Transient Abnormal Q Waves in the Course of Ischemic Heart Disease*

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The significance of transient abnormal Q waves (TAQW) observed during acute coronary insufficiency was studied in 15 patients with atherosclerotic heart disease. TAQW occurred during anginal chest pain (10 of 15), cardiogenic shock (3 of 15) and paroxysmal dyspnea (2 of 15). The anterior myocardial wall was more frequently involved (13 of 15) than the diaphragmatic wall (2 of 15). Serum enzyme levels were normal or slightly increased in 11 of 15. TAQW disappeared in four hours to nine days (mean three days). Three patients died and an autopsy was performed on two of them. It is concluded that cases with TAQW include: (1) acute episodes of myocardial ischemia, without myocardial infarction (MI) (8 of 15); (2) MI with early electrical recovery due to predominant ischemia (4 of 15); and (3) exteriorization of previous MI patterns (3 of 15). Abnormal Q waves which disappear in the MI course, reappear transiently during an acute ischemic episode. It is emphasized that electrical inertia is not synonymous with irreversible myocardial damage.

Abnormal Q waves have generally been considered an indication of permanent myocardial damage. However, this concept has been repeatedly challenged by both clinical and experimental data. Isolated reports described transient abnormal Q waves without myocardial infarction during acute coronary insufficiency, cerebral hemorrhage, bronchial asthma, cardiac surgery, selective coronary arteriography, uremia and hyperpotassemia, phosphorus poisoning, hepatic abscess, tetanus, shock and severe metabolic stress, and acute coronary insufficiency. Our purpose is to report and to discuss 15 cases of transient abnormal Q waves in patients with atherosclerotic heart disease.

METHODS

Except for one, the 15 patients were selected from 3,088 patients hospitalized in our department at Hôpital Tenon, from Oct. 1, 1970 through Jan. 1, 1972. A diagnosis of ischemic heart disease was made in the presence of angina pectoris and/or electrocardiographic evidence of chronic coronary insufficiency, including negative T waves and depression or elevation of the ST segments. In addition to clinical examination, a 12-lead ECG was recorded daily by the same technician. The instrument used was the Sanborn electrocardiograph at 25 mm sec speed. Q waves were termed "abnormal" when their duration exceeded 0.03 sec, or in the presence of a QS pattern. They were termed "transient" when they disappeared within a period arbitrarily fixed at 10 days. Serum enzyme levels (serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, lactic dehydrogenase, hydroxybutyrate dehydrogenase) were evaluated during the three consecutive days following the appearance of abnormal Q waves. Anatomopathologic studies were available in two cases from Dr. C. Seban.

RESULTS

There were ten women and five men, with an average age of 3.2 years (41 to 78 years). Abnormal Q waves were recorded during cardiac pain, ten cases; cardiogenic shock, three cases; and acute dyspnea, two cases. Abnormal Q waves were present in leads 2, 3, and aVF in two cases, from V1 to V4 in seven cases, in V3 and V4 in three cases and from V1 to V5 in one case. Thus, the anterior wall was more frequently involved than the diaphragmatic wall. Abnormal Q waves were transient. They disappeared in an average of three days (four hours to nine days). Serum enzyme levels were normal in eight cases, slightly increased in three cases and intensely increased in four cases. Three patients died. Anatomopathologic studies were available in two cases and failed to show any evidence of acute myocardial infarction, in the anatomic territory corresponding to the location of Q waves on the tracing. Pulmonary arteries were free of any emboli.

COMMENT

In the course of ischemic heart disease, transient appearance (or early disappearance) of abnormal Q waves may be due to the following: malposition of electrodes; shift in the electrical position of the...
heart; occurrence of intraventricular conduction defects; new myocardial infarction, which changes the electrical potentials balance; pulmonary embolism, which can mimic diaphragmatic or anteroseptal wall myocardial infarction. Once having excluded the aforementioned possibilities, cases with transient abnormal Q waves in atherosclerotic patients may be classified, according to our experience, into the three following categories.

First Degree: Acute Myocardial Ischemia

According to both experimental and clinical data, abnormal Q waves have been described during acute myocardial ischemia. Experimental occlusion of a coronary artery may result in early and temporary appearance of abnormal Q waves without myocardial infarction. Similarly, intravenous infusion of high doses of norepinephrine (Noradrenaline) in dogs, was reported to produce transient patterns of anterior wall myocardial infarction. At autopsy, no myocardial infarction was found; however, histochemical examination revealed severe, diffuse, but apparently reversible damage to the myocardial fibers, marked reduction of glycogen, impairment of succinodihydrogenase activity with a disturbance in distribution and dispersion of forma-granules, and hydropic degeneration in the sub-endocardial layers. Clinical data are in agreement with these experiments. Segers et al. observed transient abnormal Q waves in lead V4 during an exercise test in a patient with angina pectoris. Roessler and Dressler reported two cases with transient abnormal Q waves during anginal episodes. Klein et al. reported the occurrence of transitory abnormal Q waves in patients undergoing open heart surgery. Whenever anatomic control was available, it failed to demonstrate any evidence of gross myocardial infarction.

