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ORIGINAL ARTICLE

Premature ventricular complexes during continuous electrocardiographic monitoring in the intensive care unit: Occurrence rates and associated patient characteristics

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Abstract

Aims and objectives: This study examined the occurrence rate of specific types of premature ventricular complex (PVC) alarms and whether patient demographic and/or clinical characteristics were associated with PVC occurrences.

Background: Because PVCs can signal myocardial irritability, in-hospital electrocardiographic (ECG) monitors are typically configured to alert nurses when they occur. However, PVC alarms are common and can contribute to alarm fatigue. A better understanding of occurrences of PVCs could help guide alarm management strategies.

Design: A secondary quantitative analysis from an alarm study.

Methods: The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist was followed. Seven PVC alarm types (vendor-specific) were described, and included isolated, couplet, bigeminy, trigeminy, run PVC (i.e. VT>2), R-on-T and PVCs/min. Negative binomial and hurdle regression analyses were computed to examine the association of patient demographic and clinical characteristics with each PVC type.

Results: A total of 797,072 PVC alarms (45,271 monitoring hours) occurred in 446 patients, including six who had disproportionately high PVC alarm counts (40% of the total alarms). Isolated PVCs were the most frequent type (81.13%) while R-on-T were the least common (0.29%). Significant predictors associated with higher alarms rates: older age (isolated PVCs, bigeminy and couplets); male sex and presence of PVCs on the 12-lead ECG (isolated PVCs). Hyperkalaemia at ICU admission was associated with a lower R-on-T type PVCs.

Conclusions: Only a few distinct demographic and clinical characteristics were associated with the occurrence rate of PVC alarms. Further research is warranted to

At the time of the study, Dr. Suba was a doctoral student at UCSF School of Nursing.

examine whether PVCs were associated with adverse outcomes, which could guide alarm management strategies to reduce unnecessary PVC alarms.

Relevance to clinical practice: Targeted alarm strategies, such as turning off certain PVC-type alarms and evaluating alarm trends in the first 24 h of admission in select patients, might add to the current practice of alarm management.

KEYWORDS
alarm fatigue, arrhythmia, clinical alarm, electrocardiographic monitoring, intensive care unit, premature ventricular complex

1 | INTRODUCTION AND BACKGROUND

When electrocardiographic (ECG) monitoring was introduced into the intensive care unit (ICU) in the 1960s, premature ventricular complexes (PVCs) were identified as one of the most common arrhythmias among patients with myocardial infarction (MI) (Meltzer & Kitchell, 1966). It was generally accepted that PVCs indicated electrical irritability of the heart and might forewarn a lethal ventricular arrhythmia (i.e. ventricular tachycardia [VT] or ventricular fibrillation [VF]) (Lown et al., 1967; Meltzer & Kitchell, 1966). In addition, it was hypothesised that as the frequency of PVCs increased, so too did the likelihood of subsequent VT and/or VF (Meltzer & Kitchell, 1966). Therefore, prompt identification of PVCs followed by pharmacologic therapy (antiarrhythmics) was standard practice with the goal of improving prognosis (Lown et al., 1967). As a result, PVC detection algorithms were incorporated into hospital-based ECG monitoring devices (Meltzer & Kitchell, 1966). However, the Cardiac Arrhythmia Suppression Trial (CAST) published in 1989, showed that treatment of PVCs after acute MI with class IC antiarrhythmic drugs (i.e. encainide, flecainide and moricizine) was unexpectedly associated with a higher rate of mortality as compared to placebo (Cardiac Arrhythmia Suppression Trial, 1989). As a result, the practice of aggressive pharmacological treatment to suppress PVCs was no longer standard practice, which remains true today.

The most recent American Heart Association (AHA) Practice Standards for In-hospital ECG Monitoring identified PVCs as not 'immediately life-threatening'. Therefore, the AHA recommended that in the absence of other significant indications (e.g. origin of VT), continuous PVC monitoring is not required (Class IIb); rather, it could be 'considered' during monitoring (Sandau et al., 2017). Despite this recommendation, it is a common practice to configure bedside ECG monitors with PVC alarms enabled (audible or inaudible). Moreover, monitoring manufacturers generally offer a variety of algorithms for PVC detection. For example, common types of available PVC algorithms include isolated PVCs, bigeminy, trigeminy, couplets, run PVCs (i.e. VT >2), number/minute and R-on-T type. As a result, hospitals often configure bedside monitors to alarm for all of the available PVC types, with some made audible and others inaudible.

Enabling PVC alarms, while a common practice, can have drawbacks in some patients. For example, patients with acute coronary syndrome (ACS) (Winkler et al., 2013), structural heart disease (Lee et al., 2019; Marcus, 2020) or acute exacerbation of chronic obstructive pulmonary disease (Einvik et al., 2017), often generate frequent PVC alarms, which can contribute to alarm fatigue (i.e. desensitisation, ignoring alarms, unsafe alarm adjustments). Unfortunately, clinically important PVCs (e.g. new onset, more frequent or R-on-T) can be missed because they become buried within the 'noise' of frequent PVC alarms that are not clinically actionable. Among hospital-based investigations, two research studies (Cvach et al., 2015; Gazarian, 2014) and four quality improvement projects (De Vaux et al., 2017; Graham & Cvach, 2010; Sendelbach et al., 2015; Srinivasa et al., 2017) reported on PVC alarms but did not examine specific numbers or types. However, one comprehensive observational study identified PVCs as the most common type of alarm. In 461 consecutively enrolled ICU patients with over 2.5 million total alarms, 33% (n = 854,901) were PVCs (Drew et al., 2014). While this study illustrates the sheer number of PVC alarms in an ICU population, the investigators did not report on the specific PVC types or examine whether PVCs were associated with patient demographic and/or clinical characteristics. A better understanding of PVC occurrence rates and associated patient and/or clinical characteristics could help guide alarm management strategies in select patients.

Therefore, the purpose of this study was threefold: (1) determine the number and type of seven different PVC types generated during continuous bedside ECG monitoring in the ICU, including isolated (single), couplets, bigeminy, trigeminy, run PVCs (VT >2), R-on-T and PVCs/minute; (2) determine the distribution of the seven PVC types by patient demographic (i.e. age, sex, race/ethnicity) and clinical characteristics (i.e. medical history, diagnosis, hospital 12-lead ECG,

What does this paper contribute to the wider global clinical community?

