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Aims

This study aims to evaluate the prevalence, morphology, and clinical relevance of transient, extreme heart rate (HR) spikes on heart rate monitors (HRMs) in endurance athletes.

Methods and results

Heart rate monitor training sessions ($n = 57\,282$) from 251 endurance athletes (167 Pro@Heart; 84 Master@Heart), were analysed. Individual physiological maximal HR ($HR_{\max_{Tan}}$) was determined using the kernel density of peak HR values. Extreme HR events were defined as sessions exceeding this limit ($HR_{\max_{HRM}}$). Tracings were visually classified; paroxysmal spikes were considered to be potential tachyarrhythmia. Incidence was estimated with Kaplan–Meier analysis. Demographic, training, and device predictors were tested with a generalized linear mixed model (GLMM). Clinical and Holter data were reviewed for arrhythmia documentation. Extreme HR values occurred in 1.0% of sessions across 133 athletes (53.0%). Paroxysmal spikes represented 28.1% of cases and occurred in 0.27% of sessions and 23.9% of athletes, cumulative incidence was 33.8% (95% confidence interval [CI], 26.1–41.4%) after 291 sessions. The GLMM identified no significant predictors. Paroxysmal spikes were observed in 71.4% of athletes (10/14) with arrhythmia vs. 21.1% without (50/237) ($P < 0.001$). Nearly all $HR_{\max_{HRM}}$ spikes preceded Holter-documented arrhythmias.

Conclusion

Many characteristics point to true arrhythmias as the cause of $HR_{\max_{HRM}}$ recordings with paroxysmal spike morphology: they are rare, do not occur randomly across athletes, and cosegregate with clinically documented arrhythmias. Whether they represent malignant arrhythmias or an extreme athletic cardiac phenotype remains to be determined and is relevant to the role of HRMs as surveillance tools. $HR_{\max_{HRM}}$ tracings with spike morphology should not be dismissed as artefact but require work-up to determine clinical relevance.

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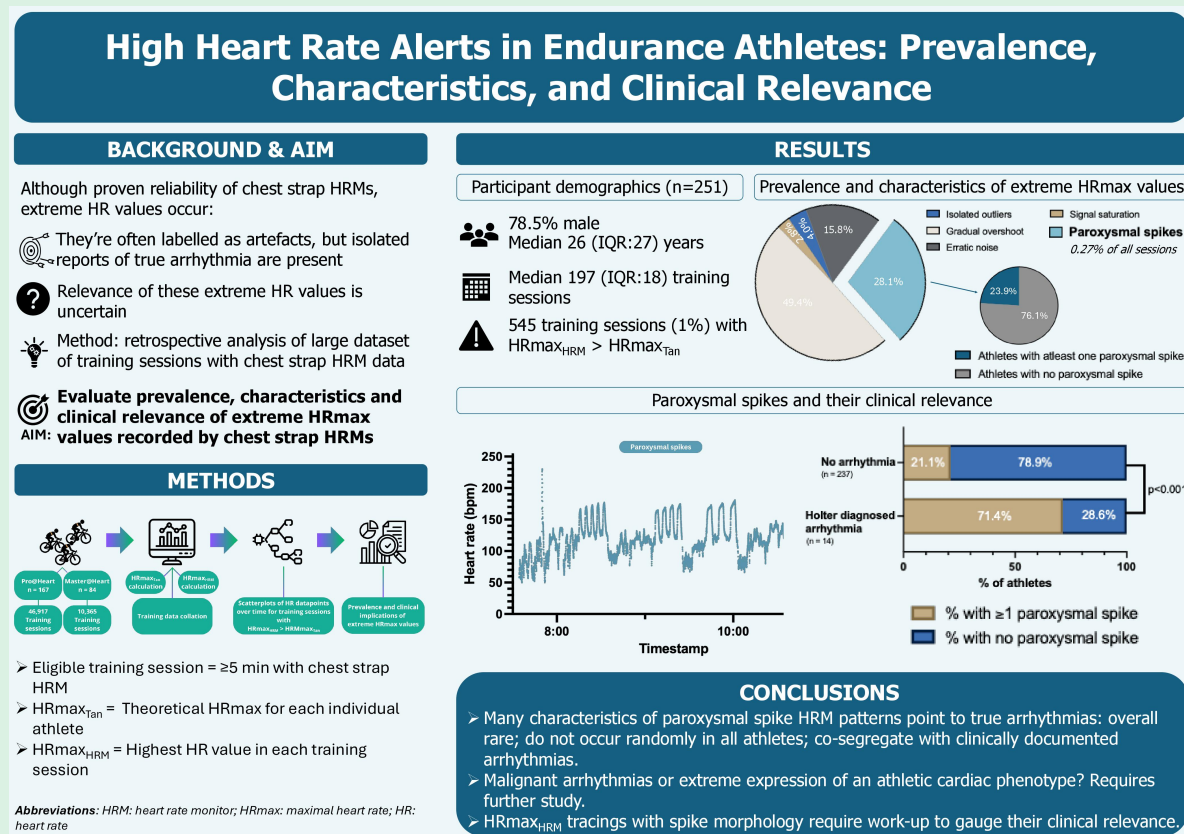
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Lay summary

Chest-strap heart rate monitors (HRMs) are commonly used to assess cardiovascular load in endurance athletes. Despite their reliability, transient, abnormally high heart rate (HR) spikes are occasionally observed. Their interpretation as artefacts or arrhythmias remains enigmatic.

- In 57 282 sessions from 251 athletes, rare yet recurrent paroxysmal ‘spike’ patterns were identified in only 0.27% of sessions but in 23.9% of athletes with a cumulative incidence of 33.8% within 291 sessions, showing no clear predictors.
- Spike morphology occurred at least once in 71.4% of athletes with an arrhythmia, compared with only 21.1% of those without.

Graphical Abstract



Keywords

Exercise • Heart rate • Wearable electronic devices • Heart rate monitors • Chest-straps • Arrhythmias • Athletes • Sports cardiology

Introduction

Commercially available chest-worn heart rate monitors (HRMs) using electrical sensors have become standard for athletes participating in endurance sports. By providing accurate, real-time assessment of cardiovascular load, they have largely replaced subjective measures of exertion and are now integral to training and performance monitoring in both amateur and elite athletes.¹ The accuracy of these devices has been validated against gold-standard Holter monitoring across multiple settings and populations.^{2–5}

Despite this proven reliability, several studies have reported transient, unusually high heart rate (HR) spikes during exercise, often exceeding physiological limits (e.g. >230 b.p.m.).^{6–8} These

episodes are usually attributed to technical artefacts, such as poor skin contact or signal interference.⁶ However, isolated evidence suggests that in rare cases, these spikes may reflect true cardiac arrhythmias. In a prospective cohort study of endurance athletes, over 45% of sessions displayed brief HR spikes on HRM recordings, with one event corresponding to atrioventricular nodal re-entry tachycardia, illustrating the potential of HRMs to uncover latent arrhythmias during routine training.⁶ Similar single-patient case reports have confirmed HRM-detected tachyarrhythmias.^{7,8}

However, no study has systematically examined the prevalence and morphology of abnormal HR patterns in unselected athletes over time. This is clinically relevant because endurance

athletes carry a higher lifetime risk of exercise-induced arrhythmia,^{9–11} yet most HR anomalies are currently dismissed as artefacts. This creates a tension between failing to recognize clinically significant events and over-interpreting them, which can lead to unnecessary investigations.

