The Electrocardiogram in Athletes Revisited

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Abstract

Cardiovascular-related sudden death is the leading cause of mortality in athletes during sport. Thus, it is of clinical importance to identify ECG changes that represent normal adaptation in athletes, and differentiate them from truly pathological findings. However, a distinction between adaptive and pathological ECG changes in athletes is not always easy. This article discusses exercise-induced ECG changes and the differential diagnosis of conditions that present with similar ECG patterns.

Keywords

Athlete's heart, early repolarization, cardiomyopathy, Brugada

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Sudden Cardiac Death in Athletes

Cardiovascular-related sudden death is the leading cause of mortality in athletes during sport.^{1,2} The incidence of sports-related sudden death of any cause in the general population is 0.5–1.7 per 100,000 persons per year.³ This is higher in professional athletes, with a reported incidence of sudden cardiac death (SCD) as 1/43,770 participants per year in the National Collegiate Athletic Association (NCAA),¹ and 1/3,100 per year among NCAA Division I male basketball players.¹ In other studies, the reported incidence of SCD ranges from 0.26 to 3.60/100,000 per year with the higher rates seen in African/Afro-Caribbean (black) athletes.^{2,4–8} For comparison, the incidence of SCD in the general population, not necessarily related to sport, is estimated at 100–200/100,000 annually and increases as a function of advancing age, being 100-fold less in adolescents and adults younger than 30 years (1/100,000) than it is in adults older than 35 years.^{9–11}

Vigorous exertion may trigger cardiac arrest or SCD, especially in untrained persons, but habitual vigorous exercise diminishes the risk of sudden death during vigorous exertion.¹² Most studies have found inverse associations between regular physical activity and SCD.¹³ Concerning sports-related sudden death in the general population, a clear diagnosis is made in <25 % of the cases, but is most frequently an acute coronary syndrome (75 %).³ In trained athletes, a diagnosis is usually made in up to 65 % of the cases and usually is cardiovascular disease such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), congenital coronary anomalies, genetic channelopathies and myocarditis, with blunt trauma, *commotio cordis* and heat stroke being less frequent (see *Table 1*).^{2–4} Recently, findings from the Race Associated Cardiac Arrest Event Registry (RACER) initiative indicate that marathons and half-marathons are associated with a low overall risk of cardiac arrest (1/184,000) or sudden death (1/259,000). However, event rates, most commonly attributable to hypertrophic cardiomyopathy or atherosclerotic coronary disease, have risen over the past decade among male marathon runners.¹⁴

A mandatory national pre-participation screening strategy with routine electrocardiograms (ECGs) is recommended by the European Society of Cardiology (ESC)¹⁵ and the International Olympic Committee,¹⁶ mainly based on the beneficial results of screening observed in the Veneto region of Italy.⁵ The American Heart Association has expressed reservations about the cost-efficiency of this approach, and does not endorse ESC screening.¹⁷ Although debatable,^{2,7} the recent report on data from the US NCAA argues for closer scrutiny.¹ Regardless, it is of clinical importance to identify ECG changes that represent normal adaptation in athletes, and differentiate them from truly pathological findings.

Exercise-induced Electrocardiogram Remodeling

A differentiation between adaptive and pathological ECG changes in athletes is not always easy.¹⁸ Recommendations have been published by the ESC,¹⁶ a group of US experts,¹⁹ and recently, by an international group of experts in sports cardiology and sports medicine (Seattle criteria, see *Tables 2–4*).²⁰

Differentiation from Pathologic Conditions

Conduction Abnormalities

Sinus bradycardia is normal. Only heart rates <30 beats per minute (bpm) and pauses 3 seconds (sec) during wake suggest sick sinus syndrome. First and second degree Type 1 (Wenckebach) blocks are benign and usually resolve with hyperventilation or exercise. Axis between -30 and +115 degrees is also normal.

