3 Benign Arrhythmias and Conduction Defects in Athletes

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3.1 Sinus Arrhythmia

3.1.1 Definition

Sinus arrhythmia is a physiological phenomenon, common in young people and related with breathing phases that cyclically change the neuro-autonomic balance of heart rhythm [1]: inspiration stimulates the sympathetic system and sinus tachycardia, while expiration stimulates the parasympathetic system and sinus bradycardia.

3.1.2 Epidemiology in Sport

Sinus arrhythmia is a consequence of an increased vagal tone [2, 3]: the P wave axis remains normal in the frontal plane and the heart rate fluctuation should resolve with the onset of physical activity or with administration of atropine [4]. Therefore, sinus arrhythmia should be considered a normal finding in athlete's ECG [2]. The physiologic mechanisms by which training may induce these intrinsic changes in the specialized conduction system of the heart are unknown and may be multifactorial [5]: an altered ionic balance across the membrane [6] as well as biochemical and mechanical effects induced by dilation and hypertrophy [7] has been proposed as possible explanations.

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3.1.3 Current Guidelines

According to current guidelines [8, 9]:

– athletes with sinus arrhythmia without symptoms can participate in all competitive athletic activities unless otherwise excluded by underlying structural heart disease or other arrhythmias.

3.2 Sinus Bradycardia

3.2.1 Definition

Sinus bradycardia is defined as a sinus rhythm with a resting heart rate <60 bpm [10].

3.2.2 Epidemiology in Sport

Moderate and asymptomatic sinus bradycardia (40–50 bpm) is one of the most common finding in sport practitioners [11], and it reflects both functional and structural adaptations [12, 13]. Its prevalence varies widely among the populations, oscillating from 4 to 8% in non-athletes, and from 40 to 90% in athletes [14]. In the absence of symptoms such as fatigue, dizziness, or syncope, heart rates ≥30 bpm are considered a normal finding in highly trained athletes. Sinus pauses >2 s during 24-h Holter ECG monitoring are also quite common in athletes and are observed in more than one-third of them [11]. Sinus bradycardia should resolve with the onset of physical activity [2, 4]. Sinus bradycardia may be attributed to increased vagal tone and receptor density reduction, especially in the inferior left ventricular wall [15, 16]. Additional mechanisms have been described, such as reduced sensitivity to catecholamines, altered neural input to the sinus node, carotid bulb and left ventricular baroreceptor stimulation due to greater contractile force, and increased afferent vagal reflex with acetylcholine release and blockade of adrenaline release [17]. However, electrophysiological studies performed in athletes and sedentary subjects with sympathetic and parasympathetic blockade showed the existence of non-autonomic influences in the athletes sinus bradycardia at rest [5]. Nevertheless, recent evidences showed that vagal hypertonia is not the main mechanism behind this physiological adaptation, but rather intrinsic structural and ionic channel remodeling of the sinus node [18–20]. Some athletes may have extreme forms of sinus bradycardia, such as a resting sinus rhythm <30 bpm or heart pauses ≥3 s [2].

Clinical evaluation includes a careful history to determine whether sinus bradycardia is related to symptoms like dizziness or syncope. Exercise stress test may be useful to verify a normal heart rate response. Symptomatic athletes and asymptomatic athletes with extreme bradycardia at rest (e.g., <30 bpm on ECG) should have 24-h Holter ECG monitoring and a cardiac work-up to exclude structural heart disease [12, 21]. Even if in a small percentage of sport practitioners, sinus bradycardia is a negative prognostic sign [22]. According to several authors, in the absence of symptoms and evidence of structural cardiac disease and adequate chronotropic response to physical exercise, these athletes should not be excluded from their usual sporting activities [2]. However, the tolerable limit of pause duration to define sports interruption, or even pacemaker implantation, is not well established [23]. Cessation of sports activity may result in resolution of symptoms and improvement in rhythm after 3–6 months [24]. In asymptomatic patients with extreme bradycardia at rest (e.g., <30 bpm on ECG) a yearly follow-up could be sufficient. In those who were symptomatic before but became asymptomatic after a detraining period, a follow-up evaluation after 6 months is recommended [12].

With significant sinus bradycardia, a junctional or ventricular escape rhythm can compete with the sinus rhythm. Sinus arrhythmia and wandering atrial pacemaker are also more prevalent in athletes compared with the general population [25].

