Evaluation of the phase-plane ECG as a technique for detecting acute coronary occlusion

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Abstract

Objective: To evaluate the phase plane (PP) ECG as a method for detecting acute coronary occlusion (ACO). Background: Balloon inflation in a coronary artery during PTCA produces acute myocardial ischemia. The sensitivity of the standard ECG for detecting ACO is approximately 50%, depending on the number of leads recorded. Methods: The standard ECG signals of 18 patients (91 leads), undergoing PTCA were sampled and converted to digital data, prior to, and during acute coronary occlusion. PPs were constructed by projecting the ECG signals and their first derivatives onto a two-dimensional plane. Standard ECG signals and PPs, prior to ACO, were compared to their respective recordings and PPs during ACO. Results: Using the standard ECG analysis, the acute occlusion was detected in 39 of 91 leads (43%), and in 15 of 18 patients (83%), whereas using the PP analysis it was detected in 82 of 91 leads (90%), and in all 18 patients (100%) (P<0.001, for leads). The median number of leads per patient demonstrating standard ECG changes was 2.0, whereas for the PP analysis it was 5.5 (P<0.001). The specificity of the PP method was 83.5%. Conclusions: The sensitivity of the PP method for detecting ACO during PTCA was superior to that of the standard ECG analysis. A smaller lead system is required to detect changes of ACO, during PTCA, when the PP method is used. The PP method is simple, low-priced, and may serve to detect acute myocardial ischemia in a number of clinical settings.

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Keywords: Phase plane; ECG analysis; Myocardial ischemia; PTCA

1. Background

During balloon inflation (BI), PTCA may serve as a unique, controlled, human clinical model for assessing the evolution of acute myocardial ischemia [1–3]. The demonstration of acute myocardial ischemia is based on indirect measurements of mechanical and electrical variables. Using echocardiography one may detect wall motion abnormalities occurring early, within seconds, after BI [4–6]. Intra-coronary ECG recording has a sensitivity of 90% for detecting ACO but is invasive and largely unavailable [3,7]. The standard body surface ECG has a relatively low sensitivity (50%) for detecting ACO [6–9]. Sophisticated methods based on cross-correlation analysis of body surface ECG increased the sensitivity for detecting BI to more than 80% [7].

This study was undertaken to evaluate the phase
plane (PP) method for detecting ACO using PTCA as a model for controlled coronary occlusion in human patients. Briefly, the PP is a geometric object constructed by plotting the first derivative of the ECG signal against the ECG (voltage) signal itself. This representation allows to examine the slopes of the signal at the voltage value where they occur. This method allows concentrating on subtle morphological changes in the ECG signal in general and the QRS complex in particular. The PP technique is commonly used in physics [10], dynamics [11,12], and was previously used in medicine by several investigators. In one study the PP was used to evaluate the effect of cocaine administration on the electrocardiograms in cats [13]. It was also used to identify vulnerability to ventricular fibrillation in dogs [14]. In a qualitative study the deterministic versus stochastic characteristics of the electrical system of the human heart were evaluated using the distribution of trajectories in PP [15]. However, the use of the PP for detecting acute myocardial ischemia during BI has not yet been described.

Since the PP is a sensitive technique for detecting subtle morphological changes in the ECG signal, it was hypothesized that this technique may be useful for detecting ACO.

2. Methods

2.1. Patient group

Body surface electrocardiograms were recorded prior to and during BI in 52 randomly selected patients with suspected obstructive coronary artery disease who were referred to angiography in our institution, between July 1997, and August 1998. Twenty-one patients underwent PTCA, whereas 31 patients did not undergo angioplasty at the same session (they were referred to other revascularization procedures, or were demonstrated to have non-significant coronary artery disease by angiography). Patients who had previously undergone bypass surgery were excluded from this study. Poor technical ECG recordings precluded analyzing the ECG signals of three patients, thus 18 patients comprised the study group. All patients gave their informed consent prior to participating in this study. The study protocol was approved by the Ethics committee of our institution.

