Errors in Electrocardiographic Parameter Estimation from Standard Leadsets

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Abstract

The term quantitative electrocardiography refers to the extraction of parameters from electrocardiographic signals that can be linked to cardiac electrophysiology and patient diagnosis. Many of these parameters rely on measurements of maximal and/or minimal values, fiducial time markers, sums, or averaged values, over a specific leadset. Among the necessary conditions are that either a) the leadset used provides enough coverage and resolution to detect accurately the global extrema or b) the error incurred by missing the true values is insignificant. We have examined these theses by comparing standard ECG leads against a more complete picture of electrocardiographic distributions acquired through body surface potential mapping in a database of patients who underwent coronary angioplasty (PTCA). In the case of ST segment deviations, for example, in one study of 470 beats recorded before, during, or after PTCA, we found that precordial leads often predicted smaller deviations — for 184 beats which had deviations below 100 μV by precordial leads, the map distributions showed 57 (31%) which actually had maximum deviations larger then 100 μV (153.0 $\pm$ 40 μV, mean $\pm$ standard deviation). Thus we conclude that specific leadsets will tend to underpredict extrema and probably misrepresent underlying electrophysiology and that the resulting errors could limit the diagnostic efficacy of the ECG.

1 Introduction

With the availability of low-cost, high-speed computers with (relatively) vast amounts of storage has come something of an explosion in the application of "quantitative electrocardiography". A search of the Medline medical reference database on the subject string "quantitative AND electrocardiography" reveals no less than 509 citations, suggesting that the term at least has gained widespread acceptance. However, while quantitative techniques are, in general, to be welcomed to clinical medicine and research, their use as a diagnostic or patient monitoring tool begs a careful examination of just what is being counted and how it is being linked to physiology. In this paper, we focus on the use of standard electrocardiographic (ECG) lead systems as the basis for quantitative patient evaluation and attempt to highlight some limitations in the ability to extract meaningful parameters with such a limited sampling of human thoracic electrical activity.

Although there seem to exist no specific definitions of quantitative electrocardiography, the words suggests a means of collecting and processing electrical information from the thoracic surface with the goal of counting something. More specifically, quantitative electrocardiography might be described as the extraction from ECG signals of parameters of frequency, time, space, or amplitude, which can be linked, ideally in a continuous dependence, to disease states. From the time signals (electrocardiograms), one may apply signal transformation or decomposition approaches (Fourier analysis, wavelet transforms, principal component analysis), or simply measure specific amplitudes or durations from each PQRST cycle. By including the spatial relationships between the surface electrodes and the shape of the torso, it is possible to extract quantitative spatial parameters as simple as QRS axis deviations and as complex as surface spatial gradients and estimated epicardial potential distributions (the electrocardiographic inverse solution).

The advantages of quantitative electrocardiography are firstly those shared by all quantitative diagnostic methods: the ability to derive parameters that differentiate between disease states and stratify extent of disease. Implicit in this notion is the possibility to set thresholds that separate sick from healthy and mild from serious levels of illness. A further, related, advantage is that quantitative methods sometimes uncover a link between the value of a parameter and a *continuous* physiological state. Grossly simplified, a reduced R-R interval suggests a more rapid rate of pacemaker activity in the SA node, and the relationship is continuous and inverse. A third advantage to quantitative approaches is that they often lend themselves to automated analysis, both at the point of data acquisition and preprocessing and for the extraction and evaluation of specific parameters for diagnosis.

As stated above, there are many reports of quantitative electrocardiography in the literature, however, many fall into a few categories. Well represented are applications in heart rate variability analysis, late potential characterization, detection of atrial enlargement and left ventricular hypertrophy, and perhaps most frequently, measurement of repolarization abnormalities and ST-segment analysis. To reflect the importance of the latter two measures in clinical cardiology, the focus of this paper will be on extracting measures of variation in repolarization through the sequential analysis of ST segment potentials as a response to transient, acute ischemia.

The principle upon which quantitative electrocardiography rests is that the link between measurable variable and underlying physiology satisfies at least some and ideally all of the following assumptions:

1. That changes in the measured (or extracted) ECG parameter *always* reflect changes in specific, known aspects of cardiac physiology. And as a corollary;

2. That changes in aspects of cardiac physiology are reflected in the ECG in a consistent manner;

3. That the natural *variation* in measured or extracted parameters can also be quantified, at least in terms of population means and variance or referenced to patient-specific values and that from this, thresholds and normals ranges can be derived;

4. That the variable, patient specific, geometrical relationship between heart and sensing electrodes can be accounted for in the acquisition of data or extraction of parameters;

5. That a recording arrangement can be determined that is not sensitive to placement error or other uncontrollable differences between repeated electrodes applications; and

6. That this sensor arrangement captures a complete picture of parameter variability, *i.e.,* that the spatial coverage and temporal resolution of the electrode system are adequate.