3.2.3 Current Guidelines

According to current guidelines [8, 9, 12]:

Eligibility for competitive sport can be granted in case of sinus bradycardia, if:

- absence of underlying cardiac disease that is incompatible with sports
- absence of bradycardia-related symptoms (syncope, asthenia, dizziness, dyspnea, effort intolerance)
- in subjects with no evidences of sinus node intrinsic dysfunction (at least 85% of max heart rate expected at exercise stress test and no heart pauses >3 s at 24-h Holter ECG)
- in highly trained aerobic athletes, also having <40 bpm and heart pauses >3 s
- in doubtful cases, after a detraining period (3–6 months) leading to a disappear of these phenomena

Athletes should be restricted from training and athletic competition while being evaluated (exercise stress test and 24-h Holter ECG). If treatment of the bradycardia eliminates symptoms, they can return to sport.

3.3 Right Bundle Branch Block

3.3.1 Definition

Right bundle branch block (RBBB) is divided into two subtypes [26]:

- complete RBBB: QRS complex >120 ms + rsR’ pattern in leads V1 and/or V2 or RR’ pattern in leads V1 and/or V2 + wide and slurred S wave in lead V6 and I (S > R duration or S wave duration >0.06 s)
- incomplete RBBB: QRS complex 100–120 ms + rsr’ pattern in leads V1 and/or V2.

3.3.2 Epidemiology in Sport

Mildly delayed right ventricle conduction in trained athletes could be linked to a spectrum of structural and physiological cardiac remodeling (i.e., right ventricle dilatation, increased cavity size, and resultant increased conduction time [13, 27]); indeed, some highly trained athletes manifested a reduction in right ventricular systolic function at rest and a higher rate of RBBB [28]. Therefore, incomplete RBBB represents a phenotype of cardiac adaptation to exercise [13], and in the absence of other features suggestive of disease does not require further evaluation [2]. Its prevalence in athletes varies between 35 and 50% [9]. Complete RBBB is detected in approximately 1% of the general population, with a range of 0.5–2.5% in several studies [29–31]. The presence of at least one of other borderline findings (left axis deviation, left atrial enlargement, right axis deviation, right atrial enlargement) does not warrant further assessment in asymptomatic athletes without a family history of premature cardiac disease or sudden cardiac death. Conversely, the presence of more than one of these borderline findings in combination should be additionally investigated [2].

3.3.3 Current Guidelines

According to current guidelines [8, 9]:

Eligibility for competitive sport can be granted in:

- absence of underlying cardiac disease that is incompatible with sports
- isolated RBBB
- RBBB + left anterior fascicular (LAFB) or posterior fascicular (LPFB) blocks only after excluding cardiac diseases

In particular, athletes with RBBB, who do not develop periods of second degree Mobitz 2 AV block or third degree AV block spontaneously or during exercise and who have no symptoms or heart disease identified by appropriate testing that otherwise precludes participation, can participate in all competitive athletics.

Eligibility for competitive sport should be denied in:

- presence of family history of sudden cardiac death
- presence of cardiac syncope
- presence of family history of Lenegre disease, Brugada syndrome, or ion channel diseases

3.4 Left Bundle Branch Block

3.4.1 Definition

Left bundle branch block (LBBB) is an electrocardiographic abnormality detected in patients affected by abnormal cardiac conduction through anterior and posterior left fascicles of the His-Purkinje system [32].

3.4.2 Epidemiology in Sport

LBBB is found in less than 1% athletes [28, 33]. On the other hand, there is a high rate of cardiomyopathy in patients with LBBB: one study comparing athletes and patients with hypertrophic cardiomyopathy showed that 5.9% of these patients had LBBB, but no athlete with normal cardiac imaging had LBBB [34]. Thus, complete LBBB always should be considered an abnormal finding and requires a comprehensive evaluation to rule out a pathological cardiac disorder [2].

3.4.3 Current Guidelines

According to current guidelines [8, 9]:

Eligibility for competitive sport can be granted in:

- absence of underlying cardiac disease that is incompatible with sports
- isolated LAFB or LPFB
- LBBB, RBBB + left anterior fascicular (LAFB), or posterior fascicular (LPFB) blocks only after excluding cardiac diseases.

In particular, athletes with permanent or rate-dependent LBBB who do not develop spontaneous second degree Mobitz 2 AV block or third degree AV block and who have no symptoms or heart disease identified by appropriate testing that otherwise precludes participation can participate in all competitive athletics.

Eligibility for competitive sport should be denied in:

- presence of family history of sudden cardiac death
- presence of cardiac syncope
- presence of family history of Lenegre disease, Brugada syndrome, or ion channel diseases.

