Transient QRS Changes Simulating 
Acute Myocardial Infarction

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SUMMARY. The purpose of this study was to determine the characteristics and incidence of abrupt occurrence of abnormal initial QRS forces that cannot be explained by acute myocardial infarction or left or right ventricular overload. Computerized data from 3175 patients with suspected acute infarction were reviewed to identify those in whom the ECGs revealed QRS complexes considered to be diagnostic (Q wave or markedly diminished R wave) in the presence of persistently normal profiles of both creatine kinase and lactate dehydrogenase isoenzymes. Lead misplacement had been minimized by obtaining multispace tracings and vectorcardiograms.

Eight patients (0.25%) were identified. The abnormal forces were confined to leads V_1, V_2 in six, V_4a, V_5 in one, and involved all precordial leads in the last. These QRS changes resolved completely within 6 days in all eight patients, which suggests that they did not have an acute infarction. This theory was supported by postmortem examination in one patient.

An extremely low incidence (0.25%) has been documented for a syndrome characterized by transient loss of initial anterior forces with persistently normal isoenzyme profiles. Although no etiology could be determined, a transient conduction block of the septal fascicle of the left bundle could have been the cause in seven of the eight patients.

THE ACUTE DEVELOPMENT of a marked abnormality of the initial portion of the QRS complex on the surface ECG is considered one of the most reliable indicators of acute myocardial infarction (MI). Epidemiologic and experimental evidence, however, has revealed that abnormal Q or R waves may be caused by right or left ventricular overload, intraventricular conduction blocks, pulmonary disease, and intracranial lesions. In such conditions, the abnormal initial forces are either permanent or transient.

In this study, the computerized medical records of consecutive patients admitted to a coronary care unit after a possible acute MI were analyzed retrospectively. All patients in whom the diagnosis of infarction was indicated by the development of abnormal initial forces but was not corroborated by abnormalities of serum isoenzymes were identified. Serial ECGs evaluated to determine the location and persistence of the QRS abnormalities. The clinical context in which these changes occurred was considered and a review of the literature was undertaken to determine the etiology of this phenomenon.

Methods

During a 6-year period, 3175 patients in the Duke Medical Center coronary care unit were evaluated for a suspected acute MI, and their data entered into the Duke University Medical Center's computerized data bank. A search of their records was conducted to identify patients with acutely abnormal initial forces on ECG but with persistently normal serum creatine kinase (CK) and lactate dehydrogenase (LDH) isoenzymes. Abnormal initial forces were defined as a Q wave $\geq 0.03$ second or an initial R wave $\leq 1$ mm in amplitude and $\leq 0.01$ second in duration in two or more leads on a standard 12-lead surface ECG. Patients with prior deformity of the QRS caused by right ventricular hypertrophy, left ventricular hypertrophy, bundle branch block (QRS $\geq 0.12$ second), or previous MI indicated on the same leads as the transient abnormalities of the acute situation were excluded from the study. Also excluded were patients with acute pulmonary embolism because the resultant cor pulmonale is a commonly recognized cause of abnormal initial forces.

The possibility that the abnormal initial forces could have been produced by variations in the placement of the ECG leads had been minimized by recording all six precordial leads one intercostal space above and below the standard locations and by obtaining Frank lead vectorcardiograms (VCGs) when the patients were admitted to the coronary care unit. The VCGs were recorded using a Hewlett-Packard 1507A vectorcardiograph or an Instruments for Cardiac Research VCG1B instrument, with the patient in the supine position and the electrodes in the fourth intercostal space. The criteria of Starr et al., with documented 95% sensitivity and 99% specificity, were used for corroboration of anterior MI. Patients with previously deformed initial QRS forces were excluded.
Both CK and LDH isoenzyme profiles were obtained to facilitate the diagnosis of acute MI. Blood samples were obtained upon admission to the coronary care unit, every 8 hours for 24 hours, and once daily during the next 2 days.

An autopsy was performed on one patient. The coronary vessels were studied by postmortem angiography and serial macroscopic and microscopic sections were taken.

Results

The computer search revealed eight patients (0.25%) who met the criteria for the study. Table 1 is a list of pertinent clinical data for these patients. The history of MI was elicited in three patients and ECG evidence of an inferior location was present in two of these. All patients were admitted to the coronary care unit, three with complaints of chest pain and with ECG changes and the remainder on the basis of ECG changes alone. Five patients had undergone an invasive diagnostic or therapeutic procedure within 2 days before transfer to the coronary care unit.

