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Bradycardia in Athletes: Prevalence, Mechanisms, and Risks

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BACKGROUND: Sinus bradycardia is a well-recognized physiological adaptation in endurance athletes, primarily attributed to sinus node remodeling or increased vagal modulation. Although genetic influences on resting heart rate (HR) have been observed, the genetic contribution to athletic bradycardia has not been elucidated.

METHODS: We phenotyped current and former elite endurance athletes in the Pro@Heart cohort study using multimodal cardiac imaging, cardiopulmonary exercise testing, and Holter monitoring. Genetic susceptibility to bradycardia was assessed using a validated HR-associated polygenic risk score (HR-PRS), in which lower scores are associated with a lower HR, and compared with healthy nonathletic controls. Clinical and genetic features of bradycardic endurance athletes with minimum HR ≤40 bpm on a Holter monitor (bradycardic athletes [BAs]) were compared with non-BAs). A healthy cohort of nonathletes from the ASPREE study (Aspirin in Reducing Events in the Elderly) were used for genetic comparisons.

RESULTS: Among 465 endurance athletes (median age, 23 [18–49] years, 75% men), 175 (38%) had a minimum HR on a Holter monitor ≤40 bpm, of whom 7 (2% of total) had a HR ≤30bpm. Pauses ≥2 s were observed in 115 (25%) athletes, of whom 12 (3% of total) had pauses ≥3 s. Mobitz I second-degree atrioventricular block was observed in 15 (3% of total) athletes. BAs were younger and fitter and exhibited greater athletic cardiac remodeling than non-BAs. Mean HR-PRS was significantly lower in all athletes compared with ASPREE nonathletes (P<0.001) and in BAs compared with non-BAs (P=0.006). When the distribution of HR-PRS within our athletic cohort was considered, athletes with scores in the bottom quartile had a lower minimum HR (median HR, 41 [35–45] bpm versus 45 [40–49] bpm, P<0.001) and higher bradycardia burden (14 [2–37]% versus 2 [0%–25]%, P<0.001) than those with scores in the top quartile. After adjusting for age, sex, fitness, and indexed right atrial volume, HR-PRS was independently associated with lower minimum HR and increased the odds of resting bradycardia by 2-fold (odds ratio [OR], 2.2 [95% CI, 1.3–3.9]; P=0.004). Neither bradycardia nor pauses were associated with increased risk of adverse outcomes over 5.5 years.

CONCLUSIONS: Resting bradycardia (HR ≤40 bpm) and pauses of 2 to 3 s are present in a significant proportion of endurance athletes and are well tolerated. Our data suggest that both fitness and genetic variation contribute to sinus node function in endurance athletes. Intriguingly, HR-PRS differed between athletes and nonathletes, raising the possibility that genetics may be a determinant of athleticism.

Key Words: arrhythmias ■ athletes ■ AV block ■ bradycardia ■ diagnostic imaging ■ exercise ■ genetics ■ heart rate

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Clinical Perspective

What Is New?

- Genetic predisposition independently predicts bradycardia in endurance athletes.
- Genetic markers of bradycardia also influence the degree of cardiac remodeling in endurance athletes independent of baseline characteristics, fitness, or training load.

What Are the Clinical Implications?

- Future assessment of bradycardia and cardiac remodeling in endurance athletes may be individually tailored based on underlying genetic predisposition.
- Both fitness and genetic predisposition contribute to sinus node function in endurance athletes, and athleticism may, in part, be genetically determined.

Nonstandard Abbreviations and Acronyms

% Predicted VO_2peak	percentage of VO_2peak relative to predicted VO_2peak
AF	atrial fibrillation
ASPREE	Aspirin in Reducing Events in the Elderly
AV	atrioventricular
BA	bradycardic athlete
CMR	cardiac magnetic resonance
HR	heart rate
LA	left atrial
LV	left ventricular
OR	odds ratio
PPM	permanent pacemaker
PRS	polygenic risk score
RA	right atrial
RAV*i*	indexed right atrial volume
RV	right ventricular
SVT	supraventricular tachycardia
VO_2peak	Peak amount of oxygen the body can utilize per kg per minute

Sinus node remodeling is a well-recognized physiological adaptation to sustained endurance exercise training, with up to 80% of endurance athletes developing sinus bradycardia and up to one-third exhibiting pauses ≥2 s.[1,2] Bradycardic athletes have an increased risk of permanent pacemaker (PPM) implantation with advancing age, especially if there are concomitant sinus pauses or Mobitz I second-degree atrioventricular (AV) block.[3] Although low heart rates (HRs) are common in asymptomatic athletes,[2] criteria for differentiating physiological versus pathological HR changes are lacking, and threshold levels for predicting complications remain poorly defined.

Various mechanisms for resting bradycardia in athletes have been proposed, including increased vagal tone[4–6] and exercise-induced sinus node remodeling independent of autonomic modulation.[7–10] Emerging data from nonathletic populations suggest that genetic factors play a significant role in determining HR,[11,12] with heritability estimates ranging from ≈22% in family studies to as high as 65% in twin cohorts.[13–15] Whether genetic factors influence sinus node function and resting HR in endurance athletes is unknown. Understanding the genetic contribution to bradycardia in endurance athletes may help distinguish physiological adaptation from early pathological changes, enhance risk stratification, and potentially guide personalized clinical investigation and management.

In this study, we comprehensively evaluated clinical and genetic correlates of resting bradycardia in a cohort of current and former elite endurance athletes. Genetic susceptibility to bradycardia was assessed using a polygenic risk score validated for HR (HR-PRS).[16] Our findings provide novel insights into the prevalence and outcomes of resting bradycardia in endurance athletes and relationships between genetic risk, fitness, and structural cardiac remodeling.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Population

The study cohort comprised current and former endurance athletes from the Pro@Heart and ProAFHeart cohort studies, enrolled from April 2015 to March 2024. The Pro@Heart study protocol has been previously detailed.[17] The ProAFHeart study is a multicenter prospective trial aiming to determine the prevalence and 2-year incidence of arrhythmias and athletic remodeling in endurance athletes 16 to 81 years of age. Athletes were recruited individually or through their sports federation or team, who were made aware of the study through advertisements, media, and scientific presentations. Participants were enrolled to undergo study investigations at 1 of 5 medical research facilities using the same research protocol: (1) St Vincent's Institute of Medical Research (Melbourne, VIC, Australia), (2) Baker Heart and Diabetes Institute (Melbourne, VIC, Australia), (3) University Hospitals (Leuven, Belgium), (4) Jessa Ziekenhuis (Hasselt, Belgium), and (5) University Hospital Antwerp (Antwerp, Belgium).

Two groups of endurance athletes were included: (1) ostensibly healthy athletes sampled from the community, and (2) athletes referred to specialist sports cardiology clinics with known or suspected arrhythmias based on symptoms. The latter group was included specifically to facilitate identification of

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proarrhythmogenic risk factors. Athletes were eligible if they were ≥14 years of age and either actively competing or had formerly competed (for a minimum of 5 years) in endurance sports at a national or international level in which aerobic conditioning is a principal component of performance (eg, cycling, rowing, distance running ≥1500 m, swimming ≥400 m, and triathlon). Athletes taking any negatively chronotropic medication (beta-blockers, nondihydropyridine calcium channel antagonists, antiarrhythmic drugs, ivabradine, or digoxin), those with intracardiac devices, or those diagnosed with cardiomyopathies, pre-excitation, or myocardial infarction were excluded. Protocols (ProAFHeart: 484/16 and ACTRN12618000711213; Pro@Heart: 335/15 and ACTRN12618000716268 and S57241; URL: https://www.clinicaltrials.gov; Unique identifier: NCT05164328) were approved by the human research ethics committee at each of the recruiting sites in Australia and Belgium, and all participants provided informed written consent. We used the Strengthening of the Reporting of Observational Studies in Epidemiology cohort checklist[18] when writing our report (Supplemental Material).

