

Ventricular tachycardia and in-hospital mortality in the intensive care unit



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BACKGROUND Continuous electrocardiographic (ECG) monitoring is used to identify ventricular tachycardia (VT), but false alarms occur frequently.

OBJECTIVE The purpose of this study was to assess the rate of 30-day in-hospital mortality associated with VT alerts generated from bedside ECG monitors to those from a new algorithm among intensive care unit (ICU) patients.

METHODS We conducted a retrospective cohort study in consecutive adult ICU patients at an urban academic medical center and compared current bedside monitor VT alerts, VT alerts from a new-unannotated algorithm, and true-annotated VT. We used survival analysis to explore the association between VT alerts and mortality.

RESULTS We included 5679 ICU admissions (mean age 58 ± 17 years; 48% women), 503 (8.9%) experienced 30-day in-hospital mortality. A total of 30.1% had at least 1 current bedside monitor VT alert, 14.3% had a new-unannotated algorithm VT alert, and 11.6% had true-annotated VT. Bedside monitor VT alert was not

associated with increased rate of 30-day mortality (adjusted hazard ratio [aHR] 1.06; 95% confidence interval [CI] 0.88–1.27), but there was an association for VT alerts from our new-unannotated algorithm (aHR 1.38; 95% CI 1.12–1.69) and true-annotated VT (aHR 1.39; 95% CI 1.12–1.73).

CONCLUSION Unannotated and annotated-true VT were associated with increased rate of 30-day in-hospital mortality, whereas current bedside monitor VT was not. Our new algorithm may accurately identify high-risk VT; however, prospective validation is needed.

KEYWORDS Ventricular tachycardia; In-hospital mortality; Intensive care unit; Continuous electrocardiographic monitoring; Alarm fatigue; Algorithm development

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Introduction

Ventricular tachycardia (VT) is a lethal arrhythmia that ranges in incidence from 2%¹ to as high as 13% among critically ill patients admitted to the intensive care unit (ICU).^{2,3} Among hospitalized stepdown unit patients with acute coronary syndrome, nonsustained VT occurs in 15%, and <1% have a malignant ventricular arrhythmia.⁴ Few hospital-based studies have characterized the risk of in-hospital mortality

associated with VT. Studies in ICU patients show that VT is associated with increased risk of mortality and other poor cardiovascular outcomes in patients with and without underlying cardiovascular disease across cardiac, medical, and surgical ICU settings.^{1,3,5} Risk of mortality also is high among patients who experience VT storm in the ICU.⁶ VT recurrences (ie, burden) during hospitalization are also associated with an increased risk of mortality.⁷

Although continuous electrocardiographic (ECG) monitoring is the noninvasive gold standard used to identify VT in hospitalized patients,^{5,8} false alarms are extremely common.^{2,9–13} In 1 study, only 1% of 1786 VT alarms were true, and none were recognized by clinicians.¹⁴ Our group

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KEY FINDINGS

- In a large study including 5679 consecutive intensive care unit patients, there was no difference in the adjusted rate of mortality among those who had a ventricular tachycardia (VT) alert from the current bedside monitor.
- VT that was identified using a new algorithm was associated with a significantly increased rate of 30-day in-hospital mortality in adjusted models.
- In addition, expert-annotated true VT was associated with a significantly increased rate of 30-day in-hospital mortality in adjusted models.
- Our new algorithm holds promise to accurately identify patients with VT who are at increased risk for mortality, although prospective validation is needed.

found that among 461 ICU patients during 1 month, there were 3861 VT alarms, of which 13% were true.^{2,12,13,15} Thus, the current positive predictive value of VT algorithms used in modern bedside monitors is poor; therefore, the risk of in-hospital mortality associated with *true* VT is largely unknown.

The primary aim of this study was to assess the rate of 30-day in-hospital mortality associated with VT alerts generated from bedside ICU ECG monitors, VT events identified via a new algorithm that was developed by our research group,¹⁶ and annotated true VT events.

Methods

Setting and study population

We conducted a retrospective cohort study among consecutive adult patients (age ≥ 18 years) admitted to the ICU at an urban tertiary care academic teaching hospital between September 2013 and April 2015. All patients received continuous bedside ECG monitoring per our hospital's standard of practice. Patients were admitted to 1 of 3 ICU types: (1) 16 bed cardiac; (2) 32 bed medical/surgical; or (3) 29 bed neurological (medical/surgical). The research reported in this paper adhered to the Declaration of Helsinki guidelines. The University of California San Francisco's Committee on Human Research approved the study (IRB No. 12-09723). Patient consent was waived because ECG and physiological (ie, vital signs) monitoring is part of routine ICU care, and the data were analyzed retrospectively and did not influence clinical care.

