Electrocardiographic factors playing a role in ischemic ventricular fibrillation in ST elevation myocardial infarction are related to the culprit artery

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BACKGROUND Sudden cardiac death caused by ischemic ventricular fibrillation (VF) associated with ST elevation myocardial infarction (STEMI) is one of the most frequent causes of death.

OBJECTIVE We hypothesized that electrocardiographic (ECG) characteristics differ between STEMI patients with and without ischemic VF.

METHODS Fifty-five first STEMI patients with at least one 12-lead ECG recorded before ischemic VF were compared with 110 first STEMI patients without ischemic VF. Patients with bundle branch blocks or high-degree atrioventricular blocks with escape rhythms were not included. ECG measurements were performed manually after scanning the ECG with the most prominent ST deviation into a software environment and magnifying it 4 times.

RESULTS Mean age was 57 ± 12 years, and 126 patients were male. No differences were present between the VF and control group regarding baseline, enzymatic, and angiographic data. In left circumflex artery and right coronary artery myocardial infarction, a longer QRS interval (109 ± 23 ms vs. 91 ± 16 ms, $P = .02$ and 107 ± 24 ms vs. 93 ± 19, $P = .02$) was present. In the latter the PR interval (211 ± 64 ms vs. 160 ± 36 ms, $P < .001$) and ST deviation score (3.6 ± 1.0 mV vs. 1.7 ± 1.5 mV, $P < .001$) were also increased. In the left anterior descending artery group no differences in conduction intervals and ST deviation score were present.

CONCLUSION Longer PR and QRS intervals in right coronary artery and left circumflex artery MI fit with the perfusion and activation pattern of the atrioventricular node and the ventricular myocardium. Myocardium perfused by the left anterior descending artery is activated earliest, hiding any intraventricular conduction delay within the QRS complex. Intramural slowed conduction could be a substrate for ischemic VF.

KEYWORDS Conduction; Electrocardiography; Fibrillation; Myocardial infarction; Tachyarrhythmias

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Introduction

Ischemia-related ventricular fibrillation (VF) is a frequent cause of sudden cardiac arrest.[1,2] Identification of risk markers is not only of pathophysiologic interest but also are important in terms of the ability to predict or to prevent the occurrence of VF. Research aimed at the predictability of VF has been focused mostly on settings outside of ischemia, such as the postmyocardial infarction stage or nonischemic conditions. In an ST elevation myocardial infarction (STEMI) population, previous work has shown that an increased number of ventricular premature beats may prelude this lethal arrhythmia.[3] More recently it was demonstrated that in a STEMI population, familial history of sudden death is an important risk factor for the occurrence of ischemic VF.[4] This suggests that genetic factors may be involved and that the predisposition to VF may differ between patients. From clinical and experimental studies, it is known that inhomogeneity of conduction velocity plays a role as a substrate for ventricular arrhythmias and sudden death during acute ischemia.[5–9]

We hypothesized that compared with ischemic patients without VF, local conduction will be impaired in ischemic patients with VF, resulting in an increased inhomogeneity of conduction velocity, and wondered whether this could be diagnosed by analyzing the 12-lead electrocardiogram (ECG) before the occurrence of ischemic VF.

Thus, the hypothesis was tested that ECGs preceding ischemia related VF are different from ECGs from ischemic patients without VF, particularly with regard to (local) conduction intervals.

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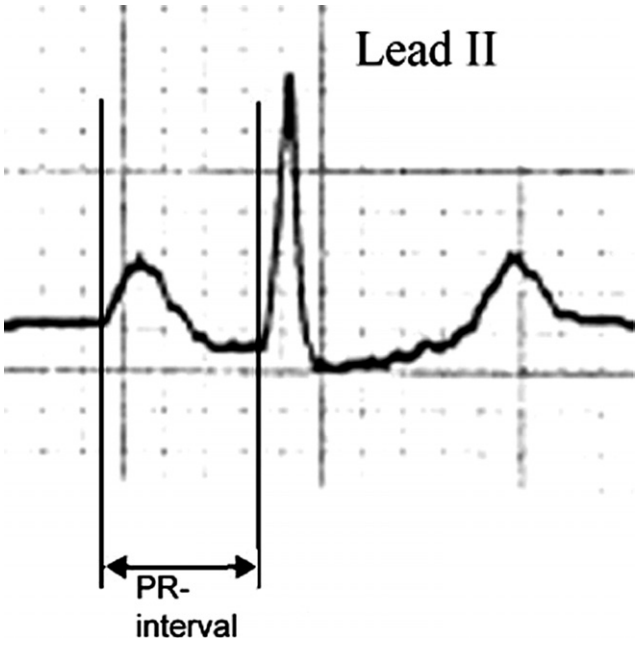

Figure 1 Measurement of the PR interval in patient A. The PR interval is measured between the beginning of the P wave and the beginning of the QRS complex.

