Ra-id Abdulla
William Bonney
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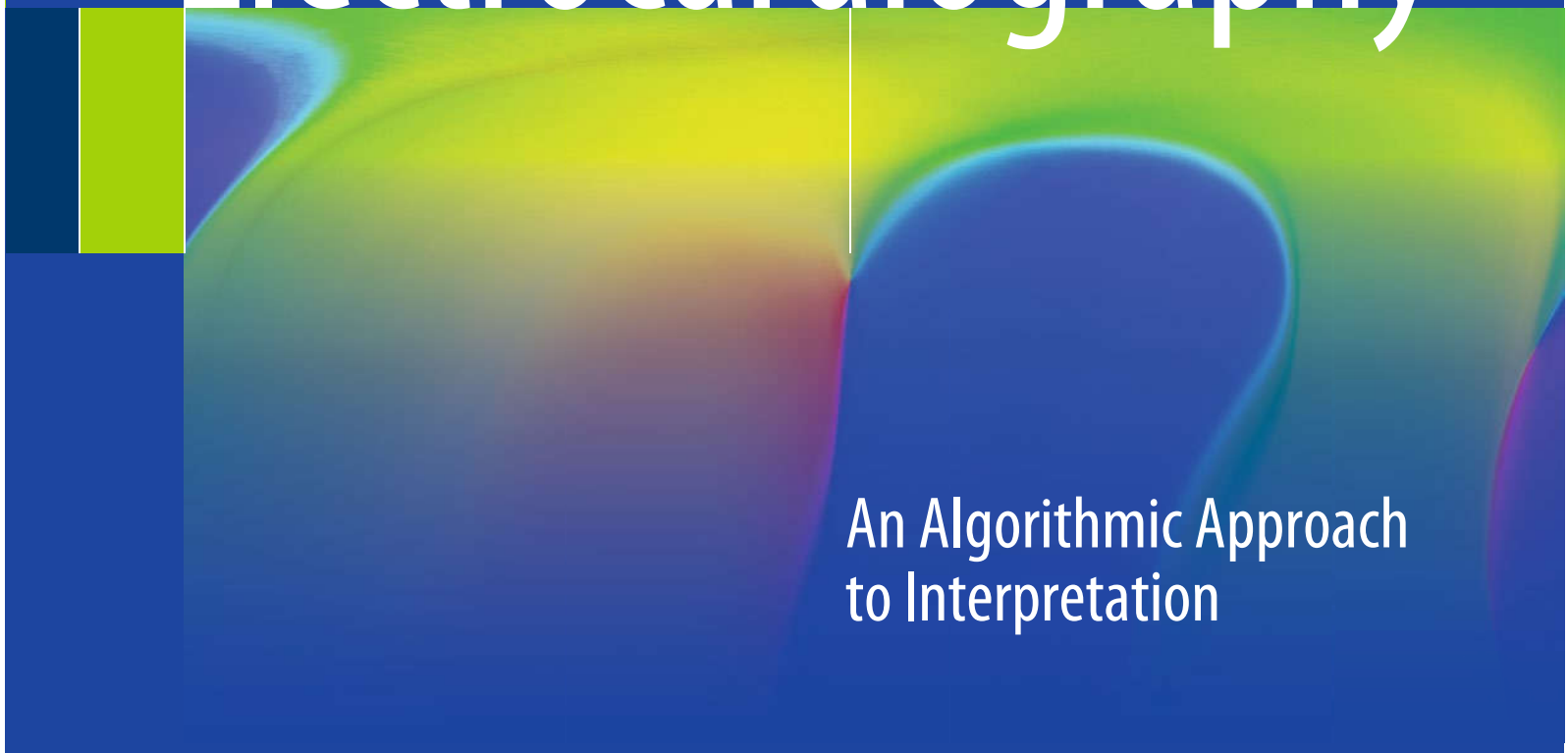
Sawsan Awad
Editors

Pediatric Electrocardiography



An Algorithmic Approach to Interpretation

Springer

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To our spouses, children, and families who define who we are.
And to our patients to whom we humbly offer our knowledge and efforts.