Eight patients of the present study were included in this group. Serum enzyme levels were normal or slightly increased. An anatomicopathologic study was available in one case; acute myocardial ischemia, without myocardial infarction was present (Fig 1). Histologic signs include dedifferentiation of myocardial fibers without an inflammatory process and transformation of myocardial fibers into fibroblasts. The significance of these facts has been extensively reviewed by De Pasquale et al. Ischemic cells are transiently compromised enough not to undergo electrical activity, but nevertheless are not dead (jeopardized but potentially salvageable ischemic myocardium); thus, loss of electromotive force is not synonymous with tissue death. However, one cannot exclude with certainty a disturbance in intraventricular conduction producing a delay in depolarization of certain areas of the myocardium. At a time when preinfarction and coronary insufficiency syndromes are approached in many places in a very aggressive way, it is emphasized that local myocardial disturbances secondary to ischemia can produce electrical manifestations indistinguishable from those following infarction.

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20947/)

**Figure 1.** A—Tracing Oct. 16, 1971 showing patterns of acute anteroseptal myocardial infarction. B—Three days later, abnormal Q waves with ST segment elevation recorded in 2, 3 and aVF. Extension of infarction to diaphragmatic wall is assumed. Right shift of QRS axis (from 15° to 60°) is probably related to loss of electromotive forces of part of left ventricle due to acute ischemia. C—Next day, abnormal Q waves have disappeared in 2, 3 and aVF. Patterns of anteroseptal myocardial infarction persist. At autopsy diaphragmatic wall was free of any infarction.
Second Degree: Recent Acute Myocardial Infarction, with Early Electrical Recovery

The longterm disappearance of abnormal Q waves in the course of a myocardial infarction is now a well established fact. Early disappearance, however, has been far less documented in the literature. Wilson et al. mentioned the disappearance of characteristic QRS changes of myocardial infarction, within a month. Kalbfleisch et al. reported that 6 of 775 patients showed regression of the pathologic Q deflections within one month, "the shortest time being six days." Goldman et al. noted in two cases "significant diminution" in the area of abnormal precordial Q waves occurring "within several days" following myocardial infarction. Haiat et al. reported a case of myocardial infarction after operation in which abnormal Q waves disappeared in two days, and normalization of the tracing occurred within a week. In the present study, four patients were included in this group (Fig 2). All had clinical symptoms of myocardial infarction and an intense increase of serum enzyme levels. No deaths occurred in this group; subsequently, anatomopathologic studies were not available.

Third Degree: Transient Exteriorization of the Patterns of an Old Myocardial Infarction

Old myocardial infarction patterns, which had long ago disappeared, may reappear on the electrocardiogram during an acute ischemic episode. The diagnosis of such cases can be made when serial electrocardiograms show the following sequence of events: myocardial infarction patterns; disappearance of abnormal Q waves and reappearance of R waves in the following months or years; transient reappearance of abnormal Q waves during an acute episode of coronary insufficiency; serum enzyme levels are normal or slightly increased. In the present study, three patients were included in this group (Fig 3). An anatomopathologic study was available in one case. It showed the presence of an old myocardial infarction, without any evidence of new myocardial necrosis. In these cases it is likely that the transient reappearance of Q waves is due to transitory ischemia of the superficial myocardial layer which surrounds the old infarcted area. During ischemia this layer becomes electrically silent; the formerly infarcted area appears then "under" the electrodes, and a QS of QR pattern is recorded.
When the superficial layer has recovered the infarcted area is no more “in contact” with the electrodes, and R waves reappear.\textsuperscript{28} Recently de la Fuente et al\textsuperscript{19} observed intermittent or transient Q waves either spontaneously or during atrial pacing in 14 patients, three of whom had acute myocardial infarction. They stated that transient Q waves may appear as an anamnestic response in remote myocardial infarction. Knowledge of these facts is of importance, since our three patients were referred to us with the wrong diagnosis (recent acute myocardial infarction). It is likely that the report of Rubin et al\textsuperscript{20} mentioning transient Q waves during bouts of tachycardia in a patient who had a healed anterior myocardial infarction, can be, at least partly, explained on this basis.

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FIGURE 3.  A—On July 2, 1970, old anterior wall myocardial infarction with left anterior hemiblock is present. True posterior wall myocardial infarction cannot be ruled out (tall R wave in V1). Because of history of typical myocardial infarction, possibility of right precordial Q waves due to left anterior hemiblock unlikely. B—April 12, 1971, 7 A.M., abnormal Q waves have disappeared in precordial leads. There have been no changes of QRS patterns in peripheral leads. C—April 12, 1971, 10 A.M. during anginal pain abnormal Q wave is now seen in V2. Tall R wave has disappeared in V1; S waves are deeper in left precordial leads. D—April 13, 1971, 24 hours later R wave is again present in V2. Deep S waves persist in left precordial leads. Patterns of old myocardial infarction were transiently exteriorized during acute episode of ischemia.

The Florentine Renaissance

While the humanists were busily immersing themselves in the cult and cultivation of Greek and Latin antiquity, the artists of the Italian Renaissance were blazing out a new vision of the emotional and intellectual world. They were engaged in the quest for the ideal beauty. This ideal and formal perfection was to rise from the congruence between its earthly representation and the ideal prototype that existed in the Platonic realm of Ideas. Ancient estheticians had laid down the fundamental laws of structure which were eternally, immutably valid for all future ages. The word "Renaissance," which we use today to describe the principal components and the universally recognized features of this time, was not really used until the sixteenth century, when the era could be observed retrospectively as past history. The word was first used to define the recent past which still inspired the present; for Vasari, who coined the word "Renaissance," did not belong to the Renaissance, but was a "mannerist" artist and architect (1511-1574). Artists and humanists alike believed that before the "Dark Ages" had shrouded Europe, a Golden Age had lit up the world, and that its art and culture had contained the monopoly of all possible truths.