- PVC alarms are frequent during continuous ECG monitoring and often are generated by a small subset of patients in the ICU.
- Assessment and evaluating whether to turn on the alarm for PVCs for individual patients is important to minimise potentially insignificant alarms.
- Nurses have a critical role in leading the effort in alarm management that is tailored to individual patients.

ejection fraction, serum potassium and magnesium at ICU admission); and (3) test whether demographic and/or clinical characteristics are associated with each PVC type.

2 | METHODS

This manuscript was prepared following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) (University of Bern, 2009) (File S1).

2.1 | Study design

This is a secondary data analysis from an alarm study previously published (Drew et al., 2014). Briefly, the study was a single-centre, prospective observational study that collected all physiologic (e.g. ECG, vital signs) and alarm data from all of the 77 adult ICU bedside monitors during a 1-month period in 2013. The 77 ICU beds (16 cardiovascular, 32 medical-surgical and 29 neurological/neurosurgery) were equipped with a 5-lead ECG monitor (Solar 8000i, version 5.4 software, GE Healthcare, Milwaukee, WI). A secure network connected all of the monitors, including the central monitoring station, to a data capture system (CARESPACE Gateway, GE Healthcare, Milwaukee, WI), which allowed the data to pass securely to an institution-approved research server for offline and retrospective analyses.

2.2 | Ethical consideration

In the primary study, the University Institutional Review Board (IRB #12-09723) approved the study with a waiver of informed consent. The study was designed to collect data from existing bedside patient physiologic monitors, which is standard of care for all ICU patients. Importantly, the data collection and storage process occurred in the background using a secure network without the need for direct contact with patients or clinicians. Therefore, the study did not interfere with patient care, and the data collected for the study was not available for clinical decision-making. The same IRB approved the secondary data analyses reported in this study.

2.3 | Premature ventricular complexes alarms

A total of seven types of PVC alarms were examined, as defined by the vendor, and included: (1) *isolated* (single PVC); (2) *couplet* (two consecutive PVCs >100 beats per minute); (3) *bigeminy* (PVC alternates with a non-ventricular beat for ≥3 cycles); (4) *trigeminy* (PVC alternates with 2 non-ventricular beats for ≥3 cycles); (5) *run PVCs* (3–5 consecutive ventricular beats ≥100 beats/min); (6) *R-on-T* (PVC falls on the ST or T-wave portion of the previous beat); and (7) *PVCs/min* based on a user-defined limit. Our hospital configured this alarm for ≥10 PVCs/minute. The algorithm adds together any of the PVC types (isolated plus couplets, etc.) to calculate PVCs/minute.

2.4 | Patient data

All consecutive patients admitted to the adult ICUs during the study period was included in the primary study. Demographic and clinical characteristics were collected from the hospital's electronic health record (EHR) (Epic, Verona, WI). Demographic data obtained included age, sex, race and ethnicity. The clinical characteristics selected for analysis were based on prior research (Dukes et al., 2015; Marcus, 2020; Nguyen et al., 2017) showing an association of these conditions and PVCs and included: a history of ischaemic heart disease (IHD), heart failure (HF), prior percutaneous coronary intervention (PCI)/stent, prior coronary artery bypass graft (CABG) surgery, stroke and atrial fibrillation. In addition, we collected the following clinical data: a hospital acquired standard 12-lead ECG obtained within 24 h of ICU admission, serum potassium and magnesium level at ICU admission, left ventricular ejection fraction (LVEF) and primary ICU diagnosis.

2.5 | Operational definitions used for clinical variables

The standard 12-lead ECG, obtained within 24 h of ICU admission, was used to identify the presence of PVCs and/or atrial fibrillation. All of the 12-lead ECGs were over-read by a board-certified cardiologist. If a 12-lead ECG had not been obtained within the first 24 h of ICU admission, a standard 12-lead ECG obtained in the emergency department or step-down/medical-surgical floor (for ICU transfer patients) within 24 h prior to ICU admission was used. Serum potassium and magnesium levels at ICU admission were also obtained. The absolute laboratory value as well as standard categorical labels were used. For potassium, the following categories included: normal (3.8–5.1 mEq/L), hypokalaemia (<3.8 mEq/L) and hyperkalaemia (>5.1 mEq/L). For magnesium, the following categories included: normal (1.8–2.4 mg/dl), hypomagnesaemia (<1.8 mg/dl), and hypermagnesaemia (>2.4 mg/dl). LVEF was obtained from an echocardiogram obtained during hospitalisation. If an echocardiogram was not available for the present admission, an echocardiogram obtained within 6 months prior to admission was used. Both the numeric LVEF (%) and categorical evaluation were obtained, and included hyperdynamic LVEF of >70%, normal 50%–70%, mild dysfunction 40%–49%, moderate dysfunction 30%–39% and severe dysfunction <30% (American College of Cardiology, 2014). Lastly, ICU diagnosis was categorised into the following: cardiovascular, medical-surgical or neurological/neurosurgical.

2.6 | Statistical analysis

The frequency of all seven types of PVC alarms was tabulated. The median and interquartile range (IQR) were calculated to describe the distribution of each PVC type across patient demographic and clinical characteristics. We report PVCs/minute in our descriptive analysis to show its occurrence rate but did not include this PVC type in the patient and clinical characteristic analyses because the PVCs for this alarm are additive; thus, would not be useful to identify specific PVC types. In clinical practice, it is not uncommon for select patients to have frequent PVCs while others may have very few or none. Therefore, we examined PVC *counts* (total number of PVCs per patient) using modelling strategies, which allowed us to account for excess zero values.

A total of 19 demographic and clinical variables were examined for associations for each of the six types of PVCs. For isolated PVCs, we conducted a negative binomial regression analysis using the R package *MASS v7.3.50* (Venables & Ripley, 2002), and tested coefficients with a non-parametric bootstrap (5000 replicates), using bias-corrected and accelerated (BCa) 95% confidence intervals (CIs) (Canty & Ripley, 2014; Davidson & Hinkley, 1997; DiCiccio & Efron, 1996). For the counts of bigeminy, trigeminy, couplets, run PVC and R-on-T type PVCs, we conducted a hurdle regression analysis (Zeileis et al., 2008) using the R package *pscl v1.5.2* (Jackman, 2017) to account for the excess zeros in these distributions. The hurdle model consisted of two components: (1) a logistic regression to examine whether or not an individual had a PVC alarm(s); and (2) a zero-truncated negative binomial regression to model the PVC counts in individuals who had one or more PVC alarms. Coefficients from this hurdle model were again tested using a non-parametric bootstrap. We first present the univariate models for each variable against each PVC type. Results were considered *significant* if they reached $p < .00044$ (i.e. alpha-level of 0.05 divided by 114) to account for 114 multiple comparisons of the six PVC types by 19 demographic and clinical characteristics.

