ST changes and temporal relation to the J point during heart rate increase and myocardial ischemia[☆]

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Abstract

There is no consensus concerning where in the ST segment to measure. We studied the relation between different J point intervals to ST results during tachycardia and ischemia.

Symptomatic (anesthetized) patients with coronary artery disease were paced at ascending incremental levels until they became ischemic. ST vector magnitude and ST vector change from baseline (STC-VM) as well as the sum of ST changes from all 12 electrocardiogram (ECG) leads (ECG ST sum) were measured at J point 0 millisecond, J + 20, J + 60, and J + 80 milliseconds for 34 patients.

ST segments increased in similar fashion during pacing and ischemia. There was no difference in ST results when measurement was performed at different time intervals for both STC-VM and ECG ST sum.

We conclude that ST assessment by ST change from baseline is not affected by different J point intervals during increased heart rate and ischemia in this clinical model of pacing-induced ischemia and vectorcardiographic ST analysis.

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Introduction

The choice of time interval from the J point for ST measurement has varied in clinical practice, and there is no consensus concerning an optimal interval. There are published recommendations for myocardial ischemia detection with the ST segment that specify a specific interval between J point and ST measurement though without references,¹ as well as guidelines for ST analysis that do not specify any interval between J point and ST measurement.^{2,3} Furthermore, in The Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction, ST measurement to be made at the J point was recommended without references.^{4,5}

Ideal ST analysis for identifying myocardial ischemia would include a simple consistent temporal relation to the J point with well-understood measurement error for the ST

point, facilitating reproducible and correct measurements for serial assessments and multiple patients. Then, ideally, ST response to ischemia would show a consistent response to myocardial ischemia. Also, ST response ideally would be highly specific for identifying ischemia. Currently, however, ST measurement challenges that confront clinicians today include that measurements close to J are dependent of the ability to set the J point with precision, which is not possible if the S wave does not end distinctly (eg, allowing identification of a vector change in vectorcardiographic [VCG] analysis). Also, ST measurement at long intervals from the J point may introduce artifact based on dynamic ST-segment shape and change as well as potential incursion of the ST measurement into the T wave at higher heart rates. These potential problems have been outlined previously in different settings where ST analysis is performed.^{5–7} There has been no conclusive examination of the effects of heart rate (HR) increases and confirmed ischemia on how to measure ST. We hypothesized that there would be a difference in ST course during increasing HR and ischemia, in relation to ST magnitudes measured with longer intervals from the J point. We aimed to test this hypothesis in a clinical study with controlled HR increases and also during myocardial ischemia using an online continuous VCG

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method and 12-lead ECG analysis for detecting change in ST segments.

Material and methods

With approval of the Umeå University Research Ethics Committee (and in conformation of the ethical guidelines of the 1975 Declaration of Helsinki) and after providing informed written consent, consecutive patients scheduled for elective coronary artery bypass surgery were enrolled into a study of electrocardiographic identification of ischemia that addressed a different scientific question.⁸ Selected from those subjects as a separate cohort for this investigation were the 35 subjects who were successfully paced to ischemia, and these VCG and ECG results were further analyzed here as a cohort concerning J point, VCG, and 12-lead ECG ST changes. Details for data study material and data collection have been reported in detail.⁸ Briefly, inclusion criteria included angina pectoris (Canadian Cardiovascular Society classification 3 and 4), positive exercise ECG test (horizontal or downsloping ST segment >0.1 mV), and coronary angiogram with “2 or 3” vessel disease including significant stenosis in the left anterior descending artery, no pathologic Q wave (defined as $\geq 25\%$ of the height of the partner R wave and/or >0.04 seconds in width and >2 mm in depth) in the preoperative ECG, and no left ventricular hypertrophy by echocardiography or ECG criteria (Solokov and Lyon criteria). Exclusion criteria included nonsinus rhythm, left or right bundle branch block, and not reaching and completing at least 3 incremental pacing steps.