ECG and physiological signal data capture system

All available ECG and physiological waveform data were collected using a closed network system that connected all 77 bedside ICU monitors (Solar 8000i Version 5.4 software, GE Healthcare, Milwaukee, WI) via a gateway system.¹⁶ Data were sent to a secure, hospital-approved server maintained by our research laboratory. The following data

were collected from each ICU monitor: (1) all available waveforms (ECG, invasive arterial blood pressure [BP], central venous pressure, intracranial pressure, plethysmograph [SpO₂]); (2) vital signs (heart rate, noninvasive BP, respiratory rate); (3) alarm settings (crisis, warning, or advisory and message/technical); and (4) audible and inaudible alarms. Although we captured all waveform and alarm data, for this study we only examined VT alerts generated from the bedside ICU monitor. In addition, we used all available ECG waveforms, SpO₂, and invasive arterial BP waveforms to process the new VT algorithm developed by our group.¹⁶

Identification of VT

Current bedside monitor VT alerts

The ICU bedside ECG monitors used a 5-electrode lead configuration and generated 7 ECG leads (I, II, III, aVR, aVL, aVF, V₁). VT was defined as ≥ 6 wide QRS complexes at a rate >100 bpm, which was the default setting in the bedside monitor. Although all VT alerts generated by the bedside ECG monitor were collected (transient, sustained, or recurrent), our analysis compared patients based on whether they had at least 1 alert. We did not annotate these alerts as true or false.

New VT algorithm alerts

The 7 ECG waveforms, SpO₂, and invasive arterial BP waveform data were processed with a new VT algorithm created by our group. The new VT algorithm, which was recently published,¹⁶ was designed to decrease false VT by addressing ECG factors identified as primary sources of false VT (ie, motion/noise artifact, bundle branch block [right or left], and/or ventricular paced rhythm).^{12,13,15,17,18} For example, false VT can occur in patients with wide QRS complexes (ie, bundle branch block or ventricular pacer) when the heart rate exceeds the standard VT criteria of 100 bpm. Current algorithms are not designed to recognize these ECG features and can lead to false alarms for VT. One algorithm strategy we have used is identification of P waves to avoid labeling wide QRS complexes associated with bundle branch block or ventricular paced rhythms as potential VT. Another algorithm strategy we used was correlation of simultaneous drops in SpO₂ and invasive arterial BP waveforms during a potential VT event, which the current bedside monitor algorithm does not do. [Figure 1](#) shows a VT alert that was generated from a bedside monitor in a patient enrolled in our study but was not generated by our new algorithm. The same definition of VT as used in the bedside monitor (>6 wide QRS complexes >100 bpm) was used by our algorithm. Our analysis compared patients based on whether they had at least 1 VT.

Annotated true VT alerts

Potential VT alerts identified by our new algorithm were annotated as true vs false. Annotations were performed by 5 PhD prepared nurse scientists who had decades of

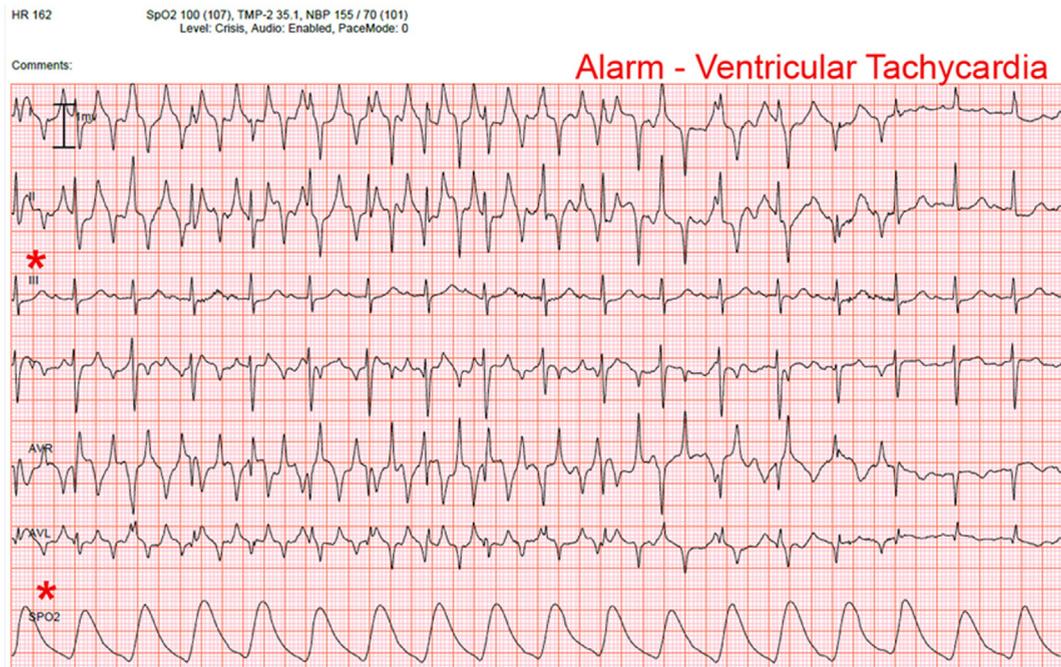


Figure 1 False ventricular tachycardia (VT) alert (heart rate [HR] 162 seen in **upper left corner**) from a bedside electrocardiographic (ECG) monitor in a patient admitted to the medical/surgical intensive care unit. Shown in order are ECG leads I, II, III, V₁, aVR, and aVL, and the pulse oximeter plethysmograph (SpO₂). In this case, both lead III and the SpO₂ waveform do not contain artifact; rather, they show a continuation of sinus tachycardia (lead III; 100 bpm) and perfusion (SpO₂ 100%) (asterisks). The bedside monitor algorithm requires a clean ECG signal in *at least 2* ECG leads and does not incorporate the SpO₂ signal; hence, a false VT alert is generated. The new algorithm tested uses a clean ECG signal in any single ECG lead with a P wave (first-degree atrioventricular block in this example) and an associated QRS in combination with a clean SpO₂ waveform to determine that this rhythm is not VT. Arterial blood pressure (not used in this patient) can also be used in the new algorithm. NBP = noninvasive blood pressure.