2.2. ECG signal acquisition

Radiolucent electrodes (CE 3M Health Care, Borken, Germany) were placed in standard body surface ECG locations. The sampling device was connected to these electrodes, via a braid of radiolucent carbon wires and a custom-made front-end piece (Atlas Research, Hod-Hasharon, Israel). ECG signals were recorded for approximately 5 min, prior to BI (baseline recording), when the patient was lying supine in the catheterization laboratory. Then, ECG signals were recorded during the first session of ACO. Recording was obtained, for as long as the procedure was deemed necessary by the operating cardiologist. The latter was blinded to the results of the analysis. Band pass filters of the ECG amplifiers were set of 1–500 Hz for all leads. The gain remained constant during both recordings. Signals were sampled at a rate of 1 kHz, digitized at 12-bit, and stored on a hard disk for later analysis (Digidata 1200A, CyberAmp, and Axoscope software, by Axon Instruments, Foster City, USA).

ECG signals were manually examined for the presence of arrhythmias or artifacts. No arrhythmias other than a limited number of premature atrial or ventricular beats were recorded, which did not interfere with the analysis. Artifacts that were disclosed were manually removed from the record. Signals were not digitally filtered after acquisition.

2.3. Standard visual examination of the ECG

In each lead, 10–15 consecutive QRS complexes were examined for conventional ischemic changes. The latter were defined as: (1) ST segment deviation (either depression or elevation), occurring 60–80 ms after the J point, or (2) T wave inversion [8,16,17]. The ST segment and T wave during ACO were compared with their counterparts prior to ACO. A visually detectable change in either one of the two segments of the complex was defined as a positive conventional ischemic change. In order to minimize the observer-related bias of standard visual ECG analysis, the two observers evaluated the ECG recordings in three stages. At the first stage, observer one evaluated the baseline ECG recordings (prior to ACO) for conventional ST-T changes, while blinded to the ECG recordings during ACO. At the same stage, observer two evaluated the ECG signals re-
corded during ACO, while blinded to the baseline recordings. At the second stage, each of the observers examined the data, which he was blinded to during the first stage. During the third stage, both observers compared their evaluations from the previous two stages. Any doubt related to the presence of a conventional change in the ECG recording was resolved by consensus between the two observers. For each lead recorded, prior to and during ACO, the result of this analysis was marked either as a ‘1’ for the presence of a standard change, or a ‘0’ for the absence of such a change.

2.4. The phase plane method of analysis

The digital voltage (V) record of a given ECG lead underwent numerical derivation via a series of computer programs, written in MATLAB (The Mathworks, Natick, MA), resulting in the first derivative of the voltage signal (V'). V, and V', were plotted in a two-dimensional space, called the phase-plane (PP) [10,12,13]. Before creating the PP, the baseline value (average value of the T-P segment prior to each individual ECG complex) was subtracted from the values of the QRS-T complex. This procedure aligned all complexes to a mutual zero baseline value, thus offsetting low frequency fluctuations of the signal. The PP is composed of trajectories, lines traversing the two-dimensional space. Each trajectory represents an individual electric activation, that is an individual ECG complex. The ECG complex is defined from the beginning of the Q wave to the end of the T wave. Time is not explicitly represented in the PP.

Phase planes prior to ACO were compared with PP during ACO. The same number of trajectories were displayed for comparison in both PPs. Of the total number of ECG complexes recorded during ACO for a given patient, one-fourth to one-third were displayed in the PP. The last third of the ECG signals was taken for comparison, since the state of ACO, and the associated myocardial ischemia, is a gradually evolving state throughout the PTCA.

Two observers visually examined the PPs, prior to and during ACO, for qualitative changes. The two observers independently documented changes in the PPs. Only changes that were independently identified by both observers were defined as PP changes during ACO. The PPs were evaluated according to three criteria: (1) changes in the amplitude of the R or S voltage values; (2) changes in the value of the maximal V' value, V_max; (3) changes occurring in the course the trajectories take in the PP. The latter may take the form of a new ‘knee’ appearing or disappearing in the PP. The result of this analysis, for each recorded lead, prior to and during ACO, was either a ‘1’ for the presence of any one of the changes in PP described above, or a ‘0’ for the absence of such a change.

