EKG Refresher for the Pediatrician

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- I have no relevant financial relationships with the manufacturers(s) of any commercial products(s) and/or provider of commercial services discussed in this CME activity
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Brief History of the EKG

- **1887** British physiologist Augustus D. Waller of St Mary's Medical School, London publishes the first human electrocardiogram. It is recorded with a capillary electrometer from Thomas Goswell, a technician in the laboratory

Brief History of the EKG

- **1893** Willem Einthoven introduces the term 'electrocardiogram' at a meeting of the Dutch Medical Association.
- **1895** Einthoven, using an improved electrometer and a correction formula, distinguishes five deflections which he names P, Q, R, S and T.

Brief History of the EKG

- **1901** Einthoven invents a new galvanometer for producing electrocardiograms using a fine quartz string coated in silver, his "string galvanometer" weighs 600 pounds.

Brief History of the EKG

- **1912** Einthoven addresses the Chelsea Clinical Society in London and describes an equilateral triangle formed by his standard leads I, II and III later called 'Einthoven's triangle'. This is the first reference in an English article to the abbreviation 'EKG'.

Lead Systems and Technique

- The standard EKG evolved from a three-lead system introduced by Einthoven to a 12 or 15-lead tracing in current use for pediatric recording.
- The two major lead groupings include the limb leads and the precordial leads.

Lead Systems and Technique

- Routine recordings are made with a chart paper speed of 25mm/sec and are usually standardized with an amplitude response of 1mV/10mm.

Interpretation

- The EKG should be read in a systemic fashion, with measurements of rate, axes, intervals, and waveform analysis, all of which must be synthesized into a final impression based on **history and physical examination.**

Interpretation

Rhythm and Rate

- Cardiac excitation arising from the SA node generates a P wave with a normal axis at a rate within the limits for age.

Interpretation

PR Interval

- The PR interval is measured from the beginning of the P wave to the initial deflection of ventricular activation (it is more precisely a PQ interval).
- A prolonged PR interval can be due to enhanced vagal tone, cardiac medications (digoxin and antiarrhythmic agents), or disease involving either the AV node or the His-Purkinje system.
- A short PR interval (less than 0.08 second) may be observed in Wolff-Parkinson-White syndrome.

Interpretation

QRS Complex

- The normal heart leaves a characteristic QRS shape in each lead of the EKG, which may change because of distorted activation sequence or hypertrophy
- The duration of the QRS complex is related to the speed of conduction within the His-Purkinje system, as well as from myocyte to myocyte within the ventricles.
- Prolongation of the QRS may be seen with block of His-Purkinje conduction (bundle branch block), slow myocyte conduction (due to muscle injury, drugs, or electrolyte disturbances), severe ventricular hypertrophy, and some cases of preexcitation.

Interpretation

T Wave

- The T wave corresponds to phase 3 repolarization of ventricular myocytes. T-wave axis should follow the same net direction as the QRS in the frontal plane within about 60 degrees, and discordance of the axis in the limb leads may suggest pathology.
- In the precordial leads, the T wave is negative over the right chest (V4R–V1), while remaining positive in the left chest leads. This pattern persists from day 7 of life until adolescence, when the T waves tend to resume an upright direction in all chest leads.

Interpretation

QT Interval

- The QT interval is a reflection of the total action potential duration for ventricular myocytes.
- Measured from the onset of the QRS to the point of T-wave termination.
- QT interval varies with heart rate (longer at slow rates, shorter at fast rates), the measurement is corrected with the formula: QT (seconds)/square root R-R (seconds).

