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Prominent negative T waves with QT prolongation indicate reperfusion injury and myocardial stunning

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Summary

To observe the clinical course after reperfusion and recovery from myocardial stunning of the left ventricular anterior wall, we prospectively reviewed and analyzed cardiac enzymes, ECG changes, echocardiograms, and cineangiograms in 8 patients with the acute ischemic syndrome who fulfilled the following criteria: 1) no history of previous myocardial infarction, 2) repeated and/or prolonged episodes of chest pain, 3) critical stenosis of the left anterior descending artery with wall motion abnormalities, 4) successful emergency percutaneous transluminal coronary angioplasty, and 5) normal wall motion on repeat cineangiography 4 to 8 weeks later.

Creatine kinase (CK) and/or its cardiac isoenzyme (CK-MB) were minimally elevated in all cases. Wall motion was normalized with the reduction of end-systolic volume (end-diastolic volume: from 139 ± 25 to 140 ± 37 m*l*, ns, end-systolic volume: from 68 ± 16 to 39 ± 13 m*l*, $p<0.001$, ejection fraction: from 51 ± 6 to $71\pm6\%$, $p<0.001$). Serial echocardiograms showed normalization of wall motion within 4 to 28 days. T wave inversion in the left precordial leads developed 30 min to 5 hours after the cessation of chest pain or successful reperfusion, and prominent negative T waves (1.6 ± 0.6 mV) with QT prolongation (0.56 ± 0.08 sec) in V_3 or V_4 reached their peak values within one to 5 days. ECG abnormalities resolved after 21 to 95 days.

These ECG findings may indicate reperfusion injury and the presence of myocardial stunning in the anterior wall of the left ventricle.

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Key words
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Stunned myocardium

Introduction

Emergency thrombolysis and percutaneous transluminal coronary angioplasty have been established as a therapeutic measure for salvaging dying myocardium in the acute ischemic syndrome[1-4]. Restoration of blood flow can be detected only by repeat coronary cineangiography, and there is no reliable noninvasive bedside procedure for assessing successful revascularization[3]. When thrombolytic agents are administered intravenously in hospitals without catheterization facilities, it would be very helpful to assess successful revascularization if such a noninvasive tool could be available.

Myocardial stunning or prolonged postischemic ventricular dysfunction is a well-known phenomenon occurring after successful revascularization in experimental animal models[5], and the importance of this phenomenon has recently been recognized in clinical studies[6-8]. The full clinical picture of myocardial stunning, however, has not been well documented. To clarify the clinical outcome of stunned myocardium and to detect the diagnostic markers of reperfusion, we prospectively evaluated serial cardiac enzymes, ECG changes, echocardiograms, and cineangiograms in patients with the acute ischemic syndrome whose coronary blood flow was restored by emergency angioplasty.

The development of prominent negative T waves in the left precordial leads has been observed in some patients with the acute ischemic syndrome. These ECG findings have been interpreted as "intramural myocardial infarction"[9] or "myocardial injury"[10] and are usually considered equivalent to subendocardial or non-Q wave infarction[10,11]. However, the mechanism and clinical significance of this abnormality are not well understood.

This report describes the characteristic ECG findings, i.e., the development of prominent negative T waves in the left precordial leads with QT prolongation during acute ischemia in patients with critical lesions in the left anterior descending artery. We consider that these ECG changes may be manifestations of reperfusion injury and of myocardial stunning in the left ventricular anterior wall.

Table 1. Patient profiles

Patient No.	Age (yrs)	Sex	History of					Family history
			AP	HT	DM	HL	Smoking	
1	58	F	+	+	−	−	+	−
2	66	F	+	+	−	−	−	−
3	65	F	+	−	+	+	−	−
4	71	M	−	+	−	−	−	−
5	70	F	+	+	+	−	+	−
6	65	F	−	−	−	−	−	−
7	68	M	+	−	−	+	+	−
8	65	M	+	+	−	−	+	+

Abbreviations: AP=angina pectoris; HT=hypertension; DM=diabetes mellitus; HL=hyperlipidemia; F=female; M=male; +=present; −=absent.