With regard to the first two of these assumptions, Fozzard has written about the T wave that "Indeed, almost every change in environmental conditions may alter the T wave, so that it is

surprising that any useful information can be obtained from it."[1] While harsh critique, this view points to the difficulty in linking uniquely changes in electrocardiographic parameters and altered physiology. Fozzard goes on to outline a set of principles and corollaries describing the broad set of environmental and physiological factors that can influence the ST segment of the ECG. These include geometrical considerations, the phenomena of cancelation of extracellular current, the links between activation and repolarization sequences, and the ambiguity of factors that influence healthy and ischemia tissue differently.[1]

Despite these difficulties, there exist numerous studies documenting, for example, the observed links between altered recovery (*eg.,* prolonged QT) and propensity to arrhythmia[2,3]. Likewise, the utility of ST segment deviations as a means of monitoring myocardial infarcts and recovery from coronary angioplasty has been well documented[4,5]. Recent studies have attempted unsuccessfully to correlate *degree* of ST-segment deviation with exercise-induced ischemia as a means of improving diagnostic efficacy[6]. Previous studies of exercise testing, however, have suggested a link between degree of ST-segment deviation and extent of coronary artery disease[7].

The common feature of all these reports that is relevant to this study is that the ECG parameter used as the predictor was derived from either standard 12-lead or standard exercise lead electrocardiograms. In many cases of ST-segment analysis, the predictive parameter was the *maximum* value of deviation over all the leads used. This dependence on either extrema or ranges of values leads back to the last three of the assumptions listed above, those that relate to the lead system and its ability to deal with geometrical variations in heart position, altered electrode placement, and perhaps the most basic issue of all: the spatial and temporal coverage and resolution of the lead system.

Body surface potential mapping (BSPM) was developed to ensure complete data acquisition from the body surface and has played a major role in characterizing quantitatively the biophysical basis of electrocardiography[8]. However, results also suggest that most BSPM leadsets contain significant amounts of redundancy, both in the spatial and the temporal sampling used. This feature has led to numerous decomposition approaches that allow the entire time-space signal to be expressed in terms of a sparse sequence of spatial and temporal basis functions. In some approaches, the basis functions themselves can be derived from a set of data measured at full resolution by means of, for example, the Karhunen-Loeve transform[9]. A similar approach based on the information content of each lead can be used to develop 30–40 lead subsets of the full array, from which complete maps can then be reconstructed[10,11]. A different means of taking advantage of redundancy in BSPM was developed by Kornreich *et al.* in which each segment of the time signal from each lead was evaluated statistically for its ability to differentiate between cardiac disease states[12].

The common result from all these studies that is relevant to quantitative electrocardiography is the repeated finding that the standard 12-lead system does not sense all the available information.

Neither the CVRTI nor the Kornreich reduced-lead systems align with the 12-lead, and even if they did, neither of these systems uses the measured signals directly to interpret cardiac state. Mirvis, in an elegant series of articles has shown numerous cases of BSPM recordings that demonstrate clearly the inadequacies of the standard ECG leadsets in a variety of pathologies[13].

In this paper we provide some new evidence that standard lead systems, even the 12-lead system with its precordial unipolar leads, do not always provide adequate coverage to sense all of the relevant information available on the torso surface. We further suggest, based on analysis of body surface mapping data from patients undergoing percutaneous transluminal coronary angioplasty (PTCA), that the errors incurred by using a standard leadset could lead to incorrect evaluation of the patient's electrophysiological state and hence potentially, the diagnosis.

2 Methods

Data Acquisition: Body surface maps from two groups of patients undergoing percutaneous transluminal coronary angioplasty (PTCA) were used for this study. In one sample of 52 patients, we recorded epochs of 10 s before, during and after inflations. The leadset used was the CVRTI 32-lead reduced array [14]. In a second sample or 15 patients, a different acquisition system allowed recording durations that spanned the entire inflation, beginning 20 s. before inflation of the angioplasty balloon and continuing until 2 minutes after it was deflated. The leadset was the Dalhousie University system consisting of 117 torso electrodes and three Mason-Likar modified limb leads [15].

