ST and Ventricular Gradient Dynamics During Percutaneous Transluminal Coronary Angioplasty

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Abstract

Diagnosis/triage in the hyperacute phase of the acute coronary syndrome (ACS) is mainly based on the ECG. There are serious limitations in the interpretation of the ST segment in the ECG in the setting of ACS, so there is need for the investigation of alternative or additional ECG features. Therefore we studied the potential role of the Ventricular Gradient (VG) in acute ischemia.

We computed, in ECGs of 84 patients (pts) recorded during elective PTCA, the maximal values of VG and ST, and the changes with respect to baseline, ΔST and ΔVG. In most pts, ΔST and ΔVG assumed the same direction and changed proportional in magnitude;55% of the pts responded to balloon inflations with ST elevation (STE) ECGs, 45% with non-STE (NSTE) ECGs. In a subset of 31pts with sestamibi area-at-risk (AAR) assessment, ROC analysis showed comparable performance of the maximal ST, ΔST, VG and ΔVG values to discriminate small and large AARs (areas under the curve >0.80, P<0.01).

In conclusion, our study shows that: 1) the VG has, in addition to ST, a potential role in detecting ischemia and in relating this to the area at risk, and 2) the STE/NSTE classification of ischemic ECGs is not very meaningful to discriminate between complete and partial occlusions.

1. Introduction

Diagnosis and triage in the hyperacute phase of the acute coronary syndrome (ACS) is mainly based on the ECG. According to the guidelines[1, 2], patients with an ST-elevation (STE) ECG are thought to have a complete occlusion of a coronary artery segment, and, hence, to be at risk of developing larger, transmural infarctions. Swift transportation to a hospital with PTCA facilities is needed to mechanically restore blood flow as soon as possible. Patients with a non-STE (NSTE) ECG are supposed to more likely have an incomplete occlusion that might lead to a smaller, non-transmural infarction. In these patients immediate in-hospital anti-thrombotic treatment is indicated as long as ACS cannot be ruled out. With insufficient therapeutic response and blood chemistry suggestive for cardiac cell death, the patient finally has to undergo PTCA as well, often first requiring transportation to a hospital with PTCA facilities. The time loss causes a larger infarcted area, moreover, NSTE may comprise high-risk conditions like posterior-wall infarction[3].

Hence, the role of the ECG in diagnosing and further specifying an impending myocardial infarction is crucial. Wang and colleagues[4] showed that of 1957 NSTE-ACS patients 528 (27%) had a complete occlusion. We found that 38.8% of 441 patients (a subgroup of patients studied previously[5]) with single vessel disease and a complete occlusion had a NSTE ECG. These observations are the logical consequence of the fact that the manifestation of ischemia in a given ECG lead as either ST depression or elevation simply depends on the projection of the ST injury vector on the lead vector. If a patient with a complete occlusion has an injury vector that is small or in the "wrong" (mainly posterior) direction, the ECG doesn't meet the STE criterium and is classified as NSTE.

The limitations in the interpretation of the ST segment in the setting of ACS prompt for the investigation of alternative or additional ECG features. While ST segment deviations are caused by phase 4 injury current (ischemic myocytes have a less negative resting membrane potential than well-perfused cells), the spatial ventricular gradient (VG, the spatial QRST integral) measures gradients between action potential duration/morphologies throughout the heart (during ischemia, myocardial action potential durations initially increase and thereafter decrease in amplitude and duration). Hence, the VG yields different information than ST. We studied the potential role of the VG in acute ischemia by analyzing a database of ECGs collected during elective PTCA.

2. Methods

ECGs were recorded in the setting of elective PTCA procedures performed in 1995 and 1996. These data (the STAFF III database) is unique because of the relatively long balloon inflation times. As such it is a model of the hyperacute phase of ACS in humans.

