Received: 18 November 2025 | Accepted: 22 March 2026

DOI: 10.1002/evj.70166

ORIGINAL ARTICLE

Equine Veterinary Journal WILEY

Exercising electrocardiograms from Thoroughbred racehorses with exercise associated sudden death

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Funding information
Horseracing Integrity and Safety Authority; National Institutes of Health (NIH) K12, Grant/Award Numbers: 1UM1TR004405-01A1, K12TR004373

Abstract

Background: Exercise associated sudden death (EASD), defined as a fatal collapse in a closely monitored and previously presumed clinically healthy horse that occurs during exercise or within approximately 1 h after exercise, is disproportionately more common in equine than in human athletes.

Objectives: To describe ECGs from EASD cases in Thoroughbred racehorses.

Study Design: Retrospective case series.

Methods: An international call for potential cases was made through direct contact with relevant racing authorities requesting that trainers of horses which had experienced EASD consent to researchers gaining access to any available ECG data recorded with a wearable device prior to or during EASD events. ECGs were evaluated by a single observer and the findings were described and summarised.

Results: Eleven horses, with a total of 24 readable ECGs (median [range] 2 [1–4]/horse) were identified. Four horses were wearing an ECG at the time of death; 3 had atrial fibrillation (AF) throughout the recording that led to malignant arrhythmias and death, and the 4th had couplets and triplets with R on T, ventricular fibrillation, and death in the late recovery period. The other seven horses had ECGs recorded 4–462 days before death. One of these horses had AF throughout the recording and died 9 days later. Late recovery arrhythmias were identified in 5 recordings from 3 additional horses.

Main Limitations: Small number of horses with EASD were sampled, and use of a single lead ECG limited information on arrhythmia origin.

Conclusions: ECG screening to detect AF before horses train or race has the potential to reduce EASD incidence. More information is needed for risk stratification of late recovery and other arrhythmias.

KEYWORDS
arrhythmia, atrial fibrillation, collapse, horse, racing

1 | INTRODUCTION

Exercise associated sudden death (EASD) accounts for approximately 20%–25% of all racing fatalities.[1–5] EASD is defined as a fatal collapse in a closely monitored and previously presumed clinically healthy horse that occurs during exercise or within approximately 1 h after exercise.[6] Arrhythmia is often suspected as a cause of EASD.[3] However, because cardiac arrhythmia is an electrical event, post-mortem

cardiac evaluation is typically unremarkable and accompanied by non-specific changes to the lungs such as pulmonary oedema, congestion, and haemorrhage.[3,7,8]

A growing body of evidence has shown that in human athletes, prolonged strenuous exercise promotes a pro-arrhythmic cardiac substrate and increases the risk for arrhythmias, including atrial fibrillation (AF).[9–12] Additionally, conditions that are largely inherited, such as ion channelopathies and arrhythmogenic cardiomyopathy, can be unmasked by exercise and are implicated in EASD.[13–15] Cardiac rhythm disturbances are commonly observed in horses engaged in high-intensity exercise such as racing, with studies reporting arrhythmias in 20%–84% of horses.[16–18] Studies investigating risk factors for EASD in horses have observed that both older[19–21] and younger[8] horses appear to be at higher risk, and familial arrhythmias have been described.[22] The risk of EASD appears to be many times higher in racehorses than in human athletes, and it is plausible that factors unique to equine physiology and arrhythmogenesis exist.[5,23]

Cardiac screening in human athletes has contributed to a substantial reduction in sports related deaths.[15] To improve horse welfare and jockey safety, a better understanding of the cardiac electrical events involved in EASD is required. A single report in the literature of a horse that died while wearing an ECG describes ventricular fibrillation following an apparent couplet of ventricular premature depolarisations.[24] Recently, wearable devices capable of ECG recording have become commonplace in racehorse training, and the evaluation of such recordings could assist in cardiac screening.[25–27] Currently, it is challenging to stratify the risk associated with observed ECG features, and while complex rhythm disturbances are thought to be more dangerous, this interpretation is based on expert opinion and not quantified.[28]

The aim of this study was to identify horses with EASD and evaluate ECGs collected during training from these horses.

2 | MATERIALS AND METHODS

Cases of EASD in Thoroughbred racehorses were identified from databases in Australia (Racing Australia), France (France Galop), the United Kingdom (British Horseracing Authority), and the United States of America (Horse Integrity and Safety Authority). Postmortem reports, when available, were reviewed and cross-referenced with the fatality list. If cases did not meet the International Federation of Horseracing Authorities EASD definition (fatal collapse of a previously presumed healthy horse during exercise or within 1 h of exercise) they were not included.[6] Identified horses were matched to the database of a wearable device that includes a single-lead ECG (Arioneo Equimetre, France), and 24 ECGs from 11 horses with EASD were analysed. ECG recordings during fast work and recovery were used for this study.