Finally, we conducted a stepwise regression model for each type of PVC where we added one variable at a time into the model. Here, we used a bootstrap multiple imputation approach (500 bootstraps, each bootstrap using 20 multiple imputations) (Schomaker & Heumann, 2018) to appropriately handle missing data. Because some patients had extreme PVC counts, which had an impact on the magnitude of the effect size for some variables, we used the following approach. To understand the effects of each variable and the influence of outliers on them, we fit the model by including all of the patients and then removing those with a standardised residual from the negative binomial model using a threshold of ≥3 (iterated until none remained). We also examined demographic and clinical characteristics in the outlier patients to help explain patient level factors associated with the extreme counts observed. All analyses were computed using R v3.4.1 (R Core Team, 2017).

3 | RESULTS

3.1 | Premature ventricular complex alarm distribution

A total of 446 ICU patients were included. Detailed patient characteristics are shown in Table 1. The mean age was 59.97 ± 17.03 years. Of the total sample, 244 were male (54.7%) and 202 were female (45.3%), 118 (26.5%) were Non-White, and 270 (60.5%) were White. A small proportion (8.3%–16.1%) had a prior history of cardiac pathology (i.e. IHD, HF, PCI/stent/CABG, stroke and atrial fibrillation). The distribution of the primary diagnosis of the 446 ICU patients was as follows: 98 cardiovascular (22%), 170 neurological/neurosurgical (38.1%) and 178 medical-surgical (39.9%). Missing data was noted for the following variables: hospital-acquired standard 12-lead ECG (n = 95, 21.3%), serum potassium (n = 4, 0.9%), serum magnesium (n = 144, 32.3%) and LVEF (n = 273, 61.2%) because these test were not done in some of the ICU patients. In patients who had a standard 12-lead ECG available, 24 (5.4%) had PVCs and 35 (7.8%) had atrial fibrillation.

There were a total of 797,072 PVC alarms during 45,271 hours of ECG monitoring, or 17.6 PVC alarms/hour of ICU monitoring. Table 2 shows the distribution of the seven types of PVC alarm. Isolated PVCs were the most frequent (n = 646,665, 81.13%), while R-on-T type PVCs were the least frequent (n = 2321, 0.29%). Table 3 shows the distribution (median, IQR) of the seven PVC types based on demographic and clinical characteristics. There was a higher median count of isolated PVCs in patients who were older or male. In addition, patients with a history of IHD, HF, prior PCI/CABG, stroke or atrial fibrillation had a higher median count of both isolated PVCs and couplets than those without these clinical histories. Also, patients with moderate/severe LVEF dysfunction had a higher median count of isolated PVCs as compared to those in the other LVEF categories. In patients with a cardiovascular diagnosis (n = 98; 22%), the median count of isolated PVCs and couplets was higher than those with a medical/surgical or neurological diagnosis.

All of the 446 patients had at least one PVC alarm. There were, however, 42 (9.4%) patients who were categorised as outliers. Six (14%) of the 42 patients had disproportionately high alarm counts, contributing to 40% (n = 320,342) of the total number (n = 797,072) of PVC alarms. One patient, in particular, had 153,347 alarms (19%), including 124,944 isolated PVCs during 744 hours of monitoring. The patient was an 80-year-old female with a significant cardiac history including diastolic HF, chronic atrial fibrillation, hypertension and mitral valve replacement.

3.2 | Factors associated with premature ventricular complex occurrence rates

Given the findings from the descriptive analysis with regards to outliers, we examined demographic and clinical associations with and

TABLE 1 Demographic, past medical history and baseline clinical characteristics at intensive care unit (ICU) admission in 446 patients

Characteristics	*N* (%)
Age, mean ± SD, year	59.97 ± 17.03
Sex	
Female	202 (45.3)
Male	244 (54.7)
Race	
Asian	76 (17)
Black or African American	34 (7.6)
Native Hawaiian or Pacific Islander	8 (1.8)
White	270 (60.5)
Unable or decline to state	58 (13)
Ethnicity	
Hispanic or Latino	49 (11)
Not Hispanic or Latino	390 (87.4)
Unable or decline to state	7 (1.6)
Past medical history	
Ischaemic heart disease	72 (16.1)
Heart failure	44 (9.9)
PCI/stent/CABG	39 (8.7)
Stroke	37 (8.3)
Atrial fibrillation	46 (10.3)
Clinical characteristics at ICU admission[a]	
12-lead ECG	
PVCs present	24 (5.4)
Atrial fibrillation	35 (7.8)
Serum potassium mEq/L, mean ± SD	4.13 ± 0.75
Normal	283 (63.5)
Hypokalaemia	125 (28)
Hyperkalaemia	34 (7.6)
Serum magnesium mg/dl, mean ± SD	1.97 ± 0.38
Normal	177 (39.7)
Hypomagnesaemia	90 (20.2)
Hypermagnesaemia	35 (7.8)
LVEF %, mean ± SD	57.86 ± 17.29
Normal	85 (19.1)
Hyperdynamic	40 (9.0)
Mild dysfunction	17 (3.8)
Moderate–severe dysfunction	31 (7.0)
Primary ICU diagnosis	
Cardiovascular	98 (22)
Medical-surgical	178 (39.9)
Neurological/neurosurgical	170 (38.1)

Abbreviations: CABG, coronary artery bypass graft; ECG, electrocardiogram; ICU, intensive care unit; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; PVC, premature ventricular complex; SD, standard deviation.

[a]The proportions, means and standard deviations are calculated excluding patients with missing data as follows: 12-lead ECGs to determine presence of PVC and/or atrial fibrillation (n = 95, 21.3%), serum potassium (n = 4, 0.9%), serum magnesium (n = 144, 32.3%) and baseline LVEF (n = 273, 61.2%). Serum potassium categories: normal (3.8–5.1 mEq/L), hypokalaemia (<3.8 mEq/L), and hyperkalaemia (>5.1 mEq/L). Serum magnesium categories: normal (1.8–2.4 mg/dl), hypomagnesaemia (<1.8 mg/dl), and hypermagnesaemia (>2.4 mg/dl). LVEF categories: hyperdynamic (>70%), normal (50%–70%), mild dysfunction (40%–49%), moderate dysfunction (30%–39%) and severe dysfunction (<30%).

without outliers (see Section 2). The number of outlier patients of the count model dataset for each PVC type were as follows: isolated PVCs n = 21 (4.7%), bigeminy n = 11 (6.4%), trigeminy n = 13 (8.7%), couplets n = 11 (2.8%), run PVCs n = 11 (3.5%) and R-on-T n = 9 (5.5%). Below are results for the regression and stepwise analysis with and without outliers.

