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Cardiac pauses in competitive athletes: a systematic review examining the basis of current practice recommendations

Tunay Senturk[1,2], Hai Xu[1], Krishna Puppala[1], Balaji Krishnan[1], Scott Sakaguchi[1], Lin Y. Chen[1], Rehan Karim[3], Oana Dickinson[1], and David G. Benditt[1]*

[1]Cardiac Arrhythmia and Syncope Center, Cardiovascular Division, Department of Medicine, University of Minnesota Medical School, Mail Code 508, 420 Delaware Street SE, Minneapolis, MN, USA; [2]The Department of Cardiology, University of Uludag Medical School, Bursa, Turkey; and [3]Division of Cardiology, Department of Medicine, Hennepin County Medical Center, Minneapolis, MN, USA

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Aims It is generally recommended that individuals aspiring to competitive sports should undergo pre-participation cardiovascular assessment, particularly including arrhythmia risk evaluation. In regard to bradyarrhythmias, the 36th Bethesda Conference suggested that asymptomatic cardiac pauses $\leq$3 s are 'probably of no significance', whereas longer 'symptomatic' pauses may be abnormal. This study focused on assessing the evidence for the '3 s' threshold.

Methods A systematic literature search was undertaken including Embase (1980–) and Ovid Medline (1950–). The following MeSH terms were used in the database searches: Cardiac.mp & pause.mp. Additionally, pertinent publications found by review of citation lists of identified publications were examined. Individuals with reversible causes of bradyarrhythmia (e.g. drugs) were excluded.

Results The study population comprised 194 individuals with cardiac pauses of 1.35–30 s. In 120 athletes, specific records for pause durations were provided, but it was not always clear whether pauses occurred at rest. Among these 120 athletes, 106 had pauses $\leq$3 s, of whom 92 were asymptomatic and 14 were symptomatic. Fourteen athletes had pauses $>$3 s, of whom nine were asymptomatic and five were symptomatic. There were no deaths during follow-up (7.46 $\pm$ 5.1 years). With respect to symptoms, the $\leq$3 s threshold had a low-positive predictive value (35.7%) and low sensitivity (26.3%), but good negative predictive value (86.7%) and specificity (91%).

Conclusion While the evidence is not incontrovertible, the 3 s pause threshold does not adequately discriminate between potentially asymptomatic and symptomatic competitive athletes, and alone should not be used to exclude potential competitors.

Keywords Cardiac • Pause • Sports • Athletes • Symptoms • Bradycardia

Introduction

It is generally accepted that individuals who aspire to competitive sports should undergo pre-participation cardiovascular assessment.[1] Nonetheless, the optimum screening strategy for prospective athletes remains unresolved.[2,3] Further, as pointed out in the 36th Bethesda Conference,[4] the published recommendations that do exist for identifying individuals at 'high risk' for adverse sport-related health issues are based mainly on expert consensus of 'what seems reasonable'.[4]

Inasmuch as there is concern that physical exertion may trigger potentially hazardous tachyarrhythmias, pre-participation screening has principally focused on tachycardia susceptibility. Bradyarrhythmias have received less attention inasmuch as they are presumed to be less likely to pose a risk during physical activity.

Certain bradyarrhythmias (first degree and Mobitz type I atrioventricular (AV) block) are common in the resting competitive athlete and are generally deemed to be of little concern in the absence of underlying structural heart disease.[5] However, a number of bradyarrhythmias may cause collapse in association with exertion,

* Corresponding author. Tel: +1 612 625 4401; fax: +1 612 624 4937. *E-mail address*: bendi001@umn.edu

What's new?

- Cardiac pauses ≤3 s, which have often been considered to be the upper limit of normal in healthy individuals, did not provide an adequate threshold to separate symptomatic and asymptomatic competitive athletes.
- A 5 s pause provided greater specificity but lacked sensitivity.
- Further assessment is need to determine diagnostic criteria for identifying susceptibility to worrisome bradyarrhythmias in competitive athletes.

including cardiac pauses during exertion (*Figure 1*) such as might be triggered by exercise-induced AV block, or sinus pauses/arrest during extreme straining (e.g. during weight lifting) or during cool-down after exertion. In any case, with respect to bradyarrhythmia screening for athletes, the 36th Bethesda Conference suggested that asymptomatic cardiac pauses of <3 s are 'probably of no significance',[4] whereas longer 'symptomatic' pauses may be of greater concern.

