CLINICAL SIGNIFICANCE OF ABNORMAL Q WAVE DISAPPEARANCE IN ACUTE TRANSMURAL MYOCARDIAL INFARCTION

KINJI ISHIKAWA, M.D., MINORU SHIMIZU, M.D., MAKOTO OHNO, M.D.
MASATAKA MORISHITA, M.D., IWAO OGAWA, M.D., TAKAHIRO HAYASHI, M.D.
YOSHIHIDE SAKAGUCHI, M.D., KEIZO YAMASHITA, M.D., HIRONARI KOKA, M.D.
NORIAKI KAMATA, M.D., AND RYO KATORI, M.D.

The clinical features of acute myocardial infarction patients in whom abnormal Q wave disappeared were analyzed. Of 201 patients, 40 (20%) (Group A) showed disappearance of Q wave in serial electrocardiograms. Regional ejection fraction of the infarcted portion improved significantly (from 24 ± 2 to $34 \pm 4\%$, $p < 0.001$) during chronic phase in Group A, but no such improvement was present in Group B patients who showed no change in the Q wave. Global ejection fraction was greater and percent akinetic segment was smaller in Group A than in Group B at chronic phase. Coronary occlusion occurred more often at segment 7 in Group A; in Group B, occlusion occurred more frequently upstream at segment 6, suggesting Group A had a smaller area of risk. Spontaneous recanalization was more often (57%) and complete occlusion was less frequent in Group A. These indicate that Group A is characterized by a smaller area of risk, smaller infarct size, earlier reperfusion, and greater improvement in wall motion. Twenty-eight patients (70%) of Group A lost Q wave within one month and 12 patients (30%), after 3 months or more. Electrical stunning of the myocardium may be a possible mechanism for the early disappearance of Q waves, and anatomical healing for the late disappearance of Q waves.

THE presence of abnormal Q waves in an electrocardiogram (ECG) is one of the most important criteria for the diagnosis of myocardial infarction (MI). It has been repeatedly shown, however, that in some cases this diagnostic Q wave, which is present initially, disappears during evolution of the MI.¹⁻¹¹ Several mechanisms have been proposed to explain this disappearance. Coronary angiography (CAG), performed

up to several hours after onset of the MI provides insight into the pathological process and aids in determining the prognosis of the evolving MI. There are only a few reports dealing with the correlation between CAG findings and Q wave loss.^{6,8-10} In this report, we analyzed CAG data during the acute phase in patients showing Q wave disappearance and we concluded that these patients are characterized by a smaller area of risk, earlier reperfusion, effective myocardial salvage, and a smaller infarct size. We believe that the Q wave can disappear within a few days if the myocardium is electrically

Key words:

Acute myocardial infarction
Abnormal Q wave in electrocardiogram
Left ventricular wall motion
Stunned myocardium

(Received January 13, 1990; accepted May 7, 1990)

First Department of Internal Medicine, Kinki University School of Medicine, Osaka, Japan

Mailing address: Kinji Ishikawa, M.D., First Department of Internal Medicine, Kinki University School of Medicine, Osaka-Sayama 589, Japan

TABLE I CLINICAL FEATURES IN THREE GROUPS OF PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

		Group A (Q disappeared) n=40 (20%)	Group B (Q no change) n=145 (72%)	Group C (Q increased) n=16 (8%)
Age	(yr)	59±2	58±1	59±3
Male / female		36 / 4	121 / 24	15 / 1
Forrester Class	I / II / III / IV	25 / 4 / 3 / 3	89 / 15 / 16 / 5	10 / 4 / 1 / 0
prev MI		6	16	3
peak CK	(U / L)	2730±378	3156±160	2817±676
t-peak CK	(hr)	16±1	19±1	18±2
Infarct-related artery				
Segment	1	1	18	1
	2	1	12	1
	3,4	1	13	0
	6	7	28*	3
	7	18	21	3
	8,9	0	3	1
	11—14	1	8	1
Recanalization: spontaneous		12	27*	3
	: 100% → UK open	6	35	2
	: 100% → 100%	3	23	2
Collateral	: acute present / absent	11 / 12	28 / 51	3 / 3
Number of vessels involved	0 / 1 / 2 / 3	2 / 14 / 7 / 4	2 / 50 / 43 / 15	0 / 4 / 3 / 3
PCW (mmHg)	acute → chronic	15±1 → 7±1§	12±1 → 7±1§	9±1 → 6±1
LVEDV (ml / m ²)	acute → chronic	101±8 → 96±6	88±6 → 113±6§	(95 → 133)
LVESV (ml / m ²)	acute → chronic	45±5 → 42±5	41±3 → 53±4§	(44 → 66)
Global EF (%)	acute → chronic	57±3 → 57±3	52±2 → 49±2*	(49 → 50)
Regional EF (%)	acute → chronic	24±2 → 34±4§	26±2 → 29±2*	(32 → 27)
LV aneurysm	present / absent	1 / 10	13 / 22*	1 / 4
% akinetic segment (%)	acute → chronic	15±4 → 5±2††	18±2 → 11±2*†	(12 → 8)
Wall motion index	acute → chronic	12±1 → 8±1§	13±1 → 11±1**§	12±2 → 9±2§