The QRS axis was normal in seven patients. Left anterior fascicular block with an axis of $-30^\circ$ was present in patient 3 both before and after the acute QRS change.

Five of the eight patients developed new Q waves and the other three met the stated criteria for R-wave reduction (fig. 1). Table 2 is a list of the ECG and VCG measurements of the eight patients. In six, the changes were localized to the anteroseptal precordial leads (V1,2). In patient 6 the ECG revealed Q waves across the entire precordium, and in patient 8 the anteropical precordial leads (V3,4) only were involved. The abnormal initial forces resolved within 2–6 days in all eight patients.

VCGs had been obtained in five of the eight patients
while changes were present on the ECG tracings. No initial anterior forces were present in four. In patient 8, the VCG showed abnormal forces consistent with an apical infarction and supportive of the findings of the ECG.

Precordial ECG recordings were obtained in the third, fourth and fifth intercostal spaces in four patients. In each patient, the ECGs continued to show the Q or minimal R wave regardless of the placement of the lead. Seven patients had had either a VCG or a three-interspace ECG. Patient 6 had neither of these controls, but his Q waves were unequivocal on 2 successive days.

The only patient examined at autopsy (no. 5) had extremely poor nutritional status during the year preceding death because of esophageal dysfunction. Postmortem findings 10 days after the documented QRS change were consistent with a nutritional cardiomyopathy. Moderate cardiomyopathy was noted on gross inspection. No coronary arterial atherosclerotic lesions were identified by either angiography or inspection. Microscopically, there was marked interstitial edema with chronic atrophic changes of the muscle fibrils. No myocytolysis or other evidence of acute MI was present.

### Discussion

Abnormal initial QRS forces have been described in both experimentally produced and clinically occurring acute MI. Several studies, however, have documented the development of Q waves without confirmation of myocardial necrosis. Altered position of the heart and rotation around its electrical axis form the Q waves that occur with emphysema. Infiltrative processes such as amyloidosis and scleroderma replace the myocardium with fibrous tissue. Anatomic variation such as left or right ventricular hypertrophy and conduction disturbances such as left anterior fascicular block and complete left bundle branch block cause abnormal septal activation. Metabolic and electrolyte disturbances, intracranial hemorrhage, and reversible coronary insufficiency may all result in a change in the QRS complex. With each of these conditions the Q wave may be transient.

By design, this study excluded the most obvious causes of abnormal initial forces, ventricular hypertrophy and common conduction abnormalities. Efforts had been made to eliminate the problem of faulty electrode placement by including VCGs and multispace leads. We also excluded patients with prior Mls indicated in the leads in which acute changes were recorded.

The Q wave of a documented acute infarct has been noted to disappear in 6.7% of cases, usually within the first 2 years after the event. However, Q waves have rarely been noted to resolve within 1 week. The resolution within 2–6 days in all of our eight patients, together with the lack of isoenzyme changes, makes the diagnosis of acute MI extremely unlikely.

Basic and clinical investigators have observed the phenomenon of transient Q waves in the absence of infarction and there are many theories as to its etiology. Bayley and Ladue noted Q-wave formation in dogs after ligating a coronary artery; shortly after release, the Q waves disappeared. In other canine experiments, Gross et al. ligated the coronary arteries and caused formation of a Q wave within 1–2 minutes; it disappeared 3–5 minutes after release of the ligatures. Histologically, no infarct was apparent.

In clinical observations, Haiat et al. noted Q waves postoperatively in a patient with mildly elevated enzymes. Subsequently, Haiat and Chiche re-
ported “transient abnormal Q waves” in 15 patients with angina and noted that the majority of ECG changes were in the anterior precordial leads. This study, however, included patients with enzyme changes. Duren and Becker\textsuperscript{24} observed ECG patterns simulating anterior infarction in two patients with intracranial lesions; enzymes were normal and autopsies revealed no necrosis.

Table 3 presents the possible etiologies of the transient Q wave. The abnormality that appears on the surface ECG might result from changes in either the myocardial or the Purkinje cell. In the former, the defect could be seen histologically as myocytolysis, whereas in the latter no change might be evident.