In athletes with concerning symptoms, an electrophysiological study (EPS) is recommended. An athlete with a normal H-V interval (time from the initial deflection of the His bundle potential and the onset of ventricular activity) and a normal atrio-ventricular (AV) conduction response to pacing can participate in all competitive sports unless otherwise restricted by their structural heart disease; athletes with abnormal AV conduction characterized by an H-V interval >90 ms or a His-Purkinje block should have pacemaker implantation.

3.5 Atrio-Ventricular Blocks

3.5.1 Definition

Atrio-ventricular blocks (AV blocks) are partial or complete interruptions of the transmission of the electrical impulse from the atrial chambers to the ventricular ones [35].

3.5.2 Epidemiology in Sport

AV conduction disorders are frequent in athletes: first degree AV block is the most common finding (from 7–10% of basal ECG, up to 25.7–40% of 24 h Holter ECG [3, 36]), followed by second degree Mobitz 1 AV block (15–22% of basal ECG [37]). Both are due to parasympathetic hypertonia and are common in endurance athletes [4], being part of physiological adaptations to exercise [13, 38]: in trained athletes indeed, there is a sympathetic decrease with relative vagal tone prevalence, and a concurrent decrease of the heart rate [2, 39]. In asymptomatic athletes, the resolution of this arrhythmia through hyperventilation or physical exercise confirms their functional origin and excludes any pathological meanings [40]. If PR interval does not shorten with physical exercise, is associated with syncopal symptoms or with a familiarity for cardiac pathology, it is advisable to perform an echocardiogram and a 24-hours Holter ECG [2]. Most of the first degree AV blocks show a PR interval ≤280 ms [24]; a PR interval ≥400 ms has to be considered significantly prolonged and therefore must necessarily be investigated [2]. More severe AV blocks are rare. Second degree Mobitz 2 AV block or third degree AV block may be present in some elite athletes, especially at night (24-h Holter ECG): their persistence during routinely ECG and their failure to disappear during exercise should be investigated to exclude heart diseases [12, 41]. However, ventricular pauses of ≥3 s or a resting heart rate ≤40 bpm due to conduction disturbances are rarely observed in leisure-time athletes, even if such disturbances have been reported in high-level athletes [42]. Therefore, these arrhythmias should never be considered as benign findings in the athlete's ECG, and should necessarily be investigated [2, 43]. Electrophysiological study is advisable in case of second or third degree AV blocks. In doubtful cases, a period of detraining (3–6 months) and a subsequent revaluation are recommended [9, 12, 39].

3.5.3 Current Guidelines

According to current guidelines [5, 9, 12]:

– athletes with asymptomatic first degree AV block or second degree Mobitz 1 AV block at rest and normalization during exercise or hyperventilation can participate in all sports.
– symptomatic athletes with second degree Mobitz 2 AV block while awake should temporarily refrain from sports. If exclusion of a structural cause and normalization within 3 months occurs, low-moderate sports can be resumed. After 4 weeks with tolerable low-moderate sports, higher intensity sports may be undertaken. Follow-up with 24-h Holter ECG is advised.
– in athletes with extreme PR prolongation first degree AV block (>300 ms), more intensified follow-up is warranted (e.g., every 6 months).
– second degree Mobitz 2 AV block or third degree AV block requires exclusion of underlying structural heart disease,

- in second degree Mobitz 2 AV block or third degree AV block without structural heart disease, a deconditioning phase of 3–6 months can be considered,
- in second degree Mobitz 2 AV block or third degree AV block with structural heart disease, a pacemaker is recommended,
- eligibility for low intensity sport can be considered in congenital AV block with narrow QRS interval, with resting heart rate >40 bpm that increase during physical activity and without effort's complex ventricular arrhythmias,
- eligibility for competitive sport should be denied in case of symptomatic bradycardia, presence of cardiac disease that is incompatible with sports and pauses >3 s (except trained athletes practicing aerobic sports).

3.6 Premature Ventricular Beats

3.6.1 Definition

A premature ventricular beat (PVB) is an extra, abnormal heartbeat, that is electrocardiographically characterized by an early onset and a morphologically abnormal QRS complex with a duration >120 ms [44, 45].