Exercise History

All participants completed a questionnaire that incorporated type of sport, number of years exercising, and the frequency, duration, and intensity of exercise sessions. Each sport was assigned a metabolic equivalent task score from the Compendium of Physical Activities[19] based on self-reported level of performance (eg, recreational versus national competition) and intensity (low, moderate, or high). Endurance exercise volume (metabolic equivalent task hours per week) was calculated by multiplying the metabolic equivalent task score by the reported weekly exercise hours as reported previously.[20,21]

Cardiopulmonary Exercise Test

Cardiopulmonary exercise testing was conducted on an electronically braked cycle ergometer (Lode, Groningen, the Netherlands) using a standardized continuous ramp protocol comprising 1 minute at 50 W followed by a progressive increase of 30 W/min until volitional fatigue. The volume of oxygen uptake and respiratory exchange ratio were measured using an automated system with a paramagnetic oxygen analyzer and an infrared carbon dioxide analyzer after calibration against a standardized gas solution (Jaeger Vyntus CPX, Vyaire Medical, Mettawa, IL). Peak oxygen uptake (VO_2peak) was defined as a 30-s rolling average of the 6 highest 5-s oxygen consumption values, confirmed by (1) a plateau or dip in VO_2 or volitional fatigue with a respiratory exchange ratio >1.15. HR and rhythm were monitored continuously throughout exercise using a 12-lead ECG (Vyntus ECG 12-lead PC-ECG; Vyaire Medical, Mettawa, IL). Percentage of predicted VO_2peak was calculated by dividing the relative VO_2peak by age- and sex-predicted norms (using the FRIEND registry [Fitness Registry of Exercise National Database] equation).[22]

ECG and Holter Monitoring

All participants underwent resting ECG and 24-hour Holter monitoring. Athletes were instructed to perform normal physical activity, including training, during the Holter monitor acquisition period. All recordings were reviewed by 2 independent cardiologists. P wave terminal force in lead V1 was calculated by multiplying the amplitude (millivolts) by the duration (milliseconds) of the terminal negative portion of the P wave in V1 on ECG (25 mm/s and 10 mm/mV). Abnormal P wave terminal force in lead V1 was defined as ≥4 mV/ms according to current consensus.[23] Minimum HR was defined as the average lowest HR sustained for ≥30 s on a Holter monitor. Resting bradycardia was defined as a minimum HR on a Holter monitor ≤40 bpm, and this threshold was used to subdivide the cohort into bradycardic athletes (BAs) and nonbradycardic athletes (non-BAs). Bradycardia burden was defined as the percentage of time with HR <50 bpm divided by total analyzed time. Pauses were defined as prolongation of the R-R interval by ≥2 s, either because of a sinus pause (absence of a P wave and corresponding QRS complex) or AV block (P wave continuing at regular intervals with intermittent/complete block of conduction to the ventricles). Atrial fibrillation (AF) and supraventricular tachycardia (SVT) were defined as those lasting ≥30 s. Nonsustained SVT was defined as any atrial arrhythmia (including AF) lasting <30 s. Nonsustained ventricular tachycardia was defined as >3 consecutive ventricular beats >100 bpm and lasting <30 s. Any sustained arrhythmia diagnosed before enrollment or detected on baseline Holter monitoring was recorded. Arrhythmias diagnosed before enrollment were verified with review of ECG or telemetry traces. All nonsustained arrhythmias, including SVT and nonsustained ventricular tachycardia, were recorded from Holter monitors only. Fridericia's formula was used for QT interval correction because of its superior accuracy over Bazzett's formula in populations with a high prevalence of bradycardia, such as athletes.[24]

CMR Imaging

Cardiac magnetic resonance (CMR) was performed using a 1.5 T or 3.0 T magnetic resonance imaging scanner (Magnetom Aera 1.5T, Prisma 3.0T or Skyra 3.0T, Siemens Healthineers, Erlangen, Germany; Ingenia, Achieva, or Ambition 1.5T, Philips Medical Systems, Best, the Netherlands). A steady-state free precession dynamic echo-gradient sequence was used to obtain cine loops during breath-hold in short-axis and 4-chamber views. Left ventricular (LV) mass (not including papillary muscles and trabeculae) and biventricular volumes and function were quantified by 2 independent experienced cardiologists using customized analysis software (Circle Cardiovascular Imaging, cvi42, Calgary, AB, Canada and SuiteHEART, Neosoft, Pewaukee, WI), with good interobserver variability between labs having been reported previously.[25] Cardiac index was calculated using resting HR recorded during CMR and CMR-derived LV stroke volume.

Echocardiography

Two-dimensional transthoracic echocardiography was performed (Vivid E9 or E95 ultrasound system, GE Healthcare, Horten, Norway) to assess atrial volumes, atrial strain, LV global longitudinal strain and diastolic function using established Doppler parameters (peak E and A wave velocities, E/A ratio, tricuspid regurgitation flow velocity, and inferior vena cava diameter to estimate right atrial [RA] pressure) and tissue Doppler parameters (septal, lateral, and average E′ and E/E′). Echocardiographic images were analyzed at 1 of 2 core laboratory facilities, both of whom use the same software (EchoPAC, GE Healthcare, Horten, Norway) and methods.

Echocardiography and CMR measurements were indexed to body surface area when appropriate.

Genetic Analysis

Peripheral blood samples were collected and deoxyribonucleic acid extracted following standard protocols.[26] Genome-wide single-nucleotide variant genotyping was conducted using the Axiom Precision Medicine Diversity Array (v2.0; Thermo Fisher Scientific, CA) as described previously.[27] Quality control procedures were implemented according to manufacturer best practices using Analysis Power Tools and in-house pipelines. Variant imputation was performed using the Michigan Imputation Server, employing Minimac4 with the Haplotype Reference Consortium panel. Genomic risk analysis was conducted with PLINK (v1.9), using both genotyped and imputed variant data, aligned to the hg19 (GRCh37) reference genome. A previously reported HR-PRS (PGS catalog ID PGS001233)[16] was derived using a subset of 13 357 variants (of the original 14 455 published variants) that were represented in both the case and control data sets. In that derivation cohort, lower HR-PRSs were associated with lower HRs. Data from athletes were also compared with a healthy reference population of 12 815 individuals of European ancestry ≥70 years of age with no history of diagnosed cardiovascular disease enrolled into the ASPREE trial (Aspirin in Reducing Events in the Elderly; URL: https://www.clinicaltrials.gov; Unique identifier: NCT01038583).[28] The ASPREE study population was genotyped using the same array and methods as the athletic cohort.

We performed 2 types of HR-PRS analyses in this study. First, we determined the proportion of athletes in our cohort with "low" HR-PRS, which was defined according to the bottom quartile of values in the ASPREE controls. Second, to examine the effects of HR-PRS on clinical parameters, we ranked the distribution of HR-PRSs within the athletic cohort and compared characteristics of athletes in the top versus bottom quartiles, quintiles, and deciles. These comparisons were performed independent of the ASPREE control group.

Follow-Up

Athletes in our cohort were followed up with a questionnaire ≈5 years after enrollment. This focused on the incidence of adverse outcomes related to bradycardia and pauses, including syncope, arrhythmias (eg, AF), implantation of cardiac devices (eg, PPM), cerebrovascular events, and sudden cardiac death. Any events were verified with review of ECGs, telemetry, device traces, or hospital/written records.

