QRS Changes During Percutaneous Transluminal Coronary Angioplasty and Their Possible Mechanisms

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**Objectives.** The purpose of the study was to describe the configuration, and investigate the mechanisms, of QRS changes occurring during percutaneous transluminal coronary angioplasty (PTCA).

**Background.** QRS changes during PTCA have been attributed to both a passive ST segment shift and conduction disturbances (peri-ischemic block). The direct relation between ST segment shift and QRS changes, however, has not been established, and the definition of conduction disturbances remains to be clarified.

**Methods.** Twelve-lead electrocardiograms (ECGs) were recorded before PTCA, at the end of 2 min of PTCA and after return to baseline values in 29 patients (left anterior descending coronary artery [LAD] in 13 patients, right coronary artery [RCA] in 14 and left circumflex coronary artery in 2). Electrocardiographic complexes before and during PTCA were superimposed to determine the amplitudes of initial, terminal and total QRS deflection; the relations of QRS changes to baseline (TP segment) and ST segment shift; and the duration of QRS and corrected QT intervals.

**Results.** 1) The direction of the initial QRS deflection was unchanged, but changes of its amplitude occurred. 2) Terminal QRS deflection changed in all patients with a ST segment shift >17% of the R amplitude, and the correlation between the decrease in the S amplitude and ST segment shift was significant (r = 0.9, p < 0.01) in patients with LAD PTCA. Correlation between changes in total QRS amplitude and ST segment shift in patients with RCA PTCA was weaker (r = 0.54, p = 0.056). 3) Transient conduction disturbance manifested by QRS widening in selected leads occurred in 2 of 29 patients.

**Conclusions.** 1) Changes in terminal QRS deflection during PTCA are proportional to the magnitude of the ST segment shift. 2) Conduction disturbances manifested by increased QRS duration occurred infrequently. We suggest that the term peri-ischemic block be applied only to changes in QRS configuration associated with QRS widening.

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Balloon occlusion of a coronary artery during percutaneous transluminal coronary angioplasty (PTCA) often produces an acute injury pattern on the electrocardiogram (ECG). The most conspicuous manifestations of this process are displacement of the ST segment and T wave and changes in the QRS configuration (1–5). Extrapolating from the animal models of acute coronary occlusion in which the ECG changes have been recorded by means of direct current (DC) coupled amplifiers (6–10), the shift of the ST segment can be explained by displacement of the TP segment (i.e., baseline shift or displacement of the ST segment itself). The differentiation between these two mechanisms is not feasible when the ECG is recorded by means of conventional alternating current (AC) coupled amplifiers, which compensate for the baseline shift.

The mechanism of QRS changes associated with acute injury pattern during PTCA is less well established. Of the two proposed hypotheses, one attributes the change in QRS configuration to the passive pull by the shifting ST segment, and the other to the intraventricular conduction disturbances caused by myocardial ischemia (4). Studies by Wagner et al. (4,5) indicate that both of these mechanisms can modify the QRS configuration.

A review of the published data suggested to us that it may be helpful to describe the QRS changes during PTCA in greater detail using a 12-lead ECG and to seek information that may contribute to an increased understanding of the mechanism of the QRS changes. Our analysis included measurements of the initial, terminal and total QRS amplitudes; QRS duration; correlation of the magnitude of the ST segment shift with the changes in the amplitude of QRS components; and corrected QT interval (QTc) changes.

**Methods**

Twenty-nine patients undergoing PTCA of one or more high grade (75% to 99%) stenotic lesions were selected at random for the study. Coronary angioplasty was performed according to the standard practice at our institution. Patients were excluded if the ECG showed deviations of the ST
Abbreviations and Acronyms

AC = alternating current
DC = direct current
ECG = electrocardiogram, electrocardiographic
LAD = left anterior descending coronary artery
LCx = left circumflex coronary artery
PTCA = percutaneous transluminal coronary angioplasty
QTc = corrected QT interval
RCA = right coronary artery

Results

Left anterior descending coronary artery (n = 10) and diagonal branch (n = 3). These groups included 10 men 45 to 79 years old and three women 69 to 83 years old, respectively. The baseline ECG was normal in five subjects and abnormal in eight patients, two of whom had Q waves compatible with a previous myocardial infarction.