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Preface

Like the heart itself, electrocardiograms (ECGs) wrap themselves with a shroud of mystery. With their squiggly lines and peaks and valleys that reflect the functions of the cardiac chambers that generate them, interpreting ECGs is perhaps the first attraction for young trainees to the field of cardiology. Daunting at first, but once the rules are learned, the hidden secrets of the heart become increasingly evident. It feels like detective's work, no clue is trivial, and the paradox can only be solved by the summation of all clues. However, it is this enigma that repels physicians in training and non-cardiologists from comprehending its benefits and reaping the most of its concealed information.

The ECG is frequently used as the first line of investigative studies when assessing a child with a symptom or sign suggesting a potential cardiac ailment. It is easy to order an ECG in the emergency room or in the outpatient office. The computerized interpretation provided in these settings is tempting to rely on, but unfortunately, it frequently leads to confusion as this particular interpretation tends to suggest pathology where none exists. All this culminates in supposition of cardiac disease, creating apprehension to patients and families and resulting in unwarranted referrals to pediatric cardiologists. This problem can be effectively averted once a reasonable level of proficiency in reading ECGs is attained through proper basic training, continued practice in reading ECGs, and having at hand a reference such as this book.

The first 4 chapters of this book discuss in details how electrical forces generated by the various cardiac chambers contribute to the normal ECG tracings. The subsequent 4 chapters review the various ECG abnormalities and the cardiac pathologies causing them. Chapter 9 details the various ECG presentations of systemic pathologies impacting the function and structure of the heart and as such resulting in aberrations of the ECG. Chapter 10 presents unique approach to ECG interpretation though algorithms. Throughout this book we have attempted to provide as many ECG illustrations and diagrams to make ECG learning effective.

Chapter 10 provides an analytical approach to electrocardiogram (ECG) reading through a practical approach of analyzing normal and abnormal findings of an ECG using a step by step methodological approach through algorithms. This process enables the formulation of competent differential diagnoses concisely and with ease when reviewing 12 lead ECGs and rhythm strips.

The illustrations used in this book are derived principally from electronically stored ECGs of patients that can be captured and reproduced for teaching purposes. In addition, many of the images presented were electronically drawn through computer programs allowing the production of clear and typical ECG findings.

Our hope in writing this book is to provide physicians, residents, students, and nurses with a concise reference for pediatric ECG and offer tools through which ECGs can be effectively and accurately read when performed in the inpatient or outpatient settings. Furthermore, we hope that our work will entice many young trainees to see the intrigue in electrocardiography and fall in love with this field, as we all did many years ago.

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The Normal Electrocardiogram

1

Carolyn M. Wilhelm and Omar Khalid

Developing a Systematic Approach

The electrocardiogram (ECG) is a graphical representation of the electrical activity of the heart. It is an important tool in the care of many patients with potential cardiovascular disease. Therefore, when approaching an ECG, it is essential to interpret each one in the same, systematic way in order to avoid missing important findings.

A typical approach to follow would include these steps:

1. Assess patient demographics (age, gender, and sex)
2. Standardization
3. Rate
4. Axis
   (a) P axis
   (b) QRS axis
   (c) T axis
5. Rhythm
6. AV conduction
   (a) P wave
   (b) PR interval
7. Ventricular conduction
   (a) QRS complex
   (b) QRS duration
   (c) ST segment and T wave
   (d) QT interval
   (e) JT Interval
   (f) U waves
8. Evaluation for chamber enlargement or hypertrophy

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Patient Demographics and Impact on Interpretation

Patient demographics are important to consider when interpreting ECGs, particularly in the pediatric population. Age, sex, race, and even body habitus can all have an effect on a patient's ECG.