EKG Interpretation in Athletes

- Electrocardiographic changes in athletes are common
- Benign structural and electrical remodeling of the heart
- Autonomic nervous system adaptations
- Physiological adaptation to physical training (athlete's heart)

EKG Interpretation in Athletes

- Regular and long-term participation in intensive exercise (4 hr/week)
- Electrical manifestations that reflect
  - increased vagal tone
  - enlarged cardiac chamber size
- Extent of changes dependent on
  - ethnicity, age, gender
  - sporting discipline, level of training

Increased Vagal Tone

Sinus bradycardia

1. P wave before every QRS complex
2. QRS complex after every P
3. Normal P wave axis (0–90°)

- In the absence of symptoms (fatigue, dizziness or syncope), a heart rate ≥30 beats/min should be considered normal in a well-trained athlete
- Increase in heart rate during physical activity

Increased Vagal Tone

Sinus arrhythmia

- Heart rate usually increases slightly during inspiration and decreases slightly during expiration
- P wave axis remains normal
- Accelerating the heart rate with physical activity normalizes the heart rhythm

Increased Vagal Tone

Junctional escape (nodal) rhythm

- QRS rate is faster than the resting P wave or sinus rate which is slowed in athletes due to increased vagal tone
- QRS rate typically <100 beats/min
- Narrow QRS complex
- Sinus rhythm resumes with increased heart rates

Increased Vagal Tone

Ectopic atrial rhythm

- P waves are present but are a different morphology compared to the sinus P wave
- atrial rate is typically <100 beats/min
- >2 different P wave morphologies is known as wandering atrial pacemaker
- Sinus rhythm resumes when heart rate is increased

Increased Vagal Tone

First-degree AV block

- PR interval is prolonged (>200 ms) the same duration on every beat
- Delay in AV nodal conduction or intrinsic AV node changes related to increase vagal activity
- Typically resolves with faster heart rates

Increased Vagal Tone

Mobitz type I (Wenckebach) second-degree AV block

- PR interval progressively lengthens from beat to beat, until there is a nonconducted P wave with no QRS complex
- Return of 1:1 conduction with exercise

Increased Vagal Tone

Early repolarization

- ST elevation and/or a J wave (distinct notch) or slur on the downslope
- Considered a benign EKG pattern in apparently healthy, asymptomatic individuals
- Common in athletes at times of peak fitness, suggesting early repolarization is a dynamic process

Increased Vagal Tone

Repolarization findings in black/African athletes

- Elevated ST segment with upward convexity ('dome' shaped), followed by a negative T wave confined to leads V1–V4
- Probably represents a specific, ethnically dependent adaption to regular exercise
- More than two-thirds of black athletes exhibit ST segment elevation and up to 25% show T wave inversions

Increased Cardiac Chamber Size

QRS voltage criteria for LVH

- Limitation of the EKG in identifying LVH is due to the reliance of measuring the electrical activity of the heart by electrodes on the surface of the body
- QRS voltage can be influenced by a variety of factors other than LV size or mass
  - Obesity, pulmonary disease, gender, ethnicity

Increased Cardiac Chamber Size

- In athletes, intensive conditioning is also associated with morphological cardiac changes of increased cavity dimensions and wall thickness
- Increased QRS amplitudes meeting EKG voltage criteria for LVH are prevalent and present in up to 45% of athletes
  - and 25% of sedentary young adults
- QRS voltage alone is a poor indicator of pathological LVH

Increased Cardiac Chamber Size

Incomplete right bundle branch block

- QRS duration <120 ms with an RBBB pattern: terminal R wave in lead V1 (rsR') and wide terminal S wave in leads I and V6
- Mildly delayed RV conduction is caused by RV remodeling, with increased cavity size and resultant increased conduction time

Normal EKG Findings in Athletes

Increased vagal tone

- Sinus bradycardia, sinus arrhythmia and early repolarization
- Junctional (nodal) rhythm or wandering atrial pacemaker
- First-degree AV block and Mobitz type I second-degree AV block

Normal EKG Findings in Athletes

Increased cardiac chamber size

- Isolated presence of high QRS voltages fulfilling criterion for LVH
- IRBBB (rSR' pattern in V1 with QRS duration <120 ms)

Normal EKG Findings in 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.