ECGs were evaluated manually and using electronic callipers by one author (CN), and automatic R markings in HRV software (Kubios version 4.1 and 4.2 for Windows; Kubios Oy, Finland) were corrected. A cut-off of R-R change of 5% was chosen to determine premature complexes based on exceeding normal variation at heart rates higher than 100 beats/min[29] expected during exercise. Premature complexes were defined as complexes for which the RR was ≥5% shorter than the preceding RR. These complexes were identified using the custom threshold beat correction setting at 0.01 and 0.02 s. As the HRV software rounds the custom setting to the closest first decimal point, electronic callipers (https://stanford.edu/~rogersaj/) were used to measure RR intervals identified with the custom setting at 0.01 s but not at 0.02 s, and calculate if the difference between this RR and the previous RR was ≥5%. Pre-processing HRV settings were set to the default values, except the detrending method, which was set as none for easier visualisation of the heart rate curve. Noise detection/correction was not used.

Arrhythmia description followed the criteria recently reported.[30] Briefly, the presence of any type and number of premature depolarisations (PDs) was defined as 'ARRHYTHMIA'. The presence of complex arrhythmia ($ARRHYTHMIA_{COMPLEX}$) was defined as the presence of multiform PDs, triplets, runs of PDs or atrial fibrillation (AF). The high intensity period of work including the rapid acceleration period was reported as 'Exercise'. The early recovery period was defined as the first 2 min after high intensity exercise that included the fast heart rate deceleration period. The late recovery period was defined as starting at the end of the early recovery period, lasting until the end of the recording (Figure 1). Premature depolarisations were described as either wide or narrow based on a subjective comparison with the appearance of the preceding QRS. Because the ECG system recorded only one lead and because P waves were inconsistently visible at high heart rates, PDs were not further classified as ventricular or supraventricular. Single PDs with or without a compensatory pause were also described. The pause category (compensatory or non-compensatory) was 'reader-determined' and obtained using manual and digital callipers on the ECG trace and the R-R tachogram. A compensatory pause was defined as a coupling interval + return cycle length equal or nearly equal to the sum of the previous 2 R-R intervals as previously described.[30] The peak heart rate during exercise (HR_{peak}) was the average rate of the 5 shortest RR intervals after excluding PDs and calculated by HRV software. While in AF all QRS complexes were considered for the calculation of HR_{peak}. Atrial fibrillation was defined as an irregular rhythm with a QRS morphology that remained unchanged in most QRS complexes compared to the normal QRS, the absence of P waves, and the presence of f waves at low heart rates. When the diagnosis of AF could not be made confidently, the rhythm was labelled as irregularly irregular tachycardia.

2.1 | Data analysis

Analyses were conducted with statistical software (R, R Foundation for Statistical Computing, Vienna, Austria). Tests of normal distribution (Shapiro–Wilk) were performed to determine the extent of skewness of continuous variables. Descriptive analyses included computation of means and standard deviations for normally distributed, and medians, interquartile ranges and ranges for non-normally

distributed continuous variables. Percentages, frequency counts and tabulation were used to describe categorical variables. Continuous outcome variables included duration of high-intensity exercise (duration from rapid heart rate acceleration to start of rapid deceleration), number of PDs during exercise, and peak heart rate (calculated over the average five shortest RR intervals). Categorical variables included arrhythmia (yes/no), arrhythmia or complex arrhythmia during high intensity exercise as described above (yes/no), arrhythmia or complex arrhythmia during early recovery (yes/no), frequent arrhythmia during late recovery (yes/no), and presence of AF (yes/no). The proportion of horses with frequent late recovery arrhythmias was described.

3 | RESULTS

Eleven cases of EASD were identified. There were 5 females and 6 geldings. At the time of death, the median age was 3 years, IQR 3–4 years [range 3–7 years]. There were 24 ECGs available: median 2, IQR 1–3 [range 1–4] ECGs/horse. ECGs were collected a median of 15.50 days, IQR 8.00–31.75 [range 0.00–462.00] days from the day of death. There were 4 ECGs from 4 horses the day of the EASD episode, 6 ECGs from 6 horses within 10 days of death, and 14 ECGs from 9 horses that were more than 10 days from the day of death.