Age

One must consider the patient's age during ECG interpretation as the normal values for the heart rate, measured intervals, axis, and voltage criteria are adjusted with age [see]. Heart rate is fastest at birth and then progressively decreases until the teenage years when patients reach their adult resting heart rate.

Davignon et al. published a study of 2,141 white Canadian children ages 0–16 years. The authors divided the patient population into 12 age groups to establish standard values for ECGs in normal children [1]. Similar trends have been noted in other recent studies [2–4]. The following observations were noted:

1. Heart rate – The mean heart rate increased from day one of life to the first month of age and then showed a slow decrease from 3 months of age on. The highest heart rate was recorded between 1 and 3 months of age with an average heart rate of 150 beats per minute.
2. QRS axis – QRS axis has been shown to vary with age as well, with the QRS axis for the first week of life on average being 135° and decreasing to 60° by 3–6 months of age.
3. P wave amplitude – The P wave amplitude in lead V2 shows a gradual decrease with age.
4. PR interval – The PR interval remains stable until approximately 3 months of age and then it gradually increases. The PR interval also increases as the heart rate decreases.

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5. R and S wave amplitudes – R wave amplitude decreases with age in leads V3R and V1 and increases in lead V7, while the S wave shows an inverse trend. This leads to an overall steady decrease in the R/S ratio in leads V3R and V1 and increased R/S ratio in leads V6 and V7 with increased age.
6. QRS duration – The QRS duration increases with age and varies with heart rate.
7. Q wave amplitude – Maximum Q wave amplitudes are seen until age 1–3 years and then gradually decrease.
8. T wave changes – The T wave in leads VI and V3R is upright at birth. Between 3 and 7 days of life, the T wave becomes negative. The T wave will remain negative until 3–8 years of age when it becomes positive.
9. QTc interval – There is a small increase with age.

Body Habitus

Body habitus can have a major effect on QRS voltages. The adipose tissue can act as a form of insulation between the heart's electrical conduction and the ECG electrodes. With an increased distance from the heart to the ECG electrode, an ECG may appear to have lower overall voltage [5]. Studies performed in the adult population have shown that patients with hypertension and obesity could potentially not meet criteria for left ventricular hypertrophy (LVH) depending on the LVH criteria used for ECG interpretation [6, 7]. In 2012, Nasir et al. reported on 55,218 adult patients age 18–35 years and found that in patients with a BMI $\geq$18.5 kg/m^2, there was a decrease in R wave voltage as BMI increased and there was an increased R wave voltage with decreased BMI in patients with BMI <18.5 kg/m^2 [8].

Gender

Davignon et al. reported differences in ECG parameters found between males and females. A significant difference was noted in R wave amplitude between the sexes and suggested this value be stratified not only by age but also by sex [1]. Rijnbeek also documented a difference in Q, R, and S wave amplitudes showing that they were all significantly larger in males compared to females. It was also noted that the QRS duration is longer in males. On the other hand, the QTc (corrected QT interval) remained relatively stable across the age spectrum, but at approximately 15 years of age, normal females have a slightly longer QTc when compared to males [4].

Race

Normal values of QRS voltage can differ by race. In 1985, a study of 15–19-year-old children revealed that African American male patients have been shown to have a higher upper limit of normal QRS voltage compared to European-descended Americans [9]. In a separate study of North American white, black, and Hispanic patients, there was also a higher limit of normal QRS voltage for African American patients compared to white patients. This was seen in both men and women, but in women, it was only evident in women >34 years of age. In comparison to white patients, Hispanic men and women were noted to have a lower limit of normal for QRS voltage [10].

Standardization

The ECG signal is standardized so that 1 mV deflection is equal to 10 mm in height (full standardization). The standardization is marked at the beginning of the ECG with a calibration signal that produces a rectangle that is two big boxes tall (10 mm) and five small boxes wide (25 mm).