Table 4 shows the univariate regression analysis of PVC count outcomes with outliers removed. A higher rate of isolated PVCs was associated with age (IRR 1.05, 95% CI 1.04–1.06), history of IHD (IRR 2.98, 95% CI 2.00–4.26), history of HF (IRR 2.30, 95% CI 1.38–3.37) and the presence of PVCs on a hospital acquired standard12-lead ECG (IRR 2.80, 95% CI 2.00–4.68). Age (IRR 1.02, 95% CI 1.01–1.03) and PVCs on the hospital acquired standard 12-lead ECG (IRR 2.18, 95% CI 1.49–3.32) were significant factors for a higher rate of couplets. Hyperkalaemia at admission was associated with a lower rate of R-on-T type PVCs (IRR 0.06, 95% CI 0.02–0.12). No associations were seen for the other PVC types (bigeminy, trigeminy and run PVC). When outliers were included, significant associations by the type of PVC were: (a) isolated PVCs: age, history of IHD and atrial fibrillation, and presence of PVCs on the standard 12-lead ECG; (b) bigeminy: age; (c) trigeminy: hyperkalaemia at ICU admission; (d) couplets: presence of PVCs on the 12-lead ECG and (e) run PVC: neurological/neurosurgical diagnosis. No associations were found for R-on-T type PVCs. Table S1 shows the results of the univariate regression analysis with the outliers included.

In the stepwise regression analysis with outliers removed from the model (Table 5), older age (IRR 1.04, 95% CI 1.03–1.05), male sex (IRR 1.94, 95% CI 1.40–2.84) and presence of PVCs on the hospital acquired standard 12-lead ECG (IRR 2.85, 95% CI 1.83–4.30) were found to be independent factors associated with higher rates of isolated PVCs. Only older age was associated with a higher rate of bigeminy (IRR 1.04, 95% CI 1.02–1.06) and couplets (IRR 1.02; 95% CI 1.01–1.03). Hyperkalaemia at ICU admission was associated with a lower rate of R-on-T type PVCs (IRR 0.07, 95% CI 0.03–0.17). There were no associations for trigeminy and run PVCs. Since LVEF was available in only 38.8% (n = 173 patients) this variable was excluded from the stepwise fit analysis. When outliers were included, significant associations by the type of PVC were: (a) isolated PVCs: age and PVC on hospital acquired 12-lead ECG; (b) bigeminy: age; (c) trigeminy: hyperkalaemia at ICU admission; (d) couplet: PVC on hospital acquired 12-lead ECG; (e) run PVCs: neurological/neurosurgical diagnosis. Table S2 shows the results of the univariate regression analysis with the outliers included.

4 | DISCUSSION

To our knowledge, this is the first comprehensive ICU-based study that has examined the occurrence rate of seven types of PVCs during continuous ECG monitoring. The main findings of our study are as follows: (1) every patient in our sample had at least one PVC alarm; (2) PVCs are frequent, with 797,072 PVC alarms during 45,271 h, or 17.6 PVC alarms/h of ICU monitoring; (3) over three-quarters of the total were isolated PVCs; (4) fewer than 1% were

TABLE 2 Distribution of 797,072 premature ventricular complex (PVC) alarms in 446 intensive care unit (ICU) patients

Alarm type	Total # of alarms (%)	Mean (Min-Max)	Median (Q_1, Q_3)	*n* patients with zero count (%)
Isolated PVC	646,665 (81.13)	1449.92 (0–124,944)	108.5 (17.25, 653.5)	2 (0.45)
Couplet	43,907 (5.51)	98.45 (0–11,778)	8 (3, 33)	47 (10.54)
Bigeminy	22,164 (2.78)	49.70 (0–8035)	0 (0, 2)	275 (61.66)
Trigeminy	18,513 (2.32)	41.51 (0–4898)	0 (0, 1)	297 (66.59)
Run PVC (VT>2)	12,595 (1.58)	28.24 (0–3866)	2 (0, 8.75)	135 (30.27)
R-on-T	2321 (0.29)	5.20 (0–372)	0 (0, 1)	283 (63.45)
PVC/minute	50,907 (6.39)	114.14 (0–7545)	6.5 (1, 53)	96 (21.52)

R-on-T type PVCs; (4) fewer than 10% of the sample ($n = 42$) generated most of the PVC alarms, with six patients generating 40% of the total number; and (5) demographic and clinical characteristics associated with PVCs include older age, being male and the presence of PVCs on a hospital acquired 12-lead ECG within the first 24 h of admission.

Our study shows that ICU clinicians will encounter frequent PVCs alarms and they will likely be concentrated in a small sub-set of patients. Our findings build on prior PVC studies (Drew et al., 2014; Sendelbach et al., 2015; Srinivasa et al., 2017); however, only one reported the total number (Drew et al., 2014), and none reported on the specific types of PVCs and none examined associated patient and/or clinical characteristics. We found that isolated PVCs were by far the most frequent type, accounting for 81% of the total number. Whereas, R-on-T type PVCs, which clinicians carefully monitor due to their potential to initial VT/VF, were rare and accounted for <1% of the total. We were somewhat surprised that the remaining PVC types were infrequent, ranging from 6% (PVC/minutes) to 2% (VT>2). However, it is worth noting that the maximum number for all types was high (Table 2), illustrating that select patients had extremely high numbers of PVC alarms. For example, one patient in our sample generated a total of 153,347 (19% of total) PVC alarms and 124,944 (81%) of these were isolated PVCs. It is noteworthy that this patient was monitored for the entire 31-day study period (744 monitoring hours), which means this patient had 206 PVC alarms/hour of monitoring. This patient had a history of mitral valve replacement and atrial fibrillation, both of which are associated with chronic PVCs (Abbadi et al., 2014; Alqarawi et al., 2018; Basso et al., 2019). Another patient had 11,778 couplet alarms, again highlighting frequent alarms in select patients. In fact, we found that almost 10% ($n = 42$) of the sample were outliers with 40% of the total PVC alarms concentrated in 14% ($n = 6$) of these patients. Prior studies have identified outlier patients for both arrhythmia and other physiologic alarms (e.g. vital signs), a problem described as 'alarm flood' (Allan et al., 2017; Drew et al., 2014; Harris et al., 2017; Nguyen et al., 2020; Yeh et al., 2020). To reduce alarm fatigue in this situation targeted alarm strategies (e.g. turning off specific PVC alarms) in these select patients should be discussed during daily clinical rounds. This problem will be compounded in patients with a long ICU stay, which should also be factored into decision making when addressing alarm reduction strategies.