A summary of the HR_{peak}, number of PDs during exercise and early recovery, proportion of horses with complex arrhythmias during exercise and early recovery, and proportion of horses with late recovery arrhythmias is presented in Table 1. Seventy percent of all recordings documented arrhythmias; 15%, 10%, and 25% of recordings presented complex arrhythmias in the 'exercise', early recovery, and late recovery period, respectively, and 17% of recordings displayed AF. Arrhythmias observed in each horse are summarised in Table S1.

3.1 | Recordings during the day of death

Four horses had ECG recordings during the fatal event. In three of these horses, the recording started at rest in AF and death occurred when the rhythm deteriorated into torsades-like ventricular tachy-

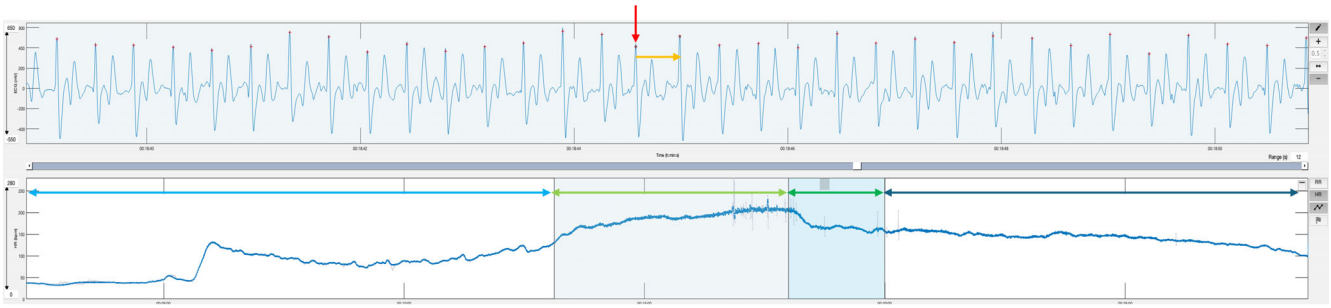

FIGURE 1 Heart rate tachogram and early recovery period electrocardiogram of a 3-year-old Thoroughbred gelding that experienced EASD 21 days after this recording. The resting and warm-up periods are marked by the light blue arrow. The acceleration and high intensity periods are marked by the light green arrow and are shaded in light blue. The early recovery period is marked with the dark green arrow and is shaded in dark blue. The late recovery period is marked by the dark blue arrow. The ECG shows a narrow PD (red arrow) with a compensatory pause (orange arrow). Complexes detected automatically as PDs using a 0.02 s custom threshold for beat correction are marked in the heart rate tachogram in grey.

TABLE 1 Summary details of the 24 ECGs obtained from 11 Thoroughbred racehorses with exercise associated sudden death (EASD).

	EASD ECGs ($n = 24$)		
Outcome	**Median**	**IQR**	**Range**
ECG duration (s)	135.5	101.8–173.2	65.0–453.0
HR_{peak} with AF (/min)	218.0	212.2–229.0	200.0–431.0
HR_{peak} without AF[a] (/min)	216.0	209.8–221.0	200.0–237.0
$nPD_{exercise}$	2.0	0.8–6.0	0.0–23.0
$nPD_{early\ recovery}$	2.0	0.0–3.0	0.0–77.0
	Number of ECGs	**Proportion**	
$ARRHYTHMIA_{exercise}$[a]	14	0.70	
$ARRHYTHMIA_{COMPLEX\ exercise}$[a]	3	0.15	
$ARRHYTHMIA_{COMPLEX\ early\ recovery}$[a]	2	0.10	
$ARRHYTHMIA_{COMPLEX\ late\ recovery}$[a]	5	0.25	
AF	4	0.17	

[a]Not including the four ECGs where the horse was in AF for the whole recording.

cardia, R on T phenomenon, and/or ventricular fibrillation (Figure 2). Two of the horses that displayed AF had 3 previous ECGs recorded 8–247 days prior to death and AF was not diagnosed in these ECGs. One of them, with recordings 8, 14, and 247 days prior to death, had 1–5 single narrow PDs during exercise and 0–2 narrow PDs in the early recovery period. The second, with recordings 17, 21, and 24 days prior to death, had 4–23 narrow PDs during exercise that included couplets (1 on day 17 and 3 on day 21 prior to death) and a run of 5 PDs (day 21 prior to death) and 2–9 PDs in the early recovery period that were single narrow PDs with the exception of one triplet of narrow PDs 21 days prior to death.