Preparation

Subjects received a standard premedication, as well as their long-term medication on the morning of surgery. The numbers of subjects taking the following types of medications were as follows: β blockers, 25; calcium-channel blockers, 12; angiotensin converting enzyme inhibitors or angiotensin II antagonists, 13; nitrates, 33; diuretics, 1; and vasodilators, 7. General anesthesia was induced, and all subjects were treated with a standardized anesthetic plan with drug doses, respiratory, and hemodynamic management with invasive blood pressure monitoring (continuous arterial and central venous pressures) provided by anesthesia personnel. The preparation and data collection were performed before the start of surgery. A coronary sinus (CS) catheter (CCS-7U-90A, Webster Labs, Baldwin Park, CA), containing a bipolar pacing electrode 2 cm from the tip, was placed into the great cardiac vein under fluoroscopic guidance, for both pacing and CS lactate measurement.

Measurements

A 12-lead ECG (Mingograph 62, Siemens-Elema AB, Solna, Sweden) was recorded for each measurement sequence. Eight VCG electrodes⁹ were used for VCG ST analysis. Averaging periods of 15 seconds were analyzed to produce a single heart cycle signal for the 3 orthogonal (X, Y, and Z) ST measurements (MIDA 1000, 2.74 software,

Ortivus Medical, Täby, Sweden). These orthogonal ST measurements were used to generate the spatial ST vector¹⁰ for both an ST vector magnitude (ST-VM) and in ST change vector magnitude from baseline (STC-VM):

$$\text{ST-VM} = \sqrt{X^2 + Y^2 + Z^2}$$

in microvolts (μV)

$$\text{STC-VM} = \sqrt{(X_i - X_0)^2 + (Y_i - Y_0)^2 + (Z_i - Z_0)^2}$$

in microvolts (μV) with “₀” (baseline reference) and “_i” (current) measurements.¹¹ ST-VM and STC-VM at the end of each pacing level were measured off-line at J + 0 millisecond, J + 20, J + 60, and J + 80 milliseconds, and these were grouped for analysis at each pacing step. J points were determined for the VCG complexes based on device manufacturer’s algorithm identifying change in vector acceleration, and for 12-lead ECGs, the J point identification and ST measurements at the above described intervals were performed manually by 2 experienced analysts. Lactate concentration was measured (Yellow Springs Lactate Analyzer 1500, Yellow Springs, Ohio, USA) immediately in the operating room at the end of each pacing step in blood from arterial and great coronary vein (GCV) blood.^{12,13}

Protocol

Pacing (Pacesetter 3077, Osypka, Rheinfeld-Herten, Germany) was initiated at an HR of 10 to 15 beats per minute (bpm) higher than the subjects’ resting HR. A baseline VCG averaged complex was determined. Measurements, including ECG and VCG, were collected or recorded at the end of each 6-minute pacing step. Incremental pacing increases of 10 bpm in HR were used with the goal of measuring at multiple pacing levels until myocardial ischemia was observed, based on ST deviation of at least 0.2 mV in 2 adjacent leads (at J + 20 milliseconds) in the 12-lead ECG or if [lactate]_{GCV} is greater than [lactate]_{arterial}, at which time the protocol was discontinued.

Calculations

Rate-pressure product was calculated as HR $\times$ systolic blood pressure. The ECG ST sum was the total of all ST changes from each 12-lead ECG recording including only those that were 0.1 mV or greater. For each subject, the ECG single lead with the most ST change during pacing was identified. Left ventricular myocardial oxygen consumption was calculated as GCV flow myocardial arteriovenous oxygen content difference ($\text{mL O}_2 \times \text{min}^{-1}$). Transcoronary lactate flux was calculated as $([\text{lactate}]_{\text{arterial}} - [\text{lactate}]_{\text{GCV}}) \times \text{GCV flow (ml} \times \text{min}^{-1})$.¹⁴ Myocardial lactate extraction (%) was calculated as $([\text{lactate}]_{\text{arterial}} - [\text{lactate}]_{\text{GCV}})/[\text{lactate}]_{\text{arterial}} \times 100 (\%)$. Myocardial ischemia by lactate analysis was defined as myocardial late extraction that was decreasing and less than 10%.

