Is Anterior ST Depression With Acute Transmural Inferior Infarction Due to Posterior Infarction?
A Vectorcardiographic and Scintigraphic Study

JHULAN MUKHARJI, MD, SUZANNE MURRAY, RN, SAMUEL E. LEWIS, MD, FACC, CHARLES H. CROFT, MD, JAMES R. CORBETT, MD, FACC, JAMES T. WILLERSON, MD, FACC, ROBERT E. RUDE, MD, FACC
Dallas, Texas

The hypothesis that anterior ST segment depression represents concomitant posterior infarction was tested in 49 patients admitted with a first transmural inferior myocardial infarction. Anterior ST depression was defined as 0.1 mV or more ST depression in leads V₁, V₂, or V₃ on an electrocardiogram recorded within 18 hours of infarction. Serial vectorcardiograms and technetium pyrophosphate scans were obtained. Eighty percent of the patients (39 of 49) had anterior ST depression. Of these 39 patients, 34% fulfilled vectorcardiographic criteria for posterior infarction, and 60% had pyrophosphate scanning evidence of posterior infarction. Early anterior ST depression was neither highly sensitive (84%) nor specific (20%) for the detection of posterior infarction as defined by pyrophosphate imaging. Of patients with persistent anterior ST depression (> 72 hours), 87% had posterior infarction detected by pyrophosphate scan. In patients with inferior myocardial infarction, vectorcardiographic evidence of posterior infarction correlated poorly with pyrophosphate imaging data. Right ventricular infarction was present on pyrophosphate imaging in 40% of patients with pyrophosphate changes of posterior infarction but without vectorcardiographic evidence of posterior infarction.

It is concluded that: 1) the majority of patients with acute inferior myocardial infarction have anterior ST segment depression; 2) early anterior ST segment depression in such patients is not a specific marker for posterior infarction; and 3) standard vectorcardiographic criteria for transmural posterior infarction may be inaccurate in patients with concomitant transmural inferior myocardial infarction or right ventricular infarction, or both.

The significance of ST segment depression in the anterior precordial leads in patients with acute transmural inferior myocardial infarction is controversial. It has been suggested that anterior ST depression with transmural inferior myocardial infarction is a marker of more extensive left ventricular dysfunction (1,2), higher rate of in-hospital complications (2), anterior ischemia (2,3), posterior infarction (1,4) and "reciprocal" electrical phenomena (5). In an earlier study (5), using the radionuclide ventriculogram in the acute phase of transmural inferior myocardial infarction, we found no differences in global or regional left ventricular function between patients with and without anterior ST depression. However, because the assessment of posterior wall motion by radionuclide ventriculography is sometimes difficult and wall motion abnormalities, in themselves, do not distinguish between infarction and ischemia, the present investigation was undertaken. Technetium pyrophosphate scintigraphy and vectorcardiography were used to test the hypothesis that anterior ST segment depression in patients with acute transmural inferior myocardial infarction is a marker for concomitant transmural posterior infarction.

Methods

Patient selection. Forty-nine consenting patients with a first acute transmural myocardial infarction were evaluated...
prospectively between August 1978 and July 1982 in the coronary care unit of Parkland Memorial Hospital, Dallas, Texas. The patients constitute part of the Multicenter Investigation of the Limitation of Infarct Size, which seeks to determine whether acute intervention with propranolol or hyaluronidase can limit the extent of myocardial necrosis in patients treated within 18 hours of the onset of acute myocardial infarction. It is a randomized, placebo-controlled, blinded study sponsored by the National Institutes of Health. All consenting patients with more than 30 minutes of ischemic chest pain and electrocardiographic changes of an acute coronary event (new Q waves, 1 mm or more ST elevation or depression in two or more limb or precordial leads or left bundle branch block) are candidates for inclusion. Patient exclusion criteria are an age greater than 75 years, onset of chest pain more than 18 hours before randomization, cardiogenic shock, pregnancy, a functioning implanted pacemaker, history of a recent (≤2 weeks) myocardial infarction or major surgery, coexistent terminal disease or major organ system failure, participation in another protocol and refusal or inability to give informed consent.

Baseline measurements are made before intervention drug therapy, and this ancillary study is based on these prerandomization measurements, which would not have been influenced by subsequent therapy. Conclusive evidence of transmural inferior myocardial infarction was determined on the basis of elevation of creatine kinase and the creatine kinase B isoenzyme (6,7) and the development of electrocardiographic evidence of new Q waves at least 40 ms wide and 0.2 mV deep in two or more inferior leads (II, III or aVF). Patients with historical or electrocardiographic evidence of previous infarction and bundle branch block were excluded from this ancillary study.

