

Transient Alterations of the QRS Complex and ST Segment During Percutaneous Transluminal Balloon Angioplasty of the Left Anterior Descending Coronary Artery

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Using continuous 3-lead electrocardiographic (ECG) recordings in 19 patients undergoing elective percutaneous transluminal coronary artery angioplasty (PTCA) of the left anterior descending (LAD) artery, this study described the dynamic changes of the ST segment and the R- and S-wave amplitudes that occur during transient myocardial ischemia. The waveforms from lead V₂ were quantified at 10-second intervals during the length of the balloon inflation that produced the greatest extent of ST-segment deviation. The simultaneous changes that occurred in leads aVF and V₅ were also observed, but not quantified. Measurements of R- and S-wave amplitudes were performed during maximal ischemia from both the PR- and the J-ST-segment baselines to determine which of these most nearly maintained its control position during ischemia. The results indicate that the R-wave amplitude is best determined from the PR-segment baseline ($p = 0.0007$), while the S wave is best determined from the J-ST-segment baseline ($p = 0.03$). However, only a portion of the QRS changes observed during PTCA could be accounted for by the baseline shift. There were additional QRS changes during ischemia in 11 of the patients (58%) suggestive of conduction disturbances in 3 endocardial sites: left septal, right septal and left anteroseptal. It is hypothesized that these changes may represent ischemia-induced delay in conduction ("periischemic block") previously thought to occur only with myocardial infarction.

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During the earliest phases of acute coronary occlusion, myocardial ischemia is known to be represented on the standard 12-lead electrocardiogram (ECG) by ST-segment deviation and accentuated T waves. Alterations in the QRS complex have been most classically associated with myocardial necrosis. However, both an increase in the amplitude of the R wave¹⁻⁴ and the development of transient Q waves⁵⁻⁷ have been observed in patients with Prinzmetal's angina and with exercise. The amplitude of the R wave has also been reported to increase with atrial pacing.⁸ Other factors known to influence the appearance of the QRS complex in the absence of necrosis include the degree of patient recumbency, interventricular conduction abnormalities, heart rate, chamber size, chest wall dimensions and lung volume.^{1,4,8,9} One-vessel percutaneous transluminal coronary angioplasty (PTCA) provides a unique clinical setting to examine the effects of transient acute coronary artery occlusion in humans. By observation of 3 continuous ECG leads during elective PTCA of the left anterior descending (LAD) coronary artery, this study describes the dynamic changes of the amplitudes of the R and S waveforms that occur during balloon inflation and examines the relation of these changes to the concomitant ST-segment deviation.

METHODS

Patient population: Nineteen patients undergoing 1-vessel elective PTCA of the LAD at Georgetown University Hospital were selected. Inclusion required: (1) a standard 12-lead ECG before the procedure with no evidence of ST-segment deviation, myocardial infarction, ventricular hypertrophy, fascicular or bundle branch block or paced rhythm; (2) no pharmaceutical interventions during the procedure; (3) control recordings of the 3 orthogonal leads approximating aVF, V₂ and V₅ immediately before the first balloon inflation showing no evidence of ischemia; and (4) evaluable continuous recordings via these leads throughout the PTCA procedure.

Study design: All patients were fitted with precali-brated 3-channel AM Holter recorders before PTCA, with radiolucent leads positioned in anteroposterior

(V₂), left to right (V₅) and inferosuperior (aVF) configurations. Playback was performed on a digitizing Marquette 8000 Holter analysis system interfaced to a DEC PDP 11/34 mainframe.

The onsets of each balloon inflation and deflation had been indicated by an electrical interference signal; thus, the periods of sequential inflations were easily isolated for analysis. The number and duration of inflations varied for each patient. While the 3 leads were examined visually, only V₂ was selected for detailed analysis.¹⁰ All subsequent measurements were performed manually to the nearest 0.5 millimeter. The J point was used as the reference for determination of ST-segment deviation.