While one could argue that because some of the PVCs we examined were configured in the bedside monitor as an inaudible text message (i.e. flash on the bedside monitor), these types of alarms/alerts do not contribute to alarm fatigue. However, in a qualitative study exploring nurses' perceptions of clinical alarms, nurses reported that flashing text alerts do catch their attention and cause them to wonder if something is wrong with the patient and if an action/intervention is indicated (Simpson & Lyndon, 2019). Based on our findings, PVC alarms are likely to be a major diversion from patient care because of their frequency. Anecdotally, we found some instances where the PVC alarms had been changed from an inaudible text alert to a low-priority alarm level (one-beep alarm). However, because of the retrospective nature of our study, we are unable to explain the rationale for these alarm adjustments. Nevertheless, this adjustment will only enhance alarm fatigue and suggests that education about alarm settings would be useful. It is worth noting that some monitoring manufacturers do not offer a configuration setting for inaudible text message alerts, rather the alarm is either on or off. In this situation, clinical teams that decide alarm configurations for hospital-based ECG monitors must weigh the potential alarm burden created by making PVC alarms audible. Future studies are needed to fully examine the impact of both inaudible and audible PVC alarms on both nurses and providers as well as inadvertent PVC alarm adjustments (i.e. changing from inaudible to audible). It should be noted that we did not examine patient outcomes; hence, future investigations are also needed to determine the clinical significance of PVC alarms, which is prudent before any broad recommendations about alarm adjustments/settings can be made.

In our study, we found that only a few distinct demographic and clinical characteristics were associated with PVC alarms. For example, older age, male sex and the presence of PVCs on a hospital acquired standard 12-lead ECG were factors associated with higher alarm counts for isolated PVCs. Older age was associated with higher alarm counts for bigeminy and couplets. Whereas, hyperkalaemia at ICU admission was associated with a lower count of R-on-T type PVCs. One consistent characteristic associated with isolated PVCs, in both univariate and the stepwise regression analysis, was the presence of PVCs on the hospital acquired standard 12-lead ECG. Although a standard 12-lead ECG only records a 10-second snapshot of information, the presence of PVCs on this

TABLE 3 Distribution of seven premature ventricular complex (PVC) types in 446 intensive care unit patients based on demographic and clinical characteristics using median and interquartile range (Q_1 and Q_3)

	Median (Q_1, Q_3)						
Characteristics	**Isolated PVC**	**Couplet**	**Bigeminy**	**Trigeminy**	**R-on-T**	**Run PVC**	**PVC/min**
Age group, years							
19–48	21 (4.8, 151)	3 (1, 16.2)	0 (0, 1)	0 (0, 0)	0 (0, 1)	1 (0, 4)	2.5 (0, 10.2)
49–61	82.5 (16.5, 530.5)	7.5 (3, 30.5)	0 (0, 1)	0 (0, 1)	0 (0, 1)	2 (1, 6)	4 (1, 33.5)
62–73	205.5 (29.2, 1116.2)	14.5 (4, 44.8)	0 (0, 4.5)	0 (0, 3)	0 (0, 1)	2 (0, 8.8)	15.5 (1, 98.2)
74+	269.5 (72.8, 1530.2)	17 (5.2, 44.2)	1 (0, 9.8)	0 (0, 5.8)	0 (0, 1)	3.5 (1, 12.8)	21 (4, 135.2)
Sex							
Female	83.5 (17.2, 421)	8 (3, 31.8)	0 (0, 2)	0 (0, 1)	0 (0, 2)	2 (0, 10)	6 (1, 33.8)
Male	135.5 (17.8, 863.2)	8 (2, 33.2)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 6.2)	7 (1, 70.5)
Race							
Asian	124.5 (18.2, 615.8)	14 (4, 42.8)	0 (0, 3.5)	0 (0, 2)	0 (0, 2)	3 (1, 14)	10 (1, 70.5)
Black or African American	182.5 (21.8, 813.8)	11 (3, 25)	0 (0, 2)	0 (0, 4)	0 (0, 2)	2.5 (1, 10.2)	11 (2, 54.2)
Native Hawaiian or Pacific Islander	188 (73, 626)	5 (3.5, 13.2)	0 (0, 3.2)	0.5 (0, 6)	0 (0, 0.2)	1.5 (0, 5.5)	8.5 (4, 60)
White	112 (15.2, 679)	8 (2, 31)	0 (0, 2)	0 (0, 1)	0 (0, 1)	1 (0, 8)	6 (1, 51.8)
Unknown or decline to state	81 (20.2, 422)	8 (3, 29.8)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 6)	5.5 (0, 36.8)
Ethnicity							
Hispanic or Latino	79 (19, 254)	8 (2, 30)	0 (0, 2)	0 (0, 0)	0 (0, 1)	3 (0, 8)	6 (0, 28)
Not Hispanic or Latino	122 (17, 717)	8 (3, 35)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 9)	7 (1, 55.8)
Unknown or not reported	78 (43, 335)	6 (3, 14)	0 (0, 3)	0 (0, 1)	0 (0, 0.5)	0 (0, 1.5)	5 (1.5, 22)
Past medical history[a]							
IHD (−)	83 (14, 441)	7 (2, 27)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	5 (1, 33.5)
IHD (+)	609 (100.5, 2451.5)	30 (8, 73.5)	1 (0, 13)	1 (0, 11.5)	0 (0, 1)	5 (1, 15)	45 (5.5, 182.5)
HF (−)	84 (14, 529)	8 (2, 29.8)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	5 (1, 42)
HF (+)	853.5 (194.8, 2641.2)	26.5 (7.8, 155)	2.5 (0, 23.8)	1.5 (0, 19.8)	0.5 (0, 4.2)	7 (1, 19)	43.5 (9.2, 322.2)
PCI/Stent/CABG (−)	93 (15, 560)	8 (2, 30)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	6 (1, 44)
PCI/Stent/CABG (+)	594 (139.5, 1795)	23 (7, 73.5)	1 (0, 6)	1 (0, 11.5)	0 (0, 1)	5 (1, 15)	38 (5, 146.5)
Stroke (−)	100 (15, 594)	8 (2, 30)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	6 (1, 51)
Stroke (+)	347 (65, 1849)	32 (7, 66)	1 (0, 20)	1 (0, 16)	0 (0, 2)	6 (1, 18)	33 (4, 216)
Afib (−)	87.5 (14, 544.8)	8 (2, 29.2)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	5 (1, 42.2)
Afib (+)	579 (145, 2522)	27 (7.5, 92.5)	3 (0, 17.5)	1 (0, 14)	0 (0, 1.8)	4.5 (1, 20.2)	41 (14, 283.2)
Characteristics at ICU admission[b]							
Baseline 12-lead ECG							
PVC absent on ECG	122 (23, 625.5)	8 (3, 32.5)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 9)	7 (1, 52)

