# Reproducibility of ST and Ventricular Gradient Vectors

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## Abstract

*In this study, we tested the reproducibility of the ST vectors at the J point and 60 ms after the J point, and the reproducibility of the ventricular gradient. The reproducibility of these ECG variables was compared with the proposed thresholds for ischemia detection by serial ECG analysis (comparison of an acute ECG suspected of the presence of acute ischemia with a previously made non-ischemic ECG in the same patient).*

*In a group of 398 patients with various cardiovascular pathology we measured the differences in the ST vectors and in the ventricular gradient in ECG pairs about one year apart. All ECGs were non-ischemic, and electively made in the outpatient clinic. ST and VG vector differences exceeded only in around 20% of the cases the proposed sensitive thresholds for ischemia.*

*The results of our study suggest that the reproducibility of the ST and VG vectors in non-ischemic ECGs is good, thus indicating that the expected amount of false positive detections of ischemia by serial ECG analysis is likely to be low.*

## 1. Introduction

In atherosclerotic disease, a stenosis in a coronary artery may cause ischemia during exercise (demand ischemia), or, when the plaque ruptures and thrombus formation causes a partly or complete occlusion, ischemia occurs during rest (supply ischemia). When supply ischemia occurs and the thrombus does not resolve spontaneously, the ischemic tissue becomes necrotic (infarction). The dynamic situation after plaque rupture is called acute coronary syndrome (ACS).

ACS may be almost symptomless and thus result in an unrecognized "silent" myocardial infarction [1]. When ACS causes symptoms, medical contact is often sought. ACS associated symptoms (notably chest pain) can have many other causes, ranging between innocent and emergency diagnoses. The initial triage at first medical contact (often with ambulance personnel) obligatory involves the making of an electrocardiogram (ECG). This ECG is, together with the symptoms and the patient history, crucial in establishing the working diagnosis of ACS. When the ECG shows ST-elevation, the current guidelines recommend immediate revascularisation by percutaneous coronary intervention (PCI), preceded by thrombolysis if there is no quick access to PCI [2]. With a non-ST elevation ECG, initial antithrombotic treatment is recommended [3]. There are, however, good arguments in favour of immediate PCI as initial treatment in all ACS patients[4]. In that case, the ECG is no longer used to discriminate between different treatment modalities, but rather to demonstrate the very existence of acute ischemia, thus helping to establish the working diagnosis of ACS.

This diagnosis of ACS should have sufficient sensitivity, because false negative decisions cause delayed access to PCI and, consequently, an increased infarct size. Also, this diagnosis should have sufficient specificity, because of the huge costs involved with false positive cathlab activations.

The current criteria to detect ischemia require ST amplitudes at the J point in the order of magnitude of 100 μV. However, sensitivity is quite low at this threshold[5]. For a lower threshold, e.g., of 50 μV, specificity goes down. The latter is caused by the fact that many persons have a pre-existing nonzero J amplitude. Serial ECG analysis (comparison of the acute ECG with a previous non-ischemic ECG of the same patient) with a low threshold would likely facilitate more sensitive ischemia detection without the drawback of low specificity.

Serial ECG would also facilitate the diagnostic use of the ventricular gradient (VG, the spatial integral of the heart vector over the QT interval[6]) for ischemia detection. Previous studies by our group have shown that ischemia detection by serial comparison of the ST amplitude and of the VG is feasible and has a high sensitivity [5,7]. This work was done in a group of patients with acute ischemia in the setting of elective PCI.

Knowledge about the dynamics in ST amplitude and VG in non-ischemic ECGs is pertinent to specificity assessment of ischemia detection by serial ECG analysis. ECGs may change on the long term, e.g., due to ageing, to changes in habitus, to developing disease and to changing medication. Additionally, ECGs are not fully

reproducible. Various factors, like differences in electrode positions, differences in heart rate, fluctuations in electrolyte concentrations, presence or absence of stressors, diurnal variations in hormone concentrations, may cause differences in subsequent ECGs. When serial ECG analysis is used to detect ischemia and the acute ECG is compared with a reference ECG, such a reference ECG should be sufficiently stable.

In our current study, we sought to answer the question if the ST vectors and the ventricular gradient in subsequent non-ischemic ECGs of stable patients are sufficiently reproducible to use one of these ECGs as a reference ECG in serial ECG analysis for the purpose of ischemia detection.