In order to evaluate the specificity of the PP method another pair of PP was constructed and compared. The PP constructed from the ECG trace recorded prior to ACO (typically from the last third of the trace) was compared with a PP constructed from the first third of the ECG trace. Thus, two baseline PP were compared (as described above) in order to assess changes occurring in the PP which are not related to ACO.

2.5. Statistics

The outcome of each of the above two methods of analysis was defined as ‘1’ or ‘0’. ‘1’ representing a change between the baseline state and the state of ACO, whereas ‘0’ stands for the absence of such a change. Four outcome combinations were possible for each ECG lead: (a) both methods of analysis demonstrated a change, (b) both methods of analysis did not demonstrate a change, (c) the standard ECG method demonstrated a change while the PP method did not, (d) the PP method demonstrated a change while the standard ECG method did not. The pairs of outcomes were organized in a 2×2 table. The McNemar test was employed to compare between the two methods of analysis [18].

3. Results

3.1. Patients

The ECG data of 18 patients were analyzed. Table 1 summarizes their clinical data.

3.2. Changes in the standard ECG during acute coronary occlusion

The upper panels of Fig. 1 present standard ECG
Table 1
Clinical characteristics of the study group

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>61.1±6.9 years</td>
</tr>
<tr>
<td>Males</td>
<td>13 (72)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>3 (17)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5 (28)</td>
</tr>
<tr>
<td>Smoking</td>
<td>8 (44)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>9 (50)</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>5 (28)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>5 (28)</td>
</tr>
</tbody>
</table>

Cause for PTCA:
- UAP: 8 (44)
- SAP: 7 (39)
- Chest pain: 3 (17)

CAD, coronary artery disease; UAP, unstable angina pectoris; SAP, stable angina pectoris; MI, myocardial infarction.

Fig. 1. Standard ECG signals (upper panels) and the corresponding phase planes (lower panels), for patient BNZ, prior to (left panels), and during (right panels) ACO.
slightly smaller during ACO than prior to ACO. The latter change is not considered as an ischemic change according to the standard criteria [8,16,17].

3.3. Changes in the phase plane during acute coronary occlusion

Fig. 1c,d displays the PP constructed from 23 consecutive complexes of the same ECG signal displayed in Fig. 1a,b. In Fig. 1c,d, the abscissa is given in a.u. of voltage, while the ordinate is given in a.u. of $V'$ (the first derivative of the voltage signal). The location in PP of the peaks of the ‘R’ and ‘S’ waves shown in Fig. 1a are shown in Fig. 1c (for a detailed description of the correlation between the ECG signal and the PP, see Appendix A). In Fig. 1c,d the baseline value was already subtracted from the ECG complexes. Therefore, the voltage values of the complexes span the interval between $+1000$ and $-400$ a.u. In the title of Fig. 1c,d, the number of complexes presented in PP is given. For example, during ACO (Fig. 1d), a total of 85 complexes were recorded, of which 24 are displayed (from complex 52 to complex 75). The complexes forming the PPs were selected from the later portions of the ECG in order to represent the ‘most’ steady states. This is especially important for the state of ACO, as this state develops gradually throughout vascular occlusion. The arrow in Fig. 1d points to a ‘knee’ formed during ACO, while this knee is absent in the state prior to ACO (Fig. 1c). This knee reflects a change in the descending limb of the ‘R’ wave occurring during
ACO (see Appendix A). In particular, one may see in the PP prior to ACO that the descent from the ‘R’ to the ‘S’ wave is smooth. That is, as the voltage value decreases from 1000 to 200 a.u., the negative derivative value increases from 0 to a maximal negative value of −200 a.u. This means that the slope of the ‘R’ wave descent becomes steeper as the voltage value approaches 200 a.u. In contrast, during ACO, the ‘R’ wave descent becomes steeper as the voltage changes from 1000 to 750 a.u. However, from a voltage value of 750 to 550 a.u. the derivative value remains constant at −100 a.u. Then, as the voltage decreases below 550 a.u., the negative derivative value increases to a maximum of −175 a.u.

Another change that can be seen between the two PPs, representing the two states, is in $V'_{\text{max}}$. The maximal negative derivative value prior to ACO (approximately −200 a.u.) is greater than its counterpart during ACO (−175 a.u.).