ICU experience and were highly skilled at interpreting hospital based-ECGs. The annotation protocol was a multitiered, multiexpert, ground truth, manual annotation, with 3-person agreement of VT.¹⁶ Again, our analysis compared patients based on whether they had at least 1 true VT. The same definition of VT as described earlier was used with additional specific criteria as listed in [Table 1](#).

Patient-level demographic and clinical data

An Epic-based electronic health record (EHR) platform (Epic 2017, Epic Systems Corporation, Verona, WI) was used to gather demographic and clinical data. EHR data were extracted by a certified data analyst using Clarity, the relational database that stores Epic's inpatient data. Data extracted included age, gender, ethnicity, primary language, need for an interpreter, insurance status, and discharge disposition. All primary and secondary *International Classification of Diseases, Ninth Revision (ICD-9)* and *International Statistical Classification of Diseases, Tenth Revision (ICD-10)* admission and discharge diagnosis codes were pulled from billing tables. To identify comorbidities associated with VT,^{19–23} we used ICD-10 codes to identify diabetes mellitus (DM), history of heart failure (HF), hypertension (HTN), coronary artery bypass graft (CABG), coronary artery disease (CAD), and myocardial infarction (MI) ([Supplemental Table 1](#)).

Statistical analysis

We compared baseline characteristics based on whether patients experienced at least 1 VT alert (bedside monitor, unannotated new algorithm, or true VT new algorithm) and whether they experienced in-hospital mortality within 30 days, using the beginning of continuous ECG monitoring as the starting point. The χ^2 test was used to evaluate associations between in-hospital mortality and presence of a VT, and other categorical variables. The difference in the distribution of patient age based on VT occurrence and in-hospital mortality was summarized using median [interquartile range] and with the Mann-Whitney rank-sum test.

Given the potential for unequal periods of observation between study subjects based on length of stay, we performed time-to-event analyses using Kaplan-Meier curves to compare the probability of 30-day in-hospital mortality between those who did and those who did not experience VT during ICU ECG monitoring by VT type (bedside monitor, unannotated new algorithm, or true VT new algorithm). Multivariable Cox proportional hazards modeling was conducted to determine the rate of 30-day in-hospital mortality associated with VT occurrence by VT type measured via the hazard ratio (HR) and 95% confidence interval (CI), controlling for age, gender, and baseline comorbidities, including DM, HF, HTN, CABG, CAD, and MI. We used the “stcox” suite of survival analysis commands in Stata 12.0 (StataCorp., College Station, TX). Because patients

Table 1 Operational definitions used by the annotators to determine true vs false VT**True VT**

Six consecutive ventricular beats defined as wide QRS complexes with clearly abnormal conduction at a rate >100 bpm. Heart rate limit applies only to the average rate, not individual R-to-R intervals (ie, 6 ventricular beats in <3.6 seconds). A fusion beat at the onset of VT counts as a ventricular beat.

QRS morphology of the VT beats should be different from those of the preceding non-VT beats to avoid labeling BBB or ventricular paced rhythms as VT.

False VT

Baseline noise or muscle artifact is present.

Periodic artifact simulating VT, which can be recognized by a heart rate too high to be “real” or a QRS width too narrow. Examine all available leads as well.

Single ECG lead.

Ventricular paced rhythm or bundle branch block; as stated above the QRS morphology of the VT beats should be different from those of the preceding non-VT beats.

Ventricular fibrillation, identified by coarse flutter waves without QRS complexes.

Presence of CPR. Heart rate waveform is similar in the thoracic impedance respiration waveform (Resp), presumably caused by true changes of thoracic impedance, or by pressure on one of the skin electrodes being used for impedance (lead II is the default lead). Typically, the pseudo-QRS complexes are >200 ms in width. Often an undisturbed lead allows for recognition of the underlying rhythm typically at a rate of 120 to 150 compressions per minute, which is our hospital’s standard CPR rate.

BBB = bundle branch block; CPR = cardiopulmonary resuscitation; VT = ventricular tachycardia.

could be admitted to the hospital multiple times during the 19-month study period, we used cluster-robust variance estimates to account for patient-level clustering.