Statistical Analyses

Data were collected and managed using REDCap and analyzed with SPSS version 29 (IBM Corp, Armonk, NY). Data distribution and normality were tested using the Shapiro-Wilk test. Continuous variables are presented as means (±SD) or as medians [interquartile range; 25th–75th percentile]. Between-group differences in continuous variables were assessed using independent *t* test or Mann-Whitney *U* test as appropriate. Dichotomous variables were compared using a χ^2 or Fisher exact test. Multivariable logistic regression, linear regression, and ANCOVA were used to examine the associations between minimum HR (modeled as both a dichotomous and continuous variable), HR-PRS, and potential confounders, including age, sex, indexed RA volume (RAV*i*), and fitness (percentage of predicted VO_2peak). Continuous covariates were standardized (*Z* scores) before entry into regression and ANCOVA models to facilitate comparison of effect sizes. When data were not available for key variables, complete-case analyses were performed. No data imputation was undertaken. Two-tailed $P<0.05$ was considered statistically significant. Given the number of preplanned pairwise comparisons by *t* tests, adjustment for the volume of comparisons was considered; however, unadjusted *P* values were retained because: (1) many of the comparisons were derived from prespecified mechanistic hypotheses about bradycardia, cardiac remodeling, and genetic risk and were subsequently tested in adjusted models; (2) the inferential conclusions of the study are based on prespecified multivariable models; and (3) many of the multiple tests probe the same underlying construct of athletic remodeling, which are physiologically associated and statistically collinear with minimum HR. Application of family-wise procedures (eg, Bonferroni-type adjustments) in this context would be overly conservative, inflate type II error, and risk obscuring biologically meaningful patterns.

RESULTS

A total of 465 current and former endurance athletes 14 to 81 years of age were investigated (Figure S1). Two hundred forty-two (52% of the cohort, 77% men) were current elite athletes <25 years of age, 83 (18% of the cohort, 68% men) were current lifelong athletes 25 to 45 years of age, and 140 (30% of the cohort, 75% men) were current and former master athletes >45 years of age. Four hundred (86%) were ostensibly healthy athletes recruited from the community, and 65 (14%) were referred with known or suspected arrhythmias on the basis of symptoms. The median age of athletes was 23 [18–49] years, 348 (75%) were men, and 463 (99.6%) were of self-reported European ancestry. Athletes predominately competed in cycling (37%), rowing (34%), running (16%), or triathlon (10%). All athletes achieved a respiratory exchange ratio >1.15, and a plateau in VO_2peak was observed at maximal exercise in 460 (99%). Twenty-two (5%) athletes had hypertension, 40 (9%) dyslipidemia, and 2 (0.4%) diabetes, and 45 (10%) were ex-smokers. Before enrollment, 60 (13%) athletes had prevalent AF, with 43 (72%) from the group recruited with known or suspected arrhythmias on the basis of symptoms. Fourteen (3%) athletes had pre-existing SVT and 3 (1%) ventricular tachycardia. All cases of ventricular tachycardia were outflow tract tachycardia in structurally normal hearts and were successfully ablated. Twenty-two (5%) athletes were taking antihypertensive medications, 29 (6%) statins, 14 (3%) antiplatelet medications, and 9 (2%) oral anticoagulants. Thirty (6%) athletes reported previous syncope, with recurrent episodes in 4 individuals and reflex syncope/orthostatic hypotension in 29.

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One athlete reported 2 episodes of arrhythmogenic syncope related to outflow tract ventricular tachycardia before successful ablation.

Baseline Characteristics and Exercise History

On Holter monitoring, 175 (38%) endurance athletes had a minimum HR ≤40 bpm (BAs), and 290 (62%) had a minimum HR >40 bpm (non-BAs). Of the total cohort, only 7 (2%) athletes had a minimum HR ≤30 bpm. Pauses ≥2 s were observed in 115 (25%) athletes, and whom pauses ≥3 s were observed in only 12 (3%) of the total cohort. In athletes with pauses, the median duration was 2.3 s (2.1–2.7 s), with the longest an asymptomatic nocturnal sinus pause of 5.4 s in a 19-year-old cyclist. Pauses were predominately sinus pauses (96%), and all pauses and incidences of AV block were asymptomatic and recorded overnight. Mobitz I second-degree AV block was observed in 15 (3%) of the total cohort. No athletes had Mobitz II second-degree or complete AV block.

Clinical characteristics and exercise history comparing BAs and non-BAs are displayed in Table 1. BAs were more likely to be men, younger, leaner, and fitter and exercised at higher volumes (metabolic equivalent task hours per week) than non-BAs. Compared with BAs, non-BAs had a greater prevalence of hypertension and dyslipidemia. There were no differences between the groups in the prevalence of syncope or arrhythmias. There was no difference in the proportion of BAs and non-BAs who were recruited from the group with known or suspected arrhythmias on the basis of symptoms (13% versus 15%, P=0.686).

ECG and Holter Monitoring

Electrocardiography and Holter monitoring findings are shown in Table 2. ECG parameters were similar between the groups. BAs tended to have a higher prevalence of abnormal P wave terminal force in lead V1 (≥4 mV/ms), although this did not reach statistical significance (P=0.056). On Holter monitoring, BAs had a significantly higher bradycardia burden (minimum HR <50 bpm) and a greater prevalence of pauses and Mobitz I second-degree AV block compared with non-BAs. Overall, there was no difference between the groups in atrial and ventricular ectopic burden or the prevalence of incident arrhythmias, except nonsustained SVT, which was more common in non-BAs.

Cardiac Imaging

Table 3 shows cardiac imaging results. On transthoracic echocardiography, BAs had a higher E/A ratio, higher average E′, larger indexed RA and left atrial (LA) volumes, higher conduit LA strain, and lower contractile LA strain than non-BAs. There was no difference between the groups in E/E′, estimated systolic pulmonary artery systolic pressure, LA reservoir strain, or average LV global longitudinal strain. On CMR, BAs had larger LV and right ventricular (RV) volumes than non-BAs, with no difference in LV and RV ejection fractions. Cardiac index was also comparable between the groups (P=0.553). LV and RV end-diastolic volumes indexed to body surface area had moderate negative correlations with minimum HR (LV end-diastolic volumes indexed to body surface area: r=–0.549, P<0.001; RV end-diastolic volumes indexed to body surface area: r=–0.523, P<0.001).

Genetics

Mean HR-PRS was significantly lower in athletes when compared with ASPREE controls (2.80±2.72 versus 3.43±2.69, P<0.001), as illustrated in Figure 1. Moreover, there was a disproportionately greater number of athletes in our cohort with "low" HR-PRS (defined by scores in the bottom quartile of ASPREE controls; 34% versus 25%, P<0.001), and relatively fewer athletes with a "high" HR-PRS (defined by scores in the top quartile of ASPREE controls; 18% versus 25%, P<0.001). As shown in Figure 2, mean HR-PRS in BAs was significantly lower compared with both non-BAs (P=0.006) and ASPREE controls (P<0.001). In addition, more BAs had "low" HR-PRS (defined by scores in the bottom quartile of ASPREE controls) compared with non-BAs (42% versus 30%, P=0.010). There were no differences in mean HR-PRS (2.78±2.72 versus 2.82±2.73, P=0.860) or the proportion of athletes with "low" HR-PRS (34% versus 35%, P=0.833) when young (<20 years of age, n=192) and older (>20 years of age, n=273) athletes were considered.

To understand the effects of HR-PRS on clinical parameters, the distribution of HR-PRSs within our athletic cohort was ranked (independent of ASPREE controls), and comparisons were made between the 116 (25%) athletes with scores in the top quartile and the 116 (25%) athletes with scores in the bottom quartile (Table 4). Baseline characteristics and exercise history were similar between these groups; however, athletes ranked in the bottom quartile for HR-PRS had a significantly lower minimum HR (P<0.001), significantly higher bradycardia burden (P<0.001), and a higher prevalence of pauses ≥2 s (P=0.009) compared with those ranked in the top quartile. There were no differences between these groups in the prevalence of Mobitz I second-degree AV block, SVT, or AF. Athletes with HR-PRSs ranked in the bottom quartile also had significantly larger atrial and ventricular volumes compared with those with HR-PRSs ranked in the top quartile. Similar results were obtained when comparing the 46 (10%) athletes ranked in the bottom decile for HR-PRS versus the 46 (10%) athletes ranked in the top decile (Table S1).