Left Ventricular Hypertrophy

Left ventricular (LV) hypertrophy and dilation occurs with isotonic exercise, whereas isometric exercise will promote LV hypertrophy alone.²¹ The single most common cardiovascular cause of these unexpected catastrophes appears to be HCM, accounting for about one-third of cases (see *Figure 1*).^{22,23} Wall thickness resembling HCM may occur in sporting disciplines that combine isometric and isotonic activity, such as cycling and rowing. Since the phenotypic expression of HCM is variable, and not uncommonly

Table 1: Common Cardiovascular Conditions Associated With Sudden Death in Athletes*

HOCM
Congenital coronary anomalies
Genetic channelopathies (Brugada, early repolarization syndrome, LQTS, SQTS, CPVT)
Coronary artery disease
ARVC
Myocarditis
Bicuspid aortic valve with stenosis or dilated aortic root
WPW
Blunt trauma
Commotio cordis

*: In sports-related sudden cardiac death in the general population, coronary artery disease is the commonest cause. Source: Katriotis, et al. 2013¹⁸

Table 2: Normal ECG 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.

1. Sinus bradycardia (≥ 30 bpm)
2. Sinus arrhythmia
3. Ectopic atrial rhythm
4. Junctional escape rhythm
5. 1° AV block (PR interval > 200 ms)
6. Mobitz Type I (Wenckebach) 2° AV block
7. Incomplete RBBB
8. Isolated QRS voltage criteria for LVH
Except: QRS voltage criteria for LVH occurring with any non-voltage criteria for LVH such as left atrial enlargement, left axis deviation, ST segment depression, T-wave inversion or pathological Q waves
9. Early repolarisation (ST elevation, J-point elevation, J-waves or terminal QRS slurring)
10. Convex ('domed') ST segment elevation combined with T-wave inversion in leads V1–V4 in black/African athletes

AV = atrioventricular; bpm = beats per minute; LVH = left ventricular hypertrophy; ms = milliseconds; RBBB = right bundle branch block. Source: Drezner JA, 2013.²⁰

includes patients with mild and localised LV hypertrophy, the differential diagnosis with athlete's heart may be difficult. Usually, however, by contrast to most patients with HCM, athletes have increased left and right ventricular cavity dimensions, and normal diastolic function.^{22–24} Physiological LV hypertrophy can also be differentiated from hypertrophic cardiomyopathy by tissue Doppler imaging velocity measurements.²⁵ In some cases, protracted deconditioning may be the only way to establish a diagnosis. LV hypertrophy certainly needs further evaluation in the presence of family history of SCD or non-voltage ECG criteria suggesting pathological ECG hypertrophy.

Q Waves

Q waves >3 millimetres (mm) in depth and/or >40 milliseconds (ms) duration in any lead except aVR, III and V1, suggest HCM. Standard criteria for myocardial infarction²⁶ in athletes should also be considered in those >40 years of age.

Early Repolarization and ST-T Abnormalities

Early repolarization is defined electrocardiographically by either a sharp well defined positive deflection or notch immediately following a positive QRS complex at the onset of the ST-segment, or slurring at the terminal part of the QRS complex (J-waves or J-point elevation, see Figure 2).²⁷ The early repolarization pattern has long been considered

Figure 1: Twelve-lead Electrocardiogram of an Asymptomatic Athlete with Hypertrophic Cardiomyopathy

The disease was suspected at pre-participation evaluation thanks to electrocardiogram abnormalities consisting of increased QRS voltages and inverted T-waves in lateral leads. Hypertrophic cardiomyopathy was diagnosed by echocardiography afterwards. Source: Corrado, et al. 2010.¹⁵

Figure 2: Patterns of Early Repolarization

Classic Definition of Early Repolarization: ST Elevation

New Definitions of Early Repolarization

In the upper panel there is 'classic definition' that describes a pattern that is common in the inferior leads of male, black athletes. Source: Perez, et al. 2012.³⁸

to be a benign ECG manifestation (6–13 % in the general population) that is seen more commonly in young healthy men and athletes (22–44 %), and its clinical significance has been questioned.²⁸ Inferolateral early repolarization may be seen in young athletes and is a dynamic phenomenon caused by exercise.²⁹

However, there are data indicating that the early repolarization pattern may be associated with a risk for ventricular fibrillation (VF) and SCD, depending on the location of early repolarization, magnitude of the J-wave and degree of any ST elevation present.^{27,30–35} A horizontal/descending type (defined as ≤ 0.1 millivolt (mV) elevation of the ST-segment within 100 ms after the J-point) in the inferior leads, as opposed to a rapidly ascending ST-segment type, may help to identify those individuals who are clearly at risk (see Figures 3 and 4).^{33,34} However, several obscure points remain with this syndrome. In the Atherosclerosis Risk in Communities (ARIC)

Figure 3: Horizontal/Descending ST-segment Patterns from Two Subjects in the General Population