To address this knowledge gap, we analysed prospectively collected, real-world HRM recordings from a large cohort of asymptomatic endurance athletes to (i) determine the prevalence of extreme or irregular HR patterns during training, (ii) characterize the morphology of those spikes, and (iii) explore contextual factors that may distinguish artefactual from potentially arrhythmogenic events. This study provides the first systematic evaluation of HRM anomalies in a large athletic population, addressing a critical gap at the intersection of sports cardiology and digital health.

Methods

Study population

The study comprised a total of 182 elite competitive endurance athletes aged 15–35 years derived from the international Pro@Heart trial ($n = 300$), conducted between 2021 and 2024, and 178 male endurance athletes aged 45–69 years from the Master@Heart study ($n = 558$), conducted between 2018 and 2021, who had data available on TrainingPeaks. Of these, 251 (P@H, $n = 167$; M@H, $n = 84$) recorded five or more training sessions that included HR data collected by an electric HR sensor-equipped chest-strap HRM (Figure 1A). The full methodological details of these trials have been described in prior publications.^{12,13}

Training data were collected during exercise sessions using commercially available HRMs and uploaded via the TrainingPeaks platform (Peakware LLC, Louisville, Colorado, USA). Heart rate monitor files were eligible for inclusion only if HR data were confirmed to have been recorded using an electrical sensor-equipped chest-strap HRM. Files collected using photoplethysmography (PPG)-based devices or those with unknown measurement methods were excluded.¹⁴ Files recorded with Tacx ($n = 186$) and 4iiii ($n = 195$) HRMs were further excluded, as these brands were represented by only a small number of sessions, precluding meaningful comparative analysis. Sessions shorter than 5 min in duration were also excluded. After applying these criteria, 57 282 of the original 100 107 files were retained for analysis (Figure 1B).

Data collection and processing

Data collection

Training data collation via TrainingPeaks

Training files were downloaded from the TrainingPeaks platform (Peakware LLC) in the *Flexible and Interoperable Data Transfer* (FIT) format, a binary file format developed by Garmin Ltd (Olathe, Kansas, USA) to collect and represent data from wearable devices.¹⁵ The uploading of training files to the platform occurred via automatic synchronization from the athletes' hub device (e.g. smartwatch and bike computer), which connects different sensors and collects various types of data (e.g. HR, power output, temperature, speed, and cadence) for collation and display purposes.¹⁶

Heart rate monitor hardware and identification

Heart rate data were exclusively obtained from electrical sensor-equipped chest-strap HRMs. Manufacturers identified were Garmin Ltd, Polar Electro Oy (Kempele, Finland), and Wahoo Fitness (Atlanta, Georgia, USA). Specific HRM models were identified, including the Garmin HRM-series (Dual, Run, Tri, Pro, 3SS),

the Polar H9 and H10, and the Wahoo TICKR and TRACKR series. For statistical analysis within the generalized linear mixed model (GLMM), devices were grouped by brand to maintain adequate statistical power. A detailed list of identified models is provided in [Supplementary material S1](#).

Data processing

HRmax calculation

Two distinct measures of maximal heart rate (HRmax) were used in this study. Physiological HRmax was calculated using the Tangent HR method, as described in detail in a previous study.¹⁴ This method estimates maximal HR by fitting a tangent to the downward slope of the kernel density plot of each individual athlete's peak training HR values, thereby identifying the inflexion point beyond which further increases are unlikely to reflect true physiological maxima. Participants were included in the dataset only if their Tangent HRmax (HRmax_{Tan}) was calculated from at least five training sessions of 5 min or longer each. The second measure of HRmax is the electrical sensor-equipped HRM measured HRmax (HRmax_{HRM}), which was defined as the highest HR value recorded during each exercise session with valid HR data.

FIT file processing

Wearable-derived activity data were processed using a custom Python script developed in-house. The script automatically extracted and aggregated key activity and physiological parameters (e.g. duration, distance, HR, cadence, and power) from FIT files. Device and sensor metadata were identified using manufacturer-specific lookup tables. A detailed description of the data extraction and processing pipeline is provided in [Supplementary material S2](#).

Identification and classification of heart rate spikes

All training sessions in which at least one recorded HRmax_{HRM} value exceeded the participant's individual HRmax_{Tan} by ≥ 1 b.p.m. were flagged for further evaluation. For each flagged session, HR data were visualized in raw (non-interpolated) format to generate scatterplots of HR vs. elapsed time (Figure 2A), avoiding artefactual visual smoothing common in most commercial software platforms. The temporal resolution of HR data varied between brands and sessions. Assessment was done by one researcher, without access to clinical records of the participants.

Flagged sessions were classified into one of five morphology categories based on the shape of the HR trace:

- *Isolated outliers*: single, isolated outlier values
- *Signal saturation*: abrupt onset or offset with a prolonged plateau of identical HR readings
- *Gradual overshoot*: smooth increases, with values only marginally exceeding Tangent HRmax.
- *Paroxysmal spikes*: steep-onset, transient HR spikes with rapid acceleration and deceleration
- *Chaotic pattern*: irregular, chaotic fluctuations not fitting other categories

Classification was performed by visual inspection of the HR traces. All morphologies were retained for descriptive analysis, but only the paroxysmal spike morphology was selected for primary statistical comparisons. This was based on its resemblance to electrocardiogram (ECG)-confirmed paroxysmal tachyarrhythmias in previous reports,^{6,7,17} making it the most clinically suspicious pattern. Subsequent group and device comparisons therefore focused exclusively on this morphology to explore its incidence in endurance athletes and potential device-related differences.