In both samples, the recorded signals were all gain corrected, baseline adjusted and averaged to form representative beats. With the CVRTI data, each epoch was averaged as recorded; with the Dalhousie system, the raw data was first segmented into 20 s epochs, then each of these was sorted into clusters of similar beats, then the majority cluster averaged. Technically bad leads (due to poor contacts or loose electrodes) were detected manually and corrected using either linear (CVRTI data) or Laplacian interpolation (Dalhousie data) [16]. Both lead systems also incorporate a reconstruction scheme permitting estimation of potentials on a "full" set of torso points; for the CVRTI system the full set contains 192 nodes, for the Dalhousie, 352.

Both "full" mapping leadsets included as a subset the standard precordial leads and the signals from these leads were extracted to form the "reduced" datasets. The same parameter extraction algorithms were then applied to both full and reduced datasets and the results compared. The following set of parameters were extracted from each lead:

1. Amplitudes of the R wave, peak-to-peak QRS, T wave, and the potential at 80 ms after the J-point.

2. Areas under the electrocardiogram (integrals) for the QRS complex, ST segment, STT segment (to end of T wave), and the QRST.

3. Duration from the onset of the QRS to the end of the T wave

We then compared the extrema of these values over the full and reduced leadsets for all of the beats. Of special interest were time series plots of parameters taken from averaged beats throughout single PTCA inflation cycles. These plots describe the change in parameter as the patient went from "normal" perfusion just before the balloon was inflated, in 20-second steps through the complete occlusion of the inflation itself, then to the deflation of the balloon and subsequent reperfusion. This tracking of parameter change over the course of time is essentially the same technique as used by Krucoff *et al.* in their online clinical ST-segment mapping system [5] and we, too, focus on ST-segment changes in this report.

3 Results and Discussion

One finding we have observed is a high degree of spatial localization of features of body surface potential distribution on the precordial surface of the thorax. The reasons for this are clearly not because cardiac electrophysiology changes only in the anterior part of the heart during ischemia. Instead, it is the proximity of the heart to the precordial chest combined with the inverse-squared dependence of electric potential with distance from the source that enhances the sensitivity and the magnitudes of the precordial potentials[17]. Focal potential patterns on the posterior or inferior aspects of the epicardium project very broadly to the back and lower torso, and hence are smaller, and appear to convey less information than the more direct projection of anterior epicardium activity to the precordium. In this study, we have observed that at least one major feature of ischemia induced electrocardiographic change can almost always be found in the precordial area; proximity to the heart ensures that this feature is larger in size then most others. This high degree of localization on the precordium also serves to enhance the ability of the standard precordial leads to reflect accurately the amplitude of global extrema.

However, the dominance of precordial features can reflect geometry more than electrophysiology and our results suggest that errors in extrema detection do arise when using precordial leads alone. An example of this can be seen in Figure 1, which shows the maximum elevation (left-hand panel) and depression (right-hand panel) of the potential measured 80 ms after the J-point over all leads, for each epoch through a single PTCA inflation/deflation cycle. The dashed line traces the value as recorded from the precordial leads, the solid line the value seen in the full leadset. The ST80 elevation revealed with full BSPM leads in the upper, solid line of the left panel is invisible to the precordial leads, which sense only a depression. ST80 depression is sensed more accurately by the precordial leads, but still with an error that ranges from zero to 130 μV.

Reasons for this discrepancy are apparent in the sequence of isopotential maps shown in Figure 2, which document the effect of PTCA-induced ischemia on ST80 potentials through the course of an inflation/deflation cycle. The top map was recorded with the guide wire in place but uninflated,