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Table 1. Baseline Characteristics and Exercise History in Athletes With and Without Resting Bradycardia

	HR ≤40 (n=175)	HR >40 (n=290)	*P* value
Age, y	21 [18–39]	32 [18–56]	<0.001
Men, n (%)	143 (82)	205 (71)	0.008
European ancestry, n (%)	174 (99)	289 (99)	1.000
Height, cm	179±8	179±8	0.524
Weight, kg	71 [64–78]	72 [65–82]	0.029
BMI, kg/m^2	22 [20–24]	23 [21–25]	<0.001
BSA, m^2	1.87 [1.76–1.99]	1.89 [1.78–2.03]	0.100
SBP, mm Hg	126 [116–135]	126 [117–135]	0.930
DBP, mm Hg	65 [59–73]	69 [62–77]	0.001
Sport, n (%)			
Cycling	78 (45)	95 (33)	0.011
Running	36 (21)	37 (13)	0.025
Rowing	35 (20)	121 (42)	<0.001
Triathlon	24 (14)	24 (8)	0.062
Swimming	2 (1)	7 (2)	0.317
Cross-country skiing	0 (0)	6 (2)	0.055
Exercise duration, y*	7 [3–20]	11 [4–26]	0.012
Exercise volume, MET h/week*	86 [60–106]	74 [48–99]	0.001
CPET†			
VO_2peak, mL/min per kg	60 [52–66]	50 [38–62]	<0.001
Percent predicted VO_2peak‡	131±17	124±20	<0.001
Power, W	417 [333–474]	349 [294–411]	<0.001
Maximum HR, bpm	182 [173–192]	183 [169–192]	0.977
Peak SBP, mm Hg	218 [195–237]	210 [194–230]	0.027
Peak DBP, mm Hg	82 [74–92]	84 [74–91]	0.505
Comorbidities & Medication, n (%)			
HTN	12 (7)	3 (1)	0.002
Dyslipidemia	19 (11)	14 (5)	0.012
Smoking history	21 (12)	17 (6)	0.020
Antihypertensive medication	12 (7)	6 (2)	0.011
Lipid-lowering medication	14 (8)	9 (3)	0.013
Syncope and arrhythmias, n (%)			
Syncope			
Reflex/orthostatic	9 (5)	20 (7)	0.449
Arrhythmogenic	0 (0)	1 (1)	0.331
Prevalent AF	19 (11)	41 (14)	0.307
Prevalent SVT (not AF)	8 (5)	6 (2)	0.134
VT	1 (1)	2 (1)	0.876
Cardiac arrest	0 (0)	0 (0)	…

Values are median (25th–75th percentile), mean±SD, or n (%). Unadjusted *P* values are reported. Antihypertensive medications are angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, angiotensin receptor-neprilysin inhibitors, nondihydropyridine calcium channel antagonists, or thiazide diuretics. Lipid-lowering medications are statins or ezetimibe. BMI indicates body mass index; BSA, body surface area (Mosteller); CPET, cardiopulmonary exercise test; DBP, diastolic blood pressure; HTN, hypertension; HR, heart rate; MET, metabolic equivalent task; SBP, systolic blood pressure; and VO_2peak, peak amount of oxygen the body can use per kilogram per minute.

*Missing exercise history data in 72 (HR≤40: 33; HR>40: 39).

†Missing cardiopulmonary exercise test data in 32 (HR≤40: 21; HR>40: 11).

‡Percentage of VO_2peak relative to predicted VO_2peak (derived from the FRIEND registry [Fitness Registry and the Importance of Exercise National Database][22]).

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Table 2. ECG and Holter Monitoring in Athletes With and Without Resting Bradycardia

ECG	HR ≤40 (n=175)	HR >40 (n=290)	*P* value
HR, bpm	46 [40–53]	55 [50–60]	<0.001
P wave duration, ms	112 [102–121]	112 [100–122]	0.977
P wave axis, degrees	55 [36–66]	55 [36–66]	0.794
PTFV1, mV/ms	1.87 [0.00–3.34]	1.89 [0.00–2.88]	0.641
PTFV1 ≥4 mV/ms, n (%)	25 (17)	25 (9)	0.056
Advanced interatrial block, n (%)	6 (4)	20 (7)	0.182
PR interval, ms	165 [147–190]	167 [146–186]	0.490
QRS duration, ms	100 [94–105]	100 [94–106]	0.703
QTc (Fridericia), ms	409 [395–425]	412 [400–425]	0.344
Abnormal TWI*	2 (1)	9 (3)	0.214
24-h Holter monitor			
Minimum HR, bpm	37 [34–39]	46 [43–49]	<0.001
Average HR, bpm	60 [55–66]	69 [64–75]	<0.001
Maximum HR, bpm	151 [126–168]	153 [129–170]	0.619
Bradycardia burden, n (%)	33 [23–43]	2 [0–9]	<0.001
Pauses, n (%)			
≥2s	78 (45)	37 (13)	<0.001
≥2.5s	30 (17)	12 (4)	<0.001
≥3s	10 (6)	2 (1)	<0.001
AV block			
Mobitz I, n (%)	10 (6)	5 (2)	0.021
Mobitz II, n (%)	0 (0)	0 (0)	…
Complete, n (%)	0 (0)	0 (0)	…
PAC/24 h	10 [3–38]	12 [4–45]	0.487
Nonsustained SVT, n (%)	8 (5)	41 (14)	0.001
SVT, n (%)	0 (0)	3 (1)	0.092
AF, n (%)	6 (3)	6 (2)	0.378
PVC/24 h	2 [0–9]	2 [0–17]	0.053
NSVT, n (%)	6 (3)	8 (3)	0.684

Values are median (25th–75th percentile), mean±SD, or n (%). AF indicates atrial fibrillation; AV, atrioventricular; deg, degrees; HR, heart rate; NSVT, nonsustained ventricular tachycardia; PAC, premature atrial complex; PTFV1, P wave terminal force in lead V1; PVC, premature ventricular complex; QTc, corrected QT interval using Fridericia's formula; SVT, supraventricular tachycardia, and TWI, T wave inversion.

*According to international guidelines: ≥1 mm in depth in ≥2 contiguous leads (excluding aVR, III, and V1) in the absence of incomplete right bundle branch block (RBBB) and complete RBBB/left bundle branch block.[55]

Table 3. Cardiac Imaging in Athletes With and Without Resting Bradycardia

	HR ≤40 (n=175)	HR >40 (n=290)	*P* value
Transthoracic echocardiogram			
E/A ratio	2.1 [1.6–2.6]	1.7 [1.2–2.2]	<0.001
Average E′, cm/s	15 [12–17]	13 [10–16]	0.002
E/E′	5.4 [4.7–6.1]	5.5 [4.8–6.5]	0.459
PAPs, mm Hg	22 [19–26]	22 [19–24]	0.832
RAV*i*, mL/m²	37 [26–46]	30 [23–38]	<0.001
LAV*i*, mL/m²	45 [40–53]	41 [34–48]	<0.001
LV GLS, %	−19 [−17 to −20]	−19 [−17 to −21]	0.187
LA Strain*			
Reservoir, %	32±7	31±6	0.317
Conduit, %	22±7	19±7	0.002
Contractile, %	10 [8–13]	12 [10–15]	0.003
Cardiac magnetic resonance†			
LVEDV*i*, mL/m²	120±19	102±19	<0.001
LVESV*i*, mL/m²	51 [43–61]	43 [37–52]	<0.001
LVSV*i*, mL/m²	68±12	58±11	<0.001
LVEF, %	58 [54–61]	56 [52–61]	0.170
LVM*i*, g/m²	79±15	67±14	<0.001
RVEDV*i*, mL/m²	134±23	115±21	<0.001
RVESV*i*, mL/m²	66±16	57±13	<0.001
RVSV*i*, mL/m²	67±12	58±12	<0.001
RVEF %	51±7	51±6	0.907
CI, L/min/m²	3.2 [2.7–3.8]	3.2 [2.6–3.6]	0.553

Values are median (25th–75th percentile), mean±SD, or n (%). CI indicates cardiac index; CMR, cardiac magnetic resonance; LA, left atrium; LAV*i*, left atrial volume indexed to body surface area; LV GLS, left ventricular average global longitudinal strain; LVEDV*i*, left ventricular end-diastolic volume indexed to body surface area; LVEF, left ventricular ejection fraction; LVESV*i*, left ventricular end-systolic volume indexed to body surface area; LVM*i*, left ventricular mass indexed to body surface area; LVSV*i*, left ventricular stroke volume indexed to body surface area; PAPs, estimated systolic pulmonary artery pressure; RAV*i*, right atrial volume indexed to body surface area; RVEF, right ventricular ejection fraction; RVEDV*i*, right ventricular end-diastolic volume indexed to body surface area; RVESV*i*, right ventricular end-systolic volume indexed to body surface area; RVSV*i*, right ventricular stroke volume indexed to body surface area; and TTE, transthoracic echocardiogram.