Source: Tikkanen, et al. 2011.³⁴

Figure 4: Rapidly Ascending (A) and Horizontal (B) ST-segment in the Leads Deploying J-waves (J-waves Marked with Arrowhead)

'Concave/rapidly ascending': when there is 0.1 mV elevation of the ST-segment within 100 ms after the J-point and the ST-segment merged gradually with the T-wave. 'Horizontal/descending': when the ST-segment elevation is 0.1 mV within 100 ms after the J-point and continues as a flat ST-segment until the onset of the T-wave. Source: Rosso, et al. 2012.³³

study, J-point elevation was associated with an increased risk of SCD in whites and in females, but not in blacks or males.³² An early repolarization pattern in the inferolateral leads occurs in 5 % of apparently healthy individuals.^{31,36} it may not be consistently seen, and even the horizontal/descending ST type was seen in 3 % of controls.^{33,34} A pattern of J-wave and/or QRS slurring (but not of ST elevation) has been associated with cardiac arrest/sudden death in athletes,³⁷ but many healthy athletes have early repolarization with a rapidly ascending pattern. Thus, interpretation of this ECG pattern is not always straightforward.

Another confounding factor is the type of ST-segment elevation encountered in well-trained athletes.³⁸ Two types predominate; an elevated ST-segment with upward concavity and positive T-wave is seen in Caucasians, and

Figure 5: 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) and (B) ST-segment Elevation with Upward Convexity (Arrows), Followed by a Negative T-wave (Arrowheads)

Source: Corrado, et al. 2010.¹⁵

Figure 6: Electrocardiogram of a Well-trained, Asymptomatic 24-year-old Soccer Player

ST-segment elevation is observed in V2–V6, but with characteristics totally different from those seen in Brugada syndrome. A coved-type ST-segment elevation is not observed. A rounded or upsloping ST elevation is seen in V2 and V3, whereas V4–V5 show a pattern resembling that commonly encountered in early repolarization syndrome. Source: Antzelevitch, et al. 2005.³⁹

an elevated ST-segment with upward convexity and negative T-wave in African-Caribbean athletes (see Figures 5 and 6). ST-segment elevation is distinguished from Brugada syndrome by an upslope rather than a downslope pattern, and by remaining largely unaffected when challenged with a sodium channel blocker (see Figures 7–9).^{39,40} African athletes display a large proportion of ECG abnormalities, including an increase in R/S-wave voltage, ST-segment elevation and inverted or diffusely flat T-waves.⁴¹ T-wave inversion in the lateral leads is not a training-related phenomenon and may represent the initial expression of underlying cardiomyopathy. However, T-wave inversions in leads V1–V4, appear to represent an ethnic variant of athletes heart.⁴² Among other conditions, this pattern may also be seen in ARVC (see Figure 10).

Right Ventricular Abnormalities

Right ventricular (RV) dilatation and increased free wall thickness have been seen in athletes who perform isotonic or isometric exercise. In

Figure 7: Electrocardiogram Abnormality Diagnostic or Suspected of Brugada Syndrome

Type 1 ECG (coved-type ST-segment elevation) is the only diagnostic ECG in Brugada syndrome and is defined as a J-wave amplitude or an ST-segment elevation of ≥ 2 mm or 0.2 mV at its peak (followed by a negative T-wave with little or no isoelectric separation). Type 2 ECG (saddle-back type ST-segment elevation), defined as a J-wave amplitude of ≥ 2 mm, gives rise to a gradually descending ST-segment elevation (remaining ≥ 1 mm above the baseline) followed by a positive or biphasic T-wave that results in a saddle-back configuration. Type 3 ECG is a right precordial ST-segment elevation (saddle-back type, coved type, or both) without meeting the aforementioned criteria. Source: Mizusawa and Wilde, 2012.⁴⁰

Figure 8: Differential Diagnosis Between Representative Right Precordial Electrocardiogram Patterns from (A) a Brugada Patient and (B) Two Trained Athletes