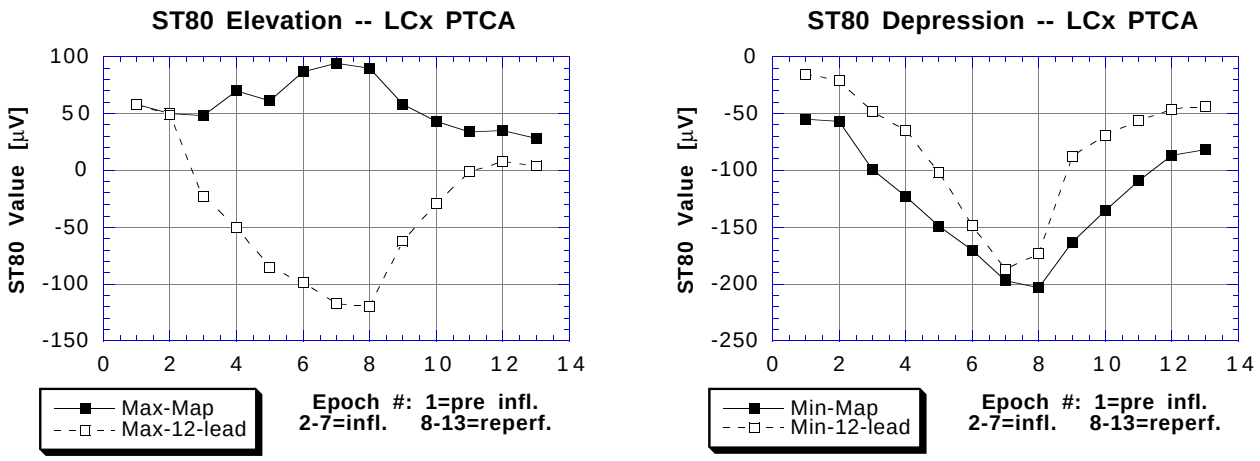

Figure 1: Maximum elevation (left-hand panel) and depression (right-hand panel) of ST80 potentials through the course of a single PTCA inflation. Solid lines show the values derived from full BSPM leadset and dashed lines (open boxes) show the values derived from standard precordial leads.

the second and third reflect early and then peak inflation, respectively, and the final two maps show a return to baseline during reperfusion. The major effect of the inflation was to depress the ST80 potentials all over the precordium, and this is detected with reasonable fidelity by the precordial leads. The ST80 elevation, however, is located on the lower back and is entirely invisible from precordial leads. While this effect is clearly smaller than that on the precordium, the maximum nevertheless shows an amplitude one-half that of minimum.

To evaluate the error in sensing maximum elevation and depression with precordial and BSPM leadsets, we also scanned 470 individual beats, recorded at various phases of PTCA from the CVRTI patient dataset. Figure 3 contains the results of this analysis for the ST80 elevation and depression, displayed as points on a scattergram with the extremum derived from the precordial leads on the abscissa and that from the BSPM leads on the ordinate. The superimposed lines mark the 50% and 100% error boundaries, *i.e.,* points that lie above these lines have greater than 50% and 100% error, respectively. Of the 470 beats analyzed, 176 (37%) lay above the 50% error line and 109 (23%) above the 100% line, suggesting that significant and frequent errors occur when extrema measurements are based on the precordial leads. Of a subset of 184 beats which had deviations less than 100 μV according to the precordial leads, 57 (31showed a mean of 153.0 μV $\pm$ 40 μV.

4 Conclusion

The motivation for this study was to examine a specific assumption of quantitative electrocardiography as it is commonly practiced in the literature. We chose an example in which amplitude parameters were extracted from BSPM data from patients undergoing PTCA. In many cases, we

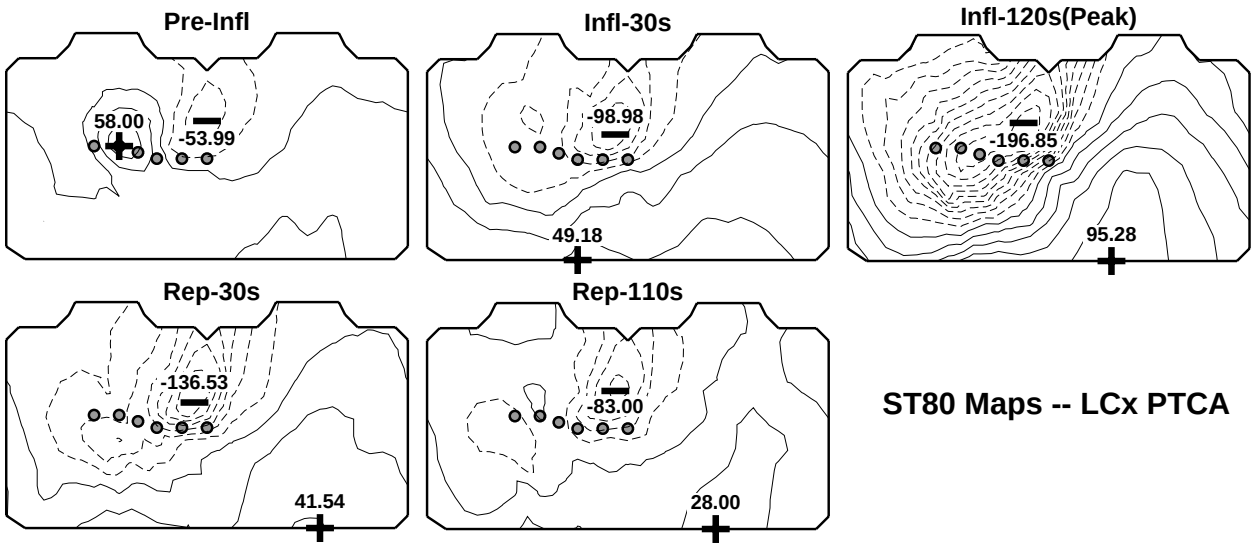

Figure 2: Body surface isopotential maps from the 80 ms after the J-point from beats recorded during a single PTCA inflation, for the same patient as in Figure 1. Each map is a projection of the body surface with the left half of the map representing the anterior surface and the outer edge of the map marking the right midaxillary line. Circles show locations of precordial leads and contours are linearly spaced at 20 μV intervals. Extrema are marked and labelled with plus and minus signs. The first map in the sequence (top panel) was recorded with the balloon in place but deflated, the rest represent 20-second epochs recorded 30 s into the inflation, at the end (peak) of the inflation, and 30 s and 110 s after release of the ballon.