mean ± SE Abbreviations prev MI: previous myocardial infarction. t-peak CK: time from onset to peak CK. 100% → UK open: 100% occluded artery was recanalized by urokinase. 100% → 100%: The artery remained 100% occluded during the acute phase. PCW: pulmonary capillary wedge pressure. LVEDV, LVESV: Left ventricular end-diastolic, end-systolic volume, respectively. EF: ejection fraction. (): Only mean value was shown, because n=2.

Statistical difference A vs B: *p<0.05, **p<0.01. acute vs chronic: †p<0.05, ††p<0.01, §p<0.001.

stunned but viable, and over months if anatomical healing processes persist.

SUBJECTS AND METHODS

We studied 201 patients who were admitted to our coronary care unit, from July 1977 to November 1987, within 6 hours of onset

of chest pain; 172 were male, 29 were female, and mean age was 58±1 years old (mean±standard error). Diagnosis of acute MI was based on the presence of typical chest pain, ST-segment elevation, increased white blood cell count, and elevated serum creatine phosphokinase activity (CK). Patients who showed intraventricular block,

TABLE II TIME TO DISAPPEARANCE OF Q WAVE

	N (%)
~1 week	6 (15.0)
~2 weeks	7 (17.5)
~1 month	15 (37.5)
~3 months	7 (17.5)
4 months~	5 (12.5)
Total	40

pulmonary embolism, atrial fibrillation, and recurrent myocardial infarction were not included. An ECG was recorded at least once a day for 7 days, once a week for 1 month, once a month for 3 months in all patients. After discharge, an ECG was recorded at least twice each year in the out-patient clinic. Those patients who died or were lost to follow up within 3 months were not included. Average follow-up time was 7.8 ± 0.7 months from onset. The Q wave was considered abnormal if it was more than 40 msec in duration and more than 25% of the R wave in amplitude. The most prominent abnormal Q wave in the ECG obtained during the first week was compared with Q waves seen in subsequent weeks. Patients were divided into 3 groups: Group A patients in whom the Q wave disappeared or did so in some leads, Group B patients in whom the Q wave did not change, and Group C patients — a

small group in whom the magnitude of the Q wave increased. Left ventriculograms and CAG were performed within 6 hours of onset in 128 of 201 cases and the same studies were performed 3 months later in 169 cases. In 99 cases, the study was done in both the 1st day (acute phase) and approximately 3 months later (chronic phase). Left ventricular end-diastolic and end-systolic volumes,¹² global ejection fraction, regional ejection fraction of the infarcted portion,¹³ and % akinetic segment¹⁴ were calculated from the left ventriculogram. Wall motion index¹⁵ was calculated from the echocardiogram. Intracoronary thrombolysis was performed in 99 of the patients who were admitted within 6 hours of onset. Recanalization by coronary thrombolysis was obtained in 43 of these patients. All data are presented as the mean $\pm$ one standard error of the mean. Intergroup comparisons were performed using Student's t-test for unpaired data.

RESULTS

Among the total group of 201 patients, 40 patients (20%) showed disappearance of Q wave (Group A), 145 (72%) retained the Q wave (Group B) and 16 (8%) showed an increase in the Q wave (Group C) (Table I). Among the 40 patients of Group A, 10 (25%) showed disappearance of all Q waves and 25 (62%) showed disappearance of Q and reappearance of r waves in some leads.