The objective of this study was to evaluate the evidence surrounding the '3 s' cardiac pause threshold. To this end, we undertook a systematic review of the published literature pertaining to the documentation of, and if available, the subsequent follow-up of cardiac pauses in active trained athletes.

Methods

This report is based on a systematic literature search undertaken between December 2013 and December 2014. The published materials comprised case reports, and both prospective and retrospective studies, in which cardiac pauses were identified in athletic individuals. Only publications in which complete text was available for review were included. Review articles were used to assure that our literature searches were complete, but were not used for data accumulation. There were no language restrictions applied to the searches.

Databases searched for this report included Embase (1980–) and Ovid Medline (1950–). The following MeSH terms were used initially in the database searches: Cardiac.mp & pause.mp & sport.mp. This combination yielded four articles. A revised search MeSH term was then employed: Asystole.mp & athlete.mp yielded a total of 11 publications. Thereafter, the MeSH term: Cardiac.mp & pause.mp resulted in 473 articles.

The searches described above resulted in a total of 488 articles. This group incorporated all articles previously located by the authors as well as those included in their personal files prior to beginning the formal searches.

The 488 articles were individually screened by title and abstract to identify 18 candidate publications (*Figure 2*). Full texts of the 18 candidate publications were reviewed in detail. Of these 18 publications, 11 had full data inclusion of athlete subjects. A data extraction form was developed and the following pre-determined findings were identified and recorded when provided from each of the 11 publications: (i) number of subjects with reported cardiac pauses, (ii) recording method, (iii) duration of pause(s), (iv) athlete age at time of arrhythmia diagnosis, (v) gender, (vi) sports participated in by athlete, (vii) athlete's past medical history, (viii) treatment, and (ix) clinical outcome and follow-up duration. When the information was provided in the publication, bradyarrhythmia-associated symptoms were recorded. Symptoms that were particularly sought out included syncope, pre-syncope, or mortality/cardiac arrest.

Results

The study population encompassed by the 11 publications identified as described above consisted of 194 individuals, of whom 10.8% were female athletes. Sports being practiced by these subjects included swimming, long-distance running, cross-country running and skiing, soccer, canoeing, mountain climbing, body-building, basketball, biathlon, and cycling. Most of these athletes were in active training and competed regularly at an intense level during the time when the original reports of cardiac pauses were obtained.

The study population, not surprisingly given its athletic background, was generally very healthy with no past medical history of concern. Other clinical characteristics are listed in *Table 1*.[5–15]

Figure 1 Figure of exercise-induced cardiac pause resulting in collapse of 23-year-old female athlete running on a treadmill. The patient was wearing a PILOT® heart rate recording instrument. The abrupt heart change is apparent at an atrial rate of >170 bpm. Subsequently, she has been limited to exercise rates <160 bpm and has been asymptomatic for 2 years.