Vertical lines mark the J-point (ST_j) and the point 80 ms after the J-point (ST_{80}) where the amplitudes of ST-segment elevation are calculated. 'Coved' type ST-segment elevation in the patient with Brugada syndrome is characterised by a 'downsloping' elevated ST-segment with a ST_j/ST_{80} ratio of 1.9. Right precordial early repolarization patterns in both athletes show an 'upsloping' ST-segment elevation with ST_j/ST_{80} ratio <1 ; 0.70 for the 'concave' toward the top (B, top) and 0.68 for the 'convex' toward the top (B, bottom) ST-segment elevation. Source: Corrado, et al. 2010.¹⁵

black athletes without concomitant symptoms or family history, T-wave inversion and RV enlargement may be a benign finding.⁴³ Recently, intensive, endurance exercise of increased duration (i.e. marathon runners) was reported to result in transient RV dysfunction that is usually reversible, although septal fibrosis was seen in athletes who undergo intensive training for prolonged periods.⁴¹ The clinical significance of this observation is uncertain. ARVC is a rare condition, with an estimated prevalence in the general population ranging from 0.10 to 0.02 %, ⁴² but it represents one of the most arrhythmogenic forms of human heart disease and a major cause of sudden death in the young⁴⁵ (see Figures 10 and 11). Arrhythmias originating from the right ventricular outflow tract (RVOT) either in the form of frequent ventricular ectopics or sustained or not-sustained ventricular tachycardia (VT), may represent idiopathic VT (a rather benign condition) or ARVC. An electrocardiographic scoring system for distinguishing RVOT arrhythmias in patients with ARVC

Figure 9: Borderline Brugada Electrocardiogram Pattern Mimicking Incomplete Right Bundle Branch Block

Unlike the 'R-wave' of RBBB, the 'J-wave' (arrows) of Brugada electrocardiogram (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, definitive diagnosis of Brugada ECG was achieved by a drug challenge with sodium channel blockers, which unmasked diagnostic 'coved-type' (arrows) pattern (V1 and V2). Source: Corrado, et al. 2010.¹⁵

Table 3: Abnormal ECG Findings in Athletes

These ECG findings are unrelated to regular training or expected physiological adaptation to exercise, may suggest the presence of pathological cardiovascular disease, and require further diagnostic evaluation.

Abnormal ECG Finding	Definition
T-wave inversion	>1 mm in depth in two or more leads V2–V6, II and aVF, or I and aVL (excludes III, aVR and V1)
ST segment depression	≥ 0.5 mm in depth in two or more leads
Pathologic Q waves	>3 mm in depth or >40 ms in duration in two or more leads (except for III and aVR)
Complete left bundle branch block	QRS ≥ 120 ms, predominantly negative QRS complex in lead V1 (QS or rS), and upright monophasic R wave in leads I and V6
Intraventricular conduction delay	Any QRS duration ≥ 140 ms
Left axis deviation	-30° to -90°
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 lead V1
Right ventricular hypertrophy pattern	$R-V1+S-V5>10.5$ mm AND right axis deviation $>120^\circ$
Ventricular pre-excitation	PR interval <120 ms with a delta wave (slurred upstroke in the QRS complex) and wide QRS (>120 ms)
Long QT interval*	QTc ≥ 470 ms (male) QTc ≥ 480 ms (female) QTc ≥ 500 ms (marked QT prolongation)
Short QT interval*	QTc ≤ 320 ms
Brugada-like ECG pattern	High take-off and downsloping ST segment elevation followed by a negative T wave in ≥ 2 leads in V1–V3
Profound sinus bradycardia	<30 BPM or sinus pauses ≥ 3 s
Atrial tachyarrhythmias	Supraventricular tachycardia, atrial-fibrillation, atrial-flutter
Premature ventricular contractions	≥ 2 PVCs per 10 s tracing
Ventricular arrhythmias	Couplets, triplets and non-sustained ventricular tachycardia

*The QT interval corrected for heart rate is ideally measured with heart rates of 60–90 bpm. Consider repeating the ECG after mild aerobic activity for borderline or abnormal QTc values with a heart rate <50 bpm. Drezner JA, et al. Electrocardiographic interpretation in athletes: the 'Seattle Criteria'. Source: Drezner, et al. 2013²⁰

from idiopathic VT has been developed and may be applied to VT or ventricular ectopics⁴⁶ (see Figure 12 and Table 5).