## 2. Methods

Data were selected from our Departmental ECG database, comprising more than 800,000 ECGs. Searching of candidate patients was done by a computer algorithm. The algorithm selected patients who had two elective ECGs, made in the outpatient clinic and 1-2 years separated in time. These ECGs were called ECG1 and ECG2. ECGs made during hospital admission or at the emergency department were excluded. Normal as well as pathological ECGs were acceptable as long as the technical quality was sufficient and as long as the ECG demonstrated regular sinus rhythm.

To further assure stability of the clinical condition of the patients in our study, patients who had a preceding ECG that was made less than one year before ECG1 were excluded, because of the increased likelihood that an acute clinical event might have occurred briefly before the selected ECG pair.

### 2.1. Clinical diagnosis

For each ECG the associated cardiologic diagnosis was noted by checking a set of diagnostic statements in the digital patient file. It was also assured in the digital patient record that no major clinical event had occurred preceding ECG1 or in between ECG1 and ECG2 (in that case, the patient was excluded).

### 2.2. ECG interpretation

All ECGs were interpreted by the Glasgow ECG Analysis Program[8], and categorized into abnormal and normal as to QRS duration, QT interval, etc.

### 2.3. ECG analysis

ECGs were analyzed by our vectorcardiographically-oriented research tool LEADS[9], using the Kors matrix for vectorcardiogram (VCG) synthesis. After computation of an averaged beat, the automatically determined onset-QRS, J-point end end-T settings were manually verified by two observers, and when necessary corrected. The J point was localized according to the procedure mentioned in the Minnesota code[10]. End of the T wave was defined in the vector magnitude signal as the time instant where the tangent to the point with the steepest slope of the descending limb of the T wave intersects the baseline. Then, three variables were computed: the ST vectors at the J point and 60 ms after the J point (ST(J+0) and ST(J+60)), and the ventricular gradient vector (VG).

Finally, for each patient, a serial comparison of ECG1 and ECG2 was done. In this comparison we computed the difference of the ST vectors at the J point, $\Delta$ST(J+0), of the ST vectors 60 ms after the J point, $\Delta$ST(J+60), and of the VG vectors, $\Delta$VG.

## 3. Results

A total of 398 patients (254/144 male/female, mean±SD age halfway ECG1 and ECG2 was 57±16.3 years) were included. Body mass index at the time of ECG1 was 26.3±4.1 $kg \cdot m^{-2}$.

### 3.1. Clinical diagnosis

Table 1 lists the prevalence of the major diagnostic categories in the study group.

Table 1. Prevalence of major diagnoses. The sum of the diagnoses exceeds the number of patients in the study group, because more than one diagnosis can apply to a single patient.

| Diagnosis | N | % |
|---|---|---|
| Systemic Hypertension | 113 | 15.1 |
| Valvular Heart Disease | 107 | 14.3 |
| Arrhythmia | 105 | 14.0 |
| Myocardial Infarction | 81 | 10.8 |
| Conduction disorders | 65 | 8.7 |
| Stable angina | 64 | 8.5 |
| Non-ischemic cardiomyopathy | 63 | 8.4 |
| M. Marfan | 56 | 7.5 |
| Diabetes mellitus | 54 | 7.2 |
| Non-cardiac diagnoses | 24 | 3.2 |
| Heart failure | 11 | 1.5 |
| Pulmonary hypertension | 7 | 0.9 |

### 3.2. ECG interpretation

According to the Glasgow ECG interpretation program, 445/798 (55.9%) of the ECGs were classified as abnormal or borderline abnormal. An overview of categories of ECG abnormalities is given in Table 2.

Table 2. Major categories of ECG abnormalities in the 796 ECGs of the 398 patients, according to the Glasgow ECG interpretation program.

| Category of ECG abnormality | N | % |
|---|---|---|
| Sinus tachycardia and bradycardia | 239 | 29.9 |
| Abnormal P wave | 66 | 8.3 |
| Abnormal AV conduction | 107 | 13.4 |
| Abnormal frontal QRS axis | 157 | 19.7 |
| Prolonged QRS duration | 168 | 21.1 |
| High QRS amplitude | 47 | 5.9 |
| Abnormal ST segment | 162 | 20.3 |
| Abnormal T wave | 223 | 27.9 |
| Long QT | 19 | 2.4 |
| Abnormal or borderline abnormal ECG | 445 | 55.8 |

## 3.3. ECG analysis

Figure 1 shows the histograms of ΔST(J+0), ΔST(J+60) and ΔVG. The proposed thresholds for serial detection of ischemia, 50 μV for ΔST and 20 mV·ms for ΔVG were 66 times (16.6%) exceeded for ΔST(J+0), 45 times (11.3%) for ΔST(J+60), and 89 times (22.4%) for ΔVG.