To conclude, the PPs of patient BNZ (Fig. 1c,d) demonstrate changes in $V'_{\text{max}}$ and a ‘knee’ formation, while ACO is induced by balloon inflation in the circumflex artery.

Fig. 2c,d displays the PP constructed from 12 consecutive complexes of the same ECG signal displayed in Fig. 2a,b. The axes are as in Fig. 1. The ‘R’ waves in Fig. 2d are greater than their counterparts in Fig. 2c. ‘R’ wave changes are not considered as standard ischemic changes according to the criteria generally used. The peaks of the ‘R’ waves are located in the PP where the trajectories cross the zero derivative line at the maximal voltage value. Thus, the major change in PP observed for this patient during ACO is in the amplitude of the ‘R’ wave. Other subtle changes are detected in the ascending limb of the ‘R’ wave. Prior to ACO, the trajectories in PP above the zero derivative line are more ‘wavy’ than during ACO.

3.4. Comparing the standard ECG analysis with the PP method

Table 2 summarizes the results of the standard ECG and PP analyses. The total number of ECG leads analyzed by both methods was 91, that is five leads per patient (on the average). An average of three leads per patient was excluded from the analysis due to poor signal quality. Prior to ACO $310\pm 120$ complexes were recorded, whereas during ACO $98\pm 29$ complexes were recorded.

Of the 91 leads analyzed, 39 (43%) had demonstrated standard ECG changes during ACO. Of the 18 patients studied, 15 (83%) showed at least one standard ECG change during ACO, that is in three patients (BNZ, HSD, BSH) the standard ECG failed to demonstrate any change during ACO. In contrast, in 82 of 91 leads (90%) PP changes were demonstrated during ACO. In particular, using the PP method it was possible to detect all patients undergoing ACO.

Table 3 summarizes the performance of the two methods of analysis for detecting ACO in each of the recorded leads. In 39 leads both methods were able to detect ACO, whereas in nine leads both methods were unable to do so. In 43 leads the PP detected ACO whereas the standard ECG analysis failed to do so. There were no leads in which the ECG analysis was able to detect ACO and the PP method failed to do so. Applying the McNemar test resulted in a significant difference between the two methods of analysis ($P<0.001$). To conclude, the PP analysis is a more sensitive method for detecting ACO during PTCA than the standard ECG analysis.

By comparing the two baseline PP, the specificity of the method was assessed. Ninety-one pairs of baseline PP were constructed from 91 baseline ECG leads. In 15 (16.5%) PP changes were demonstrated, i.e., specificity of 83.5%.

The number of leads per patient displaying changes in each of the methods was examined. Using the paired Wilcoxon signed ranks test [18], it was calculated that the median number of leads per patient showing standard ECG changes was 2.0, whereas the median number of leads showing PP changes was 5.5 ($P<0.001$).

4. Discussion

The goal of this study was to evaluate a novel, noninvasive, ECG-based method of analysis for detecting ACO during PTCA. It was accepted a priori that ACO implies the presence of acute myocardial ischemia [2,3], and therefore changes in ECG and PP detected during ACO were related to the effect of acute myocardial ischemia on the electric signal of
Table 2
Standard ECG and PP changes detected during acute coronary occlusion

<table>
<thead>
<tr>
<th>No.</th>
<th>Patient name</th>
<th>Coronary artery dilated</th>
<th>No. of ECG leads analyzed</th>
<th>No. of leads showing standard changes in the ECG</th>
<th>Amp changes of R or S waves in PP</th>
<th>V_{max} changes in PP</th>
<th>‘Knee’ detected in PP</th>
<th>No. of leads in which PP changes were detected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BNZ</td>
<td>Cx</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>BTN</td>
<td>Cx</td>
<td>5</td>
<td>1</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>PTT</td>
<td>RCA</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>KOC</td>
<td>RCA</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>1</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>GLD</td>
<td>RCA</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>BTR</td>
<td>RCA</td>
<td>6</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>LYB</td>
<td>RCA</td>
<td>6</td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>SFY</td>
<td>D1</td>
<td>6</td>
<td>5</td>
<td>6</td>
<td>2</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>PNT</td>
<td>LAD</td>
<td>7</td>
<td>2</td>
<td>6</td>
<td>2</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>10</td>
<td>HSD</td>
<td>LAD</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>LEV</td>
<td>LAD</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>12</td>
<td>BRN</td>
<td>LAD</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>13</td>
<td>GNN</td>
<td>LAD</td>
<td>6</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>6</td>
<td>6</td>
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<tr>
<td>14</td>
<td>LCT</td>
<td>LAD</td>
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<td>2</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>15</td>
<td>SYN</td>
<td>LAD</td>
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<td>6</td>
<td>2</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>16</td>
<td>ARN</td>
<td>LAD</td>
<td>6</td>
<td>2</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>17</td>
<td>FCR</td>
<td>LAD</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>18</td>
<td>BSH</td>
<td>LAD</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