Occasionally when the voltages are high, the gain can be adjusted to "half standardization." In this setting, 1 mV = 1 big box (5 mm); therefore, the voltage complex has to be multiplied by two for calculations. On the other hand, "double standardization" can be set in cases where the voltages are too small, in this setting 1 mV = 4 big boxes (20 mm). In this case, the voltage complex has to be divided by two for calculations (Fig. 1.1).

Other modes of standardization are less frequently used (Fig. 1.2). In each case of standardization, calculation must take in consideration the type of standardization and the leads it affects. The examples shown in Fig. 1.2 suggest that not all leads are standardized equally and as such calculation of actual heights and depths of ECG waves must be corrected accordingly.

Rate and Rhythm

Rate

Each ECG is printed on gridded paper, which has a speed of 25 mm per second. Each small box is 1 mm and represents 0.04 s. Thus, each large box is 5 mm and 0.2 s (Fig. 1.3).

Rate is calculated by measuring either the P-P or R-R interval. The interval should be measured from the beginning of either the P wave or the QRS wave to the beginning of the next corresponding wave. Measuring from the peak of either wave can be inaccurate in patients with abnormal conduction (Fig. 1.4).

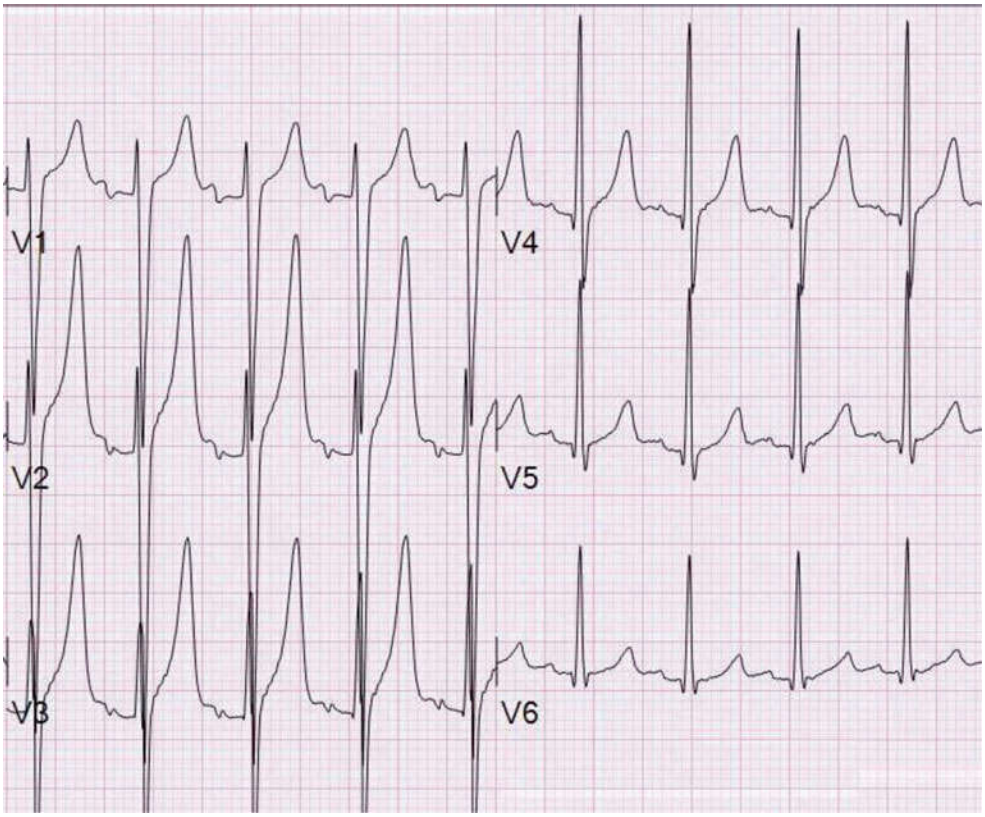

Fig. 1.18 Tall peaked T waves seen in the mid-precordial leads of a patient with diabetic ketoacidosis

