

ECG in Athletes: Limits of Normal

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When the ECG of an athlete is examined, the main objective is to distinguish between physiological patterns that should cause no alarm and those that require action and/or additional testing to exclude (or confirm) the suspicion of an underlying cardiovascular condition carrying the risk of sudden death during sports. ECG changes in athletes are common and usually reflect structural and electrical remodeling of the heart as an adaptation to regular physical training (athlete's heart).

Classification of abnormalities of the athlete's ECG

Group 1: common and training-related ECG changes: Sinus bradycardia; First-degree AV block; Incomplete RBBB; Early repolarization; Isolated QRS voltage criteria for LV hypertrophy
Group 2: uncommon and training-unrelated ECG changes: T-wave inversion; ST-segment depression; Pathological Q-waves; Left atrial enlargement; Left-axis deviation/left anterior hemiblock; Right-axis deviation/left posterior hemiblock; Right ventricular hypertrophy; Ventricular pre-excitation; Complete LBBB or RBBB; Long- or short-QT interval; Brugada-like early repolarization.

Abnormalities of the athlete's ECG: examples

Fig 1. A normal athlete's ECG demonstrates sinus bradycardia with a heart rate of 40 bpm. P wave before every QRS complex, normal P wave axis (frontal plane 0–90°s).

Fig 2. Normal athlete's ECG demonstrates sinus arrhythmia. Note the irregular heart rate that varies with respiration. The P waves are upright in leads I and aVF (frontal plane) suggesting a sinus origin.

Fig 3. ECG of a 28-year-old asymptomatic handball player demonstrating a junctional escape rhythm. Note the constant RR interval between beats. Accept as within a normal athlete's heart.

Fig 4. Brugada-ECG is confined to right precordial leads (V1 and V2) without reciprocal 'S-wave' (of comparable voltage and duration) in the leads L1

and V6 (arrowhead). (B) In this case, a definitive diagnosis of Brugada ECG was achieved by a drug challenge with sodium channel blockers which unmasked the diagnostic 'coved type' (arrows) pattern (V1 and V2).

Fig 5. Twelve-lead ECG of an asymptomatic athlete with HCM. The disease was suspected at pre-participation evaluation thanks to ECG abnormalities consisting of increased QRS voltages and inverted T-waves in lateral leads. HCM was diagnosed by echocardiography afterward.

Fig 6. Different patterns of precordial early repolarization in two healthy athletes. (A) ST-segment elevation with upward concavity (arrows), followed by a positive T-wave (arrowheads). (B) ST-segment elevation with upward convexity (arrows), followed by a negative T-wave (arrowheads).

Fig 7. Twelve-lead ECG in an asymptomatic athlete with ARVC. The athlete was referred for further echocardiographic examination and cardiac magnetic resonance because of ECG abnormalities found at pre-participation evaluation which consisted of inverted T-waves in the inferior and anteroseptal leads and low QRS voltages in the peripheral leads.

Fig 8. HCM vs Athlete's heart

Clues that distinguish a normal "athlete's heart" include:

- LV wall thickness is generally 13 mm; upper limit physiologic hypertrophy is approximately 16 mm.
- LV systolic function is normal, even if mild LV • dilatation is present.
- Absence of a systolic murmur consists of LV outflow tract obstruction.
- No personal symptoms suggesting HCM (e.g., syncope); no family hist. of S. cardiac death, HCM, etc.
- Decrease in wall thickness with cessation of athletic training.

The authors declare that there is no conflict of interest.