Figure 10: (A) Early Repolarization Pattern in a Healthy Black Athlete Characterised by Right Precordial T-wave Inversion (Arrowhead) Preceded by ST-segment Elevation (Arrow) and (B) Right Precordial T-wave Inversion in a Patient with Arrhythmogenic Right Ventricular Cardiomyopathy

Note that unlike early repolarization, in the ARVC the right precordial leads do not demonstrate any elevation of the ST-segment. Source: Corrado, et al. 2010.¹⁵

Table 4: Classification of Abnormalities of the Athlete's Electrocardiogram

Group 1: Common and Training-related ECG Changes	Group 2: Uncommon and Training-unrelated ECG Changes
Sinus bradycardia	T wave inversion
First-degree AV block	ST segment depression
Incomplete RBBB	Pathological Q waves
Early repolarization	Left atrial enlargement
Isolated QRS voltage criteria for left ventricular hypertrophy	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

European Association of Cardiovascular Prevention and Rehabilitation. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. Source: Corrado, et al. 2010¹⁵

Bundle Branch Block

Incomplete right bundle branch block (RBBB) (QRS <120 ms) is common. However, it should be differentiated from ARVC and Brugada syndrome.

Figure 11: Twelve-lead Electrocardiogram in an Asymptomatic Athlete with Arrhythmogenic Right Ventricular Cardiomyopathy

The athlete was referred for further echocardiographic examination and cardiac magnetic resonance because of electrocardiogram 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. Source: Corrado, et al. 2010.¹⁵

Table 5: Electrocardiographic Scoring System for Distinguishing RVOT Arrhythmias in Patients with ARVC from Idiopathic VT

ECG Characteristic	Points
Anterior T wave inversions (V1-V3) in sinus rhythm	3
VT/PVC:	
Lead I QRS Duration ≥ 120 msec	2
QRS Notching (multiple leads)	2
V5 Transition or later	1

Anterior T-wave inversions is defined as T-wave negativity in at least leads V1, V2, and V3. Lead I QRS duration ≥ 120 msec is defined as the duration from the initial deflection of the QRS complex to the end of the QRS complex in lead I. QRS notching in multiple leads is defined as a QRS complex deflection on the up-stroke or down-stroke of >0.5 mV that did not cross baseline occurring in at least two leads. (Figure 12). The precordial transition point is designated as the earliest precordial lead where the R-wave amplitude exceeded the S-wave amplitude. Source: Hoffmayer, et al. 2013⁴⁶.

Figure 12: Example of QRS Notching

Arrows show QRS notching in lead II, III, aVF and aVL. Source: Hoffmayer, et al. 2013⁴⁶

Complete left bundle branch block (LBBB) or RBBB with hemiblock necessitates cardiological work-up including myocardial imaging. ECG of the siblings should also be obtained to exclude genetically determined atrioventricular (AV) conduction disease. Brugada syndrome can also be masked by complete RBBB. For diagnosis in cases of clinical suspicion, relief of RBBB, demonstration of typical ST-segment elevation on repeated ECG recordings, pharmacological tests, or pacing from the right ventricle can be useful.⁴⁷

Figure 13: Summary of Recommendations for Preparticipation Examination Electrocardiogram Screening

ECG Abnormality	Criteria for Further Evaluation	Example			
Q waves	>3 mm in depth or >40 ms duration in any lead except III, aVR, aVL and V1		LBBB RBBB IVCD	Any QRS > 120 ms
ST depression	>0.5 mm below PR isoelectric line between J-junction and beginning of T waves in V4, V5, V6, I, and aVL >1 mm in any lead		QRP axis deviation	More leftward than -30° More rightward than 115°
T wave inversion	>1 mm in leads other than III, aVR and V1 (except V2 and V3 in woman <25 years)		QTc interval	>470 ms in males >480 ms in females <340 ms in any athlete
Atrial abnormalities	Right: P wave amplitude >2.5 mm Left: i) Negative portion of P wave in V1, V2 of >40 ms duration and 1 mm in depth; or ii) total P wave duration >120 ms		Brugada pattern	Presence of Type 1 pattern: coved ST segment in V1 and V2 gradually descending into inverted T wave
Right ventricular hypertrophy	>30 years: i) R wave > 7 mm in V1; or ii) R/S ratio >1 in V1; or iii) sum of R wave in V1 and S wave in V5 or V6 > 10.5 mm <30 years: above plus right atrial enlargement, T wave inversion in V2, V3, or right axis deviation > 115°		Pre-Excitation	Delta wave and PR interval <120 ms
			Ventricular extrasystoles, heart block, and supraventricular arrhythmia	Atrial fibrillation/flutter, supraventricular tachycardia, complete heart block or ≥2 PVCs in one 12 lead ECG

RAA = right atrial abnormality; LAA = left atrial abnormality; RVH = right ventricular hypertrophy; RAD = right axis deviation; RBBB = right bundle branch block; TWI = T-wave inversion; QTc = heart-rate correction of the QT interval. Source: Uberoi, et al. 2011.¹⁹

QT Interval

Suspicion of long QT (>470 ms in men and >480 ms in women) or short QT (<340 ms) should lead to evaluation by a specialist. A QTC 380 ms may also be a marker of abuse of anabolic-androgenic steroids.