*Missing left atrial strain data in 173 (HR ≤40, 82; HR >40, 91).

†Missing cardiac magnetic resonance data in 40 (HR ≤40, 13; HR >40, 27).

Predictors of Minimum HR

Univariable and multivariable logistic regression analyses examining predictors of resting bradycardia are presented in Table 5. In univariable analysis, "low" HR-PRSs (defined as scores in the bottom quartile of ASPREE controls) were associated with 1.7-fold increased odds of resting bradycardia (odds ratio [OR], 1.67 [95% CI, 1.13–2.47]; *P*=0.010). When considering HR-PRS distributions within the athletic cohort alone (independent of ASPREE controls), those with scores ranked in the bottom quartile had 2.2-fold higher odds of resting bradycardia compared with those with scores ranked in the top quartile (OR, 2.23 [95% CI, 1.29–3.86]; *P*=0.004). Similar results were obtained when comparisons were made between athletes with scores ranked in the bottom versus top quintiles (*P*=0.003) and deciles (*P*=0.042). In multivariable models adjusted for age, sex, fitness, and RAV*i*, HR-PRS remained a significant predictor of resting bradycardia. Model 1 (n=213, Nagelkerke R^2=0.185, Hosmer-Lemeshow *P*=0.836) demonstrated that athletes with HR-PRSs ranked in the

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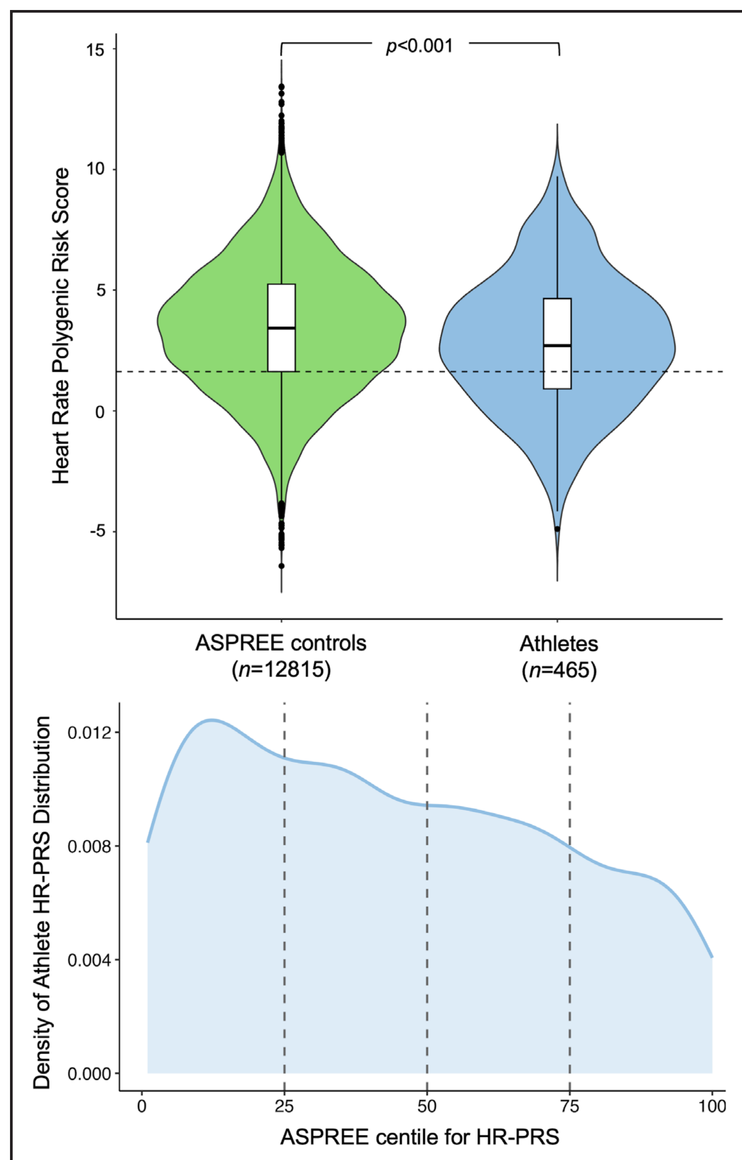

Figure 1. HR-PRS in athletes and ASPREE controls.
Top, Violin plots of heart rate–associated polygenic risk scores (HR-PRSs) in ASPREE (Aspirin in Reducing Events in the Elderly) controls and all athletes. The dotted trend line indicates low HR-PRS, which is defined as an HR-PRS in the lower quartile of ASPREE controls. **Bottom**, Kernel density plot showing the density of athlete HR-PRS distribution (*y* axis) plotted according to their position within the ASPREE control centile range (*x* axis). Dashed vertical lines indicate the 25th, 50th, and 75th percentile of the ASPREE distribution.

bottom quartile (independent of ASPREE controls) had almost 2-fold higher odds of resting bradycardia than those ranked with HR-PRSs in the top quartile (OR, 1.93 [95% CI, 1.03–3.63]; P=0.041). In Model 2 (n=186, Nagelkerke R^2=0.200, Hosmer-Lemeshow P=0.906), athletes with HR-PRSs ranked in the bottom quintile (independent of ASPREE controls) had 2.5-fold higher odds of resting bradycardia compared with those with HR-PRSs ranked in the top quintile (OR, 2.47 [95% CI, 1.19–5.15]; P=0.016). Notably, LA volumes did not predict resting bradycardia, and HR-PRS did not predict arrhythmia occurrence on multivariable analyses.

In multivariable linear regression with minimum HR as the dependent variable and age, sex, fitness (percent predicted VO_2peak), RAV*i*, and HR-PRS (lowest quartile) as independent variables, all predictors showed independent significant associations with minimum HR (F(5416)=20.36, P<0.001, R^2=0.187). As shown in Figure 3, which presents standardized coefficients (per 1 SD increase) with 95% CIs, the largest effect was for sex, followed by HR-PRS and age. An ANCOVA of the same model provided consistent findings, with significant independent associations for age (F=38.31, P<0.001, η^2=0.094), sex (F=14.96, P<0.001, η^2=0.020), RAV*i* (F=26.84, P<0.001, η^2=0.050), fitness (F=6.40, P=0.012, η^2=0.017), and HR-PRS (F=15.28, P<0.001, η^2=0.035). There were no significant interactions between HR-PRS and age (P=0.921), fitness (P=0.635), or RAV*i* (P=0.736).