Myocytolysis is the histologic change in the border zone surrounding areas of myocardial necrosis that was first described by Baroldi.\textsuperscript{22} The lesion is one of myofibrillar degeneration with initially undamaged nuclei and mitochondria. The cells die in a hypercontracted state and no exudative response is elicited; the healing is by scar formation. It is this histologic change in the absence of typical necrosis that Duren and Becker\textsuperscript{24} observed in their patients.

Histologic evidence of myocytolysis has been observed after sympathetic stimulation. Kolin and Dwasnicka\textsuperscript{7} infused norepinephrine into the inferior vena cava of dogs and recorded ECGs with depressed precordial R waves. At autopsy, “hydropic degeneration” and small foci of necrosis were observed in the anterior wall of the left ventricle. Cruickshank et al.\textsuperscript{28} found an increase in the urinary catecholamines of patients with subarachnoid hemorrhage and ECG changes. Adrenal stimulation and the resultant circulating corticosteroids may also cause cellular electrolyte aberrations.\textsuperscript{8} Ionic shifts cause potassium to move into the extracellular space and calcium into the cytoplasm, thereby sensitizing the cells to norepinephrine.\textsuperscript{24}

Five of the patients in this study underwent an invasive procedure before the ECG changes; two of these had changes in leads other than $V_{1-3}$, suggesting a more diffuse than localized effect on the heart. The adrenergic response to stress could have produced transient QRS changes through various mechanisms. This possibility cannot be excluded by the absence of histologic changes, such as myocytolysis, in the only patient studied at postmortem.

Recent detailed studies of the anatomy of the conducting pathways describe a quadrifascicular system;\textsuperscript{40} in addition to the right, left anterior, and left posterior bundles as originally proposed by Rosenbaum,\textsuperscript{31} a discrete group of fibers supplying the interventricular septum has been identified. These fibers activate the middle third and apical sectors of the interventricular septum, producing anterior and rightward orientation to the initial forces of the QRS complex.\textsuperscript{32}

Gambetta and Childers\textsuperscript{25} reported two cases of “septal fascicle block” in which transient precordial Q waves were seen in a setting of acute MI and left-axis deviation; the Q waves returned when the conduction abnormalities resolved months after the infarction. These investigators ascribed the abnormality either to the injured septal muscle or to a focal block in the septal fibers. Septal fascicular block is suggested in the six patients in our series whose changes were confined to leads $V_{1-3}$.

Hackel et al.\textsuperscript{34} documented the relative resistance of conduction tissue to infarction by showing that Purkinje fibers remain viable although surrounded by necrotic myocardial tissue. The transitory nature of the conduction defect after MI was ascribed to the effects of either the edema or the ionic changes associated with necrosis of adjacent myocardium. Alternatively, reversible ischemia to the conducting fibers themselves could cause electrical changes.

The marked loss of initial QRS forces that remains unexplained by acute processes such as MI or left or right ventricular overload is an extremely rare phenomenon. It occurred in only 0.25% of the 3175 consecutive patients we studied during a 6-year period. The syndrome is defined by its transient and characteristic involvement of the right precordial leads of the ECG. Aberration of intraventricular conduction via the septal fascicle of the left bundle may cause the ECG abnormality, but a conclusive explanation is not known.

Acknowledgment

The authors acknowledge Carolyn Lympkin for aiding the search of the computerized data bank, Dr. Raymond Ideker for consultation regarding the postmortem material and Virginia Utley for preparation of the manuscript.

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**Table 3. Possible Etiologies of the Transient Abnormal Initial Forces**

<table>
<thead>
<tr>
<th>Type of cell involved</th>
<th>Myocardial</th>
<th>Purkinje</th>
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<td>Histology</td>
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<td></td>
</tr>
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<td>1 Myocytolysis</td>
<td>1 Fibrosis of fascicle</td>
<td></td>
</tr>
<tr>
<td>2 Normal</td>
<td>2 Normal</td>
<td></td>
</tr>
<tr>
<td>Etiology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Ischemia</td>
<td>1 Ischemia</td>
<td></td>
</tr>
<tr>
<td>2 Catecholamines</td>
<td>2 Ionic environment</td>
<td></td>
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<tr>
<td>3 Other</td>
<td>3 Other</td>
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Circulation. 1980;62:975-979
doi: 10.1161/01.CIR.62.5.975

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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