Methods

1. Study population

In 1989, 8 consecutive patients among 45 who underwent emergency coronary cineangiography and 14 who underwent coronary angioplasty satisfied the following criteria: 1) no history or abnormal q waves indicating previous myocardial infarction, 2) prolonged and/or repeated episodes of chest pain longer than 15 min within 24 hours prior to admission, 3) critical stenosis (luminal narrowing >90%) of the left anterior descending artery as determined by emergency coronary cineangiography, 4) segmental wall motion abnormalities detected by biplane left ventricular cineangiography, 5) successful emergency angioplasty, and 6) normal wall motion demonstrated by repeat cineangiography 4 to 8 weeks after the emergency procedure. Five of the 8 patients were admitted to the hospital via the emergency room with the tentative diagnosis of the acute ischemic syndrome; the other 3 were transferred from other hospitals for emergency angioplasty. Three were males,and patients' ages ranged from 58 to 71 years (**Table 1**). Patients with previous myo-

Table 2. Manifestations of the acute ischemic syndrome

Patient No.	Unstable period (days)	Frequency of attacks (times)	Duration of the last pain episode (hours)	Duration from the onset of the last pain to PTCA (hours)
1	4	3	0.5	17
2	4	2	2	2
3	7	8	4	4
4	2	4	0.5	12
5	1	3	1	18
6	1	1	3	3
7	6	3	3	4
8	3	4	0.3	9

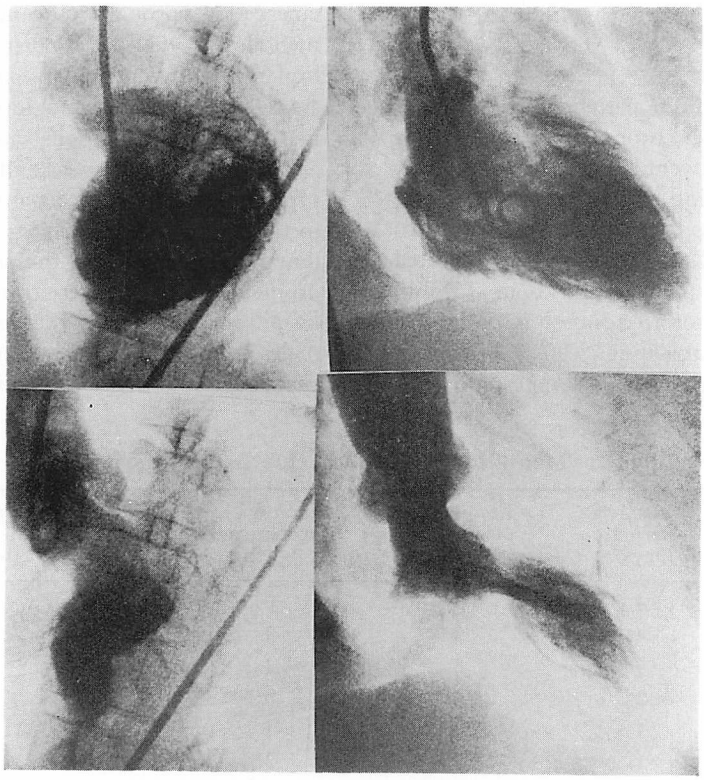
Abbreviations: PTCA=percutaneous transluminal coronary angioplasty.

Table 3. Maximum enzyme values and coronary angiographic findings

Patient No.	CK (U/L) (<160)	CK-MB (U/L) (<15)	LAD lesions (%)	Intracoronary thrombus	Residual stenosis after PTCA (%)
1	212	39	Seg 6: 99 (D+)	(+)	0
2	124	16	Seg 6: 99 (D+)	(+)	0
3	210	28	Seg 6: 100	(+)	25
4	146	25	Seg 7: 90	(−)	25
5	241	16	Seg 6: 99	(−)	25
6	545	43	Seg 6: 99 (D+)	(+)	50
7	217	24	Seg 7: 99 (D+)	(−)	50
8	114	16	Seg 7: 90	(−)	0

Abbreviations: CK=creatine kinase; CK-MB=creatine kinase isoenzyme MB band; LAD=left anterior descending artery; PTCA=percutaneous transluminal coronary angioplasty; Seg=code No. of the coronary artery according to the nomenclature of the American Heart Association[12]; D+=with delay, numbers in parentheses represent normal values of our laboratory.