Analysis

Measured values are presented as mean $\pm$ SEM. At each HR level, ST levels for all 4 J point relations

Table 1

Hemodynamic and metabolic results for each pacing step

	Baseline (n = 34)	B + 10 bpm (n = 34)	B + 20 bpm (n = 34)	B + 30 bpm (n = 34)	B + 40 bpm (n = 28)	B + 50 bpm (n = 21)
HR (bpm)	68 ± 1	78 ± 1	88 ± 1	98 ± 1	108 ± 1	118 ± 2
MAP (mm Hg)	80 ± 1	86 ± 2	86 ± 2	87 ± 2	89 ± 2	89 ± 2
CVP (mm Hg)	7 ± 0.5	6 ± 0.4	6 ± 0.4	6 ± 0.5	6 ± 0.5	6 ± 0.7
PAP (mm Hg)	14 ± 1	15 ± 1	15 ± 1	15 ± 1	16 ± 1	19 ± 1
PAOP (mm Hg)	8 ± 0.5	8 ± 0.5	8 ± 0.6	8 ± 0.6	10 ± 0.7	13 ± 1
GCVF (mL/min)	75 ± 8	87 ± 8	89 ± 9	97 ± 11	111 ± 12	123 ± 19
Lactate, flux (nmol/min)	−12.9 ± 2.2	−14.9 ± 2.6	−15.5 ± 2.9	−12.3 ± 3.2	−10 ± 3.4	−6.6 ± 4.1
Lactate extr (%)	17.9 ± 2.1	17.8 ± 2.1	17.5 ± 2.5	13.0 ± 2.7	9.3 ± 3.2	6.8 ± 3.0
RPP (mm Hg*bpm)	7251 ± 176	8797 ± 219	9946 ± 241	10,890 ± 276	11,990 ± 345	13,307 ± 390

MAP indicates mean arterial pressure; CVP, central venous pressure; PAP, mean pulmonary artery pressure; PAOP, mean pulmonary artery occlusion pressure; GCVF, great coronary venous flow; extr, extraction; RPP, rate-pressure product. Data are presented as mean ± SEM.

were grouped and analyzed for differences using analysis of variance. When differences were identified, further testing for differences between grouped J point relations was performed using Tukey “honestly significant difference” test. A *P* value of less than .05 was used for statistical significance.

Results

Thirty-five subjects (26 men and 8 women) completed at least 3 pacing-controlled HR increases before demonstrating myocardial ischemia. One subject was excluded from analysis because of technical problems in recovering the

Fig. 1. This figure demonstrates grouped ST results for J + 0 millisecond, + 20, + 60, and + 80 milliseconds. B = baseline pacing (68 bpm); B + 10 = baseline pacing + 10 bpm, and so on. Data are presented as mean ± SEM. For 1-way analysis of variance at each pacing step, # indicates *P* < .05, with a post hoc test, Tukey “honestly significant difference” test; * = *P* < .05 J + 80 vs J + 20; § = *P* < .05, J + 80 vs J + 0. STC-VM events (panel A) show no difference between the J point intervals for ST measurement at any of the heart rates during pacing and ischemia. 12-lead ST sum and the single ECG with most ST change (panels C and D) demonstrate the same pattern of response as STC-VM.

VCG signal. The mean HR at baseline was 68 ± 5.8 (SD) bpm. For baseline HR + 40 bpm, there were 28 subjects, and for baseline + 50 bpm, there were 21 subjects included for analysis. All subjects ended the protocol due to ischemia identified by CS lactate higher than arterial lactate, though in no cases was there ECG ST deviation of at least 0.2 mV in 2 adjacent leads. By myocardial lactate extraction criteria, 55 of a total of 167 measurements were conducted with myocardial ischemia present.

