Available online at www.sciencedirect.com

ScienceDirect

Journal of Electrocardiology 48 (2015) 1062 – 1068

JOURNAL OF Electrocardiology

www.jecgonline.com

ISCE Symposium Article

ECG changes on continuous telemetry preceding in-hospital cardiac arrests☆,☆☆

Duc H. Do, MD,[a,1] Justin Hayase, MD,[a] Ricardo Dahmer Tiecher, BS,[a] Yong Bai, BS,[b] Xiao Hu, PhD,[c,d] Noel G. Boyle, MD, PhD[a,*]

[a] Cardiac Arrhythmia Center, David Geffen School of Medicine at University of California, Los Angeles, CA
[b] Biomedical Engineering Graduate Program, Henry Samueli School of Engineering and Applied Science, University of California, Los Angeles, CA
[c] Department of Physiological Nursing, School of Nursing, University of California, San Francisco, CA
[d] Department of Neurosurgery, David Geffen School of Medicine, University of California, Los Angeles, CA

Abstract

Background: About 200,000 patients suffer from in-hospital cardiac arrest (IHCA) annually. Identification of at-risk patients is key to improving outcomes. The use of continuous ECG monitoring in identifying patients at risk for developing IHCA has not been studied.
Objective: To describe the profile and timing of ECG changes prior to IHCA.
Design: Retrospective, observational.
Setting: Single 520-bed tertiary care hospital.
Patients: IHCA in adults between April 2010 and March 2012 with at least 3 hours of continuous telemetry data immediately prior to IHCA.
Measurements: We evaluated up to 24 hours of telemetry data preceding IHCA for changes in PR, QRS, ST segment, arrhythmias, and QTc in ventricular tachycardia cases. We determined mechanism and likely clinical cause of the arrest by chart and telemetry review.
Results: We studied 81 IHCA patients, in whom the mechanism was ventricular tachycardia/fibrillation in 14 (18%), bradyasystolic in 21 (26%), and pulseless electrical activity (PEA) in 46 (56%). Preceding ECG changes were ST segment changes (31% of cases), atrial tachyarrhythmias (21%), bradyarrhythmias (28%), P wave axis change (21%),QRS prolongation (19%), PR prolongation (17%), isorhythmic dissociation (14%), nonsustained ventricular tachycardia (6%), and PR shortening (5%). At least one of these was present in 77% of all cases, and in 89% of IHCA caused by respiratory or multiorgan failure. Bradyarrhythmias were primarily seen with IHCA in the setting of respiratory or multiorgan failure, and PR and QRS prolongation with IHCA and concomitant multiorgan failure.
Limitations: This is a retrospective study with a limited number of cases; each patient serves as their own control, and a separate control population has not yet been studied.
Conclusions: ECG changes are commonly seen preceding IHCA, and have a pathophysiologic basis. Automated detection methods for ECG changes could potentially be used to better identify patients at risk for IHCA.

Keywords: Cardiopulmonary arrest; Telemetry; Physiologic monitoring; ECG; Arrhythmias; Critical care

Introduction

Approximately 200,000 in-hospital cardiac arrests (IHCA) occur annually in US hospitals [1]. Survival to discharge is less than 30%, and has improved only moderately since McGrath et al. published their first report in 1987 [2–4]. Identification of patients at risk for clinical deterioration and cardiac arrest has been key in improving outcomes in hospitalized patients, and is the basis of the

☆ Disclosures: none.
☆☆ Financial Support: This was an investigator-initiated study with no intramural or extramural funding source.

* Corresponding author at: UCLA Cardiac Arrhythmia Center, UCLA Health System, David Geffen School of Medicine at UCLA, 100 UCLA Medical Plaza, Suite 660, Los Angeles, CA, 90095-7392.
E-mail address: nboyle@mednet.ucla.edu
[1] Presenting Author: Duc Do, MD, UCLA Cardiac Arrhythmia Center, UCLA Health System, David Geffen School of Medicine at UCLA, 100 UCLA Medical Plaza, Suite 660, Los Angeles CA 90095-7392. Tel.: +1 310 206 2235.

http://dx.doi.org/10.1016/j.jelectrocard.2015.08.001
0022-0736/

rapid response system now utilized in many hospitals [5]. However, recognizing the at-risk patient is usually the limiting step in providing such pre-emptive care. Retrospective reviews of patients with cardiac arrests have frequently shown signs of clinical deterioration, especially mental status changes and respiratory distress that were unobserved or overlooked by nurses and physicians [6,7].

Currently, the detection of patients at risk for clinical deterioration normally occurs at time of vital sign measurements by a nurse or nursing assistant at varying intervals, depending on the hospital and unit policy [5]. This leaves significant potential for unnoticed patient deterioration during the intervening time periods [8,9]. Continuous telemetry monitoring is routine in ICUs and some non-ICU units and has been used for a variety of applications [10,11], but its utility in identifying patients at risk for cardiac arrests has not been well studied [5].