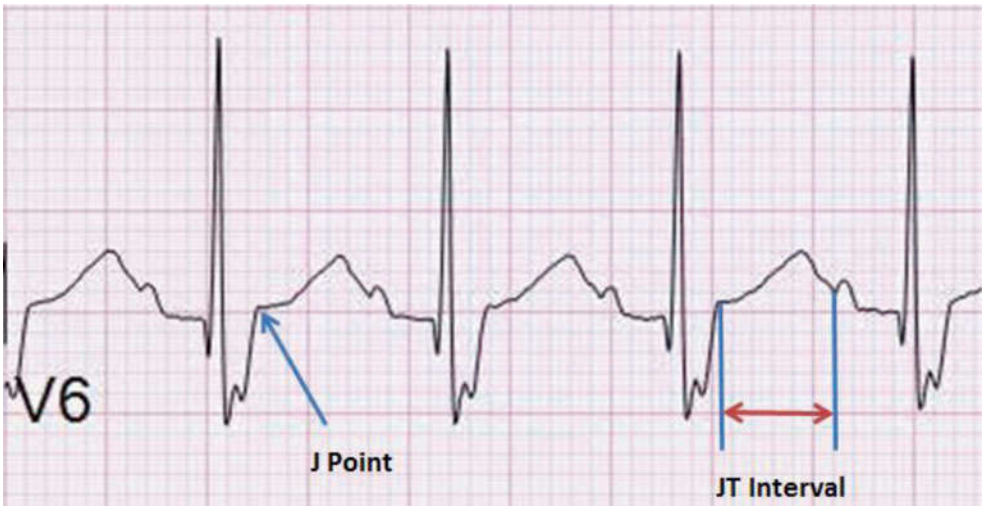

Fig. 1.19 ECG tracing demonstrating the location of the J point and JT interval

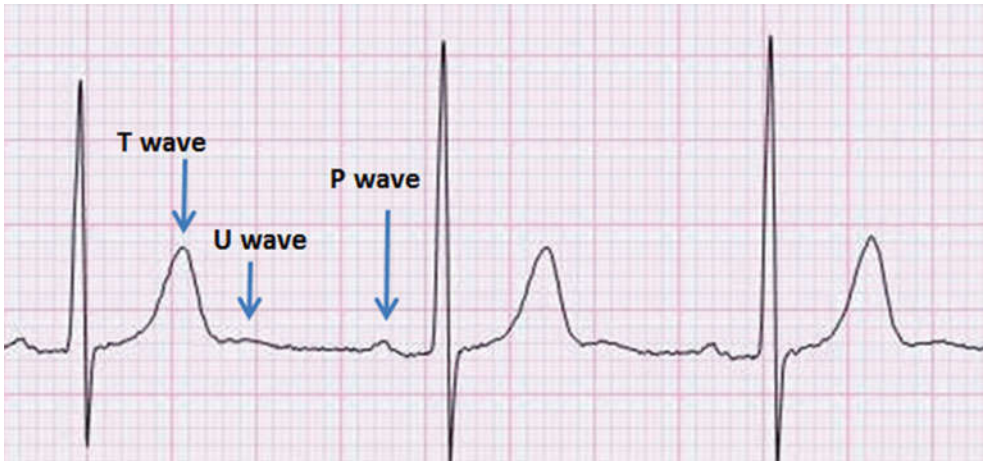

Fig. 1.20 ECG from a 16-year-old with no heart disease

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Table 3.1 Interpretation of P wave axis

P-wave axis	Origin of pacemaker	Potential causes
0–90°	Sinus node	Normal pattern
	Ectopic high right atrium	Ectopic atrial pacemaker
	Right atrium	Electronic atrial pacemaker
90–180°	Sinus node in normal position with erroneous ECG data	Wrong lead connection (placing right limb leads on left limbs and vice versa)
	Sinus node in high left-sided atrium	Heterotaxy
180–270°	Ectopic low left atrium	Ectopic atrial pacemaker
270–0°	Ectopic low right atrium	Ectopic atrial pacemaker