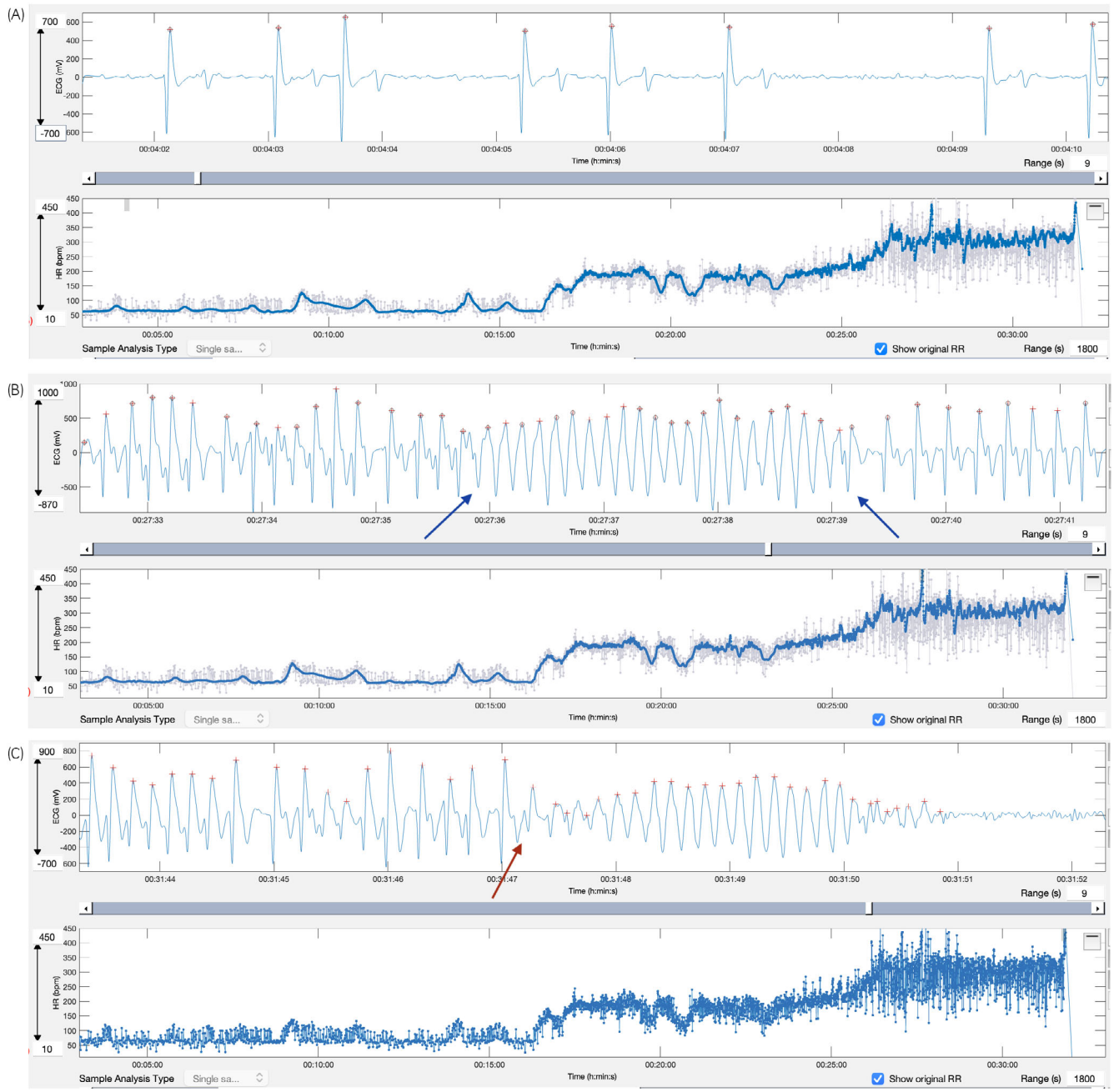

FIGURE 2 Cardiac rhythm in a 3-year-old Thoroughbred gelding that was in atrial fibrillation at the start of exercise and experienced EASD during the breeze. (A) Atrial fibrillation is apparent at the start of exercise. (B) Atrial fibrillation triggered an episode of ventricular ectopy and self-limiting torsades de pointe (between the blue arrows). (C) Atrial fibrillation triggered another episode of ventricular ectopy and torsades de pointe (starting at the red arrow) that deteriorated to ventricular fibrillation and death. The top panels are the ECGs, with the scale in mV on the Y-axis and time on the X-axis. The bottom panels are the heart rate tachograms, with heart rate in beats/min on the Y-axis and time on the X-axis. Note the difference in time scale for the ECG (9 s frame) and heart rate curve (1800 s frame). For consistency in figure formatting the 'beat correction' setting has been maintained as custom (0.02 s), while for heart rate calculations in horses in AF beat correction was set as 'none'.

