 as a teaching aid based on the information in Park MK [8]

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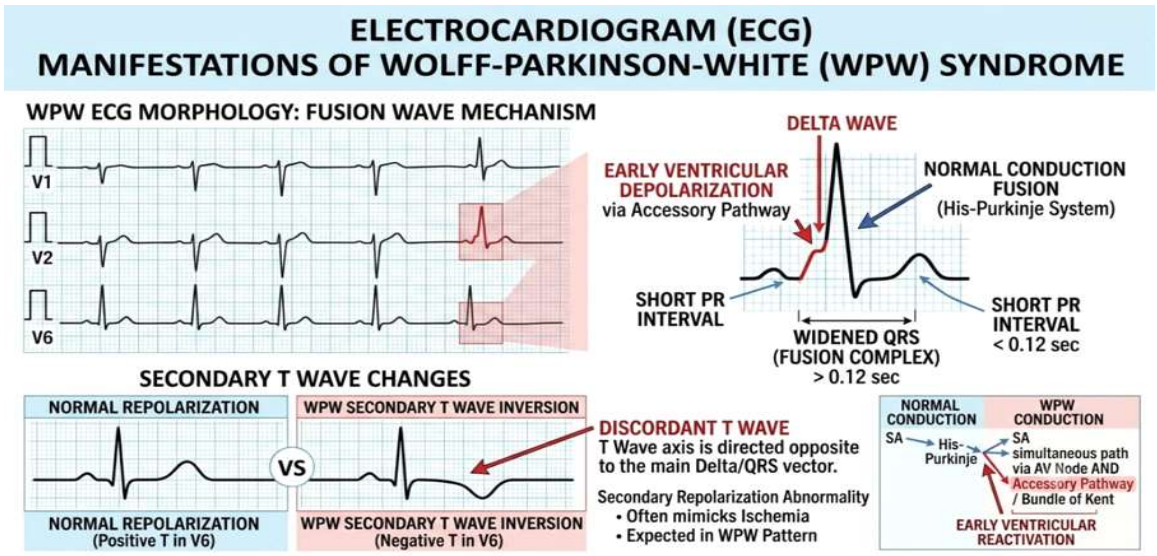


Figure 3b: T wave changes in Wolff-Parkinson-White syndrome. Schematic showing early ventricular activation with fusion of normal and pre-excited conduction leading to early ventricular depolarisation. Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