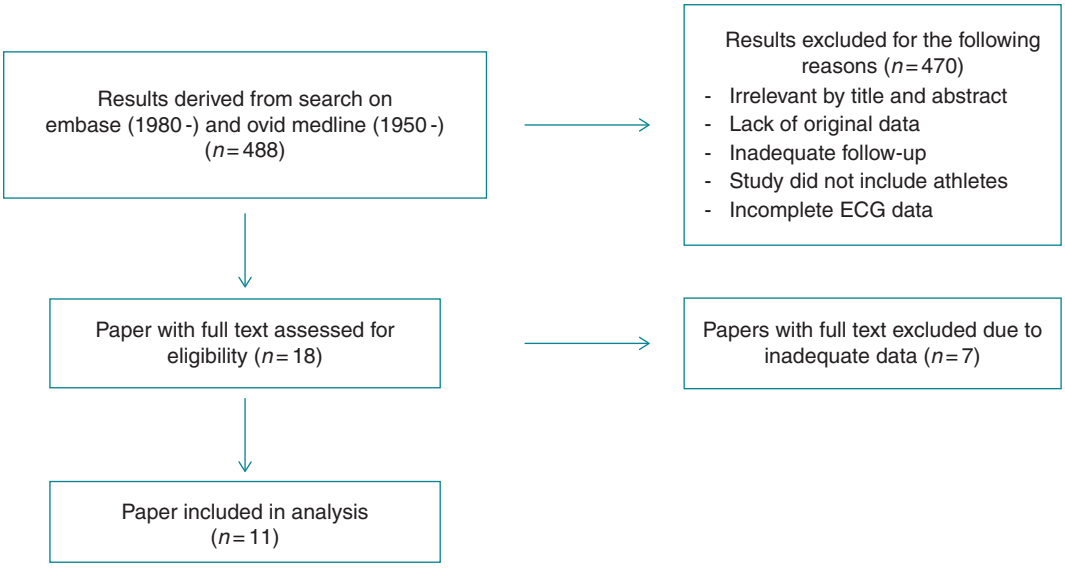

Figure 2 Flow chart of the systematic literature search used in this report.

Cardiac pause data in these reports were derived from Holter monitoring or ambulatory ECG, and in the larger set of 194 athletes ranged from 1.35 to 30 s in duration (*Table 1*). In the subset of 120 athletes in whom specific records for their individual pause durations or range of pause durations were provided, cardiac pauses <3 s comprised the majority of all reported pauses.

Among 14 athletes having pauses >3 s, 10 had sinus arrest (71.4%), 1 had both sinus arrest and AV block (7.2%), and 3 had second or third degree AV block (21.4%).[5,7–9,11,13–15] Of the 106 athletes with pauses ≤ 3 s, 80 exhibited sinoatrial block or sinus pauses (71.4%), and 26 had second or third degree AV block (21.4%).[5–8,10,11] In most cases, it was not noted whether the pauses occurred during activity or while at rest.

In the 14 athletes with pauses >3 s, 9 (9/14, 64%) were asymptomatic and 5 (5/14, 36%) were symptomatic. In the 106 athletes with pauses ≤ 3 s, 92 were asymptomatic (92/106, 86.7%) and 14 were symptomatic (14/106, 13.3%) (*Table 2*). Unfortunately, given limitations of ambulatory ECG recording systems at the time, it was not possible to provide the precise relationship of symptoms during follow-up to specific bradyarrhythmias. Further, the longer pauses tended to occur during the night while the subject was presumably sleeping, and consequently may have diminished likelihood of reporting symptoms.

The positive predictive value for symptoms in the setting of the recommended ≤ 3 s threshold was low (35.7%), as was sensitivity for symptom presence (26.3%). However, in terms of symptoms, the ≤ 3 s threshold had good negative predictive value (86.7%) and specificity (91%) (*Table 2*). If, as an alternative, a 5 s cardiac pause is used as the cutoff value, the positive predictive value for symptoms improved (66.6%), and both the negative predictive value (86.8%) and specificity (98%) remained high. However, sensitivity (21%) for detecting symptomatic patients was poor (*Table 2*).

The longest pause (30 s) was observed in a 23-year-old male athlete who had history of syncope in the past.[5] Given this prolonged pause, he ultimately received a pacemaker at the discretion of his physician. In addition, another 17 patients in this study were recorded to have received medical interventions at the time of their cardiac pauses: their treatment ranged from procedures such as pacing and cardiopulmonary resuscitation (CPR), to medications such as disopyramide and isoproterenol. Two patients refused pacemaker therapy despite having experienced a symptomatic episode.