In the fourth horse, the rhythm was normal at rest; there was 1 single narrow PD with a pause and a narrow couplet during warm-up, 3 narrow PDs with a pause during high-intensity exercise, and 4 narrow PDs with a pause during early recovery. PDs became frequent and complex in the late recovery period, ultimately deteriorating into ventricular fibrillation and death (Figure 3). This horse had

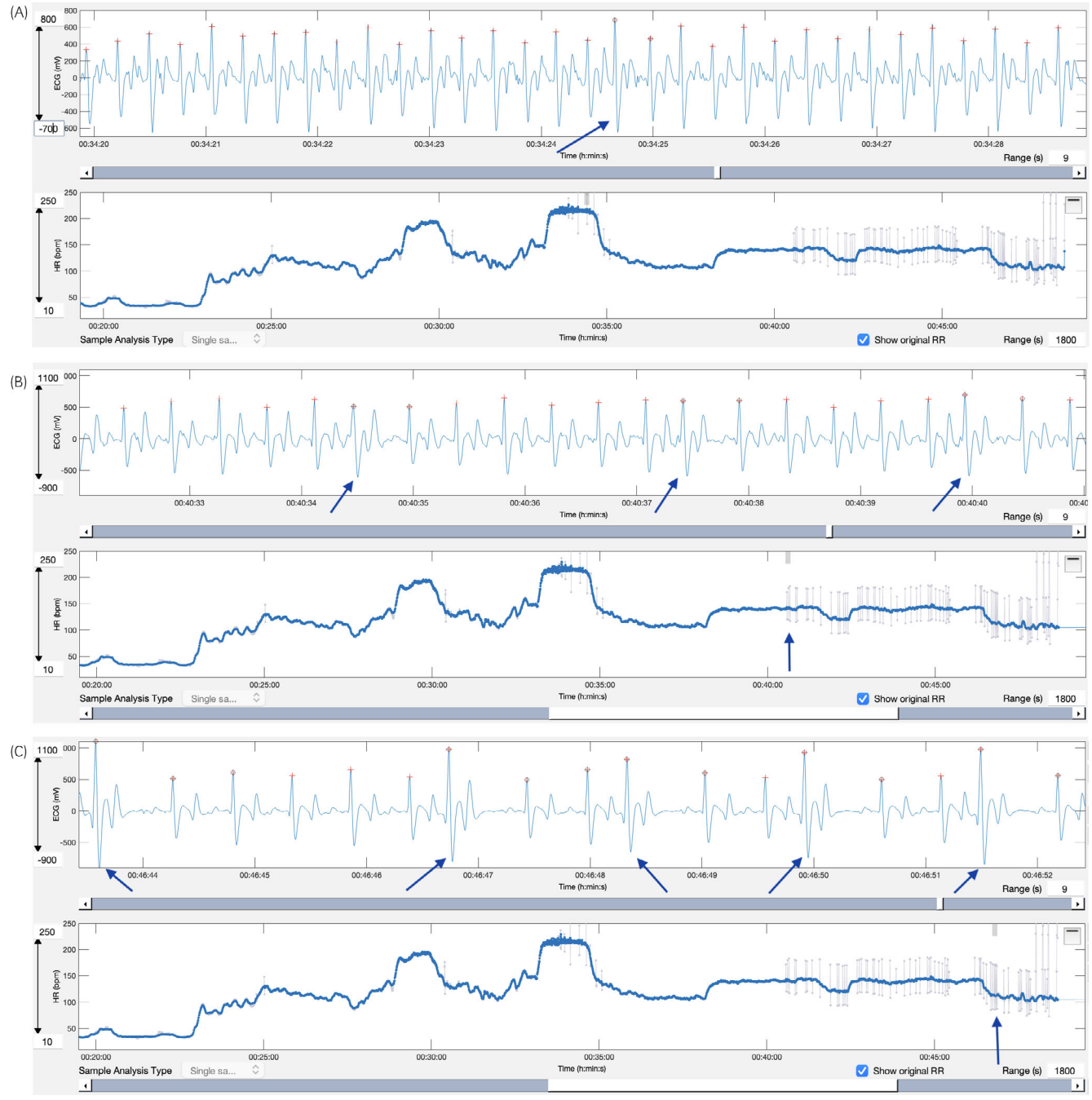

FIGURE 3 Cardiac rhythm in a 3-year-old Thoroughbred filly that experienced EASD in the late recovery period after a breeze. (A) Premature complex (blue arrow) in the late portion of the breeze. (B) Isolated premature complexes (blue arrows) are frequent in the late recovery period, beginning approximately 6 min after the breeze ended. (C) Premature complexes (blue arrows), both isolated and in a trigeminy pattern during late recovery, approximately 12 min after the breeze ended. (D) A premature depolarisation triggering a short run of self-limiting multiform wide complex tachycardia (between the red arrows) approximately 14 min after the breeze ended. (E) A ventricular premature depolarisation triggering multiform wide complex ventricular tachycardia, which deteriorated into ventricular fibrillation and death (starting at red arrow) approximately 14 min after the breeze ended (15 s after the first episode). Top panels are ECGs, with the scale in mV on the Y-axis and time on the X-axis. Bottom panels are heart rate tachograms, with heart rate in beats/min on the Y-axis and time on the X-axis. Note the difference in time scale for the ECG (9 s frame) and heart rate curve (1800 s frame). The vertical blue arrows in the bottom panels correspond to the time of the ECGs.

FIGURE 3 (Continued)

recordings 8 and 14 days prior that showed 2 narrow PDs during exercise and 3 narrow PDs in the early recovery period each. In the recording 14 days prior to death, in addition to the rhythm described above, there were many narrow and wide PDs in the late recovery period resembling the single PDs in the late recovery period seen the day of death and displayed in Figure 3.