Methods

Patient population

Between July 2004 and December 2006, ECGs recorded in the acute phase of a STEMI were collected retrospectively. Eligible patients were more than 18 years old and had presented in a coronary emergency setting in the Academic Hospital Maastricht or the Academic Medical Center Amsterdam, with a first STEMI and at least one 12-lead ECG recorded before ischemic VF. To ensure the ischemic nature of the VF, only patients with VF prior to the start of reperfusion therapy were included. Primary PCI was the primary treatment modality at both centers, from which coronary angiographic data were derived. By means of the patients' histories and ECGs, it was established that this was indeed the first STEMI in all included patients. To be able to assess intramyocardial conduction intervals, patients with bundle branch blocks or with advanced atrioventricular (AV) blocks with escape rhythms were not included. Fifty-five patients with ischemic VF (VF patients) met the inclusion criteria, and they were compared with 110 STEMI patients without ischemic VF (control patients).

ECG data

When more than one 12-lead ECG was available, the one with the most pronounced ST segment deviation was selected. For measurement of parameters related to the QRS complex and the ST segment, a single QRS-T-complex from the lead with the most pronounced ST segment deviation (either ST elevation or ST depression) was chosen because this lead is most likely the best representation of the ischemic area. For the purpose of PR interval measurement, a single P-QRS-T-complex was analyzed from the same 12-lead ECG, preferably from lead II, V1, or V2, because these leads are most suitable for P-wave recognition.

The selected complexes were scanned into a computer environment and digitally magnified by 400% using Adobe Photoshop (version 7.0, 2002, Adobe Systems Inc., San Jose, California). These magnified complexes were printed, and using these prints manual measurements were performed. ECGs were analyzed blinded to the occurrence of ischemic VF (Figures 1 and 2).

Measurements

Figures 1 and 2 show the methods of measuring the PR interval and the QRS interval, respectively. Parameters related to the extent of the ischemia are height of the peak ST deviation in millivolts, the total number of leads showing ST deviation, ST deviation score (i.e., the sum of all ST deviations), and the grade of ischemia as proposed previously.[10] ST deviation was measured 60 ms after the J point.[11] The axis of the ST segment in the frontal plane (ST deviation vector) was determined by manual measurement. QT and QTc-Fridericia intervals (QTc-Fridericia = $QT/RR^{1/3}$) were measured and calculated.

Clinical descriptors that were noted include baseline characteristics (gender, age, diabetes mellitus, hypertension, hypercholesterolemia, and history of smoking within 5

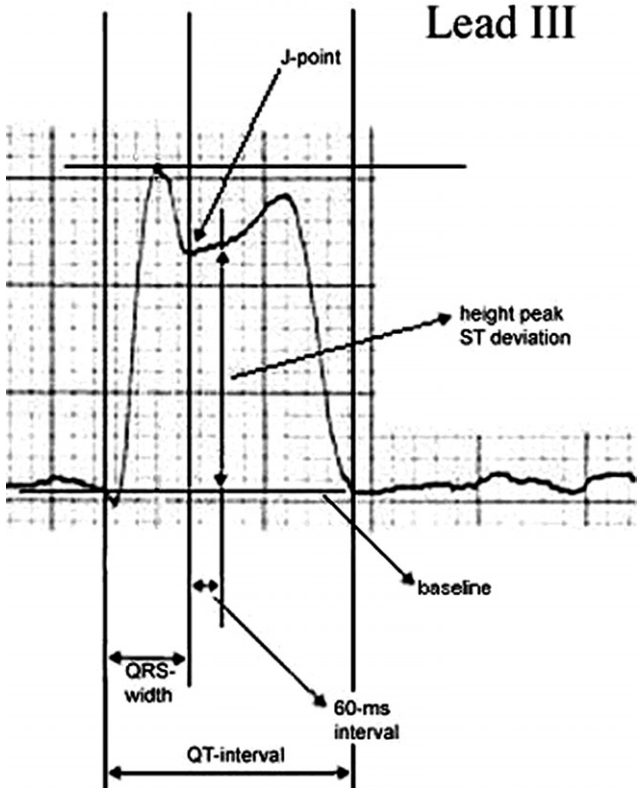

Figure 2 Measurement of QRS-related variables in patient B. The width of the QRS interval is measured between the beginning of the QRS complex and the J point. The QT interval is measured between the beginning of the QRS complex and the end of the T wave. The height of the peak ST deviation is the distance from the baseline to the ST segment, measured 60 ms after the J point.