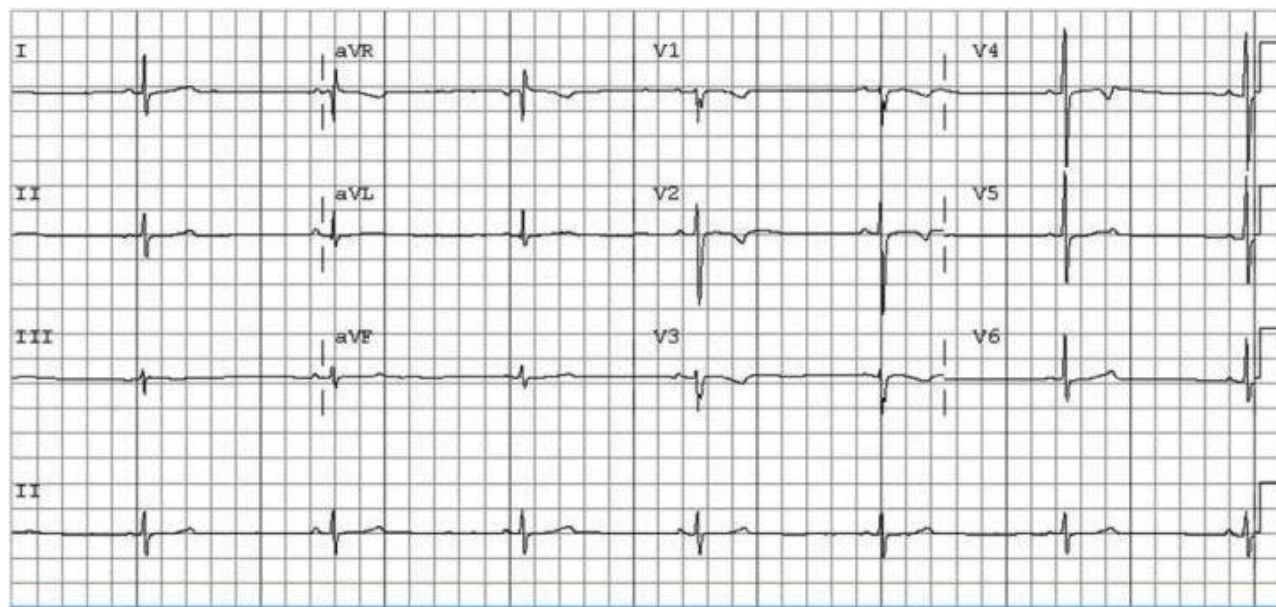


Fig 1

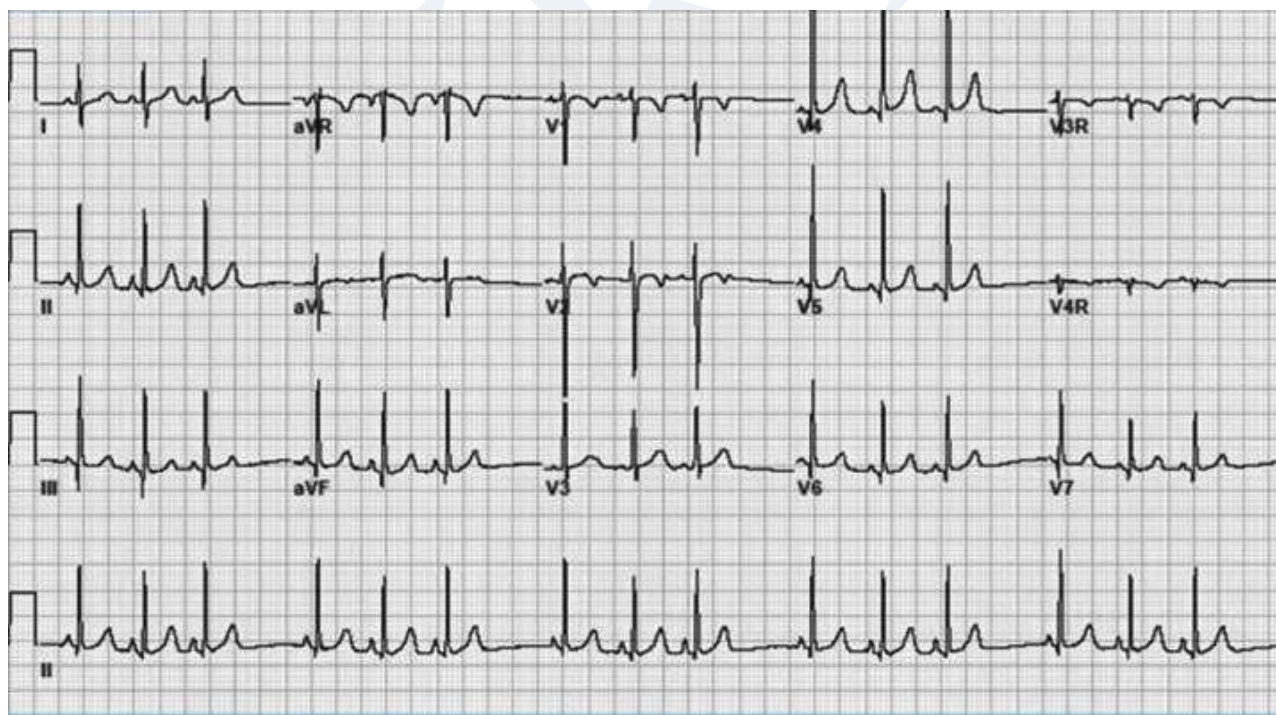


Fig 2