Recommendations for pre-participation ECG screening are presented in Figure 13. ■

- Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in national collegiate athletic association athletes. *Circulation* 2011;**123**:1594–600.
- Maron BJ, Doerer JJ, Haas TS, et al. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation* 2009;**119**(8):1085–92.
- Marijon E, Tafflet M, Celermajer DS, et al. Sports-Related Sudden Death in the General Population. *Circulation* 2011;**124**:672–81.
- Chandra N, Bastiaenen R, Papadakis M, Sharma S. Sudden cardiac death in young athletes: practical challenges and diagnostic dilemmas. *J Am Coll Cardiol* 2013;**61**:1027–40.
- Corrado D, Basso C, Pavelli A, et al. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *Jama* 2006;**296**(13):1593–601.
- Holst AG, Winkel BG, Theilade J, et al. Incidence and etiology of sports-related sudden cardiac death in Denmark—implications for preparticipation screening. *Heart Rhythm* 2010;**7**(10):1365–71.
- Roberts WO, Stovitz SD. Incidence of sudden cardiac death in Minnesota high school athletes 1993–2012 screened with a standardized pre-participation evaluation. *J Am Coll Cardiol* 2013;**62**(14):1298–301.
- Steinil A, Chundadze T, Zetser D, et al. Mandatory electrocardiographic screening of athletes to reduce their risk for sudden death: proven fact or wishful thinking? *J Am Coll Cardiol* 2011;**57**(11):1291–6.
- Kong MH, Fonarow GC, Peterson ED, et al. Systematic review of the incidence of sudden cardiac death in the United States. *J Am Coll Cardiol* 2011;**57**:794–801.
- Lloyd-Jones D, Adams RJ, Brown TM, et al. Heart disease and stroke statistics—2010 update: a report from the American Heart Association. *Circulation* 2010;**121**:e46–215.
- de Vreede-Swagemakers JJ, Gorgels AP, Dubois-Arroux WI, et al. Out-of-hospital cardiac arrest in the 1990's: a population-based study in the Maastricht area on incidence, characteristics and survival. *J Am Coll Cardiol* 1997;**30**:1500–5.
- Albert CM, Mittleman MA, Chae CU, et al. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med* 2000;**343**:1355–61.
- Deo R, Albert CM. Epidemiology and genetics of sudden cardiac death. *Circulation* 2012;**125**:620–37.
- Kim JH, Malhotra R, Chiampas G, et al. Cardiac arrest during long-distance running races. *N Engl J Med* 2012;**366**:130–40.
- Corrado D, Pelliccia A, Heidbuchel H, et al. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. *Eur Heart J* 2010;**31**:243–59.
- Bille K, Figueiras D, Schamasch P, et al. Sudden cardiac death in athletes: The Lausanne Recommendations. *Eur J Cardiovasc Prev Rehabil* 2006;**13**(6):859–75.
- Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation* 2007;**115**:1643–55.
- Katritsis DG, Gersh BJ, Camm AJ. Athlete's Heart. In: Clinical Cardiology, Current Practice Guidelines. Oxford University Press, Oxford, UK. 2013;683–8.
- Uberoi A, Stein R, Perez MV, et al. Interpretation of the electrocardiogram of young athletes. *Circulation* 2011;**124**:746–57.
- Drezner JA, Ackerman MJ, Anderson J, et al. Electrocardiographic interpretation in athletes: the 'Seattle criteria'. *Br J Sports Med* 2013;**47**:122–4.
- Baggish AL, Wood MJ. Athlete's heart and cardiovascular care of the athlete: Scientific and clinical update. *Circulation* 2011;**123**:2723–2735.
- Maron BJ. Distinguishing hypertrophic cardiomyopathy from athlete's heart physiological remodelling: clinical significance, diagnostic strategies and implications for preparticipation screening. *Br J Sports Med* 2009;**43**:649–56.
- Maron BJ. Contemporary insights and strategies for risk stratification and prevention of sudden death in hypertrophic cardiomyopathy. *Circulation* 2010;**121**:445–56.
- Pelliccia A, Maron BJ, Spataro A, et al. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N Engl J Med* 1991;**324**:295–301.