The goal of this study was to determine the profile and timing of ECG changes seen on telemetry (rhythm, morphology, intervals) in the 24 hours preceding IHCA, which, in the future, may serve as targets for automated detection algorithms to determine patients at-risk for cardiac arrest.

Materials and methods

Study design

We conducted a retrospective study utilizing data compiled by the Quality Management Service at the University of California, Los Angeles (UCLA) Ronald Reagan Medical Center, a 520-bed tertiary care hospital. Telemetry data was obtained by General Electric (GE) monitoring systems (GE Healthcare, Waukesha, WI), and pooled on remote data server via Bedmaster (Excel Medical Electronics, Jupiter, FL). Signals were sampled at 240 Hz with 12-bit representation. A total of 200 beds, including all 130 adult intensive care unit (ICU) beds, and 70 randomly selected medical–surgical unit beds were monitored with the Bedmaster system at any one time. This study received approval from the institutional review board at UCLA.

Patient population

We evaluated all 'code blues' between April 2010 and March 2012, and included all IHCA cases in patients age ≥ 18 years, with telemetry data available for at least 3 consecutive hours prior to and including the onset of IHCA. We excluded cases where cardiac arrest (defined as lack of central pulse, apnea, and unresponsiveness) was not the primary reason for the code blue, patients for whom a do not resuscitate order were in place, patients with a ventricular-paced rhythm, out-of-hospital cardiac arrest leading to current admission, IHCA in a procedural unit or operating room, and IHCA within the first 24 hours of a trauma admission. Only the first IHCA in any patient during a hospitalization was included.

For each case, we extracted telemetry data for up to 24 hours prior to the IHCA. Chart review of electronic medical records was performed to gather information regarding clinical history, comorbidities, clinically diagnosed cause of cardiac arrest, and outcomes.

Classification of cardiac arrest

The mechanism of each cardiac arrest was classified as: 1) ventricular tachycardia/fibrillation/torsade de pointes (VT/VF/TdP), 2) bradyasystole, and 3) pulseless electrical activity (PEA) (Fig. 1). VT/VF/TdP was defined by sustained ventricular tachyarrhythmia lasting greater than 30 seconds or resulting in hemodynamic instability. Monomorphic VT was categorized as VT, and polymorphic VT as TdP. Bradyasystole was defined as IHCA where the primary rhythm at onset of cardiac arrest was sinus node arrest or atrioventricular node block with asystole or slow escape rhythm (<30 bpm). PEA was defined as a continued cardiac rhythm in the absence of VT/VF/TdP associated with no pulse or perfusion. The type of arrest was determined by detailed review of the patient chart and telemetry data.

The primary cause of each cardiac arrest was classified as: 1) Respiratory failure, 2) Hemorrhagic shock, 3) Metabolic Acidosis, 4) Cardiac failure, 5) Multiorgan failure, 6) Drug-induced, and 7) Other, based on thorough chart review. Multiorgan failure was cited as the primary cause of cardiac arrest when abnormalities, due to dysfunction in at least two major organ systems resulting in failure to maintain homeostasis was present (eg. acidosis, hypoxia, hyperkalemia, prolonged hypotension), and were judged clinically as jointly responsible for precipitating the cardiac arrest.

Data analysis

Telemetry data for each patient, including ECG leads I, II, III, and V (generally in the V1 position), and waveforms for oxygen saturation and invasive blood pressure where available, were analyzed manually by two independent observers using LabChart 7 software package (AD Instruments, Colorado Springs, CO). All findings were confirmed by a cardiac electrophysiologist (NB). Time of onset of IHCA was determined as onset of a hemodynamically comprising arrhythmia in the case of bradyasystole and VT/VF/TdP cases; in PEA cases, based on onset of chest compression artifact, loss of pulse pressure on arterial line tracing where available, or at the time reported in medical records if neither were present.

The ECG data were analyzed for changes from baseline in PR interval, QRS duration, ST segment elevation or depression and new onset arrhythmias. QTc measurements were performed only in patients with VT/VF/TdP arrests, using measurements obtained in lead II and Bazett's formula ($QTc = QT/\sqrt{RR}$). All interval and duration measurements were taken at least once every hour with additional measurements at 1.5 hours, 50, 40, 30, 20, 10, 5, 2, and 1 minute(s) prior to cardiac arrest, with an average of five measurements at each time point. QRS measurements were only recorded for supraventricular rhythms to avoid bias introduced by ventricular rhythms. Findings at each time point were compared to baseline, the value of the particular parameter at the first available time point. Significant changes in the PR, QRS, and QTc were considered as a >20 ms increase from baseline that was sustained to the time of cardiac arrest, or sustained for at least 1 hour if the change did not persist to the time of cardiac arrest. Any QTc above