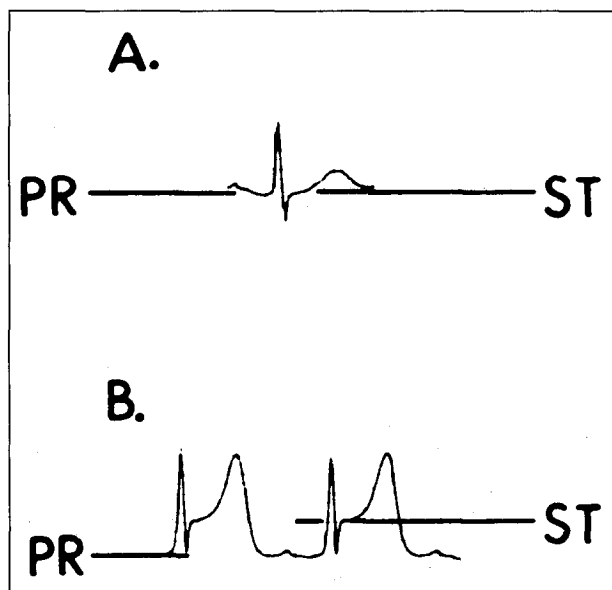


FIGURE 1. A, "control" electrocardiogram. The PR or ST segment can be used interchangeably as the baseline for determination of waveform amplitudes. B, "ischemic" electrocardiogram. The PR-segment baseline versus the shifted J-ST-segment baseline is used for determination of waveform amplitudes.

The time during any inflation that produced the greatest extent of ST-segment deviation was considered the "maximally ischemic period" for an individual patient. The ST-segment deviation was measured at 10-second intervals during this period, and a representative QRS complex ("maximally ischemic QRS complex") from the 10-second interval with the greatest amount of ST-segment deviation was selected. The R- and S-wave amplitudes from this single complex were measured from both the PR segment and from the shifted J-ST segment (Figure 1) to determine the baseline that most nearly maintained its control position during ischemia. Subsequently, the R and S waves at every 10-second interval during the maximally ischemic period were measured using the most appropriate baseline.

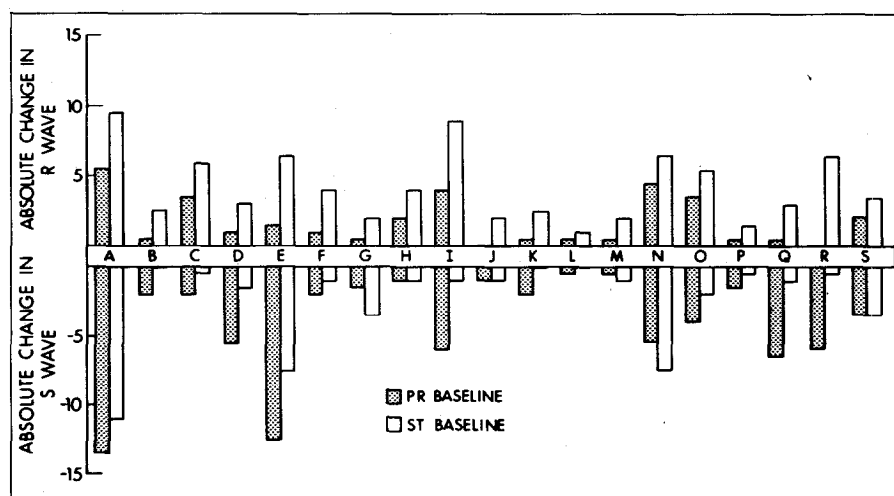
RESULTS

Fourteen men and 5 women, ages 37 to 68 years (mean 56), participated. The extent of LAD obstruction before PTCA ranged from 60 to 90% (mean 72%). The period of maximal ischemia was inflation 1 in 4 patients, inflation 2 in 7 patients and inflation 3 in 8 patients. The mean duration of this period was 78 seconds.

Figure 2 compares the R- and S-wave amplitudes of the maximally ischemic QRS complex measured from both the PR- and ST-segment baselines. The mean absolute amplitude of the R wave from the PR baseline changed only 1.7 mm compared with 4.2 mm when measured from the ST baseline ($p = 0.0007$). Conversely, the mean absolute amplitude of the S wave changed 4.1 mm from the PR baseline, versus 2.3 mm when measured from the ST baseline ($p = 0.03$).