Results

Patient demographics

There were 5370 patients with 5679 ICU admissions and 572,574 hours of continuous ECG monitoring during the 19-month study period, with an ethnic distribution representative of our institution. Mean age ($n = 5679$ admissions) was 58.0 ± 17.4 years, and 2701 (47.6%) were female. Race was identified from the EHR as follows: 25 (0.4%) American Indian or Alaska Native; 835 (14.7%) Asian; 460 (8.1%) Black or African American; 63 (1.1%) Native Hawaiian or Other Pacific Islander; 1069 (18.8%) Other/Unknown (due to acute illness, declined to state); and 3227 (56.8%) White. Patient admissions by ICU type included 965 (17.0%) cardiac; 2149 (37.8%) medical/surgical; and 2565 (45.2%) neurological. There were 503 admissions (8.9%) that resulted in 30-day in-hospital mortality, for an incidence rate of 11.1 per 1000 patient-days. Median follow-up time for the cohort was 5.6 [3.0–9.9] days. There were significant differences in age, gender, and baseline comorbidities when stratifying by 30-day in-hospital mortality (Table 2). There were significant differences in age, gender, and baseline comorbidities when stratifying by the presence of at least 1 VT alert from the current bedside monitor, unannotated, or annotated true (Table 3).

Survival analysis results

Current bedside monitor VT

In this analysis, we compared the rate of 30-day in-hospital mortality associated with having a VT based on the current bedside monitor vs not having a VT based on the current

bedside monitor. The 30-day in-hospital survival was significantly lower for those who had at least 1 VT (log-rank $P = .0291$) (Figure 2A). In unadjusted analysis, there was a 22% increased rate of 30-day in-hospital mortality among those who experienced at least 1 VT from the current bedside monitor (HR 1.22; 95% CI 1.02–1.46). However, the relationship between having at least 1 VT from the current bedside monitor and 30-day in-hospital mortality was no longer significant after adjustment for age at admission, gender, and history of HF, DM, HTN, CABG, CAD, and MI (HR 1.06; 95% CI 0.88–1.27) (Table 4). Significant risk factors for mortality from the adjusted Cox proportional hazards model included increasing age and history of MI, whereas a history of CAD was protective against mortality.

New VT algorithm (unannotated)

In this analysis, we compared the rate of 30-day in-hospital mortality associated with having a VT based on the new VT algorithm vs not having a VT based on the new VT algorithm. The 30-day in-hospital survival was significantly lower for those who had at least 1 VT (log-rank $P < .0001$) (Figure 2B). In unadjusted analysis, there was a 57% increased rate of 30-day in-hospital mortality among those who experienced at least 1 VT (HR 1.57; 95% CI 1.30–1.91). After adjustment for age at admission, gender, and history of HF, diabetes, HTN, CABG, CAD, and MI and after accounting for clustering at the patient level, there was a 38% increased rate of 30-day in-hospital mortality among those who experienced at least 1 VT based on the new algorithm (HR 1.38; 95% CI 1.12–1.69) (Table 4). In the adjusted Cox proportional hazards model, increasing age and history of MI were also associated with an increased rate of 30-day in-hospital mortality, whereas a history of CAD was protective against mortality.

Table 2 Baseline characteristics of intensive care unit patients stratified by 30-day in-hospital mortality status (n = 5679)

	Alive (n = 5176 [91%])	Died (n = 503 [9%])
Age (y)*†	57.3 ± 17.4	64.9 ± 16.3
Female	2462 (48.0)	219 (43.5)
Presence of VT from the current bedside monitor during ECG monitoring†	1416 (27.6)	295 (53.6)
Presence of VT from the new algorithm during ECG monitoring		
Unannotated	644 (12.4)	169 (33.6)
Annotated as true	517 (10.0)	143 (28.4)
Medical history		
Heart failure†	530 (10.2)	97 (19.3)
Diabetes mellitus†	846 (16.5)	129 (23.5)
Hypertension	1754 (33.9)	177 (35.2)
Coronary artery bypass graft‡	165 (3.2)	26 (5.2)
Coronary artery disease	692 (13.4)	82 (16.3)
Myocardial infarction†	60 (1.2)	27 (5.4)
Duration of monitoring in (d)§	5.6 [3.1–9.8]	5.9 [2.2–11.4]

Values are given as n (%) unless otherwise indicated.

ECG = electrocardiography; VT = ventricular tachycardia.

*Distribution given as mean ± SD. *P* value obtained using a *t* test.

†Significant difference between groups (*P* < .0001).

‡Significant difference between groups (*P* = .019).

§Distribution given as median [interquartile range]. *P* value obtained using a rank-sum test.

Annotated true VT from new algorithm

In this analysis, we compared the rate of 30-day in-hospital mortality associated with having a true VT vs not having a true VT. Based on the Kaplan-Meier curve (Figure 2C), survival was significantly lower for those who experienced at least 1 true VT (log-rank *P* < .0001). In unadjusted analysis, there was a 54% increased rate of 30-day in-hospital mortality among those who experienced at least 1 true VT (HR 1.54; 95% CI 1.26–1.88) compared to those who did not. After adjustment for age at admission, gender, and history of HF, diabetes, HTN, CABG, CAD, and MI and after accounting for clustering at the patient level, there was a 39% increased rate of 30-day in-hospital mortality among those who experienced at least 1 true VT (HR 1.39; 95% CI 1.12–1.73) (Table 4). As with the New VT algorithm, in the adjusted Cox proportional hazards model, increasing age and history of MI were also associated with an increased risk of 30-day in-hospital mortality, whereas a history of CAD was protective against mortality.