Fig. 1. Coronary angiographic findings obtained within 6 hours after onset in Groups A and B. 100-UK open: Coronary artery initially 100% occluded was recanalized by urokinase. 100-100: The occluded artery remained so even after thrombolysis with urokinase. The presence and absence of collaterals is indicated in the dotted area by (+) and (-).

Fig.2. Left ventricular (LV) volume and global ejection fraction (EF) in Groups A and B. EDV: end-diastolic volume, ESV: end-systolic volume.

In the other 5 patients of Group A, the abnormal Q wave regressed into insignificant small q waves. In 122 patients with anterior infarction, 37 (30%) showed disappearance

of the Q wave, while in the remaining 79 patients with inferior infarction, only 3 (4%) showed Q wave disappearance. The time of Q wave disappearance is shown in Table II. Six patients (15%) of Group A showed disappearance of the Q wave as early as 1 week, a total of 28 patients (70%) showed Q wave disappearance within 1 month, and 12 showed Q wave loss thereafter. There were no differences in clinical severity according to Forrester's classification¹⁶ or in previous infarction history among the 3 groups (Table I).

CAG was performed within 24 hours of onset in 21 and 85 patients in Groups A and B, respectively. This revealed that the highest incidence (57%) of spontaneous recanalization occurred in Group A (Table I, Fig. 1). The incidence of complete occlusion after thrombolysis was also lowest in Group A (only 3 patients). It is noteworthy that all of these patients had collaterals. This is in contrast to the findings in Group B: of 23 patients showing complete occlusion, only 9 (39%) had collaterals. Of the 25 patients of Group A whose occlusion was located at the left anterior descending coronary artery, 7 (28%) showed involvement at segment 6 and the rest (72%) had occlusions downstream (segment 7) (Table I). This pattern was not the same in Group B where the occluded site was rather upstream (54%) at segment 6 and 46% at segment 7, 8, or 9 (Table I). Peak CK was slightly lower in Group A than in Group B, suggesting Group A had smaller

Fig.3. Regional wall motion in Groups A and B.

infarcts. The time interval from onset to peak CK was slightly shorter in Group A than in Group B.

Left ventricular global as well as regional ejection fraction, % akinetic segment and the presence of aneurysms were evaluated only in patients with anterior infarctions because it has been shown that anterior and inferior infarctions evolve differently in terms of wall motion after reperfusion and, therefore, these patients cannot be evaluated together.¹⁷ In Group A, left ventricular end-systolic and end-diastolic volumes did not show any changes between the acute and chronic phases (Table I, Fig. 2); in Group B, the 2 volumes were greater during the chronic phase, indicating deterioration of left ventricular function. Global ejection fraction in Group A was better than that in Group B during the chronic phase since it was reduced in Group B but did not change in Group A during the chronic phase (Fig. 2). The regional ejection fraction at the infarcted portion improved during the chronic phase in Group A, while no improvement was observed in Group B (Fig. 3). % akinetic segment in Group A also diminished markedly during chronic phase; the change was less prominent in Group B (Table I, Fig. 3). Left ventricular aneurysm was rarely present (only 1 in 11 patients) in Group A; while more frequent (13/35) in Group B (Table I). Regional wall motion index was reduced at chronic phase in both groups. At chronic phase, this index was significantly lower in group A than in group B. The number of patients in Group C was limited and no statistically significant results were obtained in this group.

DISCUSSION

In the present study, 20% of transmural infarction patients showed disappearance of the Q wave. In contrast to patients in whom the Q wave persists, this group of patients is characterized by anterior (rather than inferior) infarction, smaller area of risk, the presence of collaterals, early recanalization of the occluded artery, and thus more effective myocardial salvage and smaller infarct size. There is also a greater improvement in wall motion of the infarcted portion during the chronic phase.

Incidence of Q wave disappearance It is known that abnormal Q waves often disappear in patients with transmural infarction.¹⁻¹¹ However, the reported incidence of disappearance varies considerably: 6.7%,² 11%,¹¹ 14.2%,⁴ 23%,⁷ or 34%,⁶ and Karnegis et al⁶ reported a relatively high rate of 34% in 692 patients with a mean follow-up time of 2.2 years. The criteria of the latter group for defining abnormal Q waves were more strict (Q/R amplitude ratio of 1/3 or more plus Q wave duration 0.03 seconds or more) than those in the present study and this difference might account for their greater incidence of disappearance. Kalbfleisch et al² reported that among 775 patients who had a significant Q wave initially, 6.7% showed complete disappearance of pathological Q waves by the end of 2 years. Their incidence was lower than ours. They did not include those patients showing Q wave disappearance in only some leads. In our study, complete disappearance of the Q wave was seen in 5%, which is similar to their reports. Taking into consideration the difference in the criteria of abnormal Q waves, follow-up time and patient population, it might be said that all reports are consistent and that the incidence is approximately 10 or 20%.