Figure 3 shows the ST-segment deviation at every 10-second interval during the period of maximal ischemia and the corresponding waveforms measured from the baselines as determined previously—the R wave from the PR segment and the S wave from the ST segment. Maximal ST-segment deviation ranged from 0.5 to 6.5 mm. There was no evident relation between the extent of ST-segment deviation and the extent of waveform amplitude changes. It should be noted that pa-

FIGURE 2. Absolute change in R- and S-wave amplitude for each patient from control versus maximal ischemia. Waveforms were measured from the PR-segment baseline (shaded bars) and from the shifted J-ST-segment baseline (open bars) in lead V₂.



tients A, E, G, I, R and S are examples of patients with greatly elevated ST segments, but only A, E and I have dramatic concomitant R- or S-wave changes. In contrast, while patients J, K, L and N are examples of patients with minimal ST-segment deviation, patient N has extensive R- and S-wave changes. Figure 4 further illustrates this labile relation. Of interest is the appearance of the QRS complex during inflation 1 versus inflation 3, with similar amounts of the ST-segment elevation.

There were QRS complex changes during ischemia in 11 patients (58%) that were suggestive of conduction

disturbances in 3 endocardial sites, alone or in combination (Figure 5): left septal, right septal, and left antero-superior. Examination of lead aVF was essential to determine the presence of left anterior (superior) fascicular block in patient I.

DISCUSSION

It is commonly thought that potentially reversible ischemia would have minimal effect on depolarization of ventricular muscle. Thus, no QRS complex changes would occur until the onset of the necrotic process, when the inability to depolarize would cause a shift in