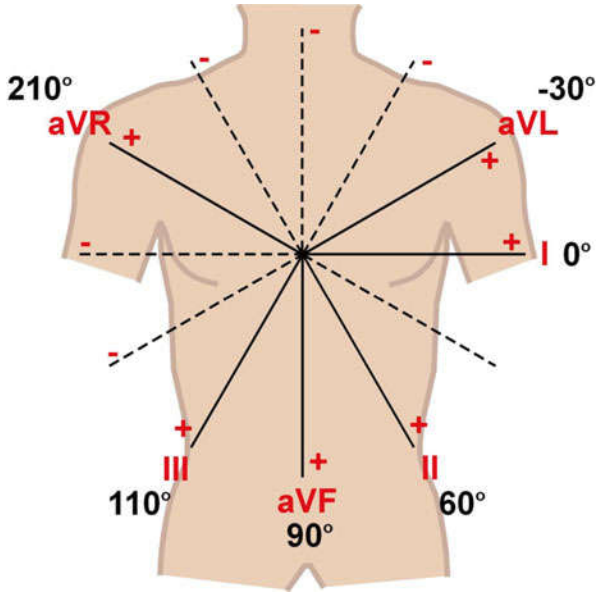

Fig. 3.1 Limb leads are leads: I, II, III, aVR, aVL, and aVF. Leads I and aVF are perpendicular to each other; using these two leads allows easy assessment of QRS axis. Lead I is at 0°, Lead II is at 60°, Lead aVF is at 90°, Lead III is at 110°, Lead aVR is at 210°, Lead aVL is at −30°

QRS Mean Vector (Axis)

The QRS mean vector is an important calculation when reading an ECG as it provides vital information regarding ventricular depolarization, conduction abnormalities, chamber hypertrophy, and other crucial data. As with the P wave, this axis is determined by the frontal plane leads [3].

The QRS mean vector between 0 and 7 days is about +135° and progressively decreases to about +60° in the age range of 3–6 months and remains stable after this age [5]. The range for the axis in the first month of a full-term neonate is +55 to +200° (Figs. 3.3 and 3.4), while the range for a premature neonate is +65 to +174°, more to the left. [4]

Lead I and aVF are perpendicular to each other and are the easiest to use when determining the QRS axis. In younger patients, aVR and Lead III may be incorporated in this process. Lead I represents a horizontal line, while Lead aVF is a vertical line perpendicular to Lead I and crossing it in a "plus"-sign fashion (Figs. 3.1 and 3.2).