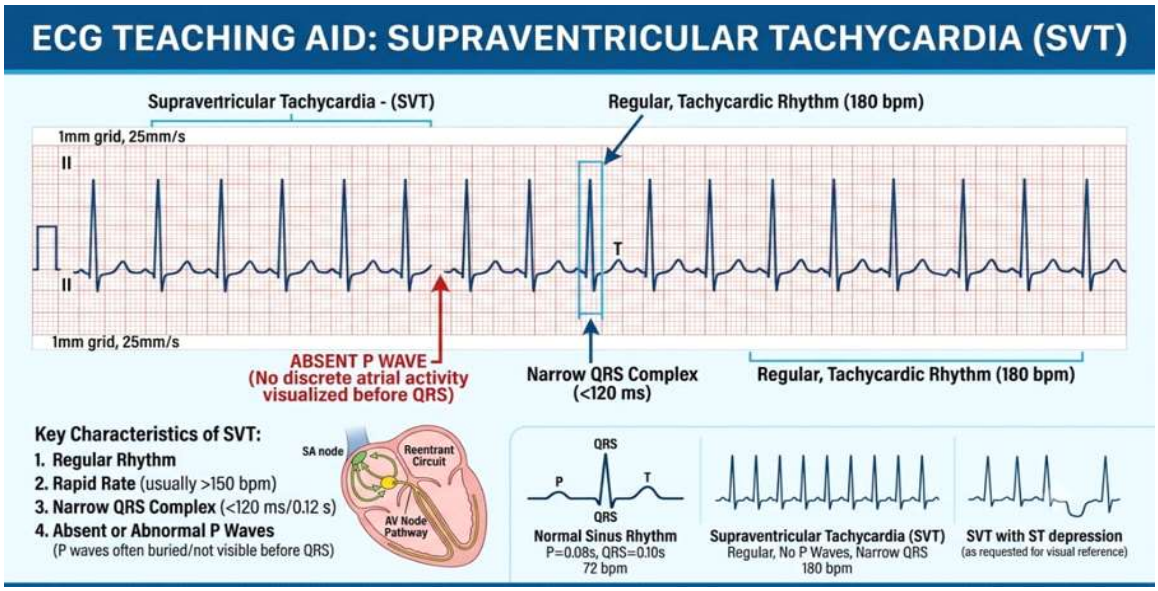


Figure 4 shows narrow QRS complexes and absent P waves seen in supraventricular tachycardia (SVT). Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

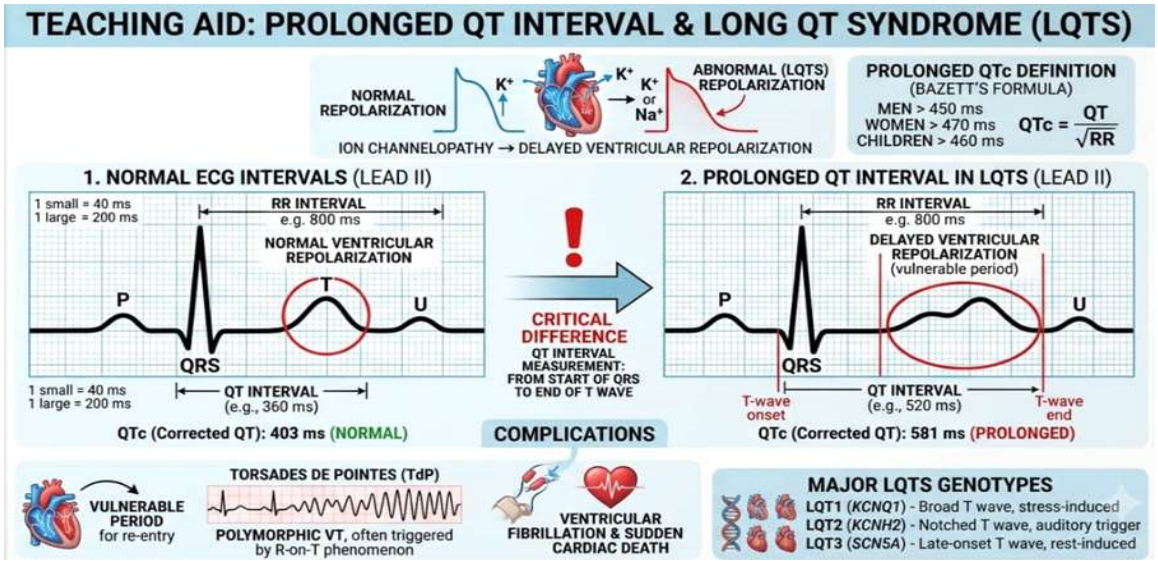


Figure 5 shows a prolonged QT interval in long QT syndrome. The image is generated with CorelDRAW 13 as a teaching aid based on the information in Park MK.[8]