Discussion

To our knowledge, this is one of the largest studies to evaluate the rate of in-hospital mortality associated with VT in adult ICU patients, with 5679 admissions and >572,500 hours of continuous ECG monitoring. In unadjusted models, there was an increased rate of 30-day in-hospital mortality for all 3 VT types (bedside monitor, unannotated new algorithm, or annotated true VT new algorithm). After adjustment for baseline demographics and comorbidities, there was no difference in the rate of 30-day in-hospital mortality among patients who had a current bedside monitor VT alert compared to those who did not. However, patients who experienced an alert based on the new VT algorithm (unannotated) had a 38% increased rate of 30-day in-hospital mortality compared to those who did not have an alert based on the new VT algorithm, and patients who experienced a true VT event had a 39% increased rate of 30-day in-hospital mortality compared to those who did not.

In our cohort, 15% of patients experienced at least 1 VT using our new algorithm (unannotated) and 10% of patients experienced at least 1 true VT, estimates that are in line with the rate reported in other studies.^{2,3,5} In contrast, 30% of the cohort experienced ≥1 VTs based on the current bedside monitor, which likely reflects an overestimate of the rate of true VT, given that current algorithms prioritize sensitivity over specificity for VT identification. Although one could argue that vital sign parameters such as BP (arterial or noninvasive) and SpO₂ could serve as an indication of altered cardiac output during VT, the occurrence rate of these vital signs alarms is much higher than the VT alarms, as shown in previous work from our group.¹² For example, during a 1-month study period there were >50,000 oxygen saturation alarms (SpO₂), nearly 200,000 arterial BP alarms, and approximately 10,000 noninvasive BP alarms. During the same 1-month study period, there were only 3861 VT alarms. Although we do not know whether the SpO₂ and/or BP alarms were true or false, the sheer number of these vital sign alarms has the potential to divert clinician attention away from direct patient care. This sets the stage for alarm fatigue in ICU clinical staff and places patients at risk for missed true events.^{10,12,13,24} Previous research has demonstrated that, of the lethal arrhythmia alarm types (asystole, ventricular fibrillation, VT), VT generates the highest number of alarms.^{2,10,12,15,25} Potential consequences of alarm fatigue include the assimilation of alarm noise into a nurses' workflow with the potential for missing alarms, delayed response to alarms, and/or unsafe alarm adjustments (ie, lowering the volume and/or completely silencing alarms). These responses place patients at risk for missed true events, which has been linked to increased morbidity and mortality. The most recent data (2005–2012) have shown >650 in-hospital alarm-related deaths.^{26,27} Several federal and national organizations in the United States have issued alerts concerning alarm fatigue, including The Joint Commission, which created a National Patient Safety Goal specific to reducing harm associated with alarms.^{27–30}

Table 3 Baseline characteristics of intensive care unit hospitalizations summarized by whether VT was identified by the current bedside monitor or by the new VT algorithm

	VT per current bedside monitor		Unannotated VT per new algorithm		Annotated true VT per new algorithm	
	No VT (n = 3968 [69.9%])	≥1 VT alert (n = 1711 [30.1%])	No VT (n = 4866 [85.7%])	≥1 VT alert (n = 813 [14.3%])	No VT (n = 5019 [88.4%])	≥1 true VT (n = 660 [10.3%])
Age (y)*	56.1 ± 17.3	62.2 ± 16.9†	57.2 ± 17.4	62.4 ± 16.8†	57.5 ± 17.5	61.4 ± 16.7†
Female	1931 (48.7)	770 (45.0)‡	2,378 (48.9)	323 (39.7)†	2440 (48.6)	261 (39.6)†
Medical history						
Heart failure	306 (7.7)	321 (18.8)†	403 (8.3)	224 (27.6)†	441 (8.8)	186 (28.2)†
Diabetes mellitus	620 (15.6)	355 (20.8)†	776 (16.0)	199 (24.5)†	823 (16.4)	152 (23.0)§
Hypertension	1337 (33.7)	594 (34.7)	1641 (33.7)	290 (35.7)	1690 (33.7)	241 (36.5)
Coronary artery bypass graft	102 (2.6)	89 (5.2)†	128 (2.6)	63 (7.8)†	145 (2.9)	46 (7.0)†
Coronary artery disease	440 (11.1)	334 (19.5)†	562 (11.6)	212 (26.1)†	604 (12.0)	170 (25.8)†
Myocardial infarction	42 (1.1)	45 (2.6)†	53 (1.1)	34 (4.2)†	63 (1.3)	24 (3.6)†
Duration of monitoring (d)¶	4.8 [2.9–7.8]	8.7 [4.7–16.3]†	5.0 [3.0–8.7]	10.9 [5.5–20.5]†	5.1 [3.0–8.8]	11.5 [5.9–21.3]†
30-day in-hospital mortality	255 (6.4)	295 (17.2)†	347 (7.1)	203 (25.0)†	376 (7.5)	174 (26.4)†

Values are given as n (%) unless otherwise indicated.