(Continues)

TABLE 3 (Continued)

Characteristics	Median (Q_1, Q_3) Isolated PVC	Couplet	Bigeminy	Trigeminy	R-on-T	Run PVC	PVC/min
PVC present on ECG	2670.5 (1247, 7077.8)	67 (24.5, 149.5)	11.5 (2.8, 287)	14 (1.8, 189.8)	1 (0, 5.2)	6 (3, 28.2)	216.5 (64.5, 410.8)
Afib absent on ECG	124.5 (22.8, 747.8)	8.5 (3, 35)	0 (0, 3)	0 (0, 1.2)	0 (0, 1)	2 (0, 9)	7 (1, 61.2)
Afib present on ECG	613 (182.5, 2415)	23 (6, 66.5)	1 (0, 7)	1 (0, 4.5)	1 (0, 4)	2 (1, 15.5)	20 (4, 260)
Normal serum potassium	100 (14.5, 624.5)	8 (2, 31)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 9)	6 (1, 54)
Hypokalaemia	129 (24, 773)	10 (4, 31)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (1, 8)	7 (1, 46)
Hyperkalaemia	152 (26.8, 624)	17 (3.8, 47.2)	0 (0, 2.8)	0 (0, 1.8)	0.5 (0, 1)	3 (1, 14.2)	14 (2.2, 53.2)
Normal serum magnesium	84 (19, 681)	8 (2, 31)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 10)	6 (1, 63)
Hypomagnesaemia	133.5 (15.2, 768)	10.5 (3, 35)	0 (0, 2.8)	0 (0, 1)	0 (0, 1)	2.5 (0, 9)	6.5 (1, 52.8)
Hypermagnesaemia	327 (109.5, 2288.5)	23 (13, 59.5)	1 (0, 8)	0 (0, 25)	1 (0, 2)	4 (1, 15.5)	21 (11, 106.5)
Normal LVEF	205 (41, 858)	14 (4, 38)	0 (0, 3)	0 (0, 3)	0 (0, 1)	3 (1, 8)	16 (3, 63)
Hyperdynamic LVEF	118 (26.8, 612.8)	13.5 (4.8, 38.2)	0 (0, 2.2)	0 (0, 1)	1 (0, 1.5)	3 (1, 12.5)	7.5 (2, 49.2)
Mild dysfunction LVEF	339 (104, 1557)	25 (7, 49)	2 (0, 33)	0 (0, 3)	1 (0, 2)	10 (1, 12)	16 (3, 70)
Moderate/severe LVEF	1069 (237, 2474)	23 (5, 141)	2 (0, 17.5)	1 (0, 17.5)	0 (0, 11)	6 (1, 16.5)	90 (5, 358)
Primary admitting diagnosis							
Cardiovascular	343 (59, 1740.2)	16 (5, 64.2)	1 (0, 7.5)	0.5 (0, 9.8)	0 (0, 1.8)	4.5 (1, 15.8)	19.5 (2.2, 161)
Medical-surgical	87.5 (16.2, 599.5)	8 (3, 30)	0 (0, 2)	0 (0, 1)	0 (0, 1)	2 (0, 8)	6 (1, 36.8)
Neurological	56 (11.2, 432.2)	6 (1, 27)	0 (0, 1)	0 (0, 1)	0 (0, 1)	1 (0, 6)	4.0 (0, 37.5)

Abbreviations: Afib, atrial fibrillation; CABG, coronary artery bypass graft; ECG, electrocardiogram; HF, heart failure; IHD, ischaemic heart disease; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; PVC, premature ventricular complex.

[a]Past medical history: (+) indicates 'Yes', (–) indicates 'No'; a patient can have more than one medical history.

[b]The proportions, means and standard deviations are calculated excluding patients with missing data (see Table 1).

brief non-invasive test at ICU admission appears to identify patients who will be prone to higher rates of PVC alarms, which is consistent with a prior study (Yang et al., 2014). This finding may suggest that the PVCs are chronic and may be tolerated by the patient even when frequent (Marcus, 2020). However, it would seem prudent to evaluate the PVC trends in these select patients during the first 24 h of admission prior to making any PVC alarm adjustments as an alarm-reduction strategy (e.g. disable alarms, widen alarm parameters). This strategy might be enhanced by also taking into account older age and male sex in these cases.

In the univariate analysis, IHD and HF were associated with a higher rate of isolated PVCs. However, these conditions did not remain significant in the stepwise regression analysis. This is inconsistent with prior studies that found a history of MI and the severity of CAD were associated with frequent PVCs (Kerola et al., 2018; Minisi et al., 1988). Of note, the other PVC types were not associated with the cardiovascular clinical history variables we included in the model in both univariate and stepwise regression analysis. We were somewhat surprised that hypokalaemia was not associated with a higher rate of PVCs, which is incongruent with prior studies (Simpson et al., 2002; Tsuji et al., 1994). In the Framingham Offspring Study, Tsuji et al. found that among participants with a 0.46 mEq/L decrease in potassium (vs. participants without potassium decrease), the odds of having complex or frequent PVC was 1.27 times higher (95% CI 1.06–1.51) (Tsuji et al., 1994). Our findings might be explained by aggressive electrolyte replacement in our sample, which is common when patients are treated in an ICU. This may explain our findings that hyperkalaemia was associated with a lower rate of R-on-T type PVCs, although we had a small sample for this PVC type due to its infrequent occurrence. One study showed that hyperkalaemia causes velocity reductions in phase 0 of cardiac depolarisation and thus, a reduction in the height of the action potential with subsequent