Total of 18 patients: LAD–10, RCA–5, Cx–2, D1–1

91 leads: 39 (43%) for PP, 71 (78%) for ECG, 35 (38%) for V_{max}, 44 (48%) for ‘Knee’, 82 (90%) for lead changes.

Amp, amplitude; PP, phase plane; V_{max}, maximum (positive or negative) derivative value; Cx, circumflex; LAD, left anterior descending artery; RCA, right coronary artery; D1, diagonal 1.

The sensitivity of the standard ECG for detecting ACO is low. Abboud et al. evaluated a number of noninvasive methods for detecting ACO during PTCA. They reported that standard ECG changes were revealed, by examining the surface ECG, in only six of 11 patients (54%) [7]. Wohlgelernter et al. studied the effect of ACO on the mechanical and electrical functions of the myocardium. They reported that only 64% of patients showed standard ECG changes on 12-lead ECG after 20 s of BI, while after 60 s of BI, ischemia was evident in 86% of the patients. It was emphasized that using a three-lead monitoring system results in decreased sensitivity for detecting ACO, compared with a 12-lead system [5,19]. Macdonald et al. studied two groups of patients, namely a group experiencing ST elevation and a group experiencing ST depression during BI. They reported that only 25 of 60 patients (42%) manifested these standard ECG changes. In their study four leads were recorded [6]. Hauser et al. studied the sequence of mechanical and electrocardiographic changes during PTCA. They reported that only eight of 18 patients (44%) demonstrated ST deviations during BI (four elevations and four depressions). They used the standard six limb leads [9].

From the above studies it follows that the use of a larger ECG lead system, increases the probability of detecting ACO by standard ECG analysis. However,
most of the laboratories for angioplasty use one to three ECG leads to monitor the patient undergoing PTCA [5,16]. The rate of detecting ACO with the use of a small lead system is relatively low. We hypothesized that the PP method may improve this rate. In the present study, using an average of five ECG leads per patient, 43% of the leads, and 83% of the patients demonstrated standard ECG changes during ACO. This result is in agreement with the reports mentioned above. However, applying the PP analysis to the same ECG data, it was possible to detect ACO in 90% of the recorded leads, and in all 18 patients studied. Thus, the PP method increases the probability of detecting ACO with a given (small) set of ECG leads.

Several factors explain the superior sensitivity of the PP method over the standard ECG analysis. First, using the PP, it is possible to evaluate the slope (derivative) of the ECG signal, at each voltage value of the signal. The PP directs our attention to changes occurring in the slope of the ECG, in addition to changes occurring in the voltage. Detecting a change in the slope of the ECG is difficult by visual examination, especially as physicians are not trained to seek such changes. In contrast, voltage changes expressed as differences in portions of the ECG (ST-segment deviation, T wave inversion) are routinely sought for. Examining the PPs during ACO allows detection of changes in both, the ECG signal and its slope.

The second factor contributing to the improved resolution of the PP is the correlation between consecutive trajectories in PP. The QRS complex is a nearly periodic signal, which appears in the same region of the PP. Thus, consecutive QRS complexes are superimposed in the PP, thereby enhancing their visualization. The third factor is related to the standard criteria defining an ischemic change. Using the standard criteria [8,16,17] ‘R’ and ‘S’ wave changes are not considered to represent acute myocardial ischemia. In previous work, ‘R’ and ‘S’ wave changes were shown to occur during ACO [20–22]. For some patients, the ‘R’ waves may increase yet for others they may decrease. The cause for ‘rejecting’ these changes as standard ischemic changes lies in the inconsistency of the occurrence of these changes during ACO [20–22]. The PP method is free of such limitation since it concentrates on change and not over its absolute direction.