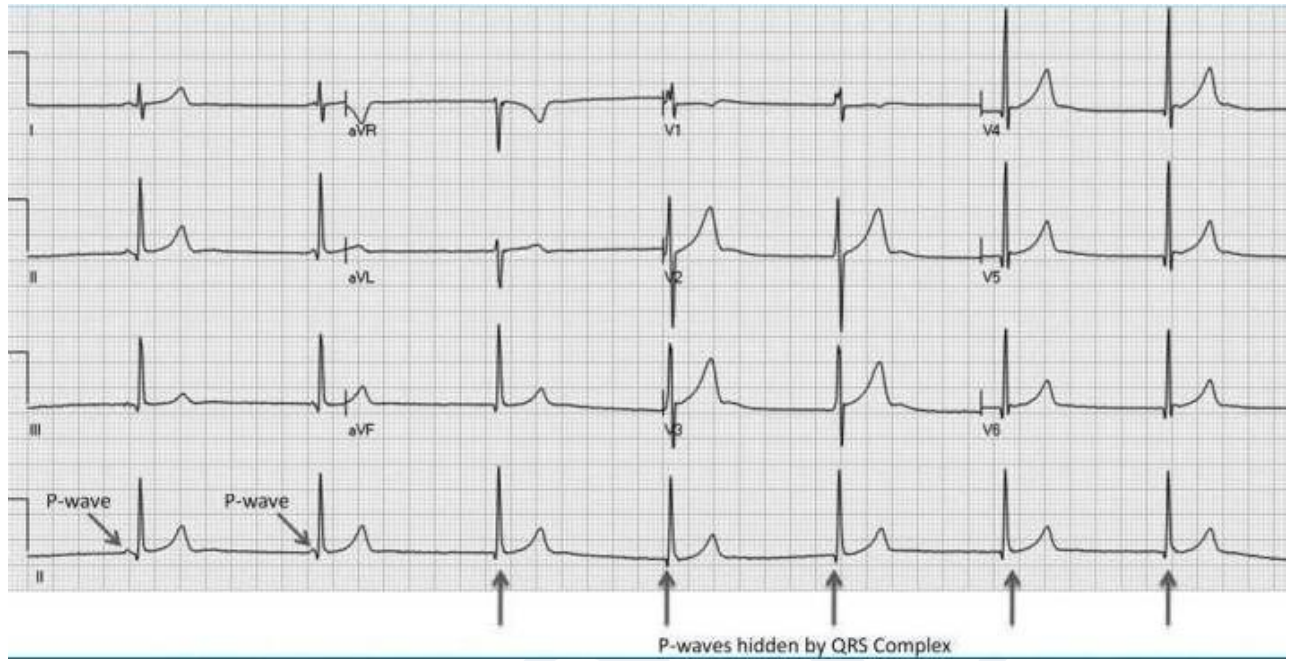


Fig 3

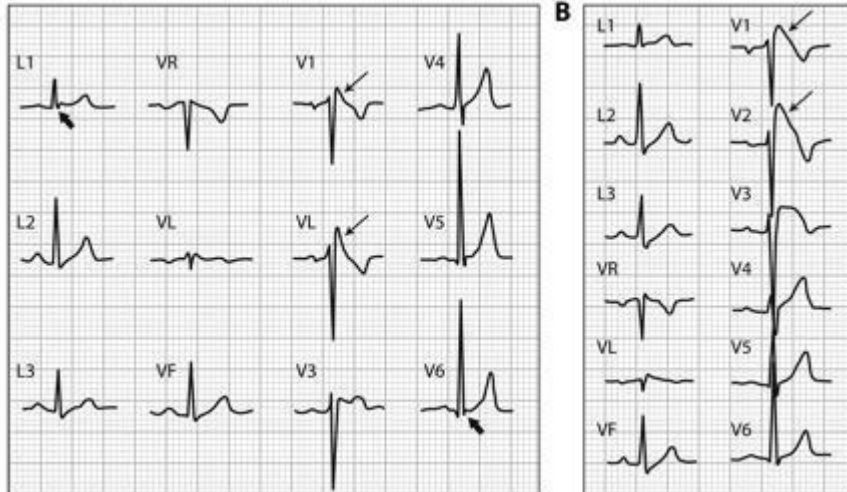


Fig 4



Fig 5