Electrocardiographic analysis and patient groups. The earliest electrocardiogram obtained from these patients (usually the emergency room tracing) was evaluated for the presence of anterior ST segment depression. This was defined as ST depression 0.1 mV or more measured 80 ms after the J point in any one or more of leads V₁, V₂ or V₃. The 49 patients were classified into two groups. Group I consisted of 39 patients with such anterior ST depression and Group II consisted of 10 patients without anterior ST depression. The initial electrocardiogram was also evaluated for evidence of transmural posterior infarction, defined by a prominent R wave (width 0.4 second or more) in lead V₁ or V₂ with an R/S ratio of 1 or more.

Vectorcardiographic analysis. Frank XYZ lead electrocardiograms and vectorcardiograms were obtained within 18 hours of the onset of symptoms (mean 7.3 hours, range 2.9 to 15.3) using an Instruments for Cardiac Research (ICR) 2001 data acquisition cart. Simultaneous 12 lead and XYZ lead electrocardiograms and vectorcardiograms were repeated 3 and 10 days after admission. The ICR instrument performs a direct analog to digital recording that is stored on a cartridge magnetic tape, and “hard copy” printouts are obtained on an XY recorder.

Figure 1. Representative transverse plane vectorcardiographic loops. Left, Normal tracing. Right, Transmural posterior myocardial infarction.
and d) total duration of the anterior QRS forces of 42 ms or more. The combined presence of any three of these criteria was considered to be vectorcardiographic evidence of transmural posterior myocardial infarction (Fig. 1).

**Pyrophosphate scintigraphy.** Technetium-99m pyrophosphate myocardial scintigraphy was performed according to a standard technique previously described from our laboratory (9,10). Studies were obtained on the third and fifth days in the anterior, modified left anterior oblique, 70° left anterior oblique and left lateral projections. The scintigrams were evaluated by two experienced observers who had no knowledge of the clinical or electrocardiographic findings. The lateral view was examined carefully for evidence of abnormal posterior wall pyrophosphate uptake. Scintigrams were graded from 1+ through 4+; 2+ or greater activity was considered abnormal (9,10). For purposes of this study, abnormal pyrophosphate uptake in the posterior wall was taken as definite evidence of posterior infarction (Fig. 2).

**Statistical analysis.** Comparison between proportions in two groups possessing a certain attribute was made using Fisher’s exact test.

**Results**

**Clinical features.** The study group consisted of 32 men and 17 women. The mean age of the group was 52.5 years and no patient had a history or electrocardiographic evidence of previous myocardial infarction. Twenty-three percent of the patients with and 26% of those without anterior ST depression were in Killip functional class II or III. No patient was classified in Killip class IV, and there were no deaths in the study group. The mean left ventricular ejection frac-

---

**Figure 2.** Technetium pyrophosphate scintigrams. A, Right ventricular and inferior wall involvement. Projections are top left, anterior; top right, 35° left anterior oblique; lower left, 70° left anterior oblique and lower right, lateral. Normal pyrophosphate uptake in the sternum and ribs is seen. Normally there is no pyrophosphate uptake by the heart, and the figure depicts abnormal uptake over the right ventricle (best seen in the modified left anterior oblique view) and inferior wall uptake without posterior involvement (left lateral view). B, Inferior and extensive posterior involvement best seen in the left lateral (L. LAT) view. ANT = anterior; LAO = left anterior oblique. A “positive” image makes areas of pyrophosphate uptake to show as dark areas, in contrast to the white areas in A.
tion determined by radionuclide angiography was 0.57 (±0.13) and 0.66 (±0.16) in patients with and without anterior ST depression, respectively. These differences were not statistically significant.

**Electrocardiographic and vectorcardiographic findings.** ST depression was present in lead V1, V2 or V3 in 39 patients (80%). It was present in lead V1 only in 13 of the 39, in leads V1 and V2 in 12 and in leads V1, V2 and V3 in 14. Twelve patients had electrocardiographic evidence of a transmural posterior myocardial infarction. Ten (83%) of these 12 had vectorcardiographic evidence of a posterior infarction, whereas 4 (11%) of 37 without electrocardiographic evidence of posterior infarction had vectorcardiographic evidence of posterior myocardial infarction (Fig. 3). This relation supports previous data on the derivation of vectorcardiographic criteria for posterior infarction (8).

**Relation between anterior ST depression and posterior infarction.** Sixty percent of patients with and 44% of patients without anterior ST depression had a posterior infarct as assessed by pyrophosphate imaging (Fig. 4). The difference was not significant (p = 0.46). The sensitivity, specificity and predictive value of anterior ST depression in identifying posterior myocardial infarction was 84, 26 and 60%, respectively. One patient with anterior ST depression had an anterior infarct assessed by pyrophosphate imaging.