The duration of follow-up in the overall population was 3 weeks to 12 years (7.46 $\pm$ 5.1 years). The majority of subjects received no treatment interventions at the time of recorded cardiac pauses, and 42 of the 62 individuals in whom follow-up data were provided remained symptom free with no need for treatment at the time of follow-up exams. No cardiac deaths were reported among subjects in whom follow-up was reported.

In one report, 8 athletes with prolonged cardiac pauses became symptom-free during 3–10 years of follow-up after stopping heavy physical training.[5] Similarly, in one other report, susceptibility to cardiac pauses were diminished in runners by reducing their average running distance.[8]

Discussion

This study provides a systematic review of published literature in which cardiac pauses were reported in competitive athletic individuals. There were three principal findings. First, cardiac pauses (>3 s) are observed in apparently healthy actively competitive athletes. Second, our findings suggest that 3 s pause threshold does not adequately discriminate between asymptomatic and potentially symptomatic competitive athletes. Finally, despite pauses well in excess of 3 s being reported, few subjects were deemed to require medical intervention at the time of diagnosis, and to the extent that follow-up was provided, the vast majority had no subsequent sequelae that could be attributed to athletic participation. To the

Table 1 Summary of the published studies and/or case reports analysing the relationship cardiac pause and symptoms in athletes

Reference	Subjects (F/M)	Age	Type of sport	Symptoms	Duration of pause (s)	Outcome
Ector *et al.*[5]	37 (M)	17–40	Runner, cyclist	None	2 s pause in 19 athletes >2 s pause in 7 athletes max pause 2.5 s	NA
Talan *et al.*[6]	20 (M)	19–28	Runner	None	Walking max: 1.35–2.55 Sleeping: 1.60–2.81	NA
Ogawa *et al.*[7]	30 (M)	23.6 ± 4	Cross-country skiers	None	<2.0 s: 10; 2.0–3.0 s: 15; >3.0 s: 5	None
Hood *et al.*[8]	1 (M)	56	Runner	None	2.6	None
	1(M)	66	Runner	None	2.5	None
	1 (M)	50	Runner	None	3.5	None
	1 (M)	72	Runner	None	2.4	None
	1 (M)	77	Runner	None	15	Pacemaker
	1 (M)	68	runner	None	2.5	None
	1 (M)	66	Runner	None	2.8	None
	1 (F)	61	Runner	None	2.5	None
Slavich *et al.*[9]	1 (F)	35	Runner	None	10	Pacemaker
Viitasalo *et al.*[10]	35 (M)	23.1 ± 6.1	Runner, skier, basketball player	None	2.0 (13/35)	NA
Bjørnstad *et al.*[11]	30 (15M/15F)	23.9	Runner, cross country skiers	None	Max 3.1 s (3/30 athletes)	NA
Milstein *et al.*[12]	2 (1F/1M)	NA	Runner, swimmer	Syncope	NA	CPR
Karadag *et al.*[13]	1 (M)	41	Body builder	Syncope	21	CPR
Ector *et al.*[5]	1 (M)	26	Soccer 9,	Syncope,	1.8	None
	1 (M)	18	Handball 1,	Adam Stokes	1.5	Isoprenalin
	1 (M)	14	Cycling 2,	attacs	1.7	None
	1 (M)	25	Athletic 1,	or both	2	None
	1 (M)	23	Rowing 1,		2.32	Belladona
	1 (M)	45	Long distance runner 1,		1.8	Pacemaker
	1 (M)	19	Swimmer 1		2.4	Refused pacemaker
	1 (M)	35			3.2	Pacemaker
	1 (M)	25			2.84	Pacemaker
	1 (M)	12			2	Isoprenalin
	1 (M)	12			1.6	Refused pacemaker
	1 (F)	16			1.4	Pacemaker
	1 (F)	17			1.6	Pacemaker
	1 (F)	22			2.32	Pacemaker
	1 (M)	16			1.6	None
	1 (M)	23			30	Pacemaker
Ito *et al.*[14]	1 (F)	16	Cross county skier	Pre-syncope	10	Disopiramide
Yerg JE 2nd *et al.*[15]	1 (M)	24	Runner	Syncope	22	None

best of our ability to determine, placement of cardiac pacemakers was primarily based on physician or patient preference.