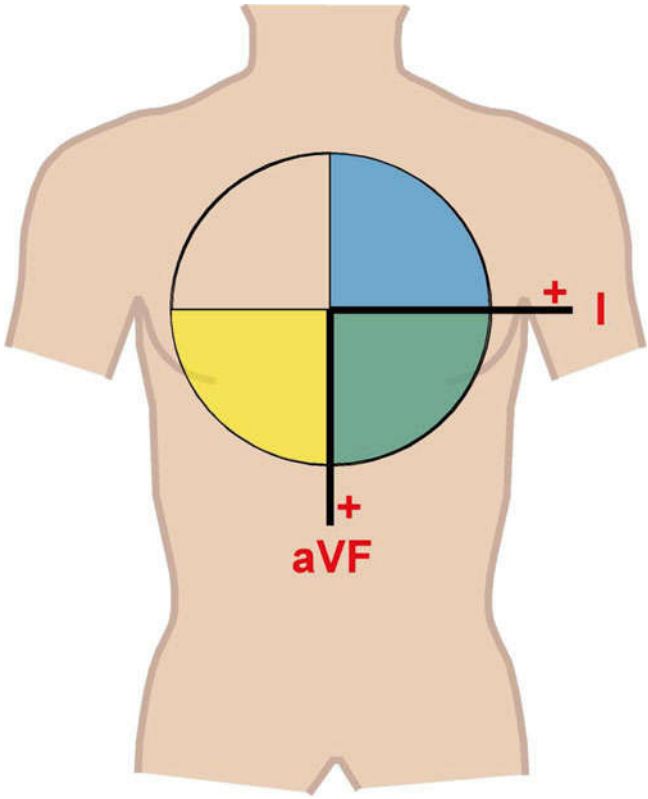

Fig. 3.2 Leads I and aVF are perpendicular to each other and divide the axis wheel into four quadrants. In this diagram green represents southeast, normal quadrant for P and RQS axes for all ages. Yellow is southwest quadrant, a normal region for QRS axis in infants and young children. Beige is northwest quadrant, QRS axis in this quadrant represents right axis deviation for all age groups. The blue quadrant is northeast quadrant where P and QRS axes in this quadrant are normal in adults if 0°–30°; otherwise it represents left axis deviation for QRS axis

There are different methods used in estimating the QRS mean vector; these include:

Quadrant-Based QRS Axis Determination

This provides a rough idea of the general direction of the QRS mean vector; although not precise, it most of the times suffices (Figs. 3.2 and 3.3).

Wide P waves:
- Left atrial enlargement

Low-voltage P wave:
- Hyperkalemia (severe)

Absent P wave:
- Hyperkalemia (severe)
- Associated with tachyarrhythmia:
  - Supraventricular tachycardia (SVT)
  - Junctional ectopic tachycardia (JET)
  - Ventricular tachycardia
- Junctional rhythm
- Ventricular rhythm
- Variable appearance of P waves: sick sinus rhythm or multifocal atrial tachycardia

PR Interval

Prolonged PR interval:
- Hypermagnesemia
- Digoxin
- Beta-blocking agents (class II)
- Amiodarone/sotalol (class III)

Short PR interval:
- Preexcitation, such as Wolff-Parkinson-White syndrome
- Class IA antiarrhythmic agents such as quinidine and procainamide
- Class IB antiarrhythmic agents such as lidocaine

PR depression:
- Pericarditis

P-QRS Relationship

AV block: AV block is of various types (see below section "Algorithmic approach to cardiac rhythm"). Most cases are idiopathic; occasionally, a cause could be detected, and this may include:

- Congenital heart disease, such as l-TGA and others
- Autoimmune disease, including maternal disease and in utero AV block
- Postoperative complication
- Post per-catheter septal occluding device
- Hyperkalemia
- Hypomagnesemia
- Digoxin
- Beta-blocking agents (class II)
- Amiodarone/sotalol (class III)
- Verapamil/diltiazem (class IV)
- Tricyclic antidepressants

QRS Axis

QRS axis 0–90°:
- Normal in all ages except neonates
- Left axis deviation in neonates:
  - LVH
  - Hypoplastic right ventricle

QRS axis 90–180°:
- Normal in neonates, infants, and young children
- Right axis deviation in adolescents and adults

QRS axis 180–270°:
- Right axis deviation:
  - Right ventricular hypertrophy
  - Hypoplastic left ventricle
- Superior axis deviation:
  - Atrioventricular canal defect
  - Heterotaxy
  - Single ventricle
  - Tricuspid atresia

QRS axis 270–0°:
- Normal in adults when within 0-30° Left axis deviation:
  - Left ventricular hypertrophy

QRS Duration

Prolonged:
- Bundle branch block; most common observation is with postsurgical closure of VSD.
- Preexcitation due to bypass tract.
- Hyperkalemia (nonspecific ventricular conduction delay or bundle branch block).
- Hypermagnesemia.
- Quinidine/procainamide.