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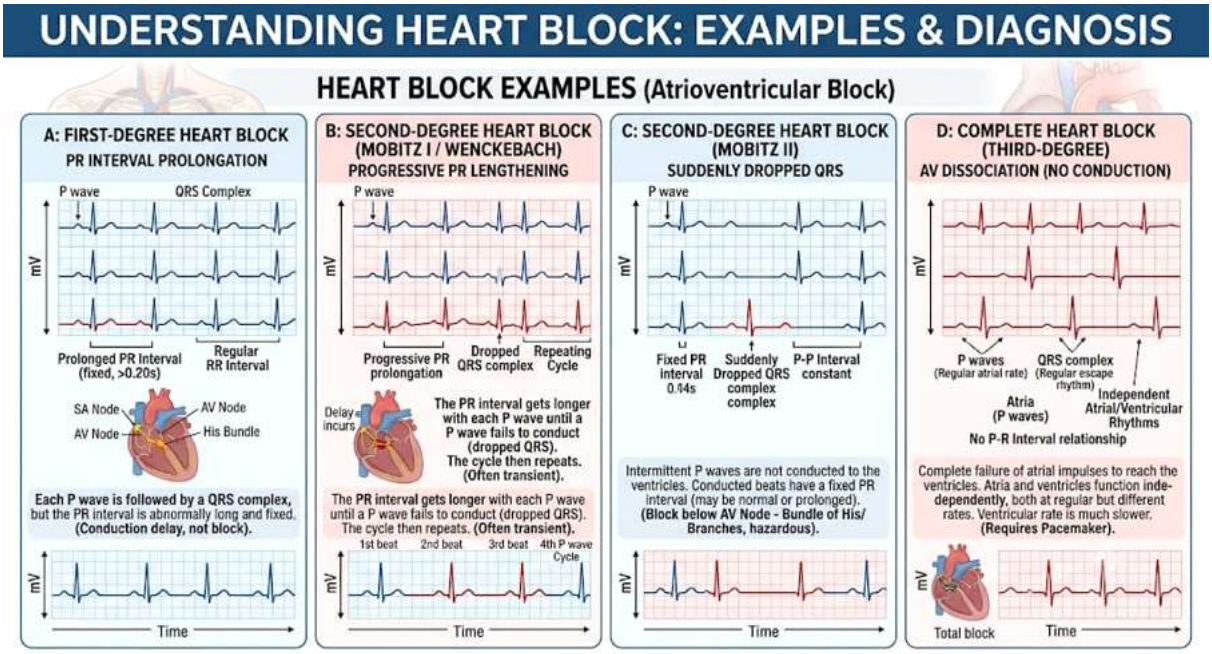


Figure 6 illustrates examples of heart block (first-degree [A], second-degree Mobitz I [B] and II [C], and complete heart block [D]). Figure redrawn by the authors using CorelDRAW 13 based on Park MK.[8]

Supraventricular Tachycardia (SVT)

SVT is the most common symptomatic tachyarrhythmia in children, with an incidence

of approximately 1 in 250–1,000. [20] Most cases are due to accessory pathways (WPW syndrome) or AV nodal re-entry (Figure 4).

ECG Features: Narrow-complex tachycardia (QRS duration normal for age) with a regular, rapid rate. P waves are often absent or difficult to discern. Rates typically exceed **220 bpm in infants** and **180 bpm in older children.** [4,12] Rates **>300 bpm in infants** should prompt consideration of atrial flutter, which may have visible flutter waves. In WPW syndrome, the baseline (sinus rhythm) ECG shows a short PR interval and a delta wave (slurred upstroke of the QRS complex), indicating ventricular pre-excitation.

Clinical Tip: SVT has an abrupt onset and termination, in contrast to sinus tachycardia, which accelerates and decelerates gradually. Haemodynamic status depends on rate, duration, and age; infants may present with poor feeding, irritability, or signs of heart failure, while older children report palpitations or chest tightness.[20]

Ventricular Hypertrophy

Right Ventricular Hypertrophy (RVH): ECG findings include (1) R wave in V1 exceeding the 98th percentile for age; (2) upright T wave in V1 between 1 week and 8 years of age; (3) pure R wave or qR pattern in V1; (4) right axis deviation for age. [12,16] Causes include pulmonary stenosis, tetralogy of Fallot, and pulmonary hypertension.