3.2 | Recordings 1–10 days before death

Six horses, including two horses that were also recorded the day of death, had ECG recordings 1–10 days before death. One horse had AF throughout the exercise period. This was 9 days before the day of death; the horse was not recorded on the day of death. In this horse, 2 additional recordings obtained 47 and 51 days before death showed frequent late recovery PDs that were sometimes in runs of irregularly irregular RR and somewhat abnormal QRS morphology. In another horse, many narrow PDs and short runs of narrow irregular complexes in which QRSs were not preceded by visible P waves while the regular complexes were observed in the recovery period (Figure 4). This was the only available recording from this horse. In the other 4 horses with ECGs recorded between 1 and 10 days before death, there were 1–2 narrow PDs with pause during high-intensity exercise and 0–3 narrow PDs with pause in early recovery.

3.3 | Recordings more than 10 days before death

There were 14 ECGs from 11 horses in this period. The horse that was recorded on the day of death to have late recovery arrhythmias leading to EASD without AF had a recording 14 days earlier with single narrow and wide complexes similar to those that triggered ventricular fibrillation in the recording on the day of death, as described above. In the other horses, there was a variable total number of PDs (0–23 during high intensity exercise and 0–9 during the early recovery period) in all except one, which had no PDs 462 days before death. In the ECGs more than 10 days prior to EASD, there were complex arrhythmias in 3/20 recordings during high-intensity exercise and 1/20 in the early recovery period.

4 | DISCUSSION

Atrial fibrillation was a common underlying cardiac rhythm disturbance in horses with EASD in this case series. Three of 4 ECGs