## 4. Discussion

We studied the reproducibility of ECGs in a group of 398 stable patients with a variety of cardiovascular pathology (Table 1), focusing on the ST vector at the J point and 60 ms thereafter, and on the ventricular gradient, VG. The ECGs in our study group showed a high degree of pathology (Table 2). The results of our study showed that the reproducibility of the ST and VG vectors is quite acceptable when this is compared to the proposed thresholds for serial ischemia detection, 50 μV for ΔST and 20 mV·ms for ΔVG. In case we would assume that ECG1 is a reference ECG, and that ECG2 is an acute ECG to be compared with ECG1 for the detection of ischemia, there would only be a false positive diagnosis in the order of magnitude of 20% (16.6% for ΔST(J+0), 11.3% for ΔST(J+60) and 22.4% for ΔVG), which would be quite acceptable.

In conclusion, this study supports the view that serial ECG analysis for ischemia detection is feasible in terms of specificity. Earlier first results, obtained in ECGs recorded during acute coronary occlusions, supported the view that serial ECG analysis for ischemia detection is feasible in terms of sensitivity[5,7]. Further research in the actual clinical setting of acute coronary syndrome is needed to corroborate these findings.

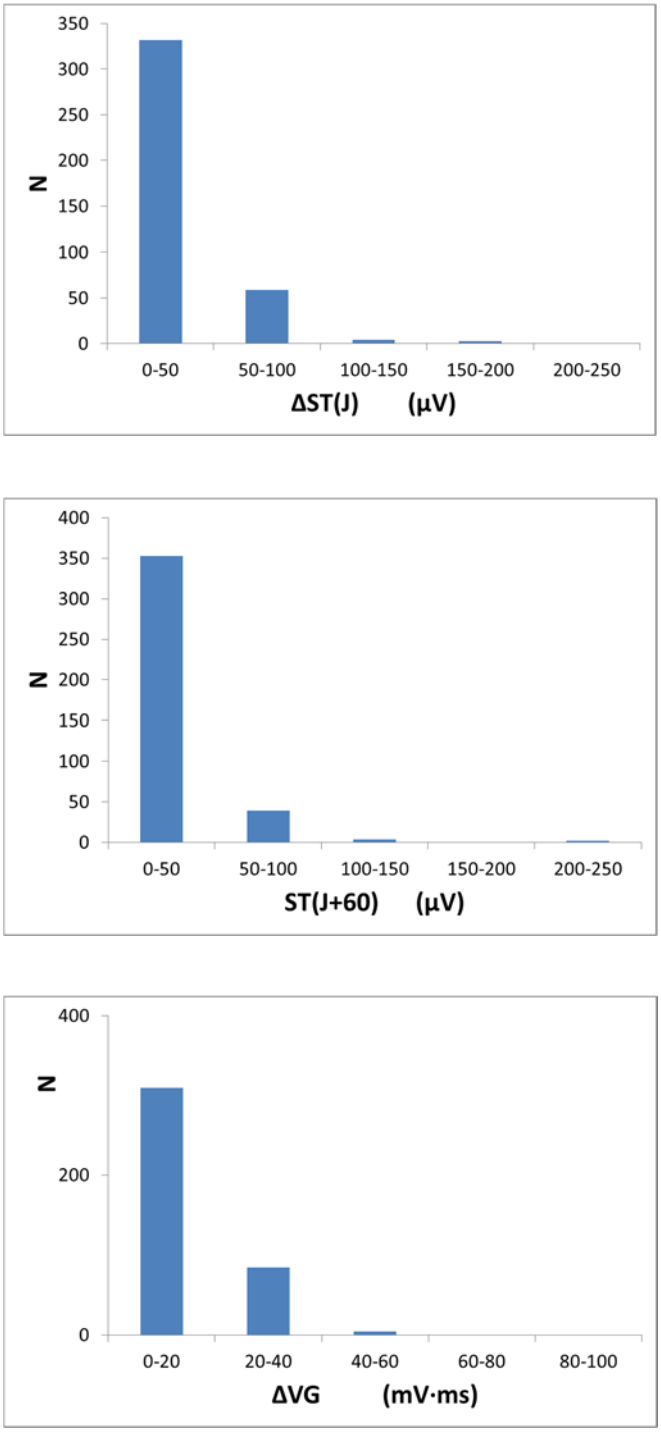


Figure 1. Histograms of ΔST(J+0), ΔST(J+60) and ΔVG. Data in the first bins (0-50 μV for ΔST and 0-20 mV·ms for ΔVG) are below the proposed ischemia threshold in serial ECG comparison.

## References


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