TABLE 4 Univariate regression analysis of PVC counts after outliers (standardised residual cutoff ≥3) removed

Variable	Isolated PVC	Bigeminy	Trigeminy	Couplet	Run PVC	R-on-T
Age	**1.05 (1.04,1.06)****	1.04 (1.02,1.07)*	1.01 (.99,1.05)	**1.02 (1.01,1.03)****	1.01 (1.00,1.02)	0.98 (.96,1.00)
Male	1.72 (1.26,2.49)*	2.25 (.97,5.10)	1.70 (.69,3.86)	1.11 (.81,1.51)	0.89 (.62,1.25)	0.82 (.38,1.58)
Race: White	1.10 (.75,1.67)	1.21 (.57,2.59)	2.19 (1.00,4.85)*	0.87 (.63,1.18)	0.80 (.55,1.18)	3.13 (1.71,6.36)*
Presence of clinical history						
IHD	**2.98 (2.00,4.26)****	1.92 (0.58,4.22)	0.85 (0.31,2.22)	1.68 (1.08,2.40)*	1.22 (0.73,1.91)	0.70 (0.23,1.48)
HF	**2.30 (1.38,3.37)****	0.71 (0.33,1.56)	1.22 (0.42,3.19)	1.60 (0.97,2.44)*	1.52 (0.80,2.55)	1.60 (0.28,4.06)
PCI/stent/CABG	2.49 (1.42,4.06)*	0.34 (0.07,0.95)	1.28 (0.31,3.56)	1.23 (0.77,1.84)	1.22 (0.53,2.26)	0.41 (0.10,1.05)
Stroke	1.79 (1.00,2.86)*	1.09 (0.28,2.55)	1.29 (0.30,3.78)	1.57 (0.86,2.51)	1.36 (0.78,2.23)	0.98 (0.29,2.12)
Atrial fibrillation	2.15 (1.30,3.30)*	2.25 (0.98,4.71)	1.32 (0.45,3.35)	2.03 (1.32,2.92)*	1.37 (0.78,2.26)	1.33 (0.35,3.67)
Clinical characteristics on ICU admission						
PVC on 12-lead ECG	**2.80 (2.00,4.68)****	4.66 (1.10,12.43)*	3.42 (0.51,9.49)	**2.18 (1.49,3.32)****	1.88 (1.01,3.19)*	1.12 (0.28,2.51)
Atrial fibrillation on 12-lead ECG	1.59 (0.98,2.32)*	0.64 (0.15,1.76)	0.62 (0.15,1.90)	1.46 (0.82,2.08)	1.31 (0.70,2.23)	1.16 (0.27,2.94)
(log) Potassium	1.67 (0.67,4.89)	11.59 (0.98,298.87)	0.56 (0.05,15.64)	1.41 (0.58,3.19)	2.75 (0.96,7.39)	0.02 (.00,0.38)*
Hypokalaemia	1.08 (0.66,1.64)	0.59 (0.22,1.33)	2.17 (0.77,5.16)	0.99 (0.71,1.34)	0.64 (0.43,0.91)*	1.55 (0.76,3.13)
Hyperkalaemia	1.04 (0.54,1.67)	1.35 (0.27,3.16)	0.29 (0.07,0.74)*	0.78 (0.44,1.26)	0.80 (0.38,1.53)	**0.06 (0.02,0.12)****
Hyperdynamic LVEF	0.58 (0.33,0.89)*	1.63 (0.28,4.53)	2.02 (0.40,5.00)	0.87 (0.52,1.22)	1.16 (0.71,1.83)	0.68 (0.23,1.45)
Dysfunction LVEF	1.99 (1.22,3.40)*	0.91 (0.46,2.62)	1.06 (0.51,3.71)	1.29 (0.83,2.43)	1.09 (0.69,2.02)	1.75 (0.62,3.97)
Hypomagnesaemia	1.10 (.67,1.51)	0.83 (0.24,1.88)	0.52 (0.20,1.14)	1.11 (0.76,1.46)	0.98 (0.68,1.55)	0.81 (0.44,1.80)
Hypermagnesaemia	1.50 (0.79,2.59)	1.07 (0.26,2.55)	4.35 (0.88,7.69)*	1.16 (0.74,1.87)	1.31 (0.68,2.16)	0.53 (0.09,1.04)
Neuro/neurosurgical diagnosis	0.70 (0.47,1.01)	0.67 (0.22,1.50)	0.80 (0.31,1.76)	0.77 (0.53,1.03)	0.87 (0.57,1.22)	0.85 (0.42,1.65)
Cardiovascular diagnosis	2.05 (1.36,2.94)*	0.80 (0.35,1.76)	0.86 (0.34,2.10)	1.70 (1.19,2.34)*	1.53 (1.00,2.32)*	1.14 (.53,2.39)

Note: Cells display the incidence rate ratios (IRRs) for the counts by PVC type and 95% confidence interval (95% CI). *, $p < .05$; **, $p < .00044$. Analysis with outliers included is shown in Table S1.

Abbreviations: CABG, coronary artery bypass graft; ECG, electrocardiogram; HF, heart failure; IHD, ischaemic heart disease; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; PVC, premature ventricular complex.

Bold values indicated statistically significant values.

TABLE 5 Stepwise regression analysis of PVC counts after outliers (standardised residual cutoff ≥3) removed

Variable	Isolated PVC	Bigeminy	Trigeminy	Couplet	Run PVC	R-on-T
(Intercept)	0.22 (0.09,0.44)**	0.00 (0.00,0.01)**	0.00 (0.00,0.05)**	0.07 (0.04,0.14)**	0.06 (0.05,0.08)**	0.00 (0.00,0.01)
Age	**1.04 (1.03,1.05)****	**1.04 (1.02,1.06)****		**1.02 (1.01,1.03)****		
Male	**1.94 (1.40,2.84)****					
Race: White						2.92 (1.66,5.70)*
History of IHD	1.75 (1.10,2.58)*					
History of HF	2.06 (1.28,3.15)*					
PVC on baseline 12-lead ECG	**2.85 (1.83,4.30)****	5.70 (1.03,15.49)*		2.16 (1.34,3.46)*	2.18 (1.10,3.63)*	
Hypokalaemia at baseline					0.65 (0.47,0.88)*	
Hyperkalaemia at baseline			0.29 (0.07,0.74)*			**0.07 (0.03,0.17)****
Cardiovascular diagnosis				1.74 (1.18,2.44)*	1.72 (1.15,2.58)*	

Note: An empty cell indicates the variable was not in the final stepwise model. Cells display the incidence rate ratios (IRRs) for the counts by PVC type and 95% confidence interval (95% CI). *, $p < .05$; **, $p < .00044$. Analysis with outliers included is shown in Table S2.