2.1. ECG processing

The ECGs were preprocessed by low-pass (100 Hz) filtering (to remove fluoroscopy-related interference) and by coarse baseline removal[6]. Further ECG processing was done in BEATS[7], our vectorcardiographically-oriented ECG analysis system. BEATS first synthesizes a vectorcardiogram (VCG), and then interactively detects beats, defines the isoelectric points of each beat, fine-corrects the baseline by piecewise linear regression through these points, and determines landmarks in time (onset QRS, J point, peak and end of the T wave) in each beat. Inherently to the recording conditions, ECGs had sometimes abundant artifacts: beats were manually deselected when signal quality was low. In the remaining beats we computed the ST vector (magnitude, azimuth and elevation at J + 60ms) and the spatial QRS, T and QRST integrals (the QRST integral is the same as VG).

We analyzed all reference ECG recordings made in the catheterization room before the actual PTCA procedure started, and the ECG recordings made during the first balloon inflation (intentionally made with some pre- and post-balloon-inflation context). We made plots of 1) the ST vector magnitude, 2) the QRS width / QTpeak / QT intervals, and 3) the QRS, T, and QRST integrals, to facilitate reliable identification of the ECG episodes of interest (the dye injections during PTCA cause – very briefly, though – ischemia in the complete perfusion area of the involved coronary artery and should, hence, be left out of consideration). If possible, we selected all episodes of interest from the PTCA ECG recordings.

Table 1. Patient characteristics of the study group.

	Number	Percentage
N	84	
Sex (male/female)	54/30	64/36
Age (mean±SD)	60±11	
Previous infarction	32	38
Aberrant conduction	3	4

For each patient we attempted to identify three ECG episodes of interest: 1) a baseline ECG; 2) an occlusion ECG; 3) a reperfusion ECG. When a baseline ECG could not be found in the PTCA recording due to dye injections immediately preceding occlusion, or because of a late start of the recording, we defined a baseline ECG episode in the reference recording. If this was impossible, e.g., because heart rate differed too much from that during PTCA, the patient was excluded. When dye injections occurred during occlusions, the related beats were excluded from analysis. Sometimes, presence of multiple dye injections during occlusion precluded further analysis of that patient. Reperfusion ECGs were defined as the episode immediately after release of the occlusion, until the ECG assumed baseline conditions. Many reperfusion ECGs could not be analyzed because of dye injections.

2.2. Study group

The above mentioned reasons for exclusion, together with some additional exclusion criteria (atrial fibrillation, misplaced ECG leads, too much missing ECG data) lead to a study group of 84 out of the 104 patients in the STAFF III database. Tables 1 and 2 show the group characteristics and the occlusion sites, respectively.

Table 2. Balloon positions during the occlusions.

Site	Number	Percentage
Left main	2	2
Proximal LAD	14	17
Prox-mid LAD	3	4
Mid LAD	6	7
LAD diagonal	2	2
Proximal RCA	17	20
Prox-mid RCA	2	2
Mid RCA	12	14
Dist RCA	10	12
Prox LCx	7	8
Mid LCx	4	5
Dist LCx	5	6

2.3. ECG diagnosis

Ten-second ECG segments from the baseline ECGs and from the terminal part of the occlusion ECGs were transferred to the LUMC departmental ECG management system and analyzed by the University of Glasgow ECG Analysis Program[7]. Thus, an ECG diagnosis was generated of the baseline ECG, and STE / NSTE classifications were established of the occlusion ECGs by using the measurement matrix data of the Glasgow program. STE was diagnosed as an elevation at the J-point of ≥ 0.2 mV in two or more contiguous leads in leads V1, V2 or V3, and of ≥ 0.1 mV in other contiguous leads. Contiguity in the frontal plane is defined in the lead sequence aVL, I, inverted aVR, II, aVF, III. When the ECG did not qualify as STE, it qualified as NSTE.