ST segment changes. ST segment elevation occurred in precordial leads V₁ to V₆ in 11 patients and in leads I and aVL in five patients and was absent in one patient. In one patient ST segment elevation occurred only in leads I and aVL. Reciprocal ST segment depression was present in leads II, III, and aVF in four patients, in leads III and aVF in two patients and in lead III only in one patient and was absent in six patients. Maximal ST segment elevation occurred in lead V₂ in seven patients, in lead V₄ in two patients and in each of leads V₃, V₅, and aVL in one patient. Maximal ST segment elevation in lead V₄ occurred in one patient with occlusion of the LAD at mid-level (Fig. 1), and in one of three patients with occlusion of the diagonal branch. Of the two patients with occlusion of the diagonal branch, the ST segment elevation was maximal in lead aVL in one patient and was absent in the other patient.

QRS changes. QRS duration decreased from 124 to 120 ms in one patient and was unchanged in the remaining 12 patients (Fig. 1 and 2). Changes in R amplitude, S amplitude and total QRS amplitude were measured in the lead with maximal ST segment elevation in nine patients and in a lead next to that with maximal ST segment elevation (lead V₃) in three patients in whom maximal ST segment elevation occurred in the lead with an R amplitude <0.05 mV. In these three patients the difference between ST segment elevation in lead V₃ and the neighboring lead with maximal ST segment elevation was <20%.

The R amplitude increased by 25% to 33% in three patients and remained unchanged in 10 patients. Before occlusion, the S amplitude measured 10% to 93% (41.6 ± 28.0) of the total QRS amplitude. During occlusion, the S amplitude decreased by 7% to 93% (47.3 ± 29.0) in 12 of 13 patients and was unchanged in one patient. The total QRS amplitude remained unchanged in one patient and increased in two patients—one with occlusion of the diagonal branch by 28% and the other with occlusion of the LAD at mid-level by 5%. In the remaining 10 patients, the QRS amplitude decreased by 9% to 73% (35.7 ± 11.5%).
Corrected QT change. The QTc interval increased in eight patients by 4 to 28 ms\(^{1/2}\) (11.8 ± 8.4), decreased by 2 to 30 ms\(^{1/2}\) (15.3 ± 11.5) in four patients and was unchanged in one patient.

Relation between ST segment elevation and change of S amplitude. The plot in Figure 3 shows a significant correlation (r = 0.9, P < 0.01) between the ST segment shift expressed in percent of R amplitude and the decrease in S amplitude in 13 patients. Of similar significance (r = 0.95, p = 0.01) was the correlation between the ST segment shift expressed in percent of total QRS amplitude and the decrease in S amplitude.

Right or posterior descending coronary artery (n = 14). This group included six men 47 to 69 years old and eight women 39 to 71 years old. The baseline ECG was normal in two patients and showed Q waves attributable to a previous myocardial infarction in eight patients, right bundle branch block in two patients and T wave abnormalities in two patients.

ST segment changes. ST segment elevation occurred in leads III and aVF in 13 patients, in lead II in 10 patients and in one or more precordial leads in three patients; no changes occurred in one patient. Reciprocal ST segment depression was present in leads I and aVL in 13 patients and in one or more precordial leads in six patients. In eight patients the ST segment elevation was maximal in lead III, in four patients in lead aVF and in one patient in lead V\(_2\).

QRS changes. The QRS duration was unchanged in 12 patients (Fig. 4) and increased in selected leads in two patients—in one from 96 to 112 ms (Fig. 5) and in the other from 100 to 132 ms. The direction of the initial QRS deflection was unchanged in all patients, but the configuration of the QRS complex changed in 12 of 14 patients. The type of
configuration change, analyzed in the lead with maximal ST segment elevation, was dependent on the initial QRS configuration. In the leads with an RS or qRS configuration (n = 4), the S amplitude decreased and the total QRS amplitude decreased by 25% to 50%; these changes were similar to the changes in the precordial leads with an RS configuration in patients undergoing LAD occlusion. In the leads with qR (n = 3) or qRs (n = 1), the r amplitude increased and the total QRS amplitude increased by 25% to 200%. In the leads with QS (n = 2), Qr (n = 1) or Qrs (n = 1), a new R wave appeared in two patients, and R amplitude increased in two patients; changes in the QRS amplitude in these patients were small. In two patients the QRS configuration did not change—in one of these the ST segment shift was absent and in the other it was 9% of the QRS amplitude. Figure 6 shows examples of three types of QRS configuration changes.

