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Evaluation of beat-to-beat ventricular repolarization lability from standard 12-lead ECG during acute myocardial ischemia

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Abstract

Background: Acute myocardial ischemia is a common cause of ventricular arrhythmias, yet recent ECG methods predicting susceptibility to ventricular tachyarrhythmia have not been fully evaluated during spontaneous ischemia. We sought to evaluate the clinical utility of alternans and non-alternans components of repolarization variability from the standard 10-second 12-lead ECG signals to risk stratify patients with acute chest pain.

Methods: We enrolled consecutive, non-traumatic, chest pain patients transported through Emergency Medical Services (EMS) to three tertiary care hospitals with cardiac catheterization lab capabilities in Pittsburgh, PA. ECG signals were manually annotated by an electrophysiologist, then automatically processed using a custom-written software. Both T wave alternans (TWA) and non-alternans repolarization variability (NARV) were calculated using the absolute RMS differences over the repolarization window between odd/even averaged beats and between consecutive averaged pairs, respectively. The primary study outcome was the presence of acute myocardial infarction (AMI) documented by cardiac angiography.

Results: After excluding patients with secondary repolarization changes (n = 123) and those with excessive noise (n = 90), our final sample included 537 patients (age 57 ± 16 years, 56% males). Patients with AMI (n = 47, 9%) had higher TWA and NARV values ($p < 0.01$). Mean RR correlated with TWA, and noise measures correlated with TWA and NARV, after adjusting for potential confounders. There was a high collinearity between TWA and NARV, and each was separately predictive of AMI after controlling for number of analyzed beats, noise measures, and other clinical variables.

Conclusions: Despite limitations imposed by signal quality, TWA and NARV are higher in patients with AMI, even after correction for potential confounders. The clinical value of TWA and NARV derived from standard ECG using our time-domain RMS method is questionable due to the small number of beats and significant noise.

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Introduction

The 12-lead ECG remains the principal tool for the initial emergency evaluation of acute myocardial ischemia and infarction in patients with chest pain. ECG changes associated with acute myocardial ischemia include alterations in the amplitude of the ST-segment, and/or the amplitude and spatial-temporal characteristics of the T-wave [1]. Our knowledge of these latter characteristics is still incomplete. Given that >60% of patients with acute myocardial infarction (AMI) manifest little or no ST elevation on their presenting ECG [2], novel methods to quantify the ischemia-induced T-wave changes from the standard 12-lead ECG might have important clinical applications.

Since acute myocardial ischemia is strongly associated with the onset of ventricular arrhythmias [3], we hypothesized that ECG methods quantifying ventricular repolarization heterogeneity for the task of arrhythmia prediction might also be useful for the task of ischemia detection. As such, we

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previously demonstrated that the principal components of the T wave, T loop morphology, and wavefront direction metrics correlate with acute myocardial ischemia [4–6]. One of the best researched methods for arrhythmia prediction that has already made its way to clinical practice is T-wave alternans (TWA). TWA has been associated with risk of ventricular arrhythmia in a wide variety of clinical settings [7]. More interestingly, Martinez et al. [8] showed that experimental myocardial ischemia induced abnormal TWA in >33% of patients within 1–2 min of balloon inflation. However, the impact of spontaneous ischemia on TWA is unknown. The clinical value of TWA from short ECG recordings is also questionable. In contrast, we recently showed that non-alternans repolarization variability (NARV) also correlates with spontaneous ventricular tachycardia and is more robust than TWA when small number of beats is analyzed [9]. Accordingly, we sought to evaluate the presence of TWA and NARV in standard 10-second 12-lead ECG signal during spontaneous myocardial ischemia in patients evaluated at the emergency department for chest pain.