Abbreviations: ECG, electrocardiogram; HF, heart failure; IHD, ischaemic heart disease; PVC, premature ventricular complex.

Bold values indicated statistically significant values.

non-excitable myocardial tissue (Watanabe et al., 1963). The authors concluded that the ventricle(s) are less susceptible to arrhythmias in this situation. A future study examining the value aggressive electrolyte replacement (e.g. sliding scale) would be of clinical relevance to support this practice.

What remains unknown is whether monitoring for PVCs is clinically meaningful. For example, an R-on-T type PVC has been shown to be a precursor of VT and/or VF (Liu et al., 2019; Noda et al., 2004; Viskin et al., 1997), and therefore, clinicians carefully monitor for these types of PVCs. In our study, we found that R-on-T type PVCs were uncommon, accounting for only 0.29% of all of the PVC alarms. Given the low occurrence rate of R-on-T type PVCs and its association with lethal arrhythmias, keeping alarms for this type of PVC turned on seems prudent. In clinical practice, clinicians closely monitor for not only R-on-T type PVCs but also new-onset PVCs and/or more frequent PVCs because they might signal electrolyte imbalance and/or myocardial irritability, both of which increase the risk of lethal ventricular arrhythmias. However, current PVC algorithms are not designed to display this type of PVC information. Enhanced PVC algorithms that could provide more clinically meaningful PVC information (e.g. new, more frequent) that are coupled with other ECG features associated with ventricular arrhythmias (e.g. QT interval) should be tested. Also, while studies suggest that a higher frequency of PVCs may actually cause HF in the community setting (Dukes et al., 2015; Limpitikul et al., 2022; Marcus, 2020), whether frequent PVCs could lead to an acute onset of HF in critical care settings requires further investigation. Finally, given the sheer volume of PVC alarms reported in our study, identifying clinically meaningful PVCs is extremely difficult for nurses and providers because they will be buried within the 'noise' of frequent PVC alarms. Therefore, there is a need for well-designed clinical research studies to help identify PVC patterns that are associated with adverse outcomes (e.g. lethal arrhythmias and/or code blue) to help not only guide alarm management strategies but new ECG monitoring algorithms.

5 | LIMITATIONS

One limitation of this study is that the PVC alarms were not annotated as true/false; hence, the accuracy of the PVC alarms is unknown. Annotation refers to manual/visual assessment (i.e. annotation) by clinical experts who review alarms, in this case PVC alarms, and use a predefined, standardised protocol to determine if the PVC alarm was true or false. One potential source of false PVC alarms is intermittent ventricular pacing. The 'pacer-mode' feature must be activated on the bedside monitor in patients with a ventricular pacemaker to avoid this problem. This feature changes the filter settings in the bedside monitor to detect pacer 'spikes'. However, if this feature is not activated, intermittent ventricular paced beats, which have wide QRS complexes like PVCs, will be incorrectly labelled as a PVC. Because we did not annotate all of the ECG data to identify this issue we cannot report on the occurrence rate of this problem. However, we did review all of the ECGs for the one outlier

patient (744 monitoring hours) who has the highest count of isolated PVCs and found that PVC detection was accurate. Importantly, our data were generated from commercially available monitors; hence, our study represents real-world data that nurses and other clinicians currently encounter at the bedside. We also do not know if the current algorithm missed any PVCs. However, in a recent review, the accuracy of PVC detection algorithms was reported to be between 86% and 99% (Nabil & Reguig, 2015).

Another limitation of our study is that our dataset had outlier patients, which may substantially impact the count regression fits and influence our ability to identify associations between demographic and clinical variables and PVC type. However, we conducted a sensitivity analysis, where we removed outliers, and we often found that variables meeting Bonferroni's significance remained in the model. Nonetheless, our data show that a small group of select patients have excessive PVC alarms. This suggests that alarm reduction strategies should be focused in these select patients, rather than making unit-wide alarm configuration settings. Algorithms that could identify outlier patients should also be explored. Lastly, our study examined only one monitoring manufacturer in a single centre, which limits the generalisability of our study.

6 | CONCLUSIONS

In this study, we found that isolated PVCs were the most common type of alarm, while R-on-T type PVCs were rare. Factors associated with a higher occurrence rate of isolated PVCs include older age, male sex and presence of PVC(s) on a hospital acquired standard 12-lead ECG. Older age was associated with a higher incidence rate of bigeminy and couplets, whereas hyperkalaemia was associated with a lower incidence rate of R-on-T type PVCs. PVCs are concentrated in fewer than 10% of ICU patients, thus, alarm management strategies should be focused in these patients. New algorithms that can identify clinically meaningful PVC patterns (e.g. new onset, more frequent) that are paired with additional physiologic data (e.g. drop in blood pressure or SpO_2) and/or other ECG waveforms (e.g. QT interval, ST segment) should be tested to identify clinically important PVCs.

7 | RELEVANCE TO CLINICAL PRACTICE

This study further amplifies the reality of the excess alarm burden that nurses and other clinicians encounter at the bedside. PVC alarms are very frequent, and whether keeping the bedside monitor alarms for PVCs is clinically meaningful remains largely unknown. This study shows that a small subset of patients often generates a large proportion of PVC alarms. Therefore, an effort to reduce the number of PVC alarms on these patients could greatly reduce alarm burden and minimise alarm fatigue. Nurses play an important role in evaluating PVC trends on these select patients, especially during the first 24 h of ICU admission for those who constantly have PVCs. Nurses should start the discussion with the care team to evaluate if PVC alarms will be critical during continuous ECG monitoring and whether there is a need to keep the alarm on (audible or inaudible) for all PVC types or only for certain types. Finally, nurses have a critical role in leading the effort in personalised alarm management that is tailored to individual patients, which will lead to improved care while at the same time minimising the risk for alarm fatigue.

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CONFLICT OF INTEREST

None to report.

DATA AVAILABILITY STATEMENT

The data that supports the findings of this study are available in the main text and supplementary material of this article.

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