Transient abnormal Q waves in various conditions Wang et al¹⁸ reported a case with antero-septal infarction in which an abnormal Q wave disappeared following acute onset of inferior infarction. In our study, patients who had recurrent myocardial infarction during convalescence were excluded. Intraventricular conduction defects can cause changes in the QRS contour and we also excluded these patients. Abnormal Q waves can occur transiently during open-heart surgery.¹⁹ Q wave disappearance has been observed in patients with acute MI in which successful coronary thrombolysis⁵ or angioplasty⁹ was performed. Abnormal Q waves after brief occlusion of the coronary artery in dogs disappears upon reperfusion.²⁰ Transient abnormal Q waves can be observed in vasospastic or exercise-induced angina pectoris.^{8,22} Besides these ischemia-related events, transient abnormal Q waves have been observed in many pathological conditions such as hypoglycemia,²³ hyperkalemia,²⁴ shock,²⁵ metabolic disturbance,²⁶ acute pancreatitis,²⁷ phosphorous poisoning,²⁸

cerebrovascular accident²⁹ and tachycardia³⁰. The reports above all suggest that the Q wave is not the hallmark of myocardial necrosis.

Smaller risk area and infarct size It is interesting to consider whether or not patients with Q wave loss have a favourable clinical outcome. Several reports^{4,11} show that mortality is not low in patients with Q wave disappearance. However, our study revealed that left ventricular motion was less impaired during the chronic phase in Group A. This is in agreement with Jaarsma et al⁷ who also showed that segmental asynergy was less in this group. Kalbfleisch et al² postulated that myocardial infarction was "less extensive" in this group of patients because elevation of SGOT was not high. Peak CK in our study was also slightly lower in Group A than in Group B. Coll et al¹¹ reported that peak CK was significantly higher in patients who retained their Q waves than in those who lost them. They indicated that although differences in mortality were not significant, congestive heart failure was prevalent among patients with permanent Q waves and concluded that Q wave loss may be related to smaller infarct size. However, they showed no CAG findings during acute phase. In our study, CAG revealed that the occluded site was located more distally in Group A, suggesting this group had a smaller area of risk. Spontaneous recanalization was prevalent in Group A. Our CAG findings indicate that Group A is characterized by a smaller area of risk, by earlier reperfusion which results in effective myocardial salvage, and by smaller infarct size.

Time course of Q wave disappearance It is important to note when the Q wave disappears because it may give some insight into the mechanisms involved in this phenomenon. There is a wide range in time, from several days^{2,5} to 6 years⁶ for Q waves to disappear. After the onset of acute MI, when the infarcted area is small, shrinkage occurs gradually by healing and viable muscle covers the scarred area^{2,31}. Adequate collateral circulation^{2,31} is no doubt important for this process. Left ventricular remodeling³² may contribute to reducing the relative size of the infarcted area. These anatomical healing processes may be important for Q wave loss but they may take

several months or years.

Haiat and Chiche²² divided Q wave disappearance into longterm and early disappearances. Kalbfleisch et al² reported that Q waves can disappear as early as 6 days after onset. Bateman et al⁵ reported a case of acute MI with a deep pathological Q wave 1 hour after onset. Coronary thrombolysis in this case resulted in the loss of chest pain and normalization of the ECG 15 min later. In our study also, 15.0% of Group A showed disappearance of Q wave within 1 week of onset and 70.0% within 1 month. These early disappearances cannot be explained by anatomical healing processes. DePasquale et al³³ concluded that there may be a loss of electromotive force (emf) from myocardial tissue without biological death. Haiat and Chiche²² also indicated that loss of electromotive force is not synonymous with tissue death. However, these authors did not show how electromotive force can be lost without tissue death. Ibba et al⁹ described this phenomenon as "electrical death" and stressed that the presence of a Q wave does not necessarily indicate a cell death. The QRS complex is the result of an action potential in the myocardium. Reversible myocardial ischemic injury will cause depletion of ATP and of the adenine nucleotide pool³⁴. These histochemical changes may cause a transient loss of electromotive force resulting in an electrical silent area. Bateman et al came to the same conclusion and described the cardiac electrical system as "stunned"^{5,8}. These histochemical changes may be more likely to cause Q wave loss in a relatively early phase.