A)



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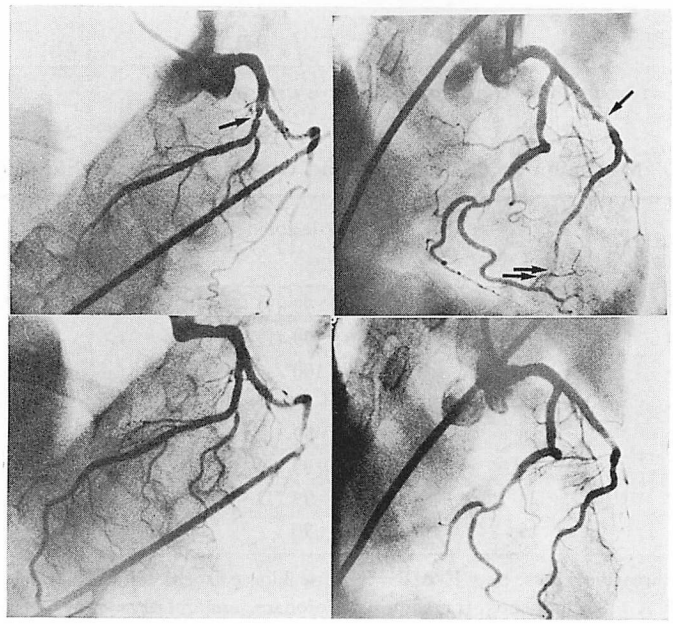

C)

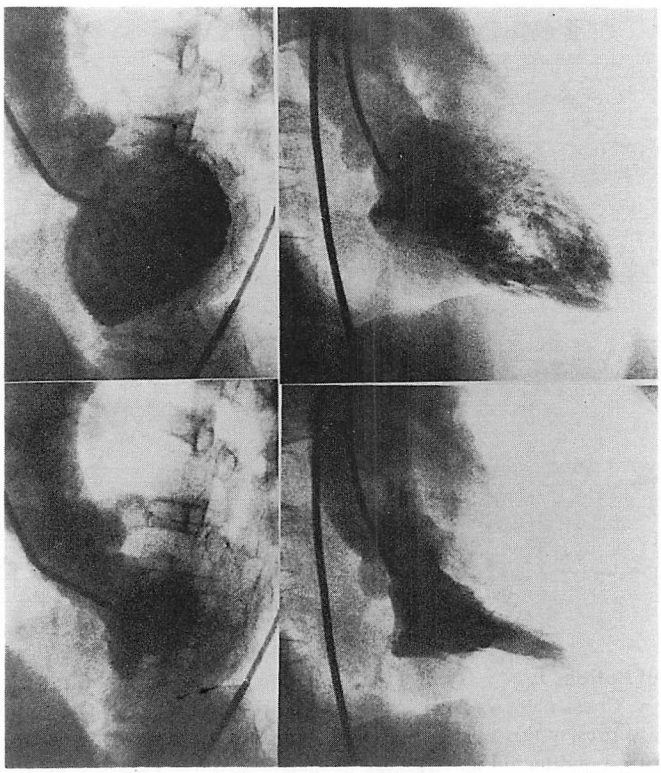

Fig. 1-A. The emergency biplane left ventricular cineangiograms of Patient 1 (Feb. 2, 1989).
The left anterior (left panel) and right anterior oblique (right panel) projections showed hypokinesis in the anterior, apical, and septal areas. Chamber volumes and ejection fraction are shown in Table 4.
Fig. 1-B. Coronary cineangiograms before (top) **and after** (bottom) **percutaneous transluminal coronary angioplasty in Patient 1** (Feb. 2, 1989).
Top: A critical stenosis was present in the left anterior descending artery with a proximal hazy filling defect which indicated intraluminal thrombus formation (single arrow), and delayed filling (double arrows). Bottom: The stenosis was eliminated by emergency angioplasty.
Fig. 1-C. Repeat biplane left ventricular cineangiograms of Patient 1 6 weeks later (March 16, 1989).
Wall motion abnormalities were not observed. There was a decreased end-systolic volume and an increased ejection fraction.

cardial infarction, critical lesions in the right coronary and/or left circumflex arteries, and those who underwent emergency aorto-coronary bypass surgery were excluded from this study because of difficulty in interpreting their ECG findings.