3.6.2 Epidemiology in Athletes

Even if there is a traditional perspective that PVBs are a consequence of the structural and neuro-autonomic remodeling of the athlete's heart [46], ventricular arrhythmias rate in young competitive athletes is low, similar to that of sedentary individuals and it is unrelated to type and intensity of sport practiced. Young athletes were found to be more likely to have isolated PVBs, compared with their sedentary counterparts (49% vs 28%) [47], but this difference was not seen in a study of middle-aged athletes and sedentary controls (53% vs 50%) [48]. PVBs at resting ECGs are uncommon: multiple PVBs can be found in less than 1.1–2.1%, and polymorphic PVBs only in 0.1% [31, 49]. Similar to the non-athletic general population, PVBs in the athletes may be associated with an underlying myocardial substrate, potentially at risk of sudden cardiac death. Therefore, the first objective in evaluating an athlete with ventricular arrhythmias is to exclude life-threatening cardiovascular diseases [50].

According to the number, morphologic pattern, complexity, response to exercise and clinical manifestation, PVBs can be classified as common and likely benign or uncommon and potentially malignant because of underlying cardiac pathology [50, 51]. A multiparametric approach is useful for differentiating between these two variants. Different studies have shown evidence that up to 30% of athletes with ≥2000 PBVs per 24 h [52], were found to have underlying structural heart disease [46, 53]. However, a high number of PVBs in itself does not confer an increased risk of malignant events [51]. For this reason, additional tests are necessary in athletes with ≥2000 PBCs per 24 h, or with episodes of non-sustained ventricular tachycardia, or with an increasing burden of PVBs during an exercise test. These additional evaluations include echocardiography, cardiac magnetic resonance imaging (MRI), and electrophysiology study.

Further evaluation is warranted when ≥2 PVBs are recorded on a resting 12-lead ECG [2]. However, even a single PVB may deserve attention especially in the presence of one or more of these five features: (1) positive family history of premature sudden cardiac death (SCD) or cardiomyopathy, (2) relevant symptoms, (3) associated ECG abnormalities, (4) uncommon PVB morphology, and (5) short coupling interval [54]. Work-up should also include a search for agents that may enhance electrical ventricular irritability, such as the use of excessive amount of alcohol, illicit drugs, or stimulants, particularly ephedrine and caffeine [25].

The assessment of the morphology of the ectopic QRS complex on the ECG helps to identify the anatomical origin of the PVBs [54]: common patterns in athletes are infundibular and fascicular ones, while atypical RBBB with QRS ≥130 ms and LBBB patterns with superior or intermediate axis are not common and should be further investigated. The prevalence of concomitant repolarization/depolarization ECG abnormalities increases the probability of an associated disease [25]. An increase in the arrhythmia at the beginning of exercise, disappearance at peak exercise, and reappearance during recovery usually suggest a benign process [46]: exercise stress test should not be stopped at the 85% of theoretical maximal heart rate but continued until the athlete is exhausted in order to increase the test sensitivity [51, 55].

3.6.3 Current Guidelines

According to current guidelines [9, 51, 56]:

- athletes with ≥2 PVBs on a baseline ECG (or ≥1 PVB in case of high-endurance athletes, or positive family history of premature SCD or cardiomyopathy, relevant symptoms, associated ECG abnormalities, uncommon PVB morphology, and/or short coupling interval) should undergo a complete evaluation to exclude underlying structural or arrhythmogenic conditions. This includes a detailed familial history, ECG (morphology suggestive of common and likely benign, or uncommon and potentially malignant VPB forms), 24-h Holter ECG monitoring possibly with a 12-lead system and including a sports session (morphology, number, and complexity of VBPs), exercise stress test (increase or decrease with exertion), and suitable imaging (echocardiography and computerized tomography and/or MRI). If no indication of familiar or structural underlying disease, all competitive and leisure-time sports activities are allowed.
- athletes with a high prevalence of asymptomatic PVBs (in absence of structural heart disease) should be re-evaluated annually (particularly in case of children and adolescents) in order to identify potential changes in the arrhythmic burden and in underlying cardiac condition. In symptomatic athletes without structural heart disease, medical treatment for PVB may be an option.

– eligibility for competitive sport can be granted in case of absence of sudden cardiac death or arrhythmia family history, absence of cardiac diseases, absence of symptoms (syncope, dizziness, palpitations), in subjects with benign pattern such as fascicular or outflow tract origins.
– eligibility for competitive sport should be denied in case of presence of sudden cardiac death or arrhythmia family history, presence of cardiac diseases not compatible with sport, presence of symptoms (syncope, dizziness, palpitations), PVBs premature and/or repetitive with short couples and/or non-sustained ventricular tachycardia, frequents PVBs with concomitant reduction of ejection fraction.

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