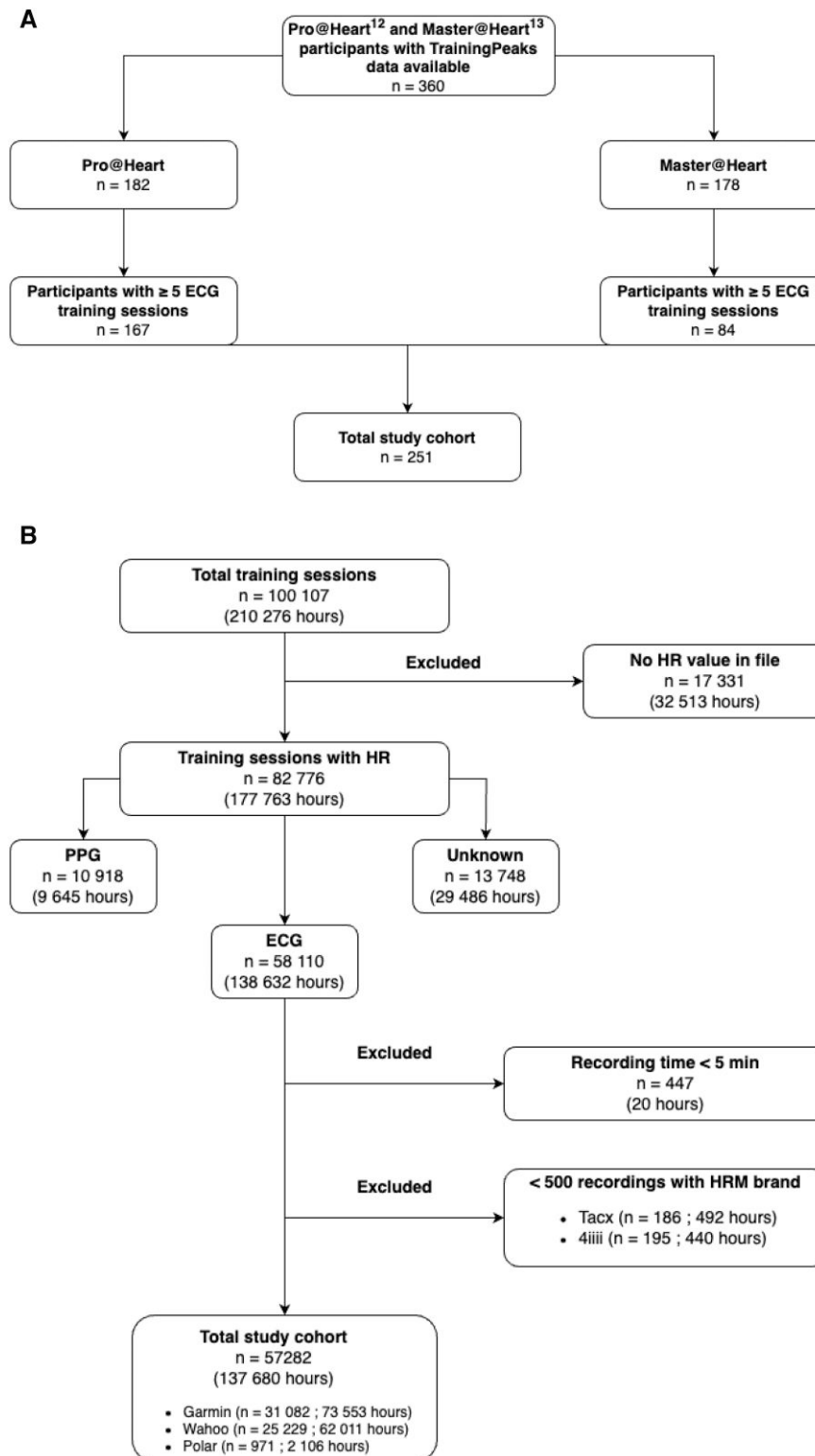


Figure 1 Study cohort: participants (A) and recorded training sessions (B) HR, heart rate; PPG, photoplethysmography; ECG, electronic sensor-equipped heart rate monitor.

Contextual analysis using power output

For sessions identified as containing paroxysmal spikes and valid power output data, further contextual assessment was carried out

and intended to explore plausible physiological triggers for the changes in HR. Heart rate and power curves were plotted together and analysed for the temporal relationship of concurrent abrupt

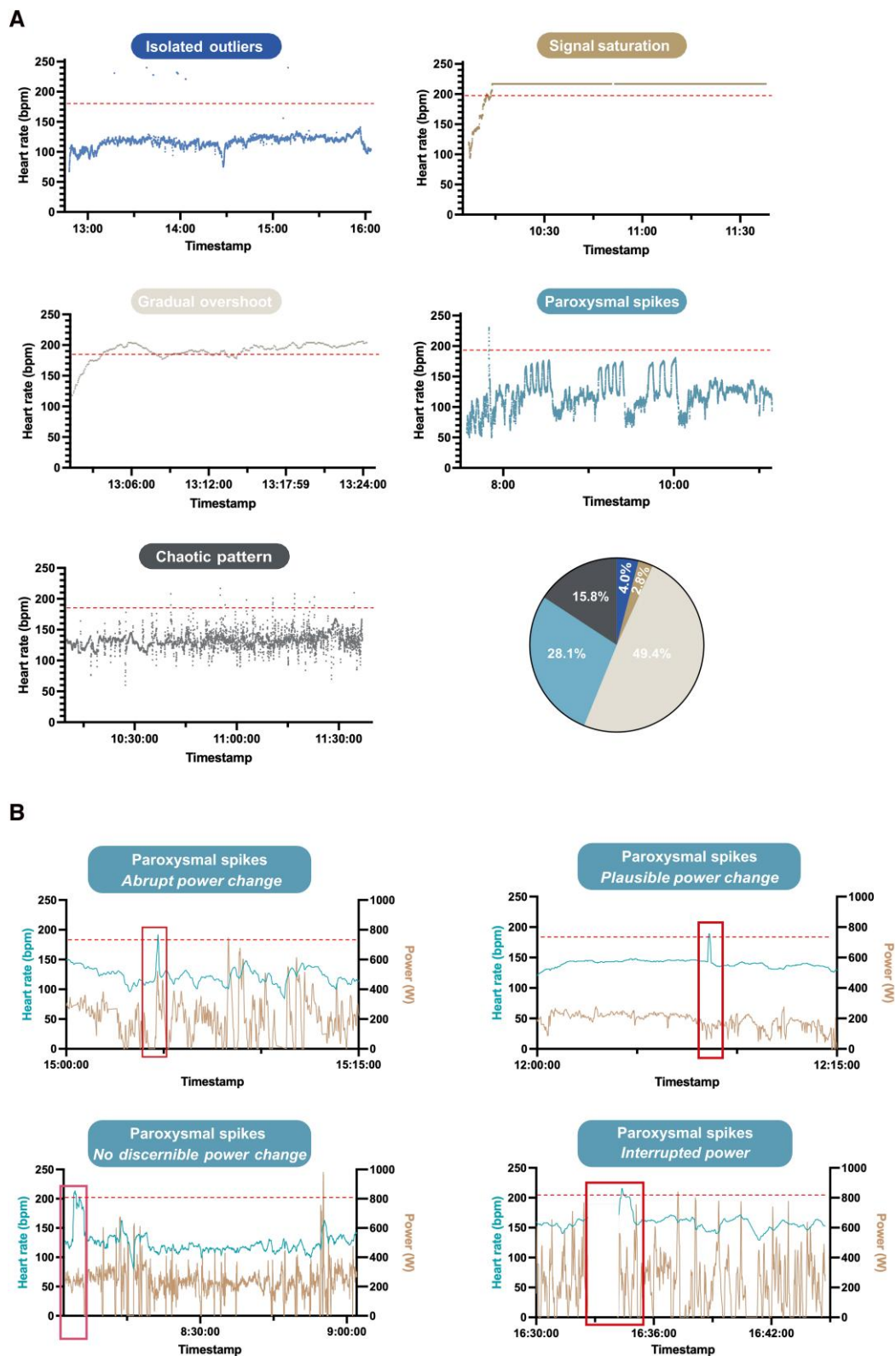


Figure 2 Different patterns of peaks in maximal heart rate values. B.p.m., beats per minute; Horizontal dashed lines indicate the participant's individual $HR_{max_{T_{an}}}$.

changes in wattage and HR (Figure 2B). Each event was categorized into one of four groups:

- *Abrupt power change*: HR spike occurred in close temporal proximity to a sudden transition from rest to exercise.
- *Plausible power change*: HR spike occurred with a plausible change in power output.
- *No discernible power change*: HR spike occurred without a significant change in power.
- *Interrupted power*: power data were present but uninterpretable due to missing values or artefacts.