2. Catheterization procedures

After the consents of the patient and the patient's family were obtained, emergency coronary cineangiography was performed via the femoral approach. Premedication consisted of intravenous diazepam (5 mg), isosorbide dinitrate (5 mg), aspirin (500 mg, Venopirin iv®), heparin (10,000 units), and oral diltiazem (60 mg). Coronary cineangiography was first recorded in multiple projections. Biplane left ventricular cineangiograms were obtained in the 30° right and 60° left anterior oblique projections after a power injection of Iopamiron 370® (30 m*l* in 3 sec). Intracoronary injection of urokinase (240,000 to 480,000 units) were administered before angioplasty in 4 patients. Percutaneous transluminal

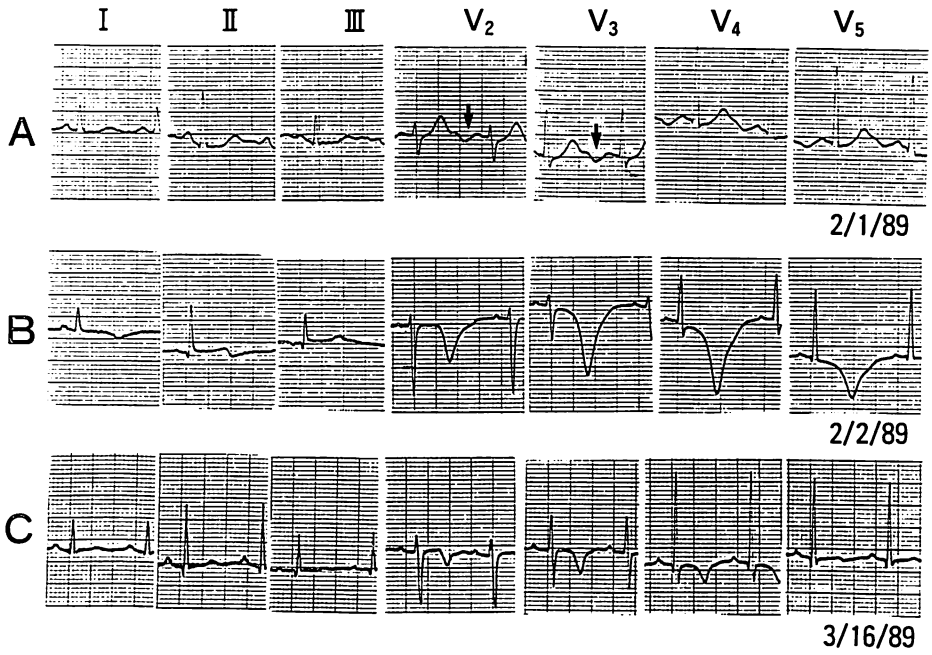

Fig. 1-D. ECG of Patient 1.

A: On admission. Chest pain resolved immediately prior to arrival at the hospital. A prominent negative U wave was present (arrow). B: Immediately before the emergency procedure, and 17 hours after admission, corresponding to Fig. 1-A. Giant negative T waves with QT prolongation comprised the characteristic findings. C: Immediately before the repeat cineangiography, corresponding to Fig. 1-C. T wave inversion was still present.

coronary angioplasty was performed in a usual manner with inflation of the balloon 3 to 4 times at a pressure of 6 to 8 atm each time. Repeat left ventricular and coronary cineangiography was performed 4 to 8 weeks after the emergency procedure. None of the patients required repeat angioplasty. Left ventricular angiograms were visually assessed according to the nomenclature of the American Heart Association[12] and the volume was calculated by the biplane area-length method using a computer (Cardio 200, Kontron, Stockholm).