- Nagueh SF, McFall J, Meyer D, et al. Tissue Doppler imaging predicts the development of hypertrophic cardiomyopathy in subjects with subclinical disease. *Circulation* 2003;**108**:395–8.
- Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. *Eur Heart J* 2012;**33**:2551–67.
- Obeyesekere MN, Klein GJ, Nattel S, et al. A clinical approach to early repolarization. *Circulation* 2013;**127**:1620–9.
- Surawicz B, Macfarlane PW. Inappropriate and confusing electrocardiographic terms: J-wave syndromes and early repolarization. *J Am Coll Cardiol* 2011;**57**:1584–6.
- Noseworthy PA, Weiner R, Kim J, et al. Early repolarization pattern in competitive athletes: clinical correlates and the effects of exercise training. *Circ Arrhythm Electrophysiol* 2011;**4**:432–40.
- Derval N, Simpson CS, Birnie DH, et al. Prevalence and characteristics of early repolarization in the CASPER registry: cardiac arrest survivors with preserved ejection fraction registry. *J Am Coll Cardiol* 2011;**58**:722–8.
- Haissaguerre M, Derval N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. *N Engl J Med* 2008;**358**:2016–23.
- Olson KA, Viera AJ, Soliman EZ, et al. Long-term prognosis associated with J-point elevation in a large middle-aged biracial cohort: the ARIC study. *Eur Heart J* 2011;**32**:3098–106.
- Rosso R, Glikson E, Belhassen B, et al. Distinguishing "benign" from "malignant early repolarization": the value of the ST-segment morphology. *Heart Rhythm* 2012;**9**:225–9.
- Tikkanen JT, Junttila MJ, Anttonen O, et al. Early repolarization: electrocardiographic phenotypes associated with favorable long-term outcome. *Circulation* 2011;**123**:2666–73.
- Wu SH, Lin XX, Cheng YJ, et al. Early repolarization pattern and risk for arrhythmic death: a meta-analysis. *J Am Coll Cardiol* 2013;**61**(6):645–50.
- Tikkanen JT, Anttonen O, Junttila MJ, et al. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med* 2009;**361**:2529–37.
- Cappato R, Furlanetto F, Giovinazzo V, et al. J wave, QRS slurring, and ST elevation in athletes with cardiac arrest in the absence of heart disease: marker of risk or innocent bystander? *Circ Arrhythm Electrophysiol* 2010;**3**:305–11.
- Perez MV, FK, Froelicher V. Semantic confusion: The case of early repolarization and the J point. *Am J Med* 2012;**125**:843–844.
- Antzelevitch C, Borggreve M, et al. Brugada syndrome: Report of the second consensus conference: Endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. *Circulation* 2005;**111**:659–670.
- Mizusawa Y, Wilde AA. Brugada syndrome. *Circ Arrhythm Electrophysiol* 2012;**5**:606–16.
- Di Paolo FM, Schmied C, Zerguini YA, et al. The athlete's heart in adolescent athletes: an electrocardiographic and echocardiographic study. *J Am Coll Cardiol* 2012;**59**:1029–36.
- Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J* 2011;**32**:2304–13.
- Zaidi A, Ghani S, Sharma R, et al. Physiological right ventricular adaptation in elite athletes of African and Afro-Caribbean origin. *Circulation* 2013;**127**:1783–92.
- La Gerche A, Burns AT, Mooney DJ, et al. Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes. *Eur Heart J* 2012;**33**:998–1006.
- Saffitz JE. Arrhythmogenic cardiomyopathy: advances in diagnosis and disease pathogenesis. *Circulation* 2011;**124**:e390–2.
- Hoffmayer KS, Bhav PD, Marcus GM, et al. An electrocardiographic scoring system for distinguishing right ventricular outflow tract arrhythmias in patients with arrhythmogenic right ventricular cardiomyopathy from idiopathic ventricular tachycardia. *Heart Rhythm* 2013;**10**:477–482.
- Aizawa Y, Takatsuki S, Sano M, et al. Brugada syndrome behind complete right bundle-branch block. *Circulation* 2013;**128**(10):1048–54.