2.4. Occlusion-related ST and VG changes

We computed the ST deviation due to ischemia as the difference vector, ΔST, between the baseline ST vector and the ST vectors for all beats in the occlusion ECG. To reduce noise, we then computed 10-beat moving averages. Analogously, we computed 10-beats moving average VG difference vectors, ΔVG. The ischemic response was characterized by the maximal magnitudes of

the ST and VG vectors and by the maximal magnitudes of the difference vectors ΔSTmax and ΔVGmax.

2.5. Area at risk assessment

In 31 of the 84 studied patients technetium sestamibi scans were done to assess the area at risk (AAR), expressed as a percentage of the left ventricular mass. In these 31 patients we performed simple linear regressions to estimate AAR from ΔSTmax and from ΔVGmax.

3. Results

Figure 1 shows a typical response of the magnitude of ΔST and of ΔVG during occlusion. Figure 2 shows the spatial orientation of ΔST and of ΔVG in de same occlusion episode. Obviously, depending on the occlusion site, the orientations of ΔST and ΔVG differ, but in most cases, like in Figure 2, they assume the same direction. Often, like in Figure 2, this direction is stable during one occlusion, but in a smaller number of cases we observed that the directions of the ΔST and ΔVG vectors change during the occlusion (example in Figure 3).

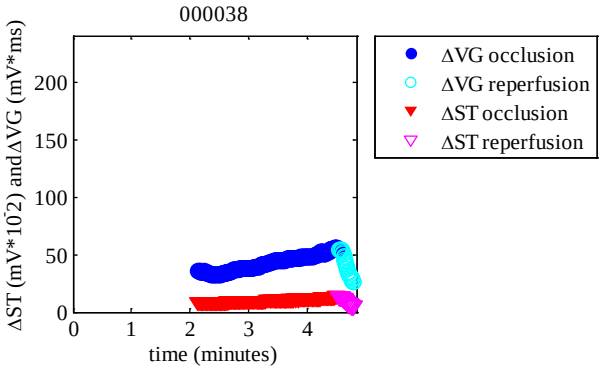

Figure 1. Subject 38. Magnitudes of the ΔST and ΔVG vectors during one occlusion-reperfusion event.

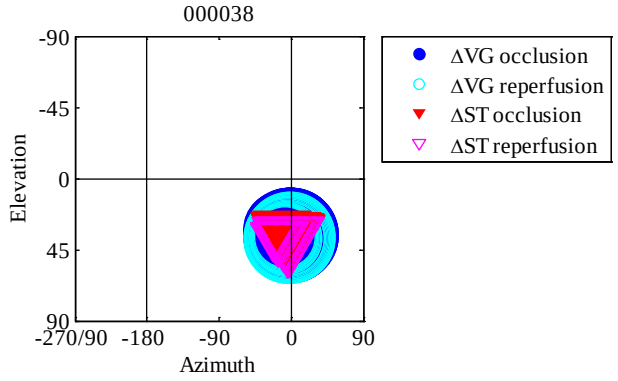

Figure 2. Same situation as in Fig. 1. Stable orientation of the ΔST and ΔVG vectors.

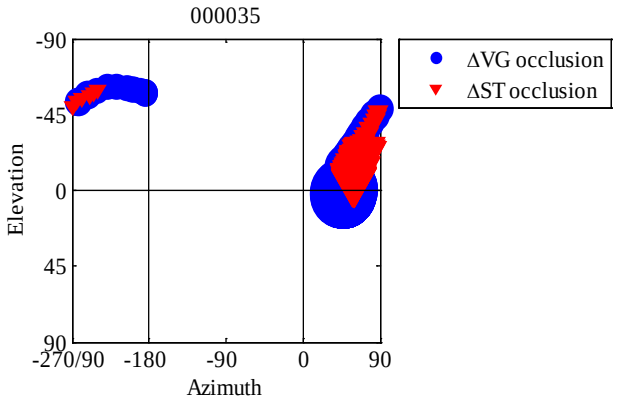

Figure 3. Subject 35. Spatial orientation of the ΔST and ΔVG vectors during an occlusion-reperfusion episode in which the orientation is not stable (minority of cases).