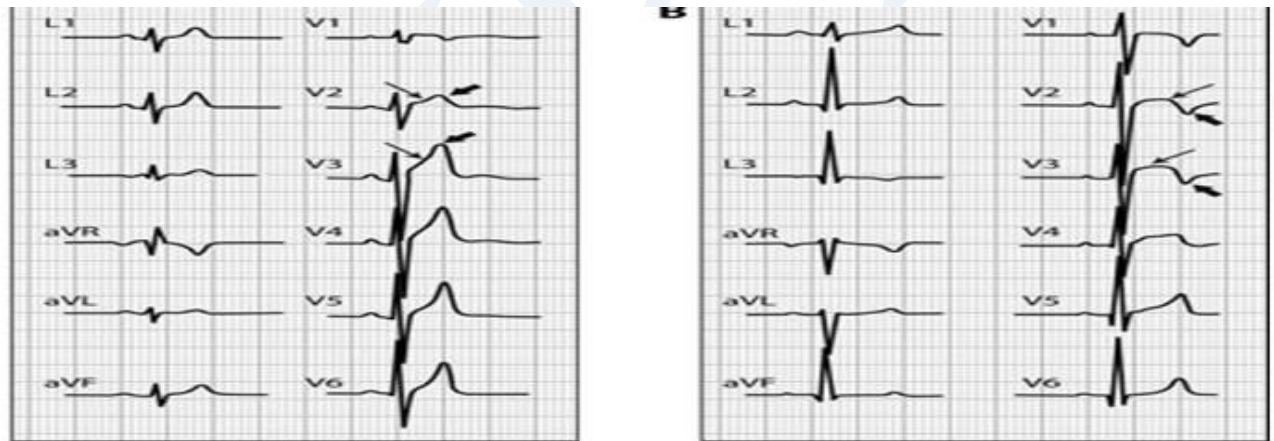


Fig 6

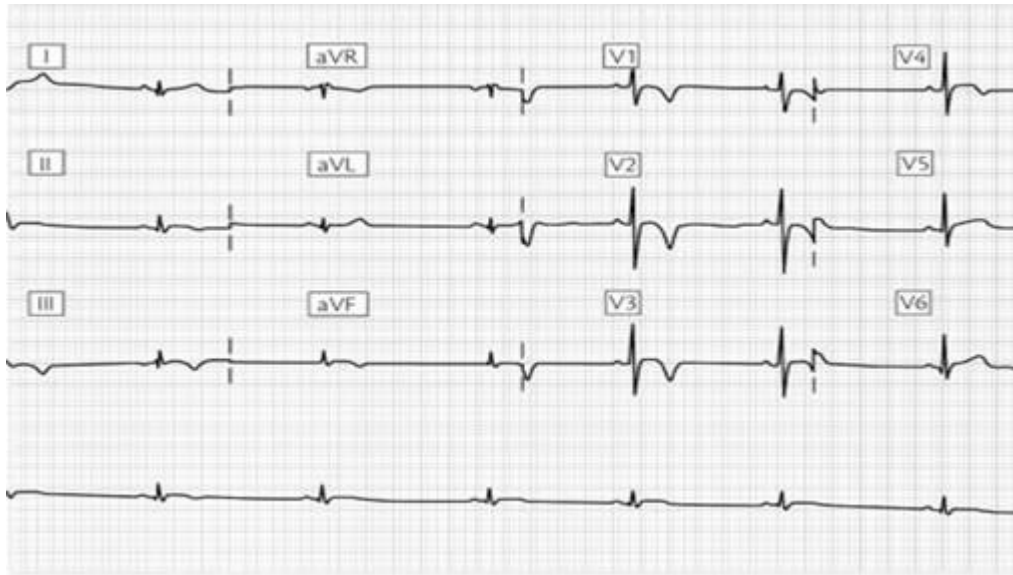


Fig 7