Clinical implications An abnormal Q wave in the early phase of acute myocardial infarction does not necessarily indicate that the infarcted area is totally necrotic. Coronary thrombolysis may be able to salvage the evolving infarction. If the Q wave disappears in the early phase, it indicates that early reperfusion was effective and a considerable amount of viable myocardium is present in the infarcted area. It may well be said in this case that the infarction is "incomplete"³⁵. Therefore, disappearance of the Q wave indicates that there is an opportunity to protect the remaining viable muscle from infarction, and coronary angioplasty or other effective measures to prevent reinfarction.

tion is warranted in this group of patients.

REFERENCES

1. RUBIN IL, GROSS H, VIGLIANO EM: Transient abnormal Q waves during coronary insufficiency. *Am Heart J* **71**: 254, 1966
2. KALBFLEISCH JM, SHADAKSHARAPPA KS, CONRAD LL, SARKAR NK: Disappearance of the Q-deflection following myocardial infarction. *Am Heart J* **76**: 193, 1968
3. MELLER J, CONDE CA, DONOSO E, DACK S: Transient Q waves in Prinzmetal's angina. *Am J Cardiol* **35**: 691, 1975
4. WASSERMAN AG, BREN GB, ROSS AM, RICHARDSON DW, HUTCHINSON RG, RIOS JC: Prognostic implications of diagnostic Q waves after myocardial infarction. *Circulation* **65**: 1451, 1982
5. BATEMAN TM, CZER LSC, GRAY RJ, MADDAHI J, RAYMOND MJ, GEFT IL, GANZ W, SHAH PK, BERMAN DS: Transient pathologic Q waves during acute ischemic events: an electrocardiographic correlate of stunned but viable myocardium. *Am Heart J* **106**: 1421, 1983
6. KARNIGIS JN, MATTS J, TUNA N, AMPLATZ K, THE POSCH GROUP: Relation between Q-QS changes on the rest electrocardiogram and left ventricular function with healed myocardial infarction. *Am J Cardiol* **58**: 399, 1986
7. JAARSMA W, VISSER CA, VAN EENIGE MJ, ROOS JP: Left ventricular wall motion with and without Q-wave disappearance after acute myocardial infarction. *Am J Cardiol* **59**: 516, 1987
8. BATEMAN T, GRAY R, MADDAHI J, ROZANSKI A, RAYMOND M, BERMAN D: Transient appearance of Q waves in coronary disease during exercise electrocardiography: consideration of mechanisms and clinical importance. *Am Heart J* **104**: 182, 1982
9. IBBA GV, TERROSO P, FRANCESCHINO V, CONTINI GM, FRAU G, SANNIA L: Disappearance of pathologic Q wave after PTCA in evolving myocardial infarction. *Am Heart J* **108**: 1538, 1984
10. ASCHER EK, STAUFFER JE, GAASCH WH: Coronary artery spasm, cardiac arrest, transient electrocardiographic Q waves and stunned myocardium in cocaine-associated acute myocardial infarction. *Am J Cardiol* **61**: 939, 1988
11. COLL S, BETRIU A, FLORES TD, ROIG E, SANZ G, MONT L, MAGRINA J, SERRA A, LOPEZ FN: Significance of Q-wave regression after transmural acute myocardial infarction. *Am J Cardiol* **61**: 739, 1988
12. DODGE HT, SANDLER H, BALLEW DW, LORD JD JR.: The use of biplane angiocardigraphy for the measurement of left ventricular volume in man. *Am Heart J* **60**: 762, 1960
13. GELBERG HJ, BRUNDAGE BH, GLANTZ S, PARMLEY WW: Quantitative left ventricular wall motion analysis: a comparison of area, chord and radial methods. *Circulation* **59**: 991, 1979
14. KITAMURA S, ECHEVARRIA M, KAY JH, KROHN BG, REDINGTON JV, MENDEZ A, ZUBIATE P, DUNNE EF: Left ventricular performance before and after removal of the noncontractile area of the left ventricle and revascularization of the myocardium. *Circulation* **45**: 1005, 1972