QRS Amplitude

Diminished:
- Hypothyroidism
- Chagas disease
- Pericardial effusion
- Myocarditis

Increased:
- Chamber hypertrophy: RVH if in right chest leads (V1, 2) and LVH in left chest leads (V5, 6)

Appendix

Table 1 Normal values for ECG parameters by age

Age	Heart rate (bpm)	QRS axis (degrees)	PR interval (ms)	QRS interval (ms)	R in V1 (mm)	R in V2 (mm)	S in V1 (mm)	R in V5 (mm)	R in V6 (mm)	S in V6 (mm)
1st week	90–160	60–180	80–150	30–80	5–26	5–30	0–23	1–20	0–12	0–10
1–3 weeks	100–180	45–160	80–150	30–80	3–21	8–29	0–16	1–23	2–16	0–10
1–2 months	120–180	30–135	80–150	30–80	3–18	8–30	0–15	10–33	5–21	0–10
3–5 months	105–185	0–135	80–150	30–80	3–20	10–31	0–15	10–34	6–22	0–10
6–11 months	110–170	0–135	70–160	30–80	2–20	12–31	0.5–20	10–31	6–23	0–7
1–2 years	90–165	0–110	80–160	30–80	2–18	7–31	0.5–21	10–33	6–23	0–7
3–4 years	70–140	0–110	90–170	40–80	1–18	6–27	0.5–21	12–38	4–24	0–5
5–7 years	65–140	0–110	90–170	40–80	0.5–14	5–25	0.5–24	15–38	4–26	0–4
8–11 years	60–130	−15–110	90–170	40–90	0–14	3–20	0.5–25	15–39	4–25	0–4
12–15 years	65–130	−15–110	90–170	40–90	0–14	3–18	0.5–21	8–35	4–25	0–4
>16 years	50–120	−15–110	90–170	40–100	0–14	3–18	0.5–23	8–35	4–21	0–4

bpm beats per minute

Table 2 PR interval by heart rate

Heart rate (bpm)	PR interval (ms)
>180	90–110
160–180	100–120
140–160	90–140
120–140	100–150
100–120	100–160
80–100	100–170
60–80	150–180
<60	160–190

bpm beats per minute

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Table 3 Calculated QTc using observed QT and heart rate (HR)

	Measured QT interval (s)						
Heart rate (bpm)	0.20	0.25	0.30	0.35	0.40	0.45	0.50
50	0.18	0.23	0.27	0.32	0.37	0.41	0.46
52	0.19	0.23	0.28	0.32	0.37	0.42	0.46
54	0.19	0.23	0.28	0.33	0.38	0.42	0.47
56	0.19	0.24	0.29	0.34	0.38	0.43	0.48
58	0.20	0.24	0.29	0.34	0.39	0.44	0.49
60	0.20	0.25	0.30	0.35	0.40	0.45	0.50
63	0.21	0.25	0.31	0.36	0.41	0.46	0.51
66	0.21	0.26	0.31	0.36	0.42	0.47	0.52
68	0.22	0.26	0.32	0.37	0.43	0.48	0.53
71	0.22	0.27	0.33	0.38	0.44	0.49	0.55
75	0.23	0.27	0.34	0.39	0.45	0.51	0.56
79	0.24	0.28	0.34	0.40	0.46	0.52	0.57
83	0.24	0.29	0.35	0.41	0.47	0.53	0.69
88	0.25	0.29	0.36	0.43	0.49	0.55	0.61
94	0.26	0.30	0.38	0.44	0.50	0.56	0.63
100	0.27	0.31	0.39	0.45	0.52	0.58	0.65
107	0.28	0.32	0.40	0.47	0.53	0.60	0.67
115	0.28	0.35	0.42	0.49	0.55	0.63	0.69
125	0.29	0.36	0.43	0.51	0.58	0.65	0.72
136	0.30	0.38	0.45	0.53	0.60	0.68	0.75
150	0.32	0.40	0.47	0.56	0.63	0.71	0.79

Example: a patient with a measured QT of 0.30 s and a heart rate of 75 bpm has a c QTc of 0.34 s
bpm beats per minute