Empirical validation of heart rate morphology via simultaneous ECG

To provide empirical validation of the identified paroxysmal spike morphology, a symptomatic athlete who presented to the cardiology department with recurrent, unexplained HR spikes was selected for single-case prospective validation. The athlete was instructed to perform exercise as usual while simultaneously wearing their own chest-strap HRM (Garmin HRM Dual) and a single-lead ECG-equipped chest-strap (Fourth Frontier X2; Fourth Frontier Technologies LLC) provided by the study team. Both devices were activated simultaneously to ensure temporal alignment of the HR data of both devices. *Post hoc* synchronization of the datasets was performed using timestamps to cross-reference the HRM-derived paroxysmal spikes with the electrophysiological rhythm.

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics (v29) and Python 3.12. A two-tailed P -value of <0.05 was considered statistically significant. Normality was assessed with the Shapiro–Wilk test. Non-normally distributed continuous variables were summarized as medians with interquartile ranges (IQRs); categorical variables were presented as frequencies and percentages. Between-group differences were analysed using the Mann–Whitney U test or the Kruskal–Wallis test. Proportional differences were analysed using a χ^2 test. A session-based Kaplan–Meier survival analysis was used to estimate the cumulative incidence of first-time paroxysmal spike occurrence among participants with a calculated $HR_{max_{Tan}}$. Time was defined as the number of training sessions, with censoring at the final available session for each participant. To identify potential predictors of paroxysmal spike occurrence, a GLMM with a binomial logit link was constructed. Participant ID was included as a random intercept to account for intra-individual clustering. Fixed effects included study group (P@H vs. M@H), HRM brand, age, sex, sport type, eTRIMP, and session duration. To investigate the influence of environmental and mechanical factors within the cycling cohort, a subgroup analysis was performed. A GLMM with a binomial logit link was constructed using paroxysmal spike occurrence as the dependent variable and participant ID as a random intercept. Fixed effects for this model included cycling sub-discipline (road, mountain bike, gravel, cyclo-cross, and indoor training), seasonal timing (Spring, Summer, Autumn, Winter), and maximal session altitude.

Results

Dataset demographics

General demographics

The median age of participants was 26 years (IQR, 21–48), reflecting the inclusion of two distinct cohorts: M@H (median, 53 years; IQR, 48–58) and P@H (median, 22 years; IQR, 20–26). The M@H cohort consisted exclusively of males, while the

P@H cohort included 54 females (32.3%). Each participant contributed a median of 197 (IQR, 111–319) valid HRM recordings (Table 1).

Maximal heart rate

As expected from the age difference, the pre-calculated $HR_{max_{Tan}}$ was significantly higher in the younger P@H group (median, 200 b.p.m.; IQR, 195–204) than in the M@H group (median, 179 b.p.m.; IQR, 170–185) ($P < 0.001$) (see [Supplementary material S3A](#)). The median recorded $HR_{max_{HRM}}$ across all sessions was 165 b.p.m (IQR, 149–179) and was likewise higher in P@H than in M@H participants [median, 169 b.p.m. (IQR, 153.00–181.00) vs. 152 b.p.m. (IQR, 139.00–163.00); $P < 0.001$] (see [Supplementary material S3B](#)).

Morphology and occurrence of extreme heart rate values in electric sensor-equipped chest-strap training sessions

Morphological patterns of extreme heart rate recordings and their incidence

A total of 545 training sessions surpassed the individual $HR_{max_{Tan}}$ threshold, accounting for 1.0% of the 57 282 chest-strap HR recordings. These episodes were identified in 133 unique participants (53.0% of all participants), comprising 246 events in the P@H group (0.52% of recordings; 85 participants, 50.9%) and 299 in the M@H group (2.88% of recordings; 48 participants, 57.1%).

The gradual overshoot morphology was the most common pattern (49.4%), followed by the paroxysmal spike morphology (28.1%), while isolated outlier, signal saturation, and chaotic pattern morphologies accounted for 4.0%, 2.8%, and 15.8% of events, respectively (Figure 3A). In total, 153 paroxysmal spike tracings were identified in 60 participants (23.9% of the cohort), with a similar distribution between P@H (27.5%) and M@H (16.7%) ($P = 0.081$).

Kaplan–Meier analysis showed that all first-time paroxysmal spike tracings occurred within the first 291 recorded sessions, with a cumulative incidence of 33.8% [95% confidence interval (CI), 26.1–41.4%]. Among participants with paroxysmal spike tracings, 25%, 50%, and 75% of events occurred within 32, 65, and 122 sessions, respectively. The median follow-up was 146 sessions (IQR, 60–253). The median number of paroxysmal spike events per affected participant was 1 (IQR, 1–3; range, 1–12), with no group difference between P@H and M@H ($P = 0.963$). Nearly half (47.5%) of affected athletes recorded ≥ 2 events, 16.4% recorded ≥ 5 , and 3.3% recorded ≥ 10 (Figure 3).

Predictors of paroxysmal spike event occurrence

In the GLMM, none of the predictors were significantly associated with paroxysmal spike occurrence. Age ($\beta = -0.001$; $P = 0.780$; odds ratio [OR] = 0.999; 95% CI, 0.988–1.009), sex ($\beta = -0.014$; $P = 0.823$; OR = 0.986; 95% CI, 0.875–1.112), eTRIMP ($\beta \approx 0.000$; $P = 0.257$; OR = 1.000; 95% CI, 0.999–1.000), session duration ($\beta = 2.65 \times 10^{-6}$; $P = 0.782$; OR = 1.000; 95% CI, 1.000–1.000), HRM brand (Polar vs. Garmin: $\beta = -0.130$; $P = 0.473$; OR = 0.878; 95% CI, 0.615–1.253; Wahoo vs. Garmin: $\beta = -0.024$; $P = 0.657$; OR = 0.976; 95% CI, 0.879–1.085), sport type (cycling vs. other: $\beta = 0.004$; $P = 0.975$; OR = 1.004; 95% CI,