VT = ventricular tachycardia.

*Distribution given as mean ± SD. *P* value obtained using a *t* test.

†Algorithm group (VT no/yes) significantly different (*P* < .0001).

‡Algorithm group (VT no/yes) significantly different (*P* = .013).

§Algorithm group (VT no/yes) significantly different (*P* = .001).

¶Distribution given as median [interquartile range]. *P* value obtained using a rank-sum test.

We found that those who had a new algorithm VT alert experienced a rate of 30-day mortality that was 38% higher than those who did not, based on the relative HR. This finding is important because it demonstrates that our new VT algorithm, even unannotated, was able to identify patients at greater risk for mortality than the current bedside monitor's VT alert. In addition, patients with the annotated alert experienced a slightly higher rate of mortality (HR 1.39), demonstrating that our algorithm likely is identifying patients at greatest risk for mortality.

Few studies have assessed the rate of true VT in ICU patients; however, our findings are similar to those of 2 previous studies. In a 2008 European multicenter study of 1341 ICU patients admitted to 1 of 26 general ICUs during a 1-month

period, sustained ventricular arrhythmias (ie, >30 seconds or requiring termination for hemodynamic collapse) were identified by 3 experts using blinded annotation. After adjustment for prognostic factors and the propensity for experiencing an arrhythmia, sustained ventricular arrhythmias were associated with an increased odds of mortality (odds ratio 3.53; 95% CI 1.19–10.42).¹ In a retrospective study at a community-based teaching hospital, a team of experts annotated all sustained arrhythmias that triggered cardiac decompensation and advanced cardiac life support procedures within the first 10 days of ICU admission among 215 medical ICU patients. In this study, ventricular arrhythmias were associated with a 93% increase in the relative risk of in-hospital mortality.³ Our data, which combine sustained and transient VTs, suggest that true VT, whether

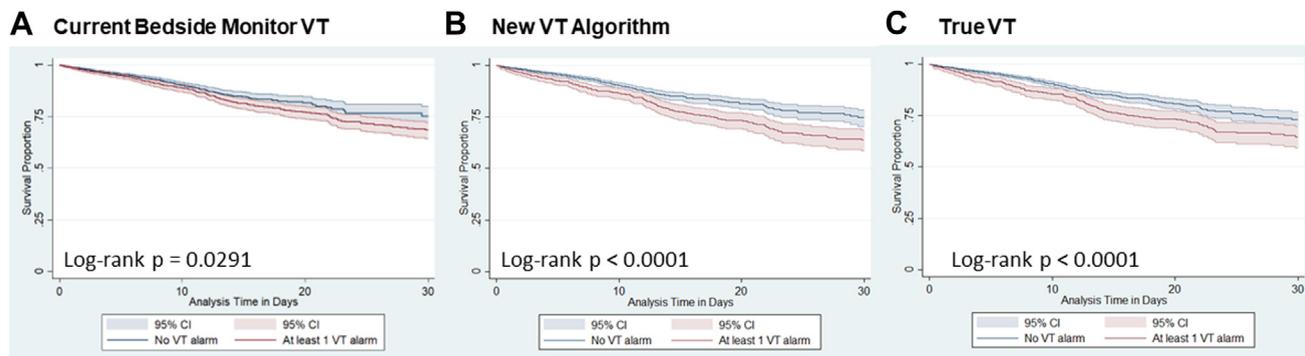


Figure 2 Unadjusted Kaplan-Meier survival curves estimating 30-day in-hospital mortality along with the 95% confidence interval (CI) stratified by presence of at least 1 ventricular tachycardia (VT) alert per current bedside monitor (A), presence of at least 1 VT alert per a new algorithm alert (unannotated) (B), and presence of at least 1 true VT identified by a new algorithm during ECG monitoring (C).

Table 4 Adjusted hazard ratios assessing the risk of 30-day in-hospital mortality among 5679 intensive care unit admissions with VT during ECG monitoring

Variable	VT alert per current bedside monitor	Unannotated VT alert per new algorithm	Annotated true VT events per new algorithm
Age (y)	1.03 (1.02–1.03)	1.03 (1.02–1.03)	1.03 (1.02–1.03)
Female	0.87 (0.73–1.05)	0.88 (0.73–1.06)	0.88 (0.73–1.06)
Medical history			
Heart failure	1.31 (1.03–1.66)	1.22 (0.95–1.55)	1.22 (0.96–1.56)
Diabetes mellitus	1.12 (0.91–1.39)	1.11 (0.89–1.37)	1.12 (0.90–1.39)
Hypertension	0.90 (0.75–1.08)	0.90 (0.75–1.08)	0.90 (0.75–1.08)
Coronary artery bypass graft	1.17 (0.73–1.86)	1.14 (0.72–1.82)	1.16 (0.73–1.84)
Coronary artery disease	0.66 (0.49–0.90)	0.66 (0.48–0.89)	0.65 (0.48–0.89)
Myocardial infarction	2.59 (1.68–3.98)	2.49 (1.60–3.86)	2.55 (1.64–3.95)
Presence of VT	1.06 (0.88–1.27)	1.38 (1.12–1.69)	1.39 (1.12–1.73)

Values are given as hazard ratio (95% confidence interval).