**Relation between vectorcardiographic and scintigraphic evidence for posterior infarction.** Twenty-four patients had pyrophosphate scan evidence of posterior infarction, but only nine (37%) had vectorcardiographic evidence of posterior myocardial infarction. Nineteen patients did not have posterior infarction detected by pyrophosphate scan, and five (26%) of them had vectorcardiographic evidence of posterior infarction (Fig. 5). The difference was not significant (p = 0.50). The sensitivity, specificity and predictive value of the vectorcardiogram in identifying posterior myocardial infarction was 37, 73 and 64%, respectively.

**Relation between right ventricular and posterior infarction.** Ten patients had concomitant right ventricular infarction defined by pyrophosphate scintigraphy. Six of them were in the subgroup of 15 patients with posterior infarction demonstrated by pyrophosphate scan but with no vectorcardiographic evidence of posterior infarction (Fig. 6). Of the patients in this subgroup, 40% had right ventricular infarction. Small numbers precluded meaningful statistical analysis of the incidence of right ventricular infarction in patients grouped according to pyrophosphate imaging and vectorcardiographic results. However, 80% of all right ventricular infarcts occurred in patients with pyrophosphate imaging evidence of posterior myocardial infarction, and 20% occurred in those without such evidence.

**Time course of anterior ST segment depression.** Thirty-nine patients had anterior ST depression on the initial
electrocardiogram. ST depression resolved in 21 patients within 24 hours. Eight patients had persistent ST depression up to 72 hours. Seven (87%) of these eight patients had posterior myocardial infarction detected by pyrophosphate imaging. Eighteen (49%) of the 37 patients without anterior ST depression at 72 hours had posterior myocardial infarction detected by pyrophosphate imaging. Of the eight patients with persistent anterior ST depression, only two had vectorcardiographic evidence of posterior infarction.

**Assessment of the ST segment vector on the Z lead.** Z lead electrocardiograms obtained during the vectorcardiographic recordings were analyzed to assess the direction of the ST segment vector. The ST segment vector was displaced posteriorly in 19 patients, 14 (77.7%) of whom had posterior infarction determined by pyrophosphate imaging. Of the eight patients with persistent anterior ST depression, only two had vectorcardiographic evidence of posterior infarction.

**Discussion**

Much has been written (1-5) about the implications of anterior ST segment depression (reciprocal changes) in patients with acute transmural inferior myocardial infarction. The purpose of this study was to address the specific issue of posterior infarction and its correlation with such reciprocal ST segment changes.

**Choice of a "gold standard."** We chose to use pyrophosphate scintigraphy as the major determinant of the presence or absence of posterior infarction for several reasons. Among the various choices available in the noninvasive armamentarium, we could use either radionuclide ventriculography, electrocardiography (vectorcardiography) or pyrophosphate imaging. Radionuclide ventriculography is valuable in the assessment of global and segmental left ventricular function, but the assessment of the posterior wall is difficult because of limited motion normally seen in this segment. Additionally, wall motion abnormalities can result from old infarction, ischemia or other myocardial diseases and, therefore, are not specific markers for acute myocardial infarction. Traditional QRS criteria developed for the diagnosis of posterior infarction by vectorcardiography stem from studies involving small numbers of patients without the benefit of acute anatomic confirmation (8). In contrast, technetium pyrophosphate scintigraphy has been determined to be a highly sensitive and specific means of identifying acute myocardial infarction in studies using creatine kinase isoenzyme correlations (11). There is also excellent correlation between the area of abnormal pyrophosphate uptake and tissue necrosis (postmortem) in both animal and human models (12,13). Hence, an infarct avid technique such as pyrophosphate scintigraphy probably affords the most direct visualization and localization of an acute infarction in the intact subject. Using this localization and a knowledge of the spatial relations of the heart to the various bony landmarks within the thorax, we were able to identify the presence of posterior wall involvement by careful assessment of the pyrophosphate image in the lateral projection.