Borderline EKG Findings in Athletes

- Left axis deviation (-30° to -90°)
- Right axis deviation (>120°)

Borderline EKG Findings in Athletes

- Left atrial enlargement
  - Prolonged P wave duration of >120 ms in leads I or II with negative portion of the P-wave ≥1 mm in depth and ≥40 ms in duration in V1
- Right atrial enlargement
  - P-wave ≥2.5 mm in II, III, or aVF

Borderline EKG Findings in Athletes

- Complete right bundle branch block
  - rSR' pattern in lead V1 and a S wave wider than R wave in lead V6 with QRS duration ≥120 ms
- Prevalence of 0.5% to 2.5% in young adult athletes
- Represents a spectrum of structural and physiological cardiac remodeling characterized by RV dilation with resultant QRS prolongation

Borderline EKG Findings in Athletes

- Presence of any one of these findings in isolation or with other recognized normal EKG patterns of athletic training does not warrant further assessment in asymptomatic athletes without a concerning family history
- Presence of more than one of these borderline findings places the athlete in the abnormal category warranting additional investigation.

Abnormal EKG Findings in Athletes

- Cardiovascular-related sudden death is the leading cause of mortality in athletes during sport.
- Many disorders associated with increased risk of sudden cardiac death, such as cardiomyopathies and primary electrical diseases, are suggested by abnormal findings present on a 12-lead ECG.

EKG Changes Suggestive of Cardiomyopathy

- HCM, ARVC, DCM and LVNC found in the majority of autopsy-positive sudden death cases in young athletes
- Characteristic EKG findings to suggest HCM and ARVC
- Less specific EKG findings in DCM, LVNC, PHTN

EKG Changes Suggestive of Cardiomyopathy

T wave inversion	>1 mm in depth in two or more leads V2–V6, II and aVF, or I and aVL (excludes leads III, aVR and V1)
ST segment depression	≥0.5 mm in depth in two or more leads
Pathological Q waves	>3 mm in depth or >40 ms in duration in two or more leads (except III and aVR)
Multiple premature ventricular contractions	≥2 PVCs per 10 s tracing

EKG Changes Suggestive of Cardiomyopathy

Complete left bundle branch block	QRS≥120 ms, negative QRS complex in lead V1 (QS or rS), and upright monophasic R wave in leads I and V6
Profound nonspecific intraventricular conduction delay ≥140 ms	Any QRS duration ≥140 ms
Epsilon wave	Small negative deflection just beyond the QRS in V1–V3

EKG Changes Suggestive of Primary Electrical Disease

Disturbances of cardiac conduction are associated with diseases predisposing to SCD in young athletes.

- Congenital long and short QT syndromes (LQTS and SQTS)
- Catecholaminergic polymorphic ventricular tachycardia (CPVT)
- Brugada syndrome (BrS)
- Ventricular pre-excitation (WPW), supraventricular tachycardias (SVT)
- Atrioventricular (AV) blocks and premature ventricular contractions (PVCs)

EKG Changes Suggestive of Primary Electrical Disease

Long QT interval	QTc ≥470 ms (male) QTc ≥480 ms (female)
Short QT interval	QTc ≤320 ms
Ventricular pre-excitation	PR interval <120 ms with a delta wave and wide QRS (>120 ms)
Profound sinus bradycardia	<30 bpm or sinus pause ≥3s

EKG Changes Suggestive of Primary Electrical Disease

Brugada-like ECG pattern	High take-off and downsloping ST segment elevation followed by a negative T wave in ≥2 leads in V1–V3
Atrial tachyarrhythmias	Supraventricular tachycardia, atrial-fibrillation, atrial-flutter
Ventricular arrhythmias	Couplets, triplets, and non-sustained ventricular tachycardia

Abnormal EKG Findings in Athletes

- Abnormal EKG findings or multiple borderline EKG findings warrant further evaluation for pathologic cardiovascular disorders.

References

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Thank You