Table 1 Demographic information

	Pro@Heart (n = 167)	Master@Heart (n = 84)	Total cohort (n = 251)	P-value (Between-group difference)
Age, years				<0.001*
Median	22	53	26	
IQR	20–26	48–58	21–48	
Gender, n (%)				<0.001**
Female	54 (32.3%)	0 (0%)	54 (21.5%)	
HRmax Tangent, b.p.m.				<0.001*
Median	200.00	179.00	196.00	
IQR	195–204	170–185	185–203	
Training sessions ECG HR ≥ 5 min				<0.001*
Median	269.00	113.50	197.00	
IQR	163.50–357.50	55.75–183.00	111.00–319.00	
Sport type, n (%)				<0.001**
Cycling	45 700 (97.41%)	8436 (81.39%)	54 136 (94.51%)	
Running	684 (1.46%)	1841 (17.76%)	2525 (4.41%)	
Training	415 (0.88%)	10 (0.09%)	425 (0.74%)	
Hiking	44 (0.09%)	1 (0.01%)	45 (0.08%)	
Swimming	0 (0%)	43 (0.41%)	43 (0.08%)	
Generic	28 (0.06%)	7 (0.07%)	35 (0.06%)	
Walking	11 (0.02%)	16 (0.15%)	27 (0.05%)	
Alpine skiing	12 (0.03%)	7 (0.07%)	19 (0.03%)	
Cross-country skiing	11 (0.02%)	4 (0.04%)	15 (0.03%)	
Mountaineering	7 (0.01%)	0 (0%)	7 (0.01%)	
Snowshoeing	5 (0.01%)	0 (0%)	5 (0.01%)	

HRmax, maximal heart rate; ECG HR, electric sensor-equipped heart rate monitor; IQR, interquartile range; *, Mann–Whitney *U* test; **, χ^2 test

0.799–1.260), and group (M@H vs. P@H: $\beta = 0.013$; $P = 0.941$; OR = 1.013; 95% CI, 0.712–1.444) yielded no significant associations. The overall model fit was non-significant [F (8, 57 655) = 0.462; $P = 0.863$]. The intraclass correlation coefficient (ICC) was 0.000, indicating negligible between-subject variance. Complete model estimates are shown in [Supplementary material S4](#).

In a subgroup analysis restricted to cycling sessions ($n = 54\ 136$), altitude, season, and cycling sub-discipline were not associated with paroxysmal spike occurrence. Compared with road cycling, no differences were observed for mountain biking ($P = 0.759$), gravel cycling ($P = 0.994$), or indoor training ($P = 0.994$). Similarly, no seasonal variation was observed compared with Summer (Autumn, $P = 0.845$; Winter, $P = 0.940$; Spring, $P = 0.935$). Detailed model estimates are provided in [Supplementary material S5](#).

Contextual factors in paroxysmal spike events

To explore the potential physiological context surrounding paroxysmal spike events, all sessions with valid power output data (113 of 153 sessions) were visually inspected ([Figure 2B](#)):

- Abrupt power change (HR spike at onset of load): 26.6%
- Plausible power change (HR spike with plausible power change): 34.5%
- No discernible power change (HR spike without power change): 26.5%
- Interrupted power (power data uninterpretable): 12.4%

Clinical events in participants with heart rate spikes

Review of clinical records in athletes with paroxysmal spike events

Among the 60 athletes with at least one paroxysmal spike tracing, 10 (16.7%) had documented arrhythmias or a history of ablation. Three athletes had confirmed non-sustained ventricular tachycardia (nsVT) on Holter monitoring: one exhibited both paroxysmal spike with abrupt power change ($n = 1$) and paroxysmal spike with interrupted power trace ($n = 1$) morphologies; another showed several paroxysmal spikes with plausible power change traces ($n = 3$), paroxysmal spike with no discernible power change ($n = 3$), and paroxysmal spikes with interrupted power traces ($n = 2$); and a third displayed only a paroxysmal spike without discernible power change ($n = 1$). One of these athletes was diagnosed with right ventricular outflow tract ventricular tachycardia (VT) and underwent successful catheter ablation.

Three athletes had supraventricular tachycardia (SVT), including one with a paroxysmal spike with abrupt power change tracing ($n = 1$), one with a paroxysmal spike with plausible power change tracing ($n = 2$), and one without available power data. Additionally, two athletes experienced both nsVT and SVT [one had a paroxysmal spike with plausible power change tracing ($n = 1$); one had no power data], and two additional athletes had a prior history of atrioventricular nodal reentry tachycardia (AVNRT) ablation. Representative examples are illustrated in [Figure 4](#).

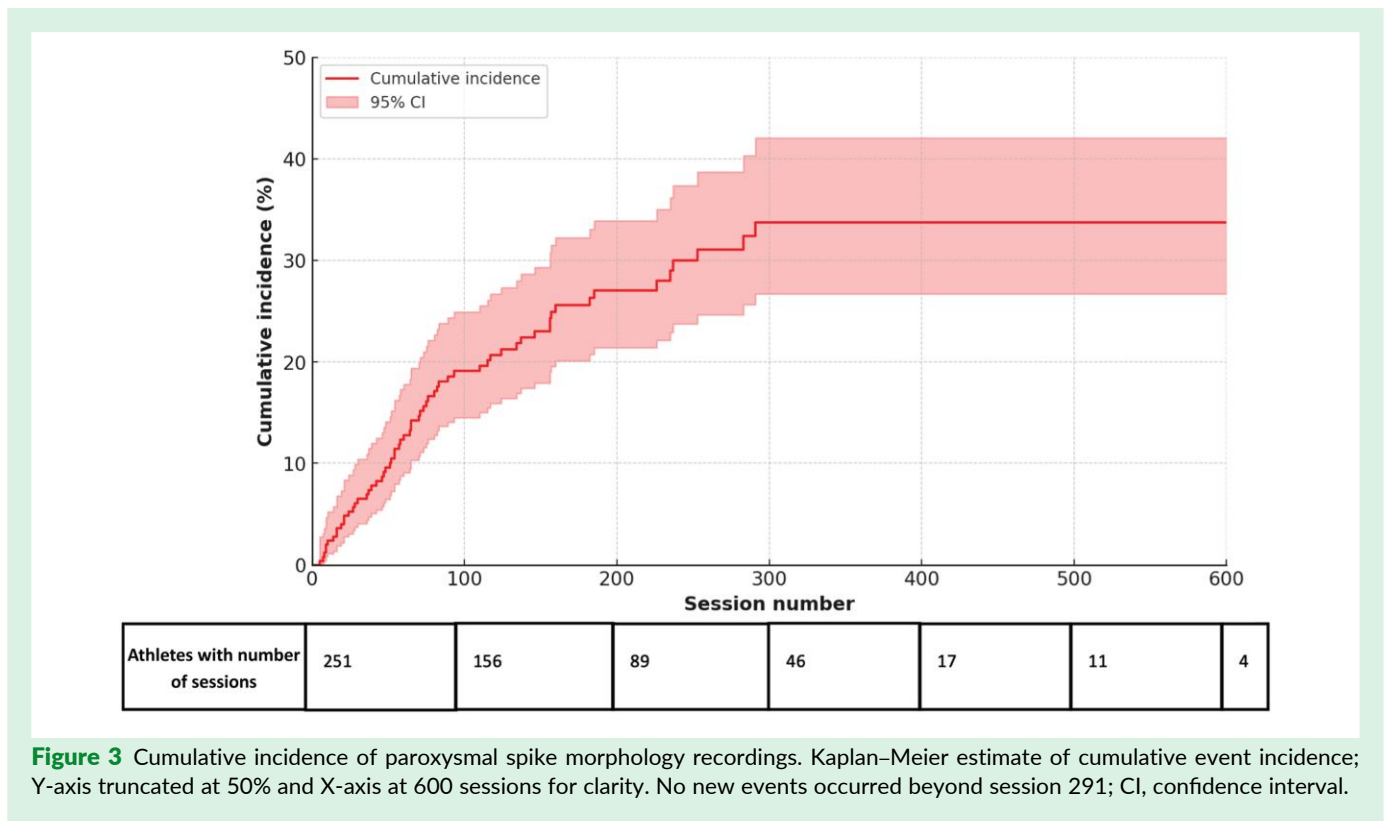


Figure 3 Cumulative incidence of paroxysmal spike morphology recordings. Kaplan–Meier estimate of cumulative event incidence; Y-axis truncated at 50% and X-axis at 600 sessions for clarity. No new events occurred beyond session 291; CI, confidence interval.

Importantly, with the exception of a single SVT episode, all paroxysmal spike events were recorded by the HRM before arrhythmia detection on Holter monitoring. The remaining 50 athletes (83.3%) with paroxysmal spike tracings had no documented arrhythmias or cardiovascular events, although some reported suggestive symptoms or had a family history of arrhythmia or cardiomyopathy.

Clinical records in all athletes with heart rate spikes

Subsequently, we reviewed clinical records and Holter data from all 133 participants who exhibited at least one HR spike, irrespective of morphology. This analysis identified four additional athletes with documented cardiac arrhythmias or a history of ablation.

One athlete with chaotic pattern tracings ($n = 2$) had nsVT on Holter monitoring and focal inferolateral myocardial fibrosis on cardiac magnetic resonance imaging, consistent with prior myocardial infarction. Another athlete with gradual overshoot recordings ($n = 17$) had SVT confirmed on Holter. In addition, one athlete with a gradual overshoot tracing ($n = 1$) underwent Kent bundle ablation for Wolff-Parkinson-White (WPW), and another with a chaotic pattern tracing ($n = 1$) underwent pulmonary vein isolation (PVI) for atrial fibrillation (AF).

Association between paroxysmal spikes and arrhythmia

Among participants with a documented arrhythmia ($n = 14$), 10 (71.4%) exhibited at least one paroxysmal spike tracing, compared with 50 of 237 participants without arrhythmia (21.1%) ($P < 0.001$) (Figure 5). The same pattern was observed for paroxysmal spike burden [median 0.5% (IQR, 0.2–1.1%) vs. 0.0% (IQR 0.0–0.0%); $P < 0.001$]. Arrhythmia was diagnosed significantly more often in patients with ≥ 1 paroxysmal spike trace (16.7%, 10/60) than in those

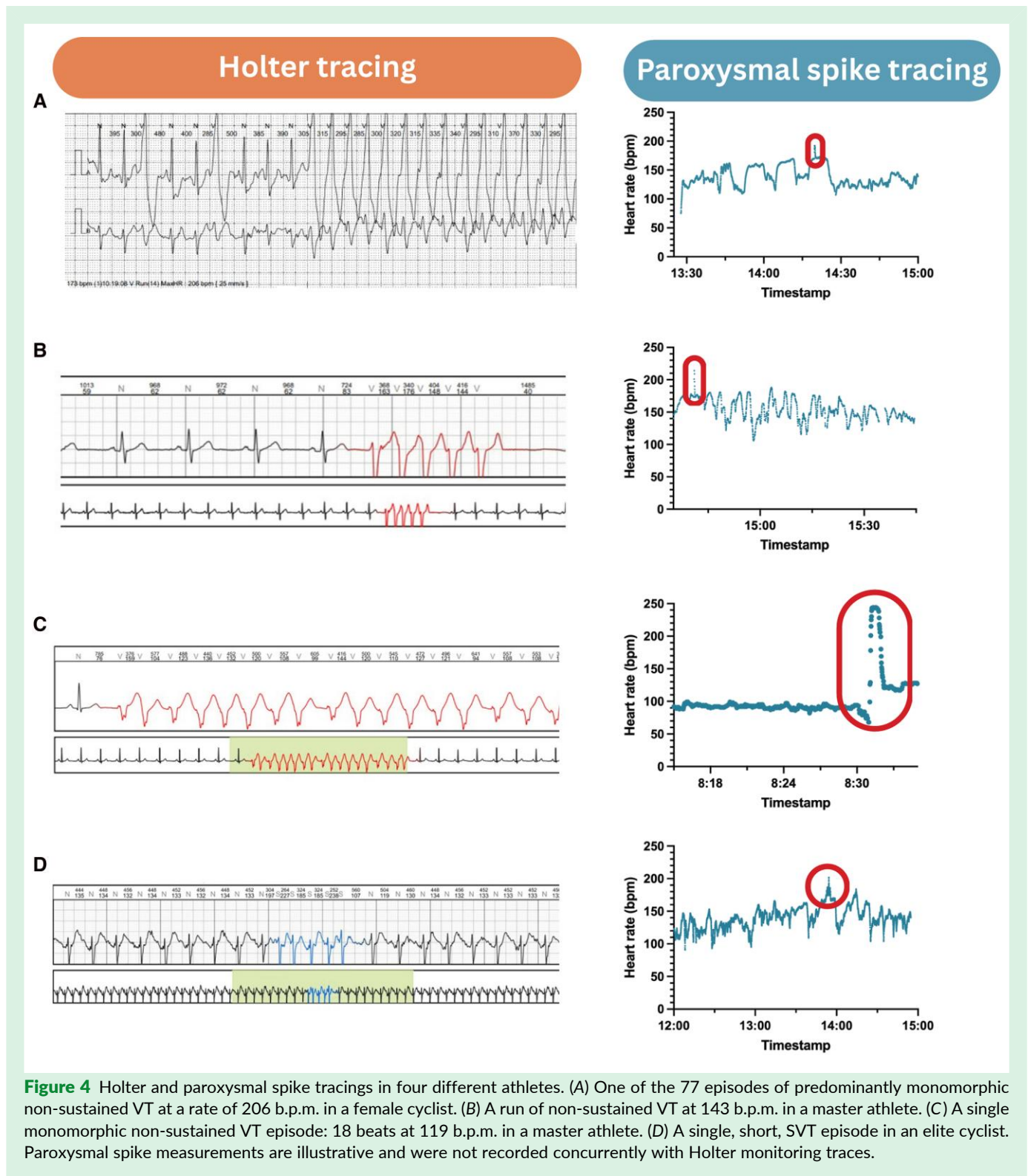
without paroxysmal spikes (2.1%, 4/191; $P < 0.001$). In the 118 participants with no morphology recordings, none had clinically diagnosed arrhythmia.

Validation of heart rate monitor spikes via simultaneous single-lead ECG

In the *post hoc* validation case, a paroxysmal spike reaching 169 b.p.m. was recorded on the commercial chest-strap HRM of the participant. Simultaneous single-lead ECG recording documented rapid atrial tachycardia lasting ~ 8 s. Notably, a temporal lag of ~ 4 s was observed between the ECG-documented onset and the HRM-recorded spike (see Supplementary material S6A). Furthermore, shorter bursts of arrhythmia in the same athlete were reflected on the HRM as isolated outliers rather than sustained spikes (see Supplementary material S6B).

Discussion

This study is the first to systematically assess the incidence, morphology, and potential clinical relevance of abnormally high maximal HR values captured by commercially available electrical chest-strap HRMs in endurance athletes. Among more than 57 000 recorded training sessions, 1.0% exceeded the expected physiological limit defined by $HR_{max_{Tan}}$. Visual analysis revealed five distinct morphological patterns associated with extreme $HR_{max_{HRM}}$ readings. The predefined morphology of interest, characterized by paroxysmal, spike-like HR surges, was rare (0.27% of sessions) but present in 23.9% of athletes; at least one such event occurred in 71.1% of those with a clinically documented arrhythmia compared with 21.1% of those without. These findings carry dual implications: they may often be artefactual noise, but in some athletes, they could indicate



genuine supraventricular or ventricular arrhythmias. Therefore, our results are relevant both for device validation and for early arrhythmia screening in athletic populations. While our primary analysis is associative, a *post hoc* validation case provides an initial proof-of-concept for the arrhythmogenic potential of these recordings.

Occurrence and morphology of extreme maximal heart rate values

The low prevalence of HRmax overshoot confirms the high reliability of chest-strap HRMs in real-world endurance training, consistent with previous validation studies against Holter ECG.^{2-5,18,19} This accuracy is specific to chest-worn electrical sensor-equipped HRMs,

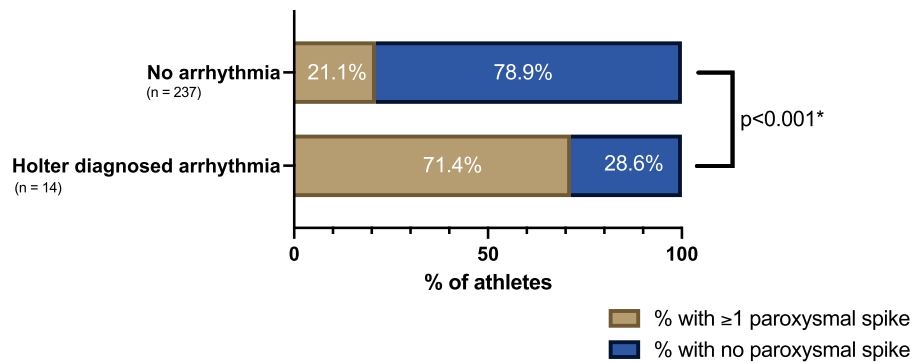


Figure 5 Proportion of athletes with ≥ 1 paroxysmal spike recording according to arrhythmia status. *, χ^2 test.

which outperform PPG-based wrist-worn devices that are susceptible to motion artefacts and signal lag, especially during dynamic or sprint-based exercise involving rapid HR fluctuations.^{20–22} Limiting our analysis to electrical sensor-equipped chest-straps reduced artefactual noise and increased confidence in the observed patterns.

Although rare, extreme HR peaks warrant closer scrutiny. We identified five distinct HR peak morphologies, with the paroxysmal spike morphology most closely resembling the sharp HR spikes previously reported in the literature and confirmed as true arrhythmias.^{6,7} Although present in only 0.27% of training sessions, such recordings were found in 23.9% of participants, raising the clinically important question of whether these are rare artefacts or real arrhythmias.

Interpretation of paroxysmal spikes: artefact or arrhythmia

Notably, 71.1% of athletes with confirmed arrhythmia exhibited paroxysmal spike tracings. This strong association supports the notion that at least a subset of these events represents genuine arrhythmias, as further suggested by correlations with Holter findings, exercise testing, and clinical history. Moreover, the absence of artefacts in many athletes despite numerous recordings argues against random signal noise as the sole explanation. Nevertheless, no significant demographic or device-related predictors of paroxysmal spike tracings were identified.

In the *post hoc* validation case, a sustained paroxysmal spike reaching 169 b.p.m. corresponded to an 8-s run of rapid atrial tachycardia documented on simultaneous ECG. Notably, a temporal lag of ~ 4 s was observed between the ECG-documented arrhythmia onset and the HRM-recorded spike. This lag is likely attributable to a moving-window averaging algorithm proprietary to the commercial chest-strap HRM, which requires a specific duration of elevated HR to ‘fill’ the calculation window and register a sustained peak. This technical factor also explains why shorter bursts of arrhythmia in the same athlete were reflected only as isolated outliers rather than paroxysmal spikes. This suggests that while we focused on sustained spikes for their high clinical correlation, even isolated outliers may, in some contexts, represent very brief paroxysmal events.

This raises a clinically relevant question: if nearly a quarter of athletes exhibit paroxysmal spike events, do these reflect a

‘physiologic’ athletic cardiac phenotype, driven by high variations in autonomic tone and ionic changes, or are they early markers of ‘pathologic’ remodelling?^{9,23} Supporting the former, 50 of the 60 athletes with one or more paroxysmal spike events had no documented arrhythmias or clinical signs, suggesting that, in many cases, these spikes, if they are not artefacts, may represent benign physiological variations. Conversely, four athletes with established arrhythmias or a history of ablation did not display paroxysmal spike patterns, but rather gradual overshoot or chaotic pattern tracings, indicating that paroxysmal spikes alone do not capture the full spectrum of clinically relevant arrhythmias.

Ambulatory 12-lead ECG studies show that ventricular ectopy is rare in healthy athletes, with $>90\%$ exhibiting ≤ 10 PVCs per 24 h and only 3% displaying complex forms.²⁴ Against this background, paroxysmal spike tracings, observed in 23.9% of athletes but only 0.27% of sessions, likely reflect sporadic but widespread transient phenomena detectable through large-scale wearable monitoring.

In a subset of training sessions with paroxysmal spikes that also included exertion data (power meter), several paroxysmal spike events were observed shortly after a relative rest (wattage = 0) and immediately following a visible surge in power output. While these periods do not represent full recovery, such early post-exertion transitions are known to involve acute autonomic recalibration and imbalance.²⁰ Although this does not imply a direct arrhythmogenic trigger, it may create the conditions for ectopic activity and re-entry and, as such, be pro-arrhythmic. Premature ventricular complexes (PVCs) occurring during recovery have been linked to elevated cardiovascular risk, particularly in individuals with underlying structural heart abnormalities.^{21,22} Conversely, artefacts could be more prevalent during abrupt intensity changes due to increased motion or sensor disruption. Although the present data do not allow definitive differentiation between physiological and artefactual origins, the recurrence of paroxysmal spikes in specific individuals underscores the need for cautious interpretation and merits targeted investigation in future studies.

Clinical correlation and diagnostic implications

The GLMM analysis confirmed negligible within-individual clustering (ICC < 0.001), indicating low recurrence of paroxysmal

spike events per athlete. However, paroxysmal spike recordings were significantly more frequent in athletes with known arrhythmias than in those without. While the number of events per affected participant was similar between groups, the overall prevalence supports the potential of paroxysmal spike tracings as a screening marker to help distinguish athletes with underlying arrhythmias from those without. In elite competitive athletes specifically, this approach may also carry implications for performance optimization, as arrhythmic events occurring during or after exercise are easily misattributed to training load, thereby circumventing the symptom-driven diagnostic pathway.

It is important to contextualize the clinical significance of the arrhythmias identified in this cohort. SVT, AVNRT, and isolated nsVT carry a favourable prognosis in athletic populations in the absence of underlying structural heart disease and are not necessarily associated with an increased risk of sudden cardiac death. Detection of a paroxysmal spike should therefore not provoke clinical alarm, but rather prompt targeted, proportionate evaluation aimed at excluding the minority of cases in whom a more serious arrhythmogenic problem may be present. This distinction is essential: neither over-investigation nor dismissal serves the athlete well, and avoiding unnecessary restriction from sport in an otherwise healthy individual remains a key clinical priority.

In summary, while paroxysmal spikes cannot yet be considered diagnostic, these may serve as a low-threshold signal warranting further evaluation and/or more focused follow-up. This aligns with the recent European Heart Rhythm Association (EHRA) Practical Guide on the use of digital devices for arrhythmia detection,²⁵ which recognizes that wearables can reliably detect atrial fibrillation and some supraventricular rhythms, but remain unvalidated for ventricular arrhythmia detection. According to EHRA, the accuracy of these devices for non-AF arrhythmias remains uncertain, and findings should therefore be interpreted with caution and clinical oversight. In line with these recommendations, we regard paroxysmal spike detections as potential screening signals that require ECG confirmation, not by itself as diagnostic evidence of arrhythmia. If validated prospectively, paroxysmal spike detections could support early risk stratification in athletes, particularly those with high training volumes, where continuous monitoring increases the likelihood of capturing rare arrhythmic events missed by standard Holter monitoring.

Limitations

Our study has several limitations. Although the P@H and M@H datasets are extensive, they were not collected initially to analyse peak HR values or evaluate HRMs from specific manufacturers. This limits control over data quality and completeness. For instance, we were unable to verify whether chest-straps were consistently worn correctly or if issues, such as static interference or clothing displacement, may have affected signal quality.⁶ Nevertheless, the large volume of training sessions analysed mitigates some of these limitations. Given that the study population comprises experienced endurance athletes, it is reasonable to assume a lower likelihood of user error in device handling. A further limitation is the absence of a questionnaire to capture subjective symptom reports during the recorded training sessions. While no participants in the current study reported symptoms associated

with these episodes during their clinical evaluations, the retrospective nature of the dataset may have led to subtle symptoms being missed or misattributed to training-related exertion. Future prospective studies should incorporate real-time symptom logging to better define the clinical burden and symptomatic correlation of these HR spikes. Crucially, the absence of simultaneous ECG for the original 57 282 sessions means the relationship between the majority of HRM spikes and specific arrhythmic substrates remains associative. However, the proof-of-concept validation provided in the results section 'Validation of heart rate monitor spikes via simultaneous single-lead ECG' demonstrates that these spikes are not universally artefactual. While the true positive and false positive predictive values for the entire population remain unknown, these results indicate that paroxysmal spikes can serve as a clinically relevant trigger for further investigation rather than mere technical noise.

Another limitation involves incomplete device metadata. While many recordings included identifiable HRM information, a significant number had to be excluded due to ambiguity about the device model or sensor type, potentially resulting in the loss of valuable data. This challenge is compounded by the binary structure of FIT files, which are inherently susceptible to corruption; a single data error can compromise an entire file.¹⁶ Moreover, different manufacturers provide information in various formats within their FIT files, making robust automated analysis challenging and manual checking time-consuming. Importantly, the retrospective nature of the study limited the range of available covariates. Several physiologically relevant factors, such as autonomic tone, training phase, and recovery state, were not available. Therefore, the lack of significant predictors in our GLMM should be interpreted with caution, as these findings may partly reflect unmeasured confounders rather than a true lack of individual susceptibility. Lastly, paroxysmal spike detection was conditional on HR_{max-Tan} exceedance, excluding potential submaximal occurrences.

Finally, the findings of this study are limited to high-volume endurance athletes and may not be generalizable to the broader, non-athletic population. To our knowledge, large-scale datasets of high-resolution HR recordings for sedentary or recreationally active individuals are currently unavailable, making it difficult to determine the baseline prevalence of paroxysmal spikes in the general population. Whether HR spikes are a specific manifestation of the electrophysiological and structural remodelling associated with the athlete's heart or a more widespread phenomenon captured here only due to high monitoring volume remains a subject for future investigation.

Future directions

Future research should focus on prospective validation of paroxysmal spike events through simultaneous HRM and ECG monitoring, combined with systematic collection of contextual variables, such as training intensity, recovery state, and environmental conditions. Such data are essential to determine in what proportion paroxysmal spikes represent true arrhythmic events or artefacts, and in which conditions and/or athlete groups their occurrence is most predictive for arrhythmic events.

In parallel, developing automated signal analysis with machine learning could allow real-time differentiation between artefactual and clinically relevant events, turning chest-strap HRMs

from basic training tools into early warning systems for arrhythmia detection. Machine learning methods trained on raw data streams from chest- straps could achieve higher specificity in event classification.

Finally, long-term follow-up studies are necessary to determine whether paroxysmal spike events forecast future malignant arrhythmic outcomes, especially in athletes with high cumulative training exposure. Establishing the prognostic value of these events will be essential to integrating wearable-derived HR data into personalized risk stratification and screening strategies for athletic populations.

Conclusions

The strong association between paroxysmal spikes and documented arrhythmias, supported by empirical validation in a proof-of-concept case, suggests that this morphology may serve as a potential screening marker for underlying rhythm disorders. Although a definitive causal link across the entire cohort cannot be established without simultaneous ECG, our findings indicate that such events are rare, do not occur randomly in all athletes, and cosegregate in athletes with clinically documented arrhythmias. Whether these events in otherwise healthy athletes represent malignant arrhythmias or an extreme expression of an athletic cardiac phenotype remains to be determined and is relevant to the role of HRMs as a surveillance tool. Early recognition and appropriate evaluation of such HR patterns may facilitate timely arrhythmia detection and risk stratification, particularly in athletes with high training volumes. These findings highlight the potential of widely used wearable devices as low-threshold screening tools in sports cardiology, warranting prospective validation with ECG.

Supplementary material

Supplementary material is available at [European Journal of Preventive Cardiology](#).

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Consent to participate

All participants in this study provided written informed consent prior to their inclusion.

Consent for publication

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Ethical approval

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Data availability

Raw data supporting the conclusions of this article will be made available by the authors upon reasonable request.

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