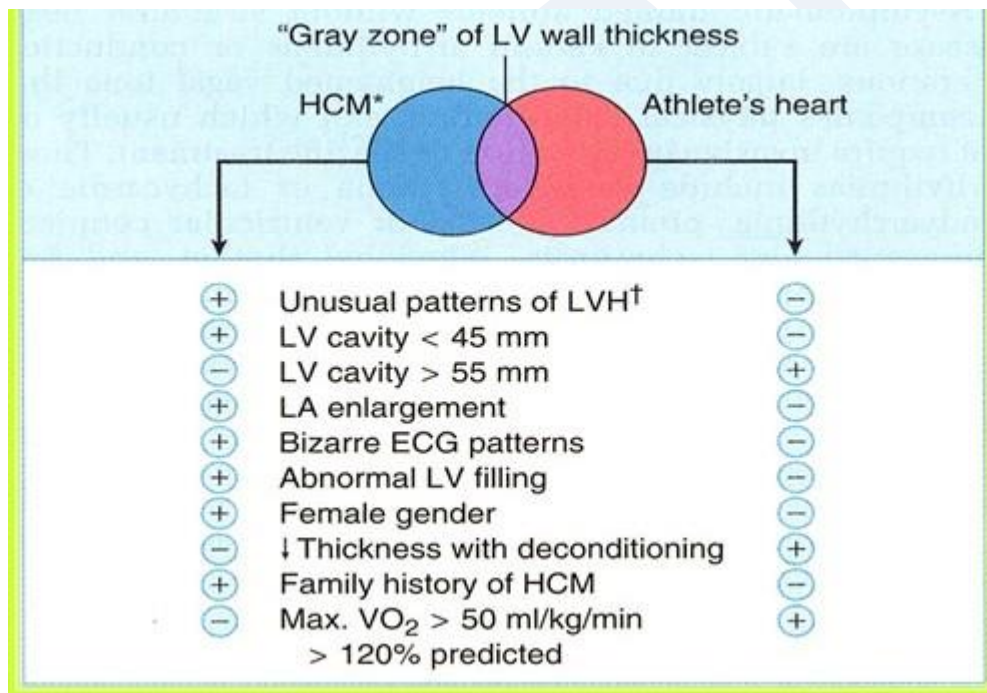


Fig 8

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