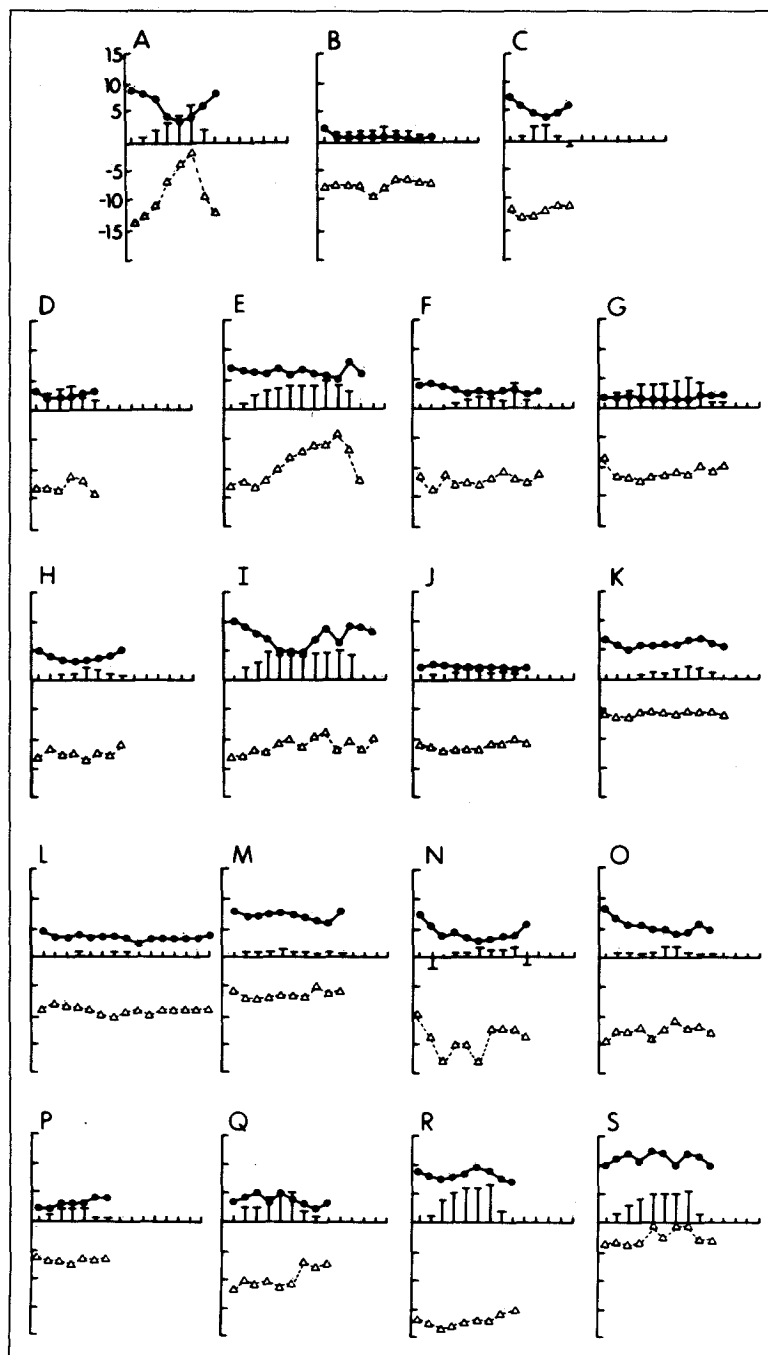


FIGURE 3. Period of maximal ischemia in lead V_2 at every 10-second interval for each patient. All numerical values are expressed in millimeters. The first interval is always the control value and the last 2 intervals always represent the time of deflation and its immediately subsequent interval. ST-segment deviation is represented by a "T" for millimeters of elevation and an inverted "T" for millimeters of depression. The R-wave values are denoted by closed circles and the S waves by open triangles. For example, in patient A, the duration of the period of maximal ischemia from control to the time of deflation is 60 seconds. The ST-segment elevation ranges from 0 to 6 mm, peaking at 50 seconds. The control R wave measures 10 mm, decreases during balloon inflation to 3 mm at 40 seconds, then gradually increases in amplitude. The control S wave is 14 mm, dramatically decreases with inflation to 2 mm at 50 seconds and then returns to its approximate control value with deflation.

summated forces away from the involved area. However, studies in experimental animals have documented significant slowing of intraventricular conduction and transient QRS complex changes during periods of ischemia.¹¹⁻¹³ There are scant reports of such changes in humans, primarily during Prinzmetal's angina.^{5-7,14} Also, alterations in the QRS complex waveforms concurrent with ST-segment elevation are visible on illustrations of several studies describing other aspects of the ischemic

process.¹⁵⁻¹⁷ A recent report of signal-averaged, high-frequency ECGs during PTCA has documented alterations of the QRS complex during balloon occlusion of the LAD.¹⁸

The "current of injury" that occurs immediately following complete coronary artery occlusion¹⁹ is reflected on the ECG by a shift in the ST segment. The pathophysiologic basis for this shift is discussed in detail in the review by Arnsdorf and Louie.²⁰ It is not known whether there is primarily movement of the ST segment toward the injured area or movement of the PR segment away from that area.^{21,22} Previous observations from our institution have noted that, during spontaneously occurring acute anterior epicardial injury, the later aspects of the QRS complex often appeared to be "carried" upward with the ST-segment elevation. This observation led to the present hypothesis that the ST segment, rather than the PR segment, may be a more appropriate baseline to measure such later aspects during an ischemic episode. To test this, the R- and S-wave amplitudes of the maximally ischemic QRS complex were quantified from the PR segment and from the shifted J-ST segment (Figure 1) to determine which baseline most nearly maintained its control position.

Although our study comprised only 19 patients, the results suggest that the R wave amplitude in lead V₂ is associated with the PR segment during anterior myocardial ischemia; conversely, the S wave is more closely associated with the ST segment (Figure 2). A recent study by Glazier et al²³ showed a similar relation between the S-wave amplitude and the ST segment occurring with subendocardial injury produced by either episodes of angina or treadmill exercise testing. Further