15. HEGER JJ, WEYMAN AE, WANN LS, ROGERS EW, DILLON JC, FEIGENBAUM H: Cross-sectional echocardiographic analysis of the extent of left ventricular asynergy in acute myocardial infarction. *Circulation* **61**: 1113, 1980
16. FORRESTER JS, DIAMOND GA, SWAN HJC: Correlative classification of clinical and hemodynamic function after acute myocardial infarction. *Am J Cardiol* **39**: 137, 1977
17. ROGERS WJ, HOOD WP JR, MANTLE JA, BAXLEY WA, KIRKLIN JK, ZORN GL, NATH HP: Return of left ventricular function after reperfusion in patients with myocardial infarction: Importance of subtotal stenoses or intact collaterals. *Circulation* **69**: 338, 1984
18. WANG K, SEGAL MS, WARD PCJ: Sudden disappearance of electrocardiographic pattern of anteroseptal myocardial infarction. Result of superimposed acute posterior myocardial infarction. *Chest* **70**: 402, 1976
19. KLEIN HO, GROSS H, RUBIN IL: Transient electrocardiographic changes simulating myocardial infarction during open-heart surgery. *Am Heart J* **79**: 463, 1970
20. BAYLEY RH, LA DUE JS: Differentiation of the electrocardiographic changes produced in the dog by prolonged temporary occlusion of a coronary artery from those produced by postoperative pericarditis. *Am Heart J* **28**: 233, 1944
21. GROSS H, RUBIN IL, LAUFER H, BLOOMBERG AE, BUJDOSO L, DELMAN AJ: Transient abnormal Q waves in the dog without myocardial infarction. *Am J Cardiol* **14**: 669, 1964
22. HAIAT R, CHICHE P: Transient abnormal Q waves in the course of ischemic heart disease. *Chest* **65**: 140, 1974
23. GOLDMAN AG, GROSS H, RUBIN IL: Transitory Q waves simulating the Q wave of myocardial infarction. *Am Heart J* **60**: 61, 1960
24. NORA JR, PILZ CG: Pseudoinfarction pattern associated with electrolyte disturbance. *Arch Intern Med* **104**: 300, 1959
25. ROSENFELD I, SILVERBLATT ML, GRISHMAN A: Allergic shock in humans: report of two cases with electrocardiographic findings. *Am Heart J* **53**: 463, 1957
26. SHUGOLL GI: Transient QRS changes simulating myocardial infarction associated with shock and severe metabolic stress. *Am Heart J* **74**: 402, 1967
27. FULTON MC, MARRIOTT HJL: Acute pancreatitis simulating myocardial infarction in the electrocardiogram. *Ann Intern Med* **59**: 730, 1963
28. PIETRAS RJ, STAVRAKOS C, GUNNAR RM, TOBIN JR Jr.: Phosphorus poisoning simulating acute myocardial infarction. *Arch Intern Med* **122**: 430, 1968
29. MENON IS: Electrocardiographic changes simulating myocardial infarction in cerebrovascular accident. *Lancet* **2**: 433, 1964
30. RUBIN IL, GROSS H, ARBEIT SR: Transitory abnormal Q waves during bouts of tachycardia. *Am J Cardiol* **12**: 659, 1963

31. PAPPAS MP: Disappearance of pathological Q waves after cardiac infarction. *Brit Heart J* **20**: 123, 1958
32. MCKAY RG, PFEFFER MA, PASTERNAK RC, MARKIS JE, COME PC, NAKAO S, ALDERMAN JD, FERGUSON JJ, SAFIAN RD, GROSSMAN W: Left ventricular remodeling after myocardial infarction: a corollary to infarct expansion. *Circulation* **74**: 693, 1986
33. DEPASQUALE NP, BURCH GE, PHILLIPS JH: Electrocardiographic alterations associated with electrically "silent" areas of myocardium. *Am Heart J* **68**: 697, 1964
34. REIMER KA, HILL ML, JENNINGS RB: Prolonged depletion of ATP and of the adenine nucleotide pool due to delayed resynthesis of adenine nucleotides following reversible myocardial ischemic injury in dogs. *J Mol Cell Cardiol* **13**: 229, 1981
35. SCHUSTER EH, BULKLEY BH: Early post-infarction angina: ischemia at a distance and ischemia in the infarct zone. *N Engl J Med* **305**: 1101, 1981