In the subset of 31 patients with AAR assessment, mean±SD AAR was 18.5±15.8%, median 14.0%, range 0.1-51.3%. STmax, ΔSTmax, VGmax and ΔVGmax correlated significantly with AAR (r=0.64, r=0.65, r=0.70 and r=0.57, respectively, P<0.01).

We arbitrarily defined the median AAR value (14%) as the separation between small and large areas (deemed to become small and large infarctions, respectively, might ischemia continue). ROC analysis revealed areas under the curve of 0.80, 0.84, 0.83 and 0.83 for STmax, ΔSTmax, VGmax and ΔVGmax, respectively (P<0.01).

In accordance with our previous findings, 38 of 84 patients (45%) had NSTE ECGs during occlusion. The distribution over the occlusion sites is given in Table 3.

Table 3. Occlusion sites and STE and NSTE ECGs.

Location	STE(N)	STE(%)	NSTE(N)	NSTE(%)
Left main	0	0	2	100
Prox LAD	14	100	0	0
Pr.md.LAD	3	100	0	0
Mid LAD	4	67	2	33
LAD diag	1	50	1	50
All LAD	22	88	3	12
Prox RCA	9	53	8	47
Pr.md.RCA	0	0	2	100
Mid RCA	7	58	5	42
Dist RCA	5	50	5	50
All RCA	21	51	20	49
Prox LCx	0	0	7	100
Mid LCx	1	25	3	75
Dist LCx	2	40	3	60
All LCx	3	19	13	81

4. Discussion

Many (45%) patients had a NSTE ECG during balloon inflation. Apparently, complete occlusions may well cause NSTE ECGs. This is fully in line with the findings by Wang et al[8], and underscores the need to reconsider the STE/NSTE stratification in the current guidelines. The site of an occlusion is an important determinant of the ischemia vector direction. As the current STE criteria favor LAD-related ischemia vector directions, LAD occlusions generate mainly STE ECGs, while LCx occlusions mainly generate NSTE ECGs (Table 3). Another striking observation is that subjects 43 and 61, both having a left main occlusion, had NSTE ECGs because of rather limited STmax values (126 and 80 μV, respectively) that brought, after projection on the relevant lead vectors, both subjects under the consensus 100 μV threshold for STE diagnosis. Sometimes, we saw NSTE ECGs in subjects with large AARs. E.g., subject 98, who had a NSTE ECG, had one of the largest AARs (30.2%).

The correlations between AAR and STmax or ΔSTmax (0.64 and 0.65, respectively) were much better than the correlation found by Andersen and colleagues[9] (0.29). A possible cause of this difference is that the data collected by Anderson et al. were recorded acute PTCA. Correlations between AAR and STmax, ΔSTmax, VGmax and ΔVGmax had the same order of magnitude. As nearly all patients had close-to-zero baseline ST vector magnitudes, the absence of a difference between the correlations with STmax and ΔSTmax is not striking. However, because VG is a non-zero variable in man, it is striking that VGmax correlates well with AAR.

The ROC analyses showed that STmax, ΔSTmax, VGmax and ΔVGmax had comparable diagnostic power in separating smaller-larger AARs. This is an important finding, because it implies that in patients with uninterpretable ST segments because of an abnormal ventricular excitation order the VG, that is independent of the excitation order[10], could discriminate between smaller and larger AARs.

5. Conclusions

Our study shows that the VG has a potential role in ischemia detection and in AAR assessment. Our data also underscore that STE-NSTE classification of ischemic ECGs is not meaningful to discriminate between complete and partial occlusions. Prospective collection of elective PTCA ECGs in patients with nonzero ST-amplitude baseline ECGs, in combination with sestamibi AAR assessment, and followed by an analysis like in our current study, could reveal if VGmax has additional value to STmax in ischemia detection and AAR assessment.

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