Models are adjusted for clustering at the patient level.

Abbreviations as in Table 2.

transient or sustained, is an important risk factor for mortality. Our study makes a significant contribution to the literature because it involved an extremely diligent annotation effort; it assessed the risk of VT in patients from all ICU settings; and it included a survival analysis of a large sample of diverse ICU patients admitted over a 19-month period.

Study limitations

Our new VT algorithm could have missed VT events. Our group is in the process of examining our data for false negatives to address this issue, which will guide future algorithm improvements. Another potential limitation is that we did not annotate the current bedside monitor VT alerts as true or false. However, we found that 74% of the true VT patients identified by our algorithm matched patients who had a VT alert generated from the current bedside monitor. Of note, in a previous study with a much smaller ICU sample, when examining VT alerts as the unit of analysis, we found that only 13% of 3861 VT alerts from the bedside monitor were true.¹² This finding illustrates that the current bedside monitor generates frequent false alarms and, thus, likely overestimates the number of patients with VT. Another limitation is that although our algorithm does use SpO₂ and/or invasive arterial BP in conjunction with the ECG waveforms to determine true vs false VT, we did not examine how often a loss of cardiac output (ie, drop in invasive arterial BP and/or SpO₂) occurred. An examination of this physiological response is an important future direction and could be useful in guiding clinical management. We did not examine whether a true VT was missed or whether an action(s) was taken or not, which should be examined in a future study. Although our dataset included a large cohort of >5600 hospital admissions, we included only ICU patients. Future studies should evaluate the risk of VT in non-ICU environments, such as stepdown units and general ward units with continuous ECG monitoring, which have fewer resources and may benefit most from an algorithm such as ours. In this head-to-head comparison sur-

vival analysis of VT alarms (current bedside monitor vs unannotated new algorithm vs true events from new algorithm), we used the first VT alert and did not consider recurrent alerts or the number of alerts per patient in our models. VT burden will be a future line of study in our cohort. Although patients were included even if they had only 1 alert, studies have shown that patients with some cardiac comorbidities are at increased risk for mortality even when they experience in-hospital nonsustained VT.³¹ Examining loss of cardiac output (ie, drop in SpO₂ and/or invasive arterial BP) associated with true VT would be another important line of inquiry to identify clinically actionable VT. We examined the ECG data of only 1 vendor, so comparing the performance of our new VT algorithm to the data of other vendors will demonstrate the generalizability of our findings. Finally, although we defined VT using the criteria currently used in clinical practice, they may be too sensitive and result in the identification of nonactionable VT events.² An unexpected finding from our study that warrants further exploration was that CAD was protective against 30-day in-hospital mortality in our cohort after controlling for other factors associated with VT. We hypothesize that patients with CAD may have undergone more optimal medical management of their condition. Other potential explanations could include multicollinearity and unmeasured confounding.

Conclusion

We found that VT identified using our new VT algorithm, even unannotated, as well as true VT are associated with an increased rate of 30-day in-hospital mortality, whereas VT identified from the current bedside monitor was not associated with an increased rate of 30-day in-hospital mortality when controlling for cardiac covariates. Our new algorithm holds promise for accurately identifying VT patients at greatest risk for mortality. Prospective validation is needed before the new algorithm can be introduced in clinical practice. In addition, a future study that examines the association

between mortality and VT burden, VT type (sustained vs nonsustained), and VT associated with loss of cardiac output is necessary to understand the clinical implications of VT, which could guide patient care.

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Patient Consent: Patient consent was waived by the Committee on Human Research because data were analyzed retrospectively, and the study did not influence clinical care.

Ethics Statement: The research reported in this paper adhered to the Declaration of Helsinki guidelines and was approved by the Committee on Human Research (IRB No. 12-09723).

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hroo.2023.09.008>.