**Corrected QT change.** The QTc decreased from 2 to 30 ms$^{1/2}$ (16 ± 10.7) in seven patients, increased from 6 to 11 ms$^{1/2}$ (9.5 ± 2.6) in four patients and remained unchanged in three patients.

**Relation between ST segment elevation and change in QRS amplitude.** As may be expected from the diversity of changes in the QRS amplitude, there was no strong correlation ($r =$...
0.54, p = 0.56) between the ST segment shift and the change in the QRS amplitude (increase or decrease) (Fig. 7).

**Left circumflex coronary artery.** In two patients occlusion of this artery produced no ST segment shift or change in the QRS configuration.

**Manifest intraventricular conduction disturbances.** In two patients in whom the QRS duration increased during PTCA, superposition of ventricular complexes before and during occlusion helped to visualize changes in the QRS configuration and duration in selected leads. In one of these patients, a wide terminal deflection appeared in the right precordial leads without any change in the QRS duration in the other leads (Fig. 8). In the other patient, appearance of a wide terminal QRS component increased the QRS duration in leads III and aVF more so than in the precordial leads (Fig. 9).

**Cardiac arrhythmias.** Arrhythmias did not occur during or immediately after PTCA in any of the patients.

**Discussion**

**Mechanism of ST segment changes.** This discussion will be limited to the sequelae of acute myocardial ischemia precipitated by occlusion of a coronary artery for ≤2 min. During this period extracellular potassium concentration in the ischemic area rises rapidly (12,13) and causes regional depolarization. Consequently, an electrotonic “current of injury” flows between the ischemic and normal tissue (8). During diastole, the injury current flows intracellularly from the ischemic toward the normal cells and emerges as a current source in the extracellular space at the normal side of the border. Then it flows back into the ischemic tissue where it enters as a current sink in the intracellular space (14). The extracellular current sources and sinks appear in the local DC amplified electrogram as positive and negative displacement of the TQ segment, respectively (8). In systole, the injury current flows in the reverse direction, giving rise to local ST segment elevation and depression, respectively (14). In the AC amplified ECG, displacement of the TQ segment cannot be differentiated from the ST segment displacement.

**Correlation between ST segment shift and QRS configuration.** The uncertainty about the mechanism of the ST segment shift does not weaken the main conclusion of our study that the magnitude of the ST segment shift correlated with the decrease of the S wave amplitude, implying that the S wave was swept upward by the shift of the ST segment. Our findings suggest that in cases in which the QRS duration remains unchanged, the decrease in the S wave amplitude can be caused by the passive shift of the ST segment. This interpretation does not rule out the possibility that other factors played a role in the alteration of the QRS configuration. Wagner et al. (4) found “no evident relation between the extent of ST segment deviation and the extent of waveform amplitude changes.” The discrepancy between our findings and those of Wagner et al. (4) could have resulted from differences between the effects of PTCA on the QRS configuration in the two studies. For instance, we found no decreases in the R amplitude, whereas in several cases Wagner et al. found the R wave amplitude to be decreased (Fig. 3 and 4 in reference 4).

In patients with RCA PTCA, the QRS pattern in the leads with ST segment elevation was more variable than the QRS pattern in leads with ST segment elevation in patients with LAD PTCA (RS pattern). Although the correlation between the ST segment shift and the total QRS amplitude change was weak, the manifested trend suggests that the mechanism of QRS changes during RCA PTCA does not differ from that of QRS changes during LAD PTCA. This conclusion is supported by the observation that in patients with RCA occlusion, the relation between the ST segment elevation and the decrease in
the S wave amplitude in the leads with an RS or qRS configuration was similar to that in the leads with a similar configuration in patients with LAD occlusion.

Similar to our results, during LAD PTCA Dellborg et al. (15) found a significant correlation between the maximal ST segment–vector magnitude (expressed in mV) and the QRS–vector difference, described as the total change “within the QRS complex” in three orthogonal leads (expressed in μVs). During RCA PTCA, however, the correlation was not significant. The method of recording used by these investigators does not reveal the details of the change in the QRS configuration.