Left Ventricular Hypertrophy (LVH): Findings include (1) S wave in V1 + R wave in V6 (or V5) > age-specific 98th percentile; (2) R wave in V6 exceeding the 98th percentile; (3) inverted T waves in V5–V6 (strain pattern); (4) left axis deviation. [12,16] A **simplified screening** is R wave in V6 >25 mm in infants or >30 mm in older children, and this should warrant formal voltage calculation. Causes include aortic stenosis, coarctation of the aorta, hypertrophic cardiomyopathy (HCM), and large left-to-right shunts such as ventricular septal defect.

Long QT Syndrome

Congenital long QT syndrome is an important cause of syncope, seizures, and sudden cardiac death in children and young adults. The hallmark ECG finding is a prolonged QTc interval (≥460 ms in children; ≥480 ms in symptomatic patients or those with a strong family history). [18] Additional features may include T-wave alternans or notched T waves. **Manual measurement in lead II or V5 is essential as automated readings are unreliable and may miss up to 40% of cases with prolonged QTc.** [18] Beta-blocker therapy and lifestyle modification are mainstays of treatment, underscoring the importance of accurate identification (Figure 5).

Myocarditis and Pericarditis

These conditions may present with chest pain, fever, and elevated cardiac enzymes. ECG findings in pericarditis include diffuse ST-segment elevation (concave up) and PR-segment depression, which later evolve into T-wave inversion. Myocarditis may produce low-voltage QRS complexes (<5 mm in limb leads), ST-T wave changes, and conduction abnormalities. [2] Low voltage with clinical suspicion of myocarditis or effusion warrants urgent echocardiography.

Arrhythmias Requiring Immediate Attention

- **Ventricular Tachycardia:** Wide QRS tachycardia (QRS duration > upper limit for age) at a rate >120 bpm or 20% faster than sinus. Requires immediate assessment and cardiology involvement.
- **Complete Heart Block:** Atrial rate normal for age but with complete AV dissociation; ventricular escape rate often slow (40–60 bpm in children, slower in infants). May be congenital (associated with maternal lupus) or acquired (post-surgical) (Figure 6).
- **Brugada Pattern:** Coved-type ST elevation in V1–V2 (type 1 Brugada pattern) is rare but important to recognise in children with syncope or a family history of sudden death. [14]

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Table 2: Quick Reference for Common Clinical Scenarios

Clinical Scenario	Key ECG Feature to Assess	ECG Findings to Document	Common Diagnoses	Action
Palpitations/ tachycardia	Rate, P waves, QRS width	Rate, regularity, P wave visibility, QRS duration	SVT (narrow complex, no P waves, rate >220 infant/>180 child); Atrial flutter (>300 bpm in infants)	Vagal manoeuvres, adenosine; refer
Syncope (exertional)	QTc, ventricular hypertrophy, pre-excitation	QTc (manual), voltages, delta waves	Long QT, HCM (LVH, deep Q waves), WPW (delta waves)	Urgent cardiology referral
Syncope (non-exertional)	Rhythm, pauses, QTc, Brugada pattern	Baseline rhythm, pauses, QTc, ST in V1-V2	Vasovagal (normal ECG), long QT, Brugada (rare)	Refer if QTc is prolonged or Brugada pattern
Chest pain	ST segments, T waves, hypertrophy	ST elevation/depression, T-wave inversion, voltages	Pericarditis (diffuse ST elevation), normal variant (early repolarisation), HCM (LVH)	Treat cause; refer if abnormal
Murmur	Ventricular voltages, axis	R and S voltages, axis	RVH (RV dominance beyond infancy), LVH (VSD, AS, HCM)	Echocardiogram if suspected
Incidental bradycardia	Rhythm, PR interval, heart rate	Rate, PR interval, AV relationship	Sinus bradycardia (athlete), heart block (AV dissociation)	Check symptoms; refer if AV block
Suspected myocarditis	Voltages, ST-T changes	QRS amplitude, ST segments	Low voltage, ST changes, conduction delay	Urgent echocardiogram; refer