Extreme Bradycardia

To evaluate more severe bradycardia thresholds, we undertook a supplemental analysis of 61 (13%) athletes who had a minimum HR on a Holter monitor ≤35 bpm (Table S2). This cohort demonstrated similar characteristics to the primary analysis but exhibited even lower mean HR-PRSs, with greater representation in the

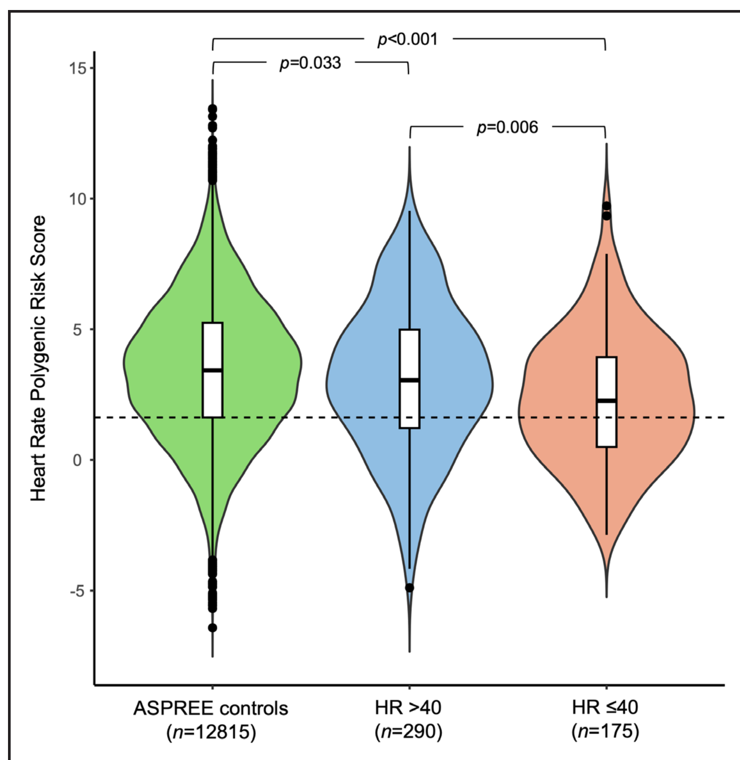

Figure 2. HR-PRS in bradycardic and nonbradycardic athletes.
Shown are violin plots of heart rate–associated polygenic risk scores (HR-PRSs) in ASPREE (Aspirin in Reducing Events in the Elderly) controls, bradycardic athletes (minimum HR ≤40 bpm), and nonbradycardic athletes (minimum HR >40 bpm). The dotted trend line indicates a low HR-PRS, which is defined as an HR-PRS in the lower quartile of ASPREE controls.

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bottom quartile, quintile, and decile of ASPREE controls. Seven athletes (2%) exhibited a minimum HR ≤30 bpm, with a young male predominance (6 of 7 men; all <20 years of age; 3 cyclists, 2 runners, and one rower), and the outlier was a 63-year-old female former rower. Six of these athletes had multiple asymptomatic nocturnal sinus pauses between 2 and 3 s, and 3 had Mobitz I second-degree AV block.

Follow-Up

Athletes were followed for a median of 5.5 (4.3–6.5) years after enrollment. Seven athletes reported syncope, with 6 experiencing reflex syncope or orthostatic hypotension. One 17-year-old male athlete (minimum HR 46 bpm on a Holter monitor, ranked in the bottom quartile among athletes for HR-PRS) reported syncope after a sustained period of tachycardia during a cycling race, ≈10 months after study enrollment. Investigations revealed the athlete's heart to be without myocardial scar on CMR. An implanted loop recorder has detected no arrhythmias to date, with ≈2.5 years of follow-up since implantation. One 76-year-old former rower (minimum HR 49 bpm on a Holter monitor, ranked in the bottom quartile among athletes for HR-PRS) had a PPM implanted for symptomatic sinus node dysfunction. Twelve athletes had newly diagnosed AF, with 3 BAs (minimum HR 31, 34, and 40 bpm) and 9 non-BAs (minimum HR ranging from 42 to 52 bpm). Among the 7 athletes with minimum HR ≤30 bpm on a Holter monitor, none had syncope, PPM implantation, or any other adverse outcomes over the follow-up period.

DISCUSSION

Sinus bradycardia is a well-recognized component of the cardiac remodeling commonly observed in endurance athletes, but the limits of normality are ill defined. In this study, using comprehensive multimodal imaging combined with ECG, Holter monitoring, cardiopulmonary exercise testing, and genetic analysis, we found that: (1) extreme bradycardia (resting HR ≤30 bpm), pauses ≥3 s, and Mobitz I second-degree AV block are uncommon, even in extreme endurance athletes with elite fitness; (2) bradycardia is associated with both greater fitness and genetic predisposition; and (3) resting bradycardia in endurance athletes does not appear to be associated with adverse outcomes over the short term. We also observed a significant difference in HR-PRS between athletes and nonathletes, with individuals genetically predisposed to bradycardia disproportionately represented among our athletic cohort. This raises the intriguing possibility that, in addition to acquired determinants of athletic conditioning, an inherited tendency toward a lower HR may also contribute to performance.

Prevalence and Outcomes of Bradycardia

Prevalence estimates of athletic bradycardia and heart block have varied greatly, and there is uncertainty as

Table 4. Comparison of Clinical Characteristics Between the Bottom vs Top Quartiles of Heart Rate–Associated Polygenic Risk Score Within the Athlete Cohort

	Bottom HR-PRS quartile (n=116)	Top HR-PRS quartile (n=116)	*P* value
Age, y	23 [17–44]	23 [18–52]	0.718
Men, n (%)	82 (71)	83 (73)	0.661
European ancestry, n (%)	116 (100)	115 (99)	1.000
BMI, kg/m²	23±3	23±3	0.897
BSA, m²	1.90±0.19	1.89±0.20	0.934
Exercise duration, y	10 [3–20]	10 [3–21]	0.970
Exercise volume, MET h/week	76 [54–97]	85 [57–101]	0.362
VO_2peak, mL/min per kg	55 [41–63]	55 [40–62]	0.823
Percent predicted VO_2peak†	126±21	124±19	0.384
24-h Holter monitor			
Minimum HR, bpm	41 [35–45]	45 [40–49]	<0.001
Average HR, bpm	64±9	69±9	<0.001
Bradycardia burden, n (%)	14 [2–37]	2 [0–25]	<0.001
Pauses ≥2 s, n (%)	42 (36)	24 (21)	0.009
Mobitz I AV block, n (%)	1 (1)	3 (3)	0.302
SVT, n (%)*	6 (5)	3 (3)	0.303
AF, n (%)*	17 (15)	13 (11)	0.433
TTE			
RAV*i*, mL/m²	33 [27–45]	28 [23–38]	0.002
LAV*i*, mL/m²	45 [39–51]	41 [32–49]	<0.001
CMR			
LVEDV*i*, mL/m²	112±20	104±23	0.007
LVEF, %	58±6	57±6	0.100
RVEDV*i*, mL/m²	126±24	116±27	0.003
RVEF %	52±6	51±6	0.061

Values are median (25th–75th percentile), mean±SD, or n (%). AF indicates atrial fibrillation; AV, atrioventricular; BMI, body mass index; BSA, body surface area; HR, heart rate; LAV*i*, left atrial volume indexed to body surface area; LVEDV*i*, left ventricular end-diastolic volume indexed to body surface area; LVEF, left ventricular ejection fraction; MET, metabolic equivalent task; PRS, polygenic risk score; RAV*i*, right atrial volume indexed to body surface area; RVEDV*i*, right ventricular end-diastolic volume indexed to body surface area; RVEF, right ventricular ejection fraction; SVT, supraventricular tachycardia; and VO_2peak, peak amount of oxygen the body can use per kilogram per minute.

*SVT and AF include prevalent and incident arrhythmias.

†Percentage of VO_2peak relative to predicted VO_2peak (derived from the FRIEND registry [Fitness Registry and the Importance of Exercise National Database][22]).