General hemodynamic results are shown in Table 1, which describe pacing steps, incrementally increased myocardial work as well as stable central circulatory conditions during conditions and, in the later pacing steps for each individual, myocardial ischemia by regional lactate production criteria. Myocardial oxygen consumption as an indicator of myocardial metabolic demand related to pacing was measured for 31 patients at baseline (7.7 ± 0.8 mL O_2 min^{-1}) and at maximal pacing level (14.0 ± 2.4 mL O_2 min^{-1}). The main finding was that although both ECG ST sum and STC-VM (Fig. 1) increased progressively during pacing increments and ischemia, there was no difference between grouped levels at each HR related to J +

0 millisecond, + 20, + 60, and + 80 millisecond measurements for either parameter. The single ECG lead with the most ST change also demonstrated the same results for HR and J point relations. Similar analysis of ST-VM groups for different J point intervals at common HR level showed only 1 instance where 2 groups differed (Fig. 1). All observations are shown in a correlation plot for ECG ST sum and STC-VM, and these demonstrated a similar and strong correlation for changes during HR increase and ischemia independent of time from J point to ST measurement (Fig. 2).

Discussion

These findings demonstrate that the time interval between the J point and ST measurement, at least for intervals 0, 20, 60, and 80 milliseconds, did not contribute significantly to variability in ST analysis in conditions of increasing HR leading to myocardial ischemia. The ST segments increased as expected during tachycardia and ischemia, and the temporal relation between the J point and ST did not affect the pattern of ST change. This is the first prospectively tested clinical material to demonstrate this with controlled HRs,

Fig. 2. Correlation plots are shown for all points with 12-lead ST sum plotted vs STC-VM for all subjects, all pacing steps ($n = 154$). J + 0 millisecond, + 20, + 60, and + 80 milliseconds are shown in the 4 panels. There is a strong agreement demonstrated between both methods of ST assessment over the range of ST elevation during J + 0 millisecond, + 20, + 60, and + 80 milliseconds.

controlled nonischemia status at start, and ischemia at the end of pacing for all subjects. Factors that might influence ST signals, such as myocardial hypertrophy, conduction disturbances, body position, lead placement, HR, and drugs, were controlled in this study design to allow study of the effect (or lack thereof) of interval between J point and ST measurement during ischemia.

STC-VM in a VCG system^{15,16} was used to study this because of its robustness as a scientific instrument. The ST vectors and change in these vectors (STC-VM) are derived from orthogonal leads, and this method presents a global or summary ST assessment. Clearly, VCG ST analysis of a summation beat differs from 12-lead ECG ST assessment where single-lead ST segments are examined individually for curve form as well as direction of change and magnitude. VCG ST analysis is very precise concerning the temporal aspects of ST change, even if for STC-VM the absolute direction of change does not matter. Therefore, if changes in the shape of ST curves occurred during development of ischemia, then this should have been identified by changes in distribution of STC-VM between different groups of J + ST times. This was not the case, and therefore, these findings support the suggestion that there was not a lot of heterogeneity in ST forms during the course of ischemia development. Furthermore, there were consistent findings of ST depression in the 12-lead ECG. A strong correlation for ST change for ECG ST sum and STC-VM for the range of measurements (J + 0, 20, 60, 80 milliseconds) reinforced the finding that time from J point for ST measurement in this model of early ischemia was not a determining factor for identification of ST changes during serial measurements. On this basis, we feel that it is valid to generalize to ST interpretation generally, when there is new onset ischemia based on increased myocardial work and tachycardia, and that the pattern of ST change does not appear to be significantly effected by the time interval between J point and ST measurement.