The factors accounting for cardiac pauses in a given athlete warrant individual evaluation prior to considering potential therapeutic options. In this regard, enhanced resting parasympathetic tone (or perhaps more accurately enhanced parasympathetic vs. sympathetic balance) is an accepted adaptation to repetitive exercise in competitive athletes.[16] For instance, Mobitz 1 AV block and sinus bradycardia are accepted normal rhythm variants in this population.[4,5] Similarly, prolonged sinus pauses, especially during rest or sleep may be an expected rhythm variant in well-trained individuals. However, high-grade AV block during exercise, chronotropic incompetence, or strain-induced pauses (e.g. during weight lifting) would reasonably be considered to be abnormal, and may signal underlying disease. In these latter cases, although accounting for only a small minority of competitive athletes, the long-term outcome should not be assumed to be benign.

Distinguishing those cardiac pauses which are worrisome and merit treatment, from more benign forms is not straightforward. As is evident from our findings, the duration of the pause may be

an inadequate marker. Even discovering that underlying structural heart disease is present cannot definitively define the risk associated with an observed pause. Until further study clarifies indications for initiating treatment for detected bradycardia, the most important diagnostic step remains the establishment of arrhythmia-symptom correlation during ambulatory cardiac monitoring.

Currently, practitioners have little guidance regarding appropriate management of athletes in whom ECG screening identifies cardiac pauses >3 s duration. The 36th Bethesda Conference avoided offering specific advice, and given the limited data available at the time, clearly admitted that the consensus statement was based on 'what seems reasonable'. Even now, an indication for pacing in this

Table 2 Assessment of cardiac pauses of 3 and 5 s for prediction of symptoms in athletes

	Pause < 3 s	Pause ≥ 3 s	Pause < 5 s	Pause ≥ 5 s
Age (range)	12–72	12–77	12–72	35–77
Duration of pause Median (min–max)	2 (1.35–2.84)	10 (3.1–30)	2.32 (1.35–3.5)	21 (10–30)
Asymptomatic	92	9	99	2
Symptomatic	14	5	15	4
PPV	35.7%		66.6%	
NPV	86.7%		86.8%	
Sensitivity	26.3%		21%	
Specificity	91%		98%	

PPV, positive predictive value; NPV, negative predictive value.