4.1. Study limitations

The PP method relies on visual examination of trajectories in PP. Visual assessment is bound to inter- and intra-observer variability, that should be quantified if this method is to become practical. In this study, we tried to avoid such bias by evaluating the data sets in a semi-blinded fashion.

Using many trajectories in PP reflects longer time intervals and may obscure the geometric change. However, using a small number of trajectories may not reflect a true steady state. This limitation applies to the standard ECG analysis as well, where a small number of complexes are examined for standard changes. It is also acknowledged that PP changes may be formed by only a few trajectories, and may not actually represent the whole ECG record. Therefore, it is recommended to verify whether the pattern in PP is related to a part, or all of the ECG record.

As signal-to-noise ratio decreases, the visible subtleties of changes may be lost in the PP. However, if standard recording quality is kept, this should not limit the use of this method [23].

4.1.1. QRS versus ST-T

The use of the PP, as demonstrated here, allows better detection of changes occurring primarily in the QRS complex of the ECG. Since the voltage and derivative values of the P and T waves are usually small relative to those of the QRS, their reflection in PP is located around point (0,0). However, if desired, it is possible to ‘window’ any part of interest in the ECG signal, and construct a PP for that specific part.

4.2. Summary

A novel noninvasive method to detect ACO during PTCA is presented. The sensitivity of the PP method for detecting ACO was shown to be superior to that of the standard ECG analysis. This method is easy to apply, low-priced, and may detect ACO using less ECG leads than are required for detecting ACO using the standard ECG. Other clinical settings of acute
myocardial ischemia may be studied using the PP method.

Acknowledgements

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Appendix A

The zero horizontal and vertical lines divide the PP into four quadrants. The upper right quadrant contains trajectories, which are characterized by a positive derivative and voltage values, whereas, the lower right quadrant contains trajectories, which are characterized by a positive voltage value, and negative derivative value. The lower left quadrant holds trajectories characterized by a negative voltage and derivative values, whereas the upper left quadrant holds trajectories with negative voltage value and positive derivative. PP is interpreted by following the trajectories. A systematic way to follow a trajectory is by examining its course from a distinguished point, e.g., maximum or minimum voltage coupled with zero derivative. Table A.1 relates distinct points and quadrants in PP, to possible ECG waves.

Table A.1 Correlating the PP trajectories with the ECG signal

<table>
<thead>
<tr>
<th>Points and quadrants in PP</th>
<th>Possible ECG wave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper right quadrant</td>
<td>Ascending limb of the ‘R’ wave.</td>
</tr>
<tr>
<td>Intersection of trajectories with positive voltage value with the zero derivative line</td>
<td>Peak of ‘R’ wave (see ‘R’ in Fig. 1ac).</td>
</tr>
<tr>
<td>Lower right quadrant</td>
<td>Descending limb of the ‘R’ wave.</td>
</tr>
<tr>
<td>Intersection of trajectories with negative derivative value with the zero voltage line</td>
<td>The point where the descending ‘R’ wave changes to the ‘S’ wave.</td>
</tr>
<tr>
<td>Lower left quadrant</td>
<td>Descending limb of the ‘S’ wave.</td>
</tr>
<tr>
<td>Intersection of trajectories with negative voltage value with the zero derivative line</td>
<td>Trough of ‘S’ wave (see ‘S’ in Fig. 1a,c).</td>
</tr>
<tr>
<td>Upper left quadrant</td>
<td>Ascending limb of the ‘S’ wave toward the ‘j’ point.</td>
</tr>
<tr>
<td>The intersection of the horizontal and vertical zero lines—point (0,0).</td>
<td>This area of PP, surrounding point (0,0) contains the waves with low voltage, low derivative values, such as: the ‘P’ and ‘T’ waves.</td>
</tr>
</tbody>
</table>

References