Red Flags and Indications for Referral

While many paediatric ECGs are normal, certain findings mandate discussion with or referral to a paediatric cardiologist:

1. Any ECG from a child with exertional syncope, chest pain, palpitations, or a family history of sudden cardiac death (first-degree relative <50 years) or cardiomyopathy in a family member**,** irrespective of whether the ECG appears normal. [1]
2. Sustained arrhythmias, including SVT, ventricular tachycardia, or complete heart block.
3. **QTc prolongation** (≥460 ms in children, ≥450 ms in infants under 6 months) or QTc shortening (<340 ms).
4. **Ventricular hypertrophy** criteria, particularly if associated with symptoms or abnormal examination.
5. **Pathological Q waves** suggesting myocardial ischaemia or cardiomyopathy.
6. **ST-segment changes** suggestive of ischaemia or pericarditis.
7. Unexplained conduction abnormalities such as left bundle branch block, Mobitz II second-degree block, or advanced first-degree block (PR >200 ms in older children) with symptoms.
8. **Extreme axis deviation** (north-west axis, –90° to ±180°) or left axis deviation in a young infant (< –30°).
9. **Low-voltage QRS** (<5 mm in limb leads) with suspected effusion or myocarditis.
10. **Brugada pattern** (type 1) in any child.

Key Points for Clinicians

- Paediatric ECG interpretation **must be age-adjusted** as normal values for rate, axis, and intervals change continuously from birth through adolescence.
- A **systematic approach** (rate, rhythm, axis, intervals, waves/complexes) reduces the risk of missing clinically significant abnormalities.
- Manual interpretation is recommended over sole reliance on machine-generated reports. At minimum, clinicians should manually verify the heart rate in young children, check for delta waves, and carefully measure the QTc interval. Automated readings are unreliable for QTc measurement; therefore, manual confirmation is essential, as QTc prolongation (defined as ≥460 ms in children and ≥450 ms in infants under 6 months) carries significant clinical implications for the risk of sudden cardiac death.
- **Recognised normal paediatric variants to avoid overdiagnosis**: Right ventricular dominance, juvenile T-wave patterns, and sinus arrhythmia are **normal findings** in children and should not be mistaken for pathology.
- Supraventricular tachycardia (SVT) is the most common paediatric tachyarrhythmia; rates exceeding 220 bpm in infants and 180 bpm in children are highly suggestive; rates >300 bpm in infants should prompt consideration of atrial flutter.
- Any ECG with abnormalities suggestive of structural heart disease, or obtained from a child with exertional symptoms or a concerning family history (sudden death in a first-degree relative <50 years, cardiomyopathy in a family member), warrants **senior cardiology review and management** regardless of the tracing's appearance.
- **Technical quality and clinical context determine**

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ECG accuracy: Correct lead placement, minimisation of artefact, and calibration (half or double standardisation) are crucial to interpretation. A normal ECG does not exclude structural heart disease and should never replace comprehensive clinical assessment.

Conclusion

Paediatric ECG interpretation is a core skill for general practitioners and frontline clinicians caring for children. The key to accurate interpretation lies in understanding the profound age-related changes that characterise the normal paediatric ECG and applying a systematic, stepwise approach to analysis. Age-specific normative data are indispensable references and should be readily accessible in all clinical areas where children are assessed. Attention to technical quality, i.e., correct lead placement, recognition of artefact, verification of calibration, and repetition of poor-quality tracings, is an essential prerequisite to accurate interpretation. By recognising both common normal variants and red-flag abnormalities that signal underlying pathology, frontline clinicians can confidently distinguish between benign and life-threatening conditions, ensuring timely specialist referral when indicated. As with all clinical investigations, the ECG must be interpreted within the full context of the history and examination; a normal tracing does not exclude cardiac disease in a symptomatic child, and an abnormal tracing in an asymptomatic child requires careful correlation before decisive action is taken.

Conflict of Interest Statement

The authors declare no conflicts of interest.

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