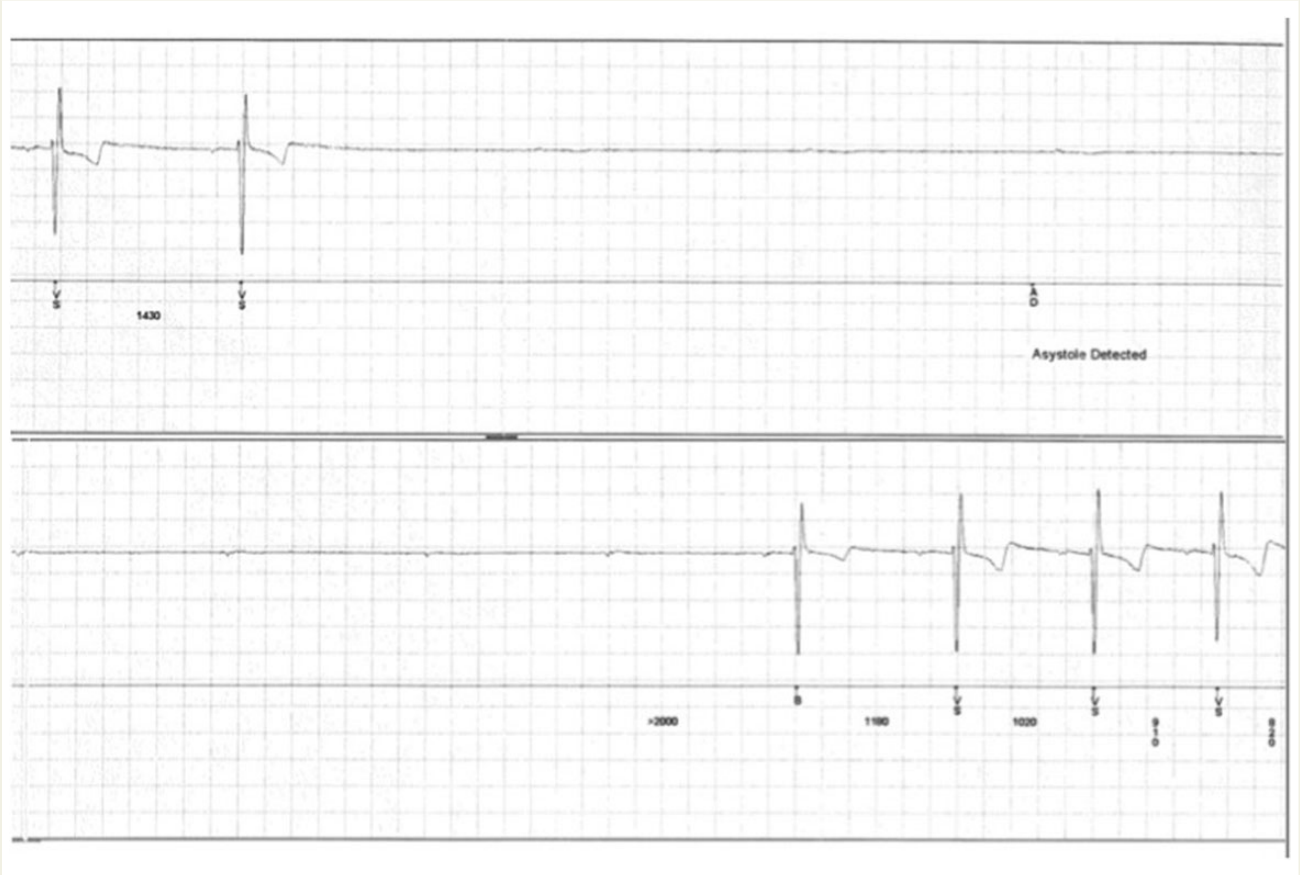

Figure 3 Rhythm strip from asymptomatic 25-year-old male athlete at rest. He had been referred for assessment of slow heart rate during routine examination. Pause duration is ~13.8 s. This patient continues to exhibit pauses 3 years later but remains untreated, asymptomatic, and athletically very active.

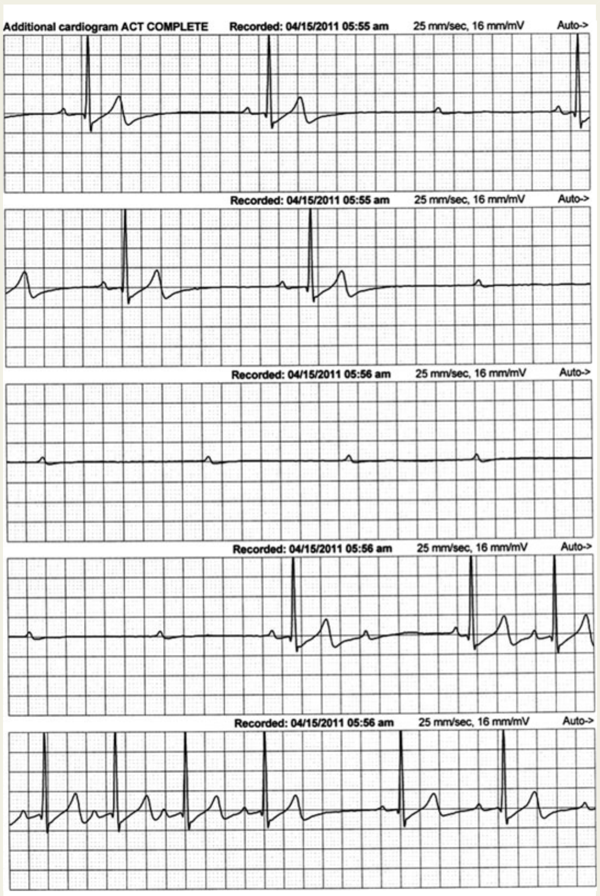

Figure 4 Electrocardiographic recording of a night-time cardiac pause >13 s in a 21-year-old female competitive college rower. The subject had multiple nocturnal pauses of 7–13 s duration. She has been asymptomatic during 3.5 years follow-up, and continues to be able to exercise to heart rates >190 bpm without problems.