The initial QRS deflection was altered by balloon occlusion less frequently than the terminal portion of the QRS complex. We did not observe any changes in the direction of the initial QRS deflection, but the amplitude of the R wave increased in two of 13 patients during LAD occlusion, and the Q wave amplitude decreased in four patients with QS, Qr or QRS patterns during RCA occlusion (Fig. 5). In the study of Feldman et al. (16), the R wave amplitude tended to decrease during the last 10 s of balloon occlusion.

Intraventricular conduction disturbances. The superposition of the ECG before and during occlusion was helpful in showing that the QRS duration was unchanged in 25 of 27 patients. This is in agreement with the findings of Spekhorst et al. (17), who constructed QRS integral maps during PTCA using 62 precordial leads. These investigators found no change in the total QRS duration, but their subtraction maps suggested regional conduction delays during the inscription of the QRS complex. Also, Abboud et al. (18) frequently found zones of reduced amplitude in the high frequency QRS complex during various stages of PTCA, and attributed this to slow conduction induced by ischemia.

We found only two cases in which the apparent slowing of conduction was detectable after the end of the precocclusion QRS complex. We believe that the term “peri-ischemic block,” cited by Wagner et al. (4) and by Dellborg et al. (15), is suitable for such cases.* In both cases of such peri-ischemic block observed by us, the pattern was present only during occlusion and was unequally expressed among the 12 leads. In one of these cases (Fig. 5 and 8), a terminal QRS deflection simulating an incomplete right bundle branch block appeared in the right precordial leads but was not detectable in the left precordial leads. Such absence of detectable deflection in more than one lead was more likely caused by the remoteness of the left precordial electrodes from the site of conduction delay than by the perpendicular orientation of the late deflection to the lead axes. The terminal late QRS deflection in this case was apparently caused by ischemia of the right ventricular myocardium supplied by the branch of the RCA.

In the other case, QRS widening was detectable in all 12 leads but was more pronounced in the indirect limb leads than in the semidirect precordial leads facing the myocardium at some distance from the inferior wall supplied by the occluded RCA (Fig. 9).

In our study, the incidence of peri-ischemic block, manifested by widening of the QRS complex (7.4%), was lower than in the studies by Wagner et al. (4,5). These investigators reported QRS changes suggestive of peri-ischemic block in a three-lead ECG in 11 of 20 patients during PTCA of the LAD and in 2 of 20 patients during PTCA of the RCA and LCx. The description of the QRS change in the cases of Wagner et al. does not make it clear whether the peri-ischemic block was associated with widening of the QRS complex or mere changes in the QRS amplitude or configuration, or both, without increased QRS duration. Pending a broader consensus about the acceptance of the term and its definition, we suggest that the term peri-ischemic block be limited to transient widening of the QRS complex during ischemia and not to changes in the QRS configuration in the absence of QRS widening, because the latter changes are more likely to be caused by the passive shift of the ST segment. The slowing of myocardial conduction during acute ischemia may be responsible for the subtle QRS changes detected by the subtraction maps (17) and the reduced amplitude of high frequency QRS components (18). The detection of such changes in the conventional, nonsignal-averaged ECG, however, may be difficult or impossible.

Corrected QT changes. In our study, acute ischemia produced small and variable changes in the duration of the QTc interval. Lengthening occurred more often than shortening during LAD occlusion, whereas shortening occurred more often than lengthening during RCA occlusion. Similar differences between the sites of balloon occlusion were reported by Cohen et al. (1), who found an average lengthening of 40 ms$^{1/2}$ in the leads with the longest QTc interval during LAD occlusion but no significant change in the QTc interval during RCA occlusion.

During acute myocardial ischemia, the duration of the ventricular action potential in the ischemic zone is known to lengthen slightly at the onset (19,20) and then to shorten (6,8,20). The shortening of the QT interval may be explained by the electrotonic interaction of normal myocardium with ischemic myocardium, in which the action potential duration is shortened. Lengthening of the QT interval may occur if the slowed conduction in the ischemic myocardium delays depolarization and the ensuing repolarization (21).

References


*We were not able to trace the origin of the term “peri-ischemic block.” It is referred to in reference 4, which did not cite a reference, and in reference 15, citing “Hamlin R, et al. Am J Physiol 1968;215:1032,” but we did not find it in this report.