**Significance of reciprocal anterior ST depression.** The most striking feature of the phenomenon of reciprocal ST changes is that it is very transient. Of the 39 instances of anterior ST depression on the emergency room electrocardiogram, 21 resolved within 18 hours. When comparing the incidence of posterior myocardial infarction in patients grouped according to the findings on the initial electrocardiogram, we found no correlation with the presence or absence of anterior ST depression. However, eight patients continued to have persistent reciprocal ST depression for 72 hours, and seven (87%) of these eight patients did have posterior infarcts. In comparison, 49% of the patients without anterior ST depression at 72 hours had evidence of posterior infarction. Thus, persistent ST depression in leads V1, V2 or V3 in the presence of an acute transmural inferior infarction appears to be a fairly specific indicator of concomitant posterior involvement, although it is not very sensitive. It is possible, however, that ST changes evaluated over a 72 hour period could have been influenced by drug intervention in this series of patients. Therefore, we advocate caution in interpreting this portion of our results regarding the significance of persistent ST depression. Acute anterior ST depression, on the other hand, is usually transient and commonly accompanies transmural inferior infarction, without necessarily connoting posterior wall involvement.
Vectorcardiographic identification of posterior infarction. Standard vectorcardiographic criteria for posterior infarction are determined on the basis of changes in the initial QRS loop and correlate well with R wave abnormalities in right precordial leads on the 12 lead electrocardiogram, as we were able to confirm in this study. Surprisingly, we found that the vectorcardiogram was neither sensitive nor specific in identifying posterior infarction. The relative insensitivity of vectorcardiographic identification of posterior myocardial infarction agrees with some previous studies. Howard et al. (14), using electrocardiographic criteria similar to ours, reported a sensitivity of 55% in their series. However, their study differed from ours in that they did not study patients with acute infarction.

Wolff and Gandhi (15) addressed the issue of posterior infarction and contended that changes in the distal rather than the initial segment of the transverse QRS loop are critical in identifying posterior necrosis. However, they did not offer any standard quantitative criteria, and most of their patients had more than three separate infarctions. In our study, it is possible that the discrepancy between vectorcardiographic and pyrophosphate scan determination of posterior myocardial infarction was, at least in part, due to the fact that the vectorcardiogram identifies transmural infarcts, whereas scintigraphy is a more sensitive technique, identifying any necrosis constituting more than 3 g of myocardium, without distinguishing transmural from transmural infarction (12). Another possible cause for lack of agreement between the two techniques is the individual variation in the position of the heart and, hence, variable alignment of the posterior wall in relation to an assumed horizontal plane. Because both vectorcardiographic and electrocardiographic criteria for posterior myocardial infarction are founded on such an assumption, results could conceivably vary with posture, thoracic configuration and the orientation of the heart in the chest. To address these considerations, the ST segment vector was evaluated. Assessment of the ST segment vector revealed that there was close agreement between posterior ST displacement and posterior infarction (78%). Also, six of the eight patients with persistent anterior ST depression had posterior ST displacement. Thus, posterior ST displacement in the transverse vector plane is a reasonably specific though not sensitive marker for posterior involvement in patients with concomitant transmural inferior infarction.

Right ventricular infarction—a confounding element? During the course of the study, it became evident that a substantial number of patients had also sustained right ventricular damage defined by pyrophosphate scintigraphy (16,17). Eighty percent (8 of 10) of the right ventricular infarctions occurred in accompaniment with posterior infarction, and 60% occurred in the subgroup with false negative vectorcardiograms. The significance of this finding remains unclear, but it is conceivable that right ventricular infarction, causing loss of anterior forces, alters the QRS loop in a manner which precludes attainment of the quantitative criteria suggested by Hoffman et al. (8). Certainly, exclusion of patients with right ventricular infarction would have improved agreement between our pyrophosphate imaging and vectorcardiographic findings.

Conclusion. In the acute phase of transmural inferior infarction, anterior ST depression is a frequent and usually transient phenomenon, which does not necessarily signify concomitant transmural posterior infarction. However, anterior ST depression, which persists for up to 72 hours, may be a specific though insensitive marker for posterior myocardial infarction. Standard QRS loop vectorcardiographic criteria are relatively inaccurate in identifying posterior involvement in patients with acute transmural inferior myocardial infarction. This could be the result of concomitant right ventricular infarction, causing alteration in the QRS forces. It is also likely that nontransmural posterior infarcts are not reliably identified by vectorcardiographic criteria. Posterior displacement of the ST segment vector in the transverse plane is probably a better vectorcardiographic indicator of posterior infarction than are standard criteria derived from changes in the initial segment of the QRS loop. Right ventricular infarction occurs in 20% of patients with transmural inferior infarction and usually occurs in association with posterior wall involvement; it may interfere with diagnosis of acute posterior myocardial infarction by standard vectorcardiographic criteria.

We gratefully acknowledge C. Gunnar Blomqvist, MD for his invaluable help in reviewing this manuscript, Paulette Newingham for preparing the manuscript and the many nurses, medical house officers and cardiology fellows in the coronary care unit at Parkland Memorial Hospital, Dallas, Texas.

References