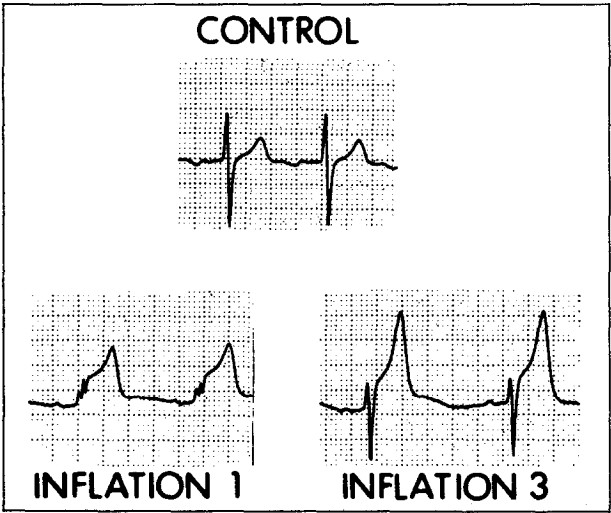


FIGURE 4. Lead V₂ electrocardiograms taken during angioplasty from patient A illustrate the great variation in QRS change from inflation 1 to inflation 3, although the extent of ST-segment elevation is similar. A "perispheric block" was probably present during inflation 1 but absent during inflation 3.

FIGURE 5. Tracings from the "control" and "maximally ischemic" electrocardiograms illustrate the types of conduction delays observed. Leads aVF, V₂ and V₅ were simultaneously acquired. The probable anatomic sites hypothesized to produce such delays are blackened and labeled 1, 2 and 3 in the top example. Isochrones reflecting every 10 seconds of the activation sequence are shown for reference. In the subsequent examples, the region of myocardium affected by each site is shown by the hatched area. Examples are from patients R (none), N (left septal), S (right septal), A (left septal + right septal) and I (left septal + right septal + anterior). See Figure 3 for additional data on these patients.

	aVF		V ₂		V ₅		ANATOMY
	CONTROL	ISCHEMIA	CONTROL	ISCHEMIA	CONTROL	ISCHEMIA	
NONE							
L. SEPTAL							
R. SEPTAL							
L + R. SEPTAL							
L + R. SEPTAL + ANTERIOR							

studies that carefully quantify the changes of each of the sequential portions of the QRS complex during the development and regression of both transmural and subendocardial injury currents will be required to fully understand these relations.

However, the present study shows that only a portion of the QRS changes occurring with ischemia can be accounted for by the baseline shift. Even when measured from the most appropriate baseline, extreme changes in the R and S waveforms are often present (Figures 3 and 4). Further, there is no evident relation between these changes and the extent of concomitant ST-segment deviation. It is likely that these residual changes may be caused by 1 of 2 types of ischemia-induced intraventricular conduction disturbances previously thought to occur only during developing necrosis. In the first type, the intraventricular Purkinje system is affected directly, resulting in complete right or left bundle branch block or left fascicular block. Because pathophysiologic studies have shown that the Purkinje cells are less susceptible to ischemia than are myocardial cells,^{24,25} it is unlikely that this type of conduction disturbance would occur with transient ischemia.

In the second type, the intramyocardial conduction from the Purkinje network is detoured around an area of subendocardial necrosis to the intact mid- and subepicardial regions. This has been termed "periinfarction block." The subendocardial myocardium is the most distal aspect of the perfusion bed of the coronary circulation and would therefore be most affected by the complete proximal arterial occlusion produced by PTCA. Experimental studies have demonstrated that reversible ischemia is capable of slowing intramyocardial conduction sufficiently to alter the QRS complex.¹¹⁻¹³ Therefore, the changes in R- and S-wave appearance that are not caused by the baseline shift are hypothesized in this study to be the result of "periischemic block."

The septal perforating branches of the LAD supply the proximal aspects of both the left and right bundles and the corresponding myocardium into which their fascicles insert. Conduction delay due to transient occlusion of the LAD, as during PTCA, would most likely occur in 1 or more of these 3 subendocardial regions—the left septal, the right septal or the left anterosuperior wall near the base of the superior papillary muscle (Figure 5). Future studies using standard and more sophisticated ECG methods will be needed to validate the relations between these types of subendocardial conduction delay and the documented sites of arterial occlusion.

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