Methods

Study population

This is a secondary analysis of the Electrocardiographic Methods for the Prompt Identification of Coronary Events Study (EMPIRE). The methods of EMPIRE have been previously described in detail [10]. In short, EMPIRE is an ongoing prospective, observational cohort study designed to risk stratify patients with chest pain transported by ambulance to three tertiary care UPMC-affiliated hospitals in Pittsburgh, PA. This study is approved by the institutional review board of the University of Pittsburgh, with a waiver of informed consent to enroll eligible patients. Inclusion criteria include age > 18; chief complaint of non-traumatic chest pain; and availability of prehospital 12-lead ECG. Patients are followed during hospitalization to collect American College of Cardiology's recommended key data elements and definitions for measuring the clinical management and outcomes of patients with acute coronary syndromes [11]. This analysis is based on the first cohort recruited between May 2013 and August 2014 (n = 750).

Outcome data

The primary study outcome was the presence of AMI defined as a spontaneous myocardial infarction related to ischemia due to a primary coronary event (i.e., acute culprit lesion). AMI was identified according to the universal definition of MI [12], plus the presence of >70% occlusion in at least one main coronary artery during cardiac catheterization performed in the indexed visit. This outcome was coded by a reviewer who was independent from the investigative team and who was blinded from the ECG analyses of TWA and NARV. To make proper assessments, the reviewer was granted full access to patient index and discharge records, serial ECG reports, results of cardiac diagnostic tests (e.g., lab values, perfusion imaging scans, and catheterization lab reports), and other information pertinent to the course of hospitalization (e.g., interventions, procedures, and prescribed medications). This primary outcome was coded as yes or no.

ECG analysis

Analysis was based on the prehospital 10-second 12-lead ECGs, which were obtained by paramedics in the field using HeartStart MRx defibrillators with DXL 12-lead ECG algorithm (Philips Healthcare, NV, USA). Philips Healthcare exported the raw ECG signal into XML file format (500 samples per second, 5 μV per least significant bit; 0.05–150 Hz). Then, a cardiac electrophysiologist (J.N.) blinded to the clinical data manually annotated each ECG file. ECG files with pacing, or predominant rhythm other than sinus rhythm were excluded (n = 123/750, 16%). ECG files non-analyzable due to excessive noise or artifact were also excluded (n = 90/750, 12%). Obvious atrial or ventricular ectopic beats were manually deleted. At least 5 consecutive sinus beats of acceptable quality were required for inclusion.

Next, the ECGs were analyzed using a custom-written software created for this purpose in C++ (Microsoft Visual Studio, Microsoft Corp., Redmond, WA, USA) by one of the investigators (J.N.) [9]. Briefly, the signals were manually reviewed for quality, ectopic beats were deleted, and consecutive normal QRS-T complexes were manually selected for analysis. After R peak detection, the signal was low-pass filtered at 20 Hz with a 3-pole Butterworth filter. The baseline wander was minimized by subtraction of smooth cubic spline passing through fiducial points in the PR segments (80 ms before R wave peak in most cases). TWA was calculated by separately averaging odd and even beats, then taking the root-mean-square (RMS) of the differences between the two averaged-beats over the repolarization window (Fig. 1A). NARV was calculated by first averaging each pair of consecutive beats, then taking the RMS of differences between these averaged pairs over the same repolarization window (Fig. 1B). Averaging consecutive pairs eliminates alternans variability from NARV and removes contamination by TWA. Both TWA and NARV values were normalized to mean QRS amplitude. TWA and NARV values were computed in each of the 12 ECG leads, the reviewer selected and reported the largest values from the ECG lead with least noise and artifact. It is worth noting that our methods depend on the time-domain RMS of differences between odd/even averaged beats or consecutive averaged pairs, which is different from the FDA-cleared Modified Moving Average (MMA) and Spectral Methods for TWA analysis [13].

To control for residual noise in the signal, we computed and reported the HF noise (i.e. high-frequency noise — RMS of the difference between the raw signal and the low-pass filtered signal), fiducial point lability (i.e., RMS of fiducial point signal amplitudes), and R peak lability (i.e., RMS of signal amplitudes at R wave maxima). These noise measures were used as covariates in statistical analyses. In addition, to account for residual baseline error caused by respiration artifact and to distinguish such error from beat-to-beat repolarization changes, we computed and reported the vital signs-derived heart rate-to-breathing ratio (HR/BR), including odd vs. even ratio, and controlled for this ratio in the multivariate analyses.

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