found that the 12-lead electrocardiography was, indeed, able to reveal good approximations of the true parameter ranges and extrema. There were, on the other hand, a significant number of cases in which the lack of spatial resolution and lack of coverage of the standard leads resulted in major errors.

The focus of this study was an examination of just one aspect of quantitative electrocardiography, that of finding global extrema of various amplitude parameters. While this is perhaps the most intuitive aspect of the ECG signal to analyze, the spatial relationships between the heart, regional disease state, and body surface electrodes contain additional information that could assist in diagnosis. Standard leadsets are certainly more restricted in their ability to resolve such spatial information than BSPM lead arrays. Extraction of clinically useful information from spatial detail has been shown to be successful in localizing accessory conduction pathways[18] and determining the artery under treatment during PTCA[19,15]. However, broader application in important areas such as myocardial infarctions and exercise stress testing are topics of ongoing research. What this study shows is that simple correlations between, for example, ST-segment deviation levels as derived from standard leads and extent of ischemia will fail not only because of the electrophysiological ambiguity (assumptions 1–4 of quantitative electrocardiography), but because of an inadequate measurement

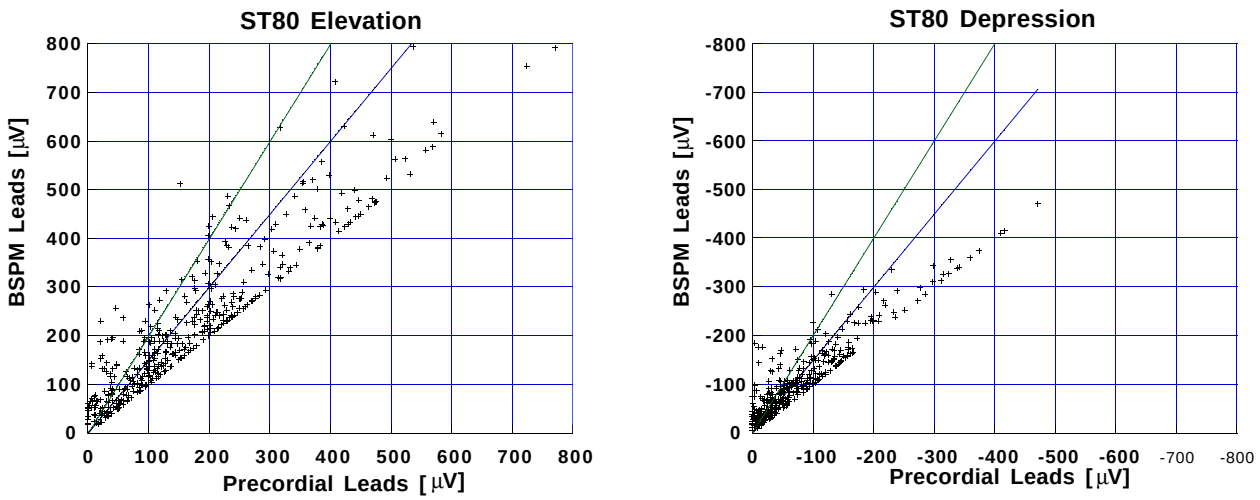

Figure 3: Scatter plot of maximum elevation (left-hand panel) and depression (right-hand panel) of the ST80 potential from all leads for each of 470 beats recorded at various time during PTCA procedures on 52 patients. Each point on the graph represents one beat with the abscissa value that extracted from standard precordial leads and the ordinate extraced from a full BSPM lead array. The lines bound the 50% and 100% error levels.

system (assumptions 5 and 6).

While we have not repeated any of the prospective clinical studies which have used ST segment mapping to great advantage, we would suggest, based on our limited findings, that further improvement in those results might be realized if more, and better placed, leads had been used. We did not report on the issue of whether another selected subset of the full mapping leadset would be adequate for detecting altered ST-segment deviation. We have, however, examined the ability of selected leads to *predict* complete body surface distributions of ST-segment potentials and report those results elsewhere in this journal.

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