Table 5. Logistic Regression for Resting Bradycardia

Univariable	OR	95% CI	*P* value
Age, y	0.98	0.97–0.99	<0.001
Sex, male	1.85	1.17–2.93	0.008
BMI, kg/m²	0.88	0.82–0.94	<0.001
Exercise duration, y	0.98	0.96–0.99	0.005
Exercise volume, MET h/week	1.01	1.01–1.02	<0.001
Fitness, % predicted VO_2peak†	1.02	1.01–1.03	<0.001
RAV*i*, mL/m²	1.03	1.01–1.04	<0.001
LAV*i*, mL/m²	1.04	1.02–1.06	<0.001
RVEDV*i*, mL/m²	1.04	1.03–1.05	<0.001
LVEDV*i*, mL/m²	1.05	1.04–1.07	<0.001
Bottom vs top HR-PRS quartiles*	2.23	1.29–3.86	0.004
Bottom vs top HR-PRS quintiles*	2.62	1.40–4.92	0.003
Bottom vs top HR-PRS deciles*	2.64	1.03–6.76	0.042
Model 1: age, sex, % predicted VO_2peak,† RAV*i*, bottom vs top HR-PRS quartiles			
Age, y	0.97	0.96–0.99	0.003
Sex, male	1.78	0.85–3.70	0.125
Fitness (% predicted VO_2peak)†	1.02	1.00–1.04	0.016
RAV*i*, mL/m²	1.03	1.01–1.06	0.012
Bottom vs top HR-PRS quartiles*	1.93	1.03–3.63	0.041
Model 2: age, sex, % predicted VO_2peak,† RAV*i*, bottom vs top HR-PRS quintiles			
Age, y	0.97	0.95–0.99	0.006
Sex, male	2.19	0.92–5.21	0.075
Fitness (% predicted VO_2peak)†	1.02	1.01–1.05	0.016
RAV*i*, mL/m²	1.02	0.99–1.05	0.148
Bottom vs top HR-PRS quintiles*	2.47	1.19–5.15	0.016

BMI indicates body mass index; HR-PRS, heart rate–associated polygenic risk score; LAV*i*, left atrial volume indexed to body surface area; LVEDV*i*, left ventricular end-diastolic volume indexed to body surface area; MET, metabolic equivalent task; OR, odds ratio; RAV*i*, right atrial volume indexed to body surface area; RVEDV*i*, right ventricular end-diastolic volume indexed to body surface area.

*Refers to HR-PRS distribution within the athletic cohort (independent of ASPREE controls).

†Percentage of VO_2peak relative to predicted VO_2peak (derived from the FRIEND registry [Fitness Registry and the Importance of Exercise National Database][22]).

to what threshold of resting HR should be considered sufficiently extreme to warrant consideration of underlying pathology.[1,29,30] Although HRs <35 bpm during waking hours are less common,[31] extreme endurance athletes have been reported to exhibit HRs ≤30 bpm during sleep, often associated with junctional rhythms, sinus pauses, and AV block.[32] According to the 2018 American College of Cardiology/American Heart Association/Heart Rhythm Society guidelines focusing on the evaluation of bradycardia in the general population,[33] in the absence of symptoms or suspicion of structural heart disease, reassurance is appropriate for any degree of sinus bradycardia. On the other hand, athlete-specific guidelines have suggested that further evaluation may be appropriate for an HR <30 bpm regardless of symptoms,[2,34–36] but to date, there is no clear evidence linking sinus bradycardia with adverse clinical events. Large data registries have raised some concern by demonstrating that male endurance athletes have a higher risk of PPM implantation compared with nonathletes, with sinus node dysfunction the main indication.[37]

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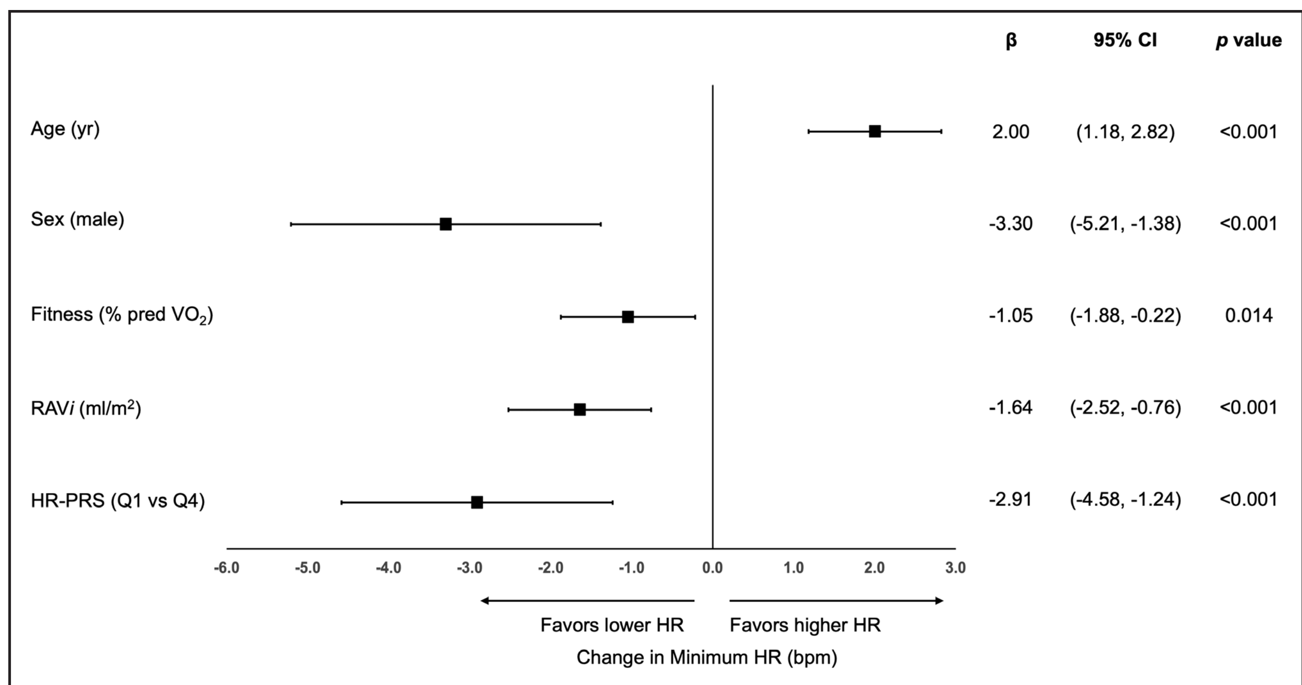

Figure 3. Predictors of minimum heart rate.
A forest plot shows standardized effect sizes from multivariable linear regression analysis, with minimum heart rate (HR) as a continuous variable. HR-PRS indicates heart rate–associated polygenic risk score; RAV*i*, right atrial volume indexed to body surface area; and % pred VO_2, percentage of VO_2peak relative to predicted VO_2peak (derived from the FRIEND registry [Fitness Registry and the Importance of Exercise National Database][22]).

In contrast to previous studies,[32] we observed a low prevalence of endurance athletes with HR ≤30 bpm, even during sleep. Both current and former athletes across a broad age range were studied, and this may have contributed to the lower-than-expected prevalence of "extreme" bradycardia. However, 52% of the athletes in our cohort were young elite athletes competing at national or international levels of competitive sports. To some extent, the low prevalence of extreme bradycardia may be attributable to our rigorous definition of minimum HR on Holter monitoring, which averaged the lowest HR over ≥30 s. By incorporating such a definition, we eliminated artificially low minimum HRs from transient events like isolated sinus pauses or postectopic or postreversion pauses.

Prevalence and Outcomes of Pauses

In addition to a 38% prevalence of resting bradycardia (HR ≤40 bpm), we found that approximately one-quarter of athletes exhibited pauses ≥2 s. However, parallelling our observations for HRs ≤30 bpm, the prevalence of pauses ≥3 s was low (only 3% of the total cohort). Such findings are consistent with previous studies that have demonstrated that pauses ≥2 s are common, whereas pauses ≥3 s are much less common.[38–40] In keeping with previous reports,[38,41] pauses in our cohort were not associated with adverse outcomes such as syncope, PPM implantation, or tachyarrhythmias. In addition, we observed a low overall prevalence of Mobitz I second-degree AV block (also only 3% of the total cohort). Although Mobitz I second-degree AV block was more common in BAs, our data challenge the notion that Mobitz I second-degree AV block "commonly" accompanies resting bradycardia, even overnight during periods of high vagal modulation. Importantly, none of our athletes had Mobitz II second-degree or complete AV block, reinforcing the current consensus that those are not abnormalities associated with endurance training.