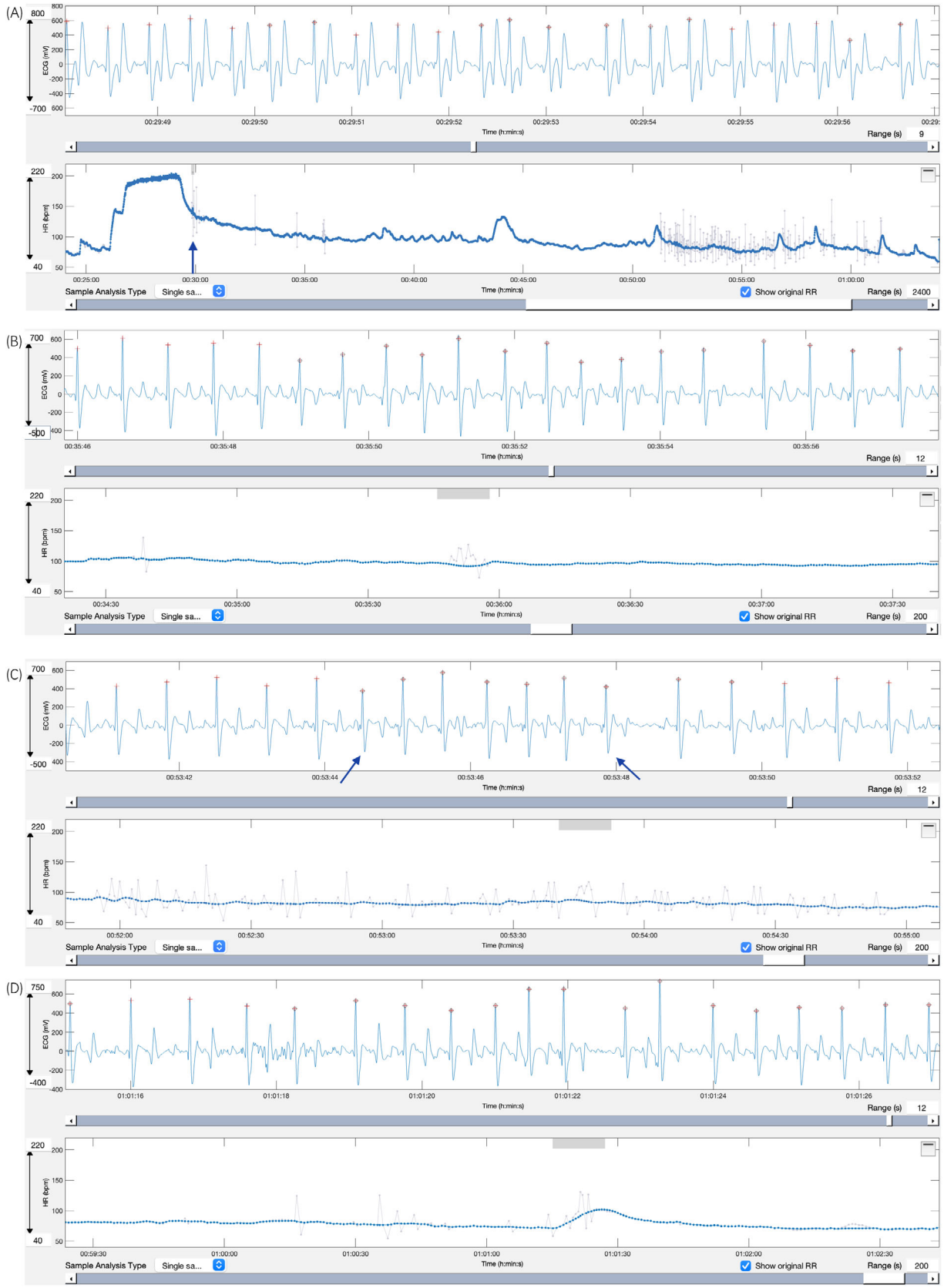

FIGURE 4 Legend on next page.

obtained during the fatal exercise event and one ECG from a different horse 9 days before death demonstrated AF throughout the recordings. Although AF is commonly observed in racehorses,[31–34] the high proportion of fatalities associated with pre-existing AF that was present at rest but unnoticed on the day of death was an unexpected finding. Publications of horses exercising on the treadmill or during lungeing while in AF have not reported fatalities associated with exercise.[35–38] However, abnormal QRS morphology is frequently observed in horses with AF, including during ridden exercise.[35,37–40] The origin of abnormal morphology is likely concurrent ventricular ectopy or aberrant conduction of a supraventricular impulse and is potentially associated with an increased risk for collapse.[38] Abnormal QRS morphology was seen more commonly after racing in horses with AF at heart rates exceeding 150 beats per minute[39] than at lower heart rates, and it has been suggested exercise inducing heart rates higher than 220/min should be avoided in horses with AF.[28]

Although EASD is an uncommon outcome in AF, horses with persistent AF, or those at high risk for recurrence, are recommended to have an evaluation for the presence of underlying disease, concomitant arrhythmias, and heart rate during exercise, if they are to continue their athletic career.[28] Horses that exercise at high intensities are unlikely to be able to do so successfully and safely while in AF. Screening of horses for AF prior to high-intensity exercise is potentially a high-yield effort in preventing incidents of collapse and EASD. Auscultation by veterinarians and trained personnel can detect AF. Smartphone-based ECG devices can obtain a resting ECG, and AI-based rhythm detection algorithms have shown satisfactory performance in identifying AF in horses[38,41] and show promise as a screening tool that can be applied by untrained personnel.