3. Cardiac enzymes, ECG, and echocardiograms

The standard 12-leads ECG, the serum creatine kinase level and its cardiac isoenzyme (CK-MB) were determined every 4 hours during the initial 24 hours. The ECG was then recorded every day for one week, and once or twice per week thereafter. Echocardiograms were recorded on the ward after the procedure using commercially-available equipment (Toshiba SSH-60, Tokyo) when the patient's condition had stabilized. Echocardiography was repeated several times until asynergy resolved. Wall motion abnormalities were judged by interpreting the cineangiograms qualitatively[12].

4. Data presentation

All data are presented as the mean and one standard deviation of the mean (Mn±SD). The paired Student's t-test was used for the statistical analysis.

Results

1. Clinical profiles

Histories of chronic stable angina were elicited in 6 patients (**Table 1**). All but one patient (patient 6) had repeated episodes of chest pain

Table 4. Left ventricular cineangiographic findings

Patient No.	Before PTCA				4 to 8 weeks after PTCA		
	EDV (ml)	ESV (ml)	EF (%)	Regional asynergy (area code)†	EDV (ml)	ESV (ml)	EF (%)
1	139	66	53	2, 3, 6: hypo	137	35	75
2	109	45	59	3: hypo	167	29	76
3	121	67	44	3, 6: a, 2: dys	110	44	62
4	178	82	54	3: hypo, 6: a	186	58	69
5	107	48	55	3: hypo	90	21	77
6	160	90	44	3, 6: hypo, 2: dys	105	31	70
7	148	82	44	1, 2, 3, 6: hypo	138	52	63
8	148	62	58	3, 6: hypo	185	48	74
	139±25	68±16	51±6		140±37	39±13*	71±6*

Abbreviations: EDV=end-diastolic volume; ESV=end-systolic volume; EF=ejection fraction; †=regional asynergy was expressed by a coding system according to the recommendations of the American Heart Association[12]; hypo=hypokinesis; dys=dyskinesis; a=akinesis; *=$p<0.001$ compared to the initial emergency angioplasty.

during the 24 hours preceding the procedure, indicative of the acute ischemic syndrome (**Table 2**). Patient 6 was transferred to the catheterization laboratory from another hospital 2 hours after the initial onset of chest pain. Emergency angioplasty was performed immediately for chest pain in 4 patients (Nos. 2, 3, 6, and 7). The time lag between the last episode of chest pain and angioplasty ranged from one to 17 hours for the remaining 4 patients. Cardiac enzymes were significantly elevated (more than twice the upper limit of normal) in patients 1 and 6, and minimally elevated in the remaining 6 patients (**Table 3**). Based on the results of these enzyme and ECG findings (described below), it was difficult, even retrospectively, to conclude that the patients had subendocardial infarction. The clinical courses in all patients were uneventful, and no patient had additional episodes of chest pain or arrhythmias after the emergency angioplasty.

2. Coronary and left ventricular cineangiographic findings

All patients had critical stenoses of the proximal portion of the left anterior descending artery (**Table 3**). Intracoronary thrombus, which was defined as a hazy filling defect, was detected in 4 patients (**Table 3, Fig. 1**). After angioplasty residual stenosis was reduced to less than 50% in all cases, none had significant restenosis on repeat angiography. Biplane ventricular cineangiograms showed segmental wall motion abnormalities in all patients with reduced ejection fractions (**Table 4, Figs. 1, 2**). These regional wall motion abnormalities had resolved by the times of re-evaluation. Com-

Table 5. Echocardiographic observations

Patient No.	Acute phase (area code)†	Normalization (days)
1	2, 3, 6: hypokinesis	17< <28
2	3: hypokinesis	<14
3	3: dyskinesis	21< <28
4	6: akinesis	<15
5	3: hypokinesis	4
6	2, 6: akinesis	
	3: dyskinesis	21< <28
7	3, 6: akinesis	5
8	3: akinesis	7

†=see Table 4.