The ST vector change from baseline, if the baseline measurement is reliably taken during nonischemia, is probably the most specific ST variable available for ischemia detection during serial measurements with the possible exception of body surface mapping. This study design used this methodological strength by analyzing subject material that was not ischemic at the start of the pacing protocol but then progressed to ischemia. This was an optimal means for comparing ST pattern and progress for the different J point–ST measurement intervals. Because the main goal of ST analysis is the diagnosis of ischemia, testing of our hypothesis was necessary in subjects with coronary artery disease and both during nonischemic and ischemic conditions.

There can be variability in the J point that is not related to the ST segment, which can cast doubt on how reliable J + 0 milliseconds may be when used generally for ST measurement. In our material, which admittedly was designed for optimal conditions for ECG or VCG recording with subjects with no intraventricular conduction delay, ST results with J + 0 agreed with the other J point intervals. This does not mean that this type of ST analysis is optimal

for all clinical settings, but just that in controlled conditions, J + 0 seems to agree with the other intervals including J + 80 milliseconds concerning change related to HR or ischemia. These findings can support the recommendation for ST measurement at J + 0 millisecond as presented in recent guidelines.^{4,5}

Other factors that theoretically can affect the ST segment and which we presume were not present in these subjects include benign early repolarization and pericarditis. There is also a theoretical concern that ST-segment measurement can be affected by later phases of atrial repolarization,¹⁷ and the likelihood of this occurring (although low) is more if the ST measurement occurs at or a few milliseconds after the J point. To be certain that atrial repolarization is not present during ST measurement, a more invasive and localized form of atrial electrophysiologic measurement would be needed and that was beyond the scope of this study.

J + 80 milliseconds demonstrated no apparent disturbances at higher HRs with ischemia (highest pacing levels), and ST-segment measurements at J + 80 milliseconds did not incur on the T wave. The HRs that were achieved in this group of subjects were not all that high, baseline + 40 bpm (<150 bpm) but were within the clinical range that is common in our coronary care unit setting. Theoretically, at very high HRs, at least higher than those measured by this protocol, the ST measurement might be able to incur on the T wave, though adaptations in repolarization rates during higher HRs may make it difficult to predict at which HR this might occur. In patients with recent or ongoing myocardial ischemia, very high HRs are considered a serious problem and are treated independently.

The subjects in this study were exposed to myocardial ischemia in the immediate presurgical period, under controlled conditions. They were selected because of their clinical condition of easily inducible ischemia, and the protocol was designed to incorporate both HR incremental increases and at least one measurement during “demand” ischemia. It is a strength in the study design that both HR increases and ischemia can be assessed as far as J point relation to ST measurement. As is the case in clinical practice, it was not possible with this study design to clearly separate the effects of HR from those of ischemia on ST-segment magnitude.⁸ This clinical study implemented controlled conditions, with subjects who were anesthetized and lying supine. Some of the presumed difficulties in serial ST analysis may have to do with body position in awake patients.^{18,19} Clinical implications of these findings probably need to be limited to the resting coronary care unit patient where ECG and body position are monitored.

In some clinical circumstances, the J point can be very difficult to establish with precision, which is part of the perceived practical need to measure the ST segment at some interval from the J point. Ischemic ST segments were not analyzed separately for shape or form. There may be cases where J point level and ST-segment level/shape may have separate patterns that diverge, though these were not identified in this material. More analysis of this relationship

in specific clinically relevant settings other than (pacing-induced) tachycardia is needed, including, for example, during exercise testing or during myocardial infarction.

In summary, ST segments were measured at different intervals from the J point, ranging from J + 0 millisecond to J + 80 milliseconds in patients with ischemic heart disease who were paced by incremental HR steps from a resting nonischemic condition to ischemia. ST results showed no effect of different intervals from J point to ST measurement during pacing or during ischemia using both 12-lead ECG, the single ECG with most ST change, and VCG; and furthermore, there was strong agreement between 12-lead ECG and VCG. We conclude that the choice of a time interval 0 to 80 milliseconds from J point to ST measurement does not generate artifact problems in serial ST assessment, at least at the HRs associated with the onset of ischemia observed in this study.

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