population is not firmly established and as was the case in the publications cited here, the advice for pacing remains largely at the discretion of the attending physician.

Prior reports have observed cardiac pauses >2 s in endurance athletes and long distance runners,[5,9] but few offer long-term follow-up to permit assessment of the clinical implications. However, Ogawa *et al.*[7] examined outcomes in 30 highly trained athletes who underwent Holter monitoring: 10 athletes had <2 s pause, 15 had 2.0–3.0 s pause, and 5 had >3 s cardiac pause. The longest ventricular pause was 3.4 s. All but one of the 30 athletes continued vigorous training.[7] During 3-year follow-up, none of the athletes reported bradyarrhythmia-related symptoms, and their outcomes did not differ from those in athletes without pauses. In our study, 14 athletes had pauses ≥3 s and most of these individuals (9/14, 64%) were asymptomatic. Six athletes had pauses ≥5 s, of whom four (4/6, 66.6%) were symptomatic. Although a small number of athletes received pacemakers based on physician advice, thereby undermining interpretation of the natural history in these cases, the vast majority of athletes were untreated and there were no deaths or exercise-related adverse events in these individuals. Consequently, we found no evidence of subsequent harm associated with pauses well in excess of 3 s. Furthermore, our own recent experience (*Figures 3* and *4*), as well as published literature,[9] suggests that even pauses in excess of 10 s (which usually occur at rest and most often at night) may be observed in seemingly very healthy individuals without adverse sequelae.

In our assessment of the clinical characteristics of symptomatic athletes who received pacemaker therapy in the published literature, the majority had devices placed for cardiac pauses <3 s. In the case of elite athletes, placing a pacemaker may substantially alter the types of sports that they are permitted to compete in and thereby substantially adversely affect their lifestyle. As such, in the absence of definitive symptom-arrhythmia concordance, pacing intervention would not seem to be warranted. On the other hand, the published evidence base is limited and future reports should focus on follow-up of such patients over many years in order to more clearly establish outcomes.

Limitations

Interpretation of our findings is subject to important limitations. First, the number of reported cases with sufficient bradyarrhythmia detail is small. Second, there was a lack of unified protocol for follow-up evaluations. In fact, most patients had their follow-up studies conducted after only a few months of initial screening diagnosis, and only a few had serial follow-up years later to assess late-stage sequelae. Third, the study population was heterogeneous and consisted of a broad age range of subjects from different sports. Fourth, it was not usually evident whether the pauses were recorded while the athlete was awake or during sleep. Fifth, the literature does not provide adequate detail to determine whether subsequent symptoms during follow-up were definitively associated with bradyarrhythmias, or whether there was a relation between the level of exercise training and susceptibility to subsequent symptoms. Finally, our observations incorporated data from a number of case study reports. While these reports presented dramatic cardiac pauses from certain subjects, there is an inevitable reporting bias inasmuch as only the most dramatic pauses are likely to be reported, and the denominator remains unknown. Nevertheless, to the extent that such cases represent the 'worst case', the observations provide an element of prognostic insight.

Conclusions

While available clinical evidence is not incontrovertible, the 3 s pause threshold does not adequately discriminate between potentially asymptomatic and symptomatic competitive athletes, and alone should not be used as a determining factor to exclude potential competitors. Further, the 3 s pause threshold does not appear to warrant either exercise restriction or early therapeutic intervention. Future studies should focus on longer-term follow-up to determine if there is a pause threshold that may be useful for identifying high symptomatic risk due to bradyarrhythmias in this population.

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