References

- Annane D, Sébille V, Duboc D, et al. Incidence and prognosis of sustained arrhythmias in critically ill patients. *Am J Respir Crit Care Med* 2008;178:20–25.
- Pelster MM, Suba S, Sandoval C, et al. Actionable ventricular tachycardia during in-hospital ECG monitoring and its impact on alarm fatigue. *Crit Pathw Cardiol* 2020;19:79–86.
- Valderrábano RJ, Blanco A, Santiago-Rodríguez EJ, et al. Risk factors and clinical outcomes of arrhythmias in the medical intensive care unit. *J Intensive Care* 2016;4:9.
- Winkler C, Funk M, Schindler DM, Hemsey JZ, Lampert R, Drew BJ. Arrhythmias in patients with acute coronary syndrome in the first 24 hours of hospitalization. *Heart Lung* 2013;42:422–427.
- Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Heart Rhythm* 2018;15:e190–e252.
- Ninni S, Layec J, Brigadeau F, et al. Incidence and predictors of mortality after an electrical storm in the ICU. *Eur Heart J Acute Cardiovasc Care* 2022;11:431–439.
- Samuel M, Elsokkari I, Sapp JL. Ventricular tachycardia burden and mortality: association or causality? *Can J Cardiol* 2022;38:454–464.
- Sandau KE, Funk M, Auerbach A, et al. Update to practice standards for electrocardiographic monitoring in hospital settings: a scientific statement from the American Heart Association. *Circulation* 2017;136:e273–e344.
- Bonafide CP, Localio AR, Holmes JH, et al. Video analysis of factors associated with response time to physiologic monitor alarms in a children's hospital. *JAMA Pediatr* 2017;171:524–531.
- Cvach M. Monitor alarm fatigue: an integrative review. *Biomed Instrum Technol* 2012;46:268–277.
- Cvach M, Kitchens M, Smith K, Harris P, Flack MN. Customizing alarm limits based on specific needs of patients. *Biomed Instrum Technol* 2017;51:227–234.
- Drew BJ, Harris P, Zègre-Hemsey JK, et al. Insights into the problem of alarm fatigue with physiologic monitor devices: a comprehensive observational study of consecutive intensive care unit patients. *PLoS ONE* 2014;9:e110274.
- Harris PR, Zègre-Hemsey JK, Schindler D, Bai Y, Pelster MM, Hu X. Patient characteristics associated with false arrhythmia alarms in intensive care. *Ther Clin Risk Manag* 2017;13:499–513.
- Rosman EC, Blaurock AD, Menco A, Trope R, Seiden HS. What are we missing? Arrhythmia detection in the pediatric intensive care unit. *J Pediatr* 2013;163:511–514.
- Nguyen SC, Suba S, Hu X, Pelster MM. Double trouble: patients with both true and false arrhythmia alarms. *Crit Care Nurse* 2020;40:14–23.
- Pelster MM, Carey MG, Al-Zaiti S, et al. An annotated ventricular tachycardia (VT) alarm database: toward a uniform standard for optimizing automated VT identification in hospitalized patients. *Ann Noninvasive Electrocardiol* 2023;28:e13054.
- Pelster MM, Fidler R, Hu X. Research: association of low-amplitude QRSs with false-positive asystole alarms. *Biomed Instrum Technol* 2016;50:329–335.
- Suba S, Sandoval CP, Zègre-Hemsey JK, Hu X, Pelster MM. Contribution of electrocardiographic accelerated ventricular rhythm alarms to alarm fatigue. *Am J Crit Care* 2019;28:222–229.
- Mithun R, Rekha MC. A prospective study of risk of arrhythmias in patients with myocardial infarction in a tertiary care center. *J Assoc Physicians India* 2022;70:11–12.
- Cheng N, Gao C, Wang R, Yang M, Zhang L. New-onset ventricular arrhythmias in patients with left ventricular dysfunction after coronary surgery: incidence, risk factors, and prognosis. *Heart Surg Forum* 2018;26:21:E117–E123.
- Afzal MR, Savona S, Mohamed O, Mohamed-Osman A, Kalbfleisch SJ. Hypertension and arrhythmias. *Heart Fail Clin* 2019;15:543–550.
- Tran HV, Gore JM, Darling CE, Ash AS, Kiefe CI, Goldberg RJ. Hyperglycemia and risk of ventricular tachycardia among patients hospitalized with acute myocardial infarction. *Cardiovasc Diabetol* 2018;17:136.
- Alvarez CK, Cronin E, Baker WL, Kluger J. Heart failure as a substrate and trigger for ventricular tachycardia. *J Interv Card Electrophysiol* 2019;56:229–247.
- Winters BD, Cvach MM, Bonafide CP, et al. Technological distractions (part 2): a summary of approaches to manage clinical alarms with intent to reduce alarm fatigue. *Crit Care Med* 2018;46:130–137.
- Cvach M, Rothwell KJ, Cullen AM, Nayden MG, Cvach N, Pham JC. Effect of altering alarm settings: a randomized controlled study. *Biomed Instrum Technol* 2015;49:214–222.
- Ruskin KJ, Hueske-Kraus D. Alarm fatigue: impacts on patient safety. *Curr Opin Anaesthesiol* 2015;28:685–690.
- Joint Commission. Medical device alarm safety in hospitals. *Sentinel Event Alert* 2013;1–3.
- Mascioli S, Carrico CB. Spotlight on the 2016 National Patient Safety Goals for hospitals. *Nursing* 2016;46:52–55.
- Keller JP. Clinical alarm hazards: a “top ten” health technology safety concern. *J Electrocardiol* 2012;45:588–591.
- The Joint Commission, Hospital: 2022 National Patient Safety Goals, <https://www.jointcommission.org/standards/national-patient-safety-goals/hospital-national-patient-safety-goals/>. Accessed July 1, 2023.
- Katritsis DG, Zareba W, Camm AJ. Nonsustained ventricular tachycardia. *J Am Coll Cardiol* 2012;60:1993–2004.