Clinical Associations With Bradycardia in Endurance Athletes

The mechanisms underpinning sinus bradycardia in athletes remain unclear. Although often attributed to enhanced vagal tone,[4–6,42] studies using dual autonomic blockade have demonstrated that the intrinsic HR is lower in athletes and that mechanosensitive ion channels within the atria may be a more important determinant of HR.[7–10] We found that clinical factors such as younger age, male sex, greater fitness, and larger RAV*i* independently predicted a lower HR. The effect of age on resting HR in athletes remains controversial, with some studies associating lower HRs with higher parasympathetic activity in younger, fitter athletes,[43] whereas others report increased bradycardia prevalence with age and prolonged training.[44,45] Our study is not well suited to isolating the effect of age given differences in fitness

and training status between the younger and older athletes. Consistent with our findings, previous studies have demonstrated that male athletes exhibit lower resting HRs and a higher prevalence of bradycardia (<50/min) than women.[46,47] Furthermore, body mass index was lower in BAs, consistent with previous reports of an association between lean body composition and bradycardia.[48] However, there is significant collinearity between body composition, age, and sex that makes interpretation challenging.

Unsurprisingly, there was a strong association between fitness and bradycardia. BAs were fitter and exhibited greater athletic cardiac remodeling and superior diastolic function compared with non-BAs. Consistent with previous studies,[42] we found moderate negative correlations between minimum HR and ventricular volumes. We also found an association between atrial volumes and bradycardia. Interestingly, RA volumes, rather than LA volumes, remained an independent predictor of bradycardia. This supports various mechanistic links with stretch-related remodeling of the sinoatrial node.[7,10]

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Is Bradycardia in Endurance Athletes Genetically Predetermined?

There is accumulating evidence that genetic variation is a key determinant of HR. In addition to rare pathogenic variants associated with familial forms of sinus node dysfunction,[49] genome-wide association studies in large case-control series have also identified >400 chromosomal loci associated with HR from which HR-PRSs have been derived, allowing individual-level assessment of genetic susceptibility.[16,50] To our knowledge, our study is the first to demonstrate a genetic contribution to bradycardia in endurance athletes. Athletes with resting bradycardia had a significantly lower mean HR-PRS than non-BAs. The strongest associations between a given PRS and a clinical trait are typically seen for the tails of the distribution curves. In accordance with this, we found that the proportion of BAs increased when progressing from the bottom quartile, quintile, and decile of HR-PRSs. This "bottom-heavy" distribution was also apparent when the bradycardia threshold was shifted, and those in the lowest decile of HR-PRSs were more than twice as likely to have a minimum HR ≤35 bpm (Table S2). In multivariable models, the independent relationship between low HR-PRS and minimum HR appeared to be consistent across different ages, fitness levels, and degree of RA remodeling.

The confirmation of both genetic and environmental influences on athletic bradycardia is new but perhaps not entirely unexpected. On the other hand, it was remarkable to find that the HR-PRS was lower in endurance athletes than in a healthy reference (ASPREE) cohort. The average HR-PRS was lower among the endurance athletes, and there was a greater proportion of athletes with a HR-PRS score lower than the bottom quartile score derived from ASPREE (Figure 1). The orthodox view of bradycardia in athletes is that habitual physical training induces cardiac and electrical remodeling that results in bradycardia. The polymorphic variants that determine the HR-PRS are inherited traits that cannot be modified by training, suggesting that this genetic signature somehow influences an individual's likelihood of becoming an athlete. This raises the possibility of an entirely new paradigm in which variation in an individual's genes could determine HR, cardiac remodeling, and athletic capacity. Rather than a construct in which HR is modified by 1 to 2 hours of exercise training per day (only 4%–8% of the total time), it may be that a genetically determined HR results in greater cardiac filling at all times, resulting in greater cardiac remodeling and greater cardiac outputs during exercise. Consistent with a previous study[45] and recent work from our group,[51] a majority of the former endurance athletes continue to exhibit low resting HRs. This suggests that exercise-induced sinus node remodeling might persist after detraining or reflect a lifetime phenotype associated with genetic predisposition or gene-environment interactions. In other words, the capacity for athletic remodeling may be genetic and may be reflected in traits such as HR that predate and postdate intensive athletic training.

Limitations

Our current analysis relies primarily on cross-sectional data, and long-term prospective studies are needed to better evaluate the consequences of resting bradycardia in endurance athletes. HR variability provides insights into autonomic regulation. Although HR variability data were lacking in this study, there remains controversy with how these metrics are interpreted in the context of training-induced bradycardia.[52] We also lacked data on sinus node remodeling, though such remodeling has only been observed in small animal models to date. Whereas RAV*i* was used as a surrogate marker, the association between exercise and intrinsic sinus node remodeling is likely far more complex than atrial size alone, given the various proposed mechanisms for these changes in animal models.[31] Our genetic analysis did not include evaluation of rare variants, which can have profound effects on HR in individual cases. In ongoing work (A.M. Mitchell, unpublished data, 2025), we have found that the prevalence of rare variants in genes associated with cardiomyopathies and arrhythmias in athletes is very low. In contrast, HR-PRS provides a more widely accepted metric of genetic bradycardia risk. Recruitment of athletes with known or suspected arrhythmias based on symptoms introduces a potential for referral bias. Whereas this likely contributed to the overall higher prevalence of AF in our cohort, there was no difference in the proportion of

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BAs and non-BAs recruited from this group. In addition, inclusion of both current and former athletes of varying ages with comorbidities such as prevalent AF enhances the clinical applicability and generalizability of our findings to real-world scenarios. We did not systematically capture detailed training loads in the weeks immediately before testing. It is possible that more strenuous training resulting in overreaching and overtraining may have influenced HR responses in some individuals. We also did not collect data on other lifestyle factors that might influence HR, such as yoga, meditation, and occupational stress. Finally, only 25% of endurance athletes were women, and 99.6% were of European ancestry. Menstrual regularity/amenorrhea were also not captured. These demographic constraints limit inference for female-specific physiological statues and reduces the generalizability of our results to female endurance athletes and to populations of diverse ancestry.

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Future Directions

Collectively, our data indicate that resting bradycardia and sinus pauses are frequent findings in endurance athletes and are likely related to exercise training as well as clinical factors and genetic predisposition. A better understanding of the natural history of bradycardia and its potential complications in athletes with and without high genetic risk will clarify the role of genetic testing in clinical management. It is possible in the future that HR-PRS might be one of multiple variables incorporated into a bradycardia risk score and may assist clinicians in determining whether an athlete's resting HR is appropriate or inappropriate and how closely they may require surveillance. This could be especially relevant in older athletes who are at higher risk of heart block and syncope leading to PPM implantation. The potential long-term clinical sequelae of athletes with high or low HR-PRS also require further investigation. For example, athleticism and lower HRs have been associated with risk of AF.[53,54] The degree to which predisposition to arrhythmias and heart block are associated with intersecting environmental and genetic risk remains an important evolving question.

Conclusions

Resting bradycardia (HR 30–40 bpm) and pauses of 2 to 3 s are common in endurance athletes and do not appear to be associated with short-term adverse outcomes. Extreme bradycardia (HR <30 bpm), pauses ≥3 s, and Mobitz I second-degree AV block are less frequently observed. Supporting the notion that bradycardia in athletes may have a multifactorial mechanism,[52] we demonstrated a novel independent association between genetic predisposition and resting bradycardia in endurance athletes. Furthermore, a low bradycardia PRS was disproportionately more common among endurance athletes than in the general population, raising the possibility that a tendency of low HR in athletes may be an inherited trait rather than a fully acquired phenomenon.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Figure S1
Tables S1 and S2

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