Arrhythmias after fast heart rate deceleration (i.e., in the late recovery period) were an interesting pattern present in one ECG on the day of death and in 4 ECGs from 3 horses (including the horse with late recovery period arrhythmias the day of death) before the day of death. These arrhythmias may also happen in horses that do not die suddenly. This finding was surprising, as arrhythmias in the recovery period, including complex arrhythmias, were found frequently in Standardbred racehorses recorded during and after racing that did not have any clinical signs or EASD.[16] These findings could reflect differences between breeds or disciplines, or that the early and late recovery periods may have different physiology affecting arrhythmogenesis. Arrhythmias in the recovery period were found to be more predictive of structural heart disease in humans than arrhythmias at rest or during peak exercise.[42] More information about late recovery arrhythmias is needed to make definitive statements about risk stratification in horses.

Limitations of this study include that the ECGs evaluated were single-lead recordings taken with a wearable device, and changes in morphology not obvious in a single lead that uses motion artefact correction, or some arrhythmias such as accessory pathways, could be missed. The speed, duration, and relationship of the recordings to the time of death were variable. In addition, the size of the group is too small to draw a conclusion about risk stratification of arrhythmias with the possible exception of AF detected before exercise. Different types of analysis, such as complexity analysis,[43] restitution analysis,[44–46] or heart rate variability (HRV)[47–49] could be interesting additions to the rhythm evaluation we performed.

In conclusion, a high percentage of this group of horses with EASD displayed AF on the day of death, and arrhythmias in the late recovery period were an overrepresented pattern before the day of death. A workflow that enables the easy and scalable detection of AF in horses participating in high-intensity athletic activities may be a high-yield effort. Risk stratification for other types of arrhythmias with particular attention to the late recovery period needs further evaluation.

FUNDING INFORMATION

This study was supported by the HISA—Horseracing Integrity and Safety Authority. A National Institutes of Health (NIH) K12 award supported by 1UM1TR004405-01A1 Blazar (PI) and K12TR004373 Ingbar (PI) (salary support to S. Durward-Akhurst).

ACKNOWLEDGEMENTS

We thank Guillaume Dubois (Arioneo), Theo Niefer (Arioneo), Valentin Rapin (Arioneo), Karen Hassan (Horseracing Integrity and Safety Authority), Grace Forbes (Racing Australia), Sally Taylor (British Horse Racing Authority), Eoin Vaughan (British Horse Racing Authority), and Alberic Thery (France Galop).

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

C. Navas de Solis: Conceptualization; investigation; funding acquisition; writing – original draft; methodology; validation; writing – review and editing; data curation; project administration. **M. Durando:** Conceptualization; investigation; writing – original draft; methodology; writing – review and editing; validation; data curation. **L. Nath:**

FIGURE 4 Cardiac rhythm post-exercise in a 7-year-old Thoroughbred gelding. This horse experienced EASD in a race 4 days after this exercise ECG recording. In this recording, in the recovery period, he had a prominent paroxysmal, narrow, irregularly irregular tachyarrhythmia. (A) Early recovery showing paroxysmal, irregularly irregular tachyarrhythmia. The vertical blue arrow in the bottom panel corresponds to the time of the ECG. (B–D) Late recovery showing short runs of irregularly irregular, narrow complex tachycardia that did not have visible P waves. The horizontal shaded grey bars at the top of the heart rate tachograms correspond to the time of the ECGs. Top panels are the ECGs, with the scale in mV on the Y-axis and time on the X-axis. Bottom panels are the heart rate tachograms, with heart rate in beats/min on the Y-axis and time on the X-axis. Note the difference in time scale for the ECG (9 s frame) and variable scales for the heart rate tachograms. The heart rate tachogram scales are displayed at the bottom right (200–2400 s).

Conceptualization; investigation; writing – original draft; writing – review and editing; methodology; validation; data curation. **S. Durward-Akhurst:** Conceptualization; investigation; funding acquisition; writing – original draft; methodology; validation; writing – review and editing; formal analysis; project administration; data curation.

DATA INTEGRITY STATEMENT

S. Durward-Akhurst and C. Navas de Solis had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of data analysis.

ETHICAL ANIMAL RESEARCH

The study was approved under the University of Minnesota IACUC protocol 2406-42168A and the University of Pennsylvania protocol POAP 806975.

INFORMED CONSENT

Trainers gave consent for the use of anonymised data for research purposes.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1001/evj.70166.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available upon reasonable request from the corresponding author. Open data sharing exemption granted by the editor.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Navas de Solis C, Durando M, Nath L, Durward-Akhurst S. Exercising electrocardiograms from Thoroughbred racehorses with exercise associated sudden death. Equine Vet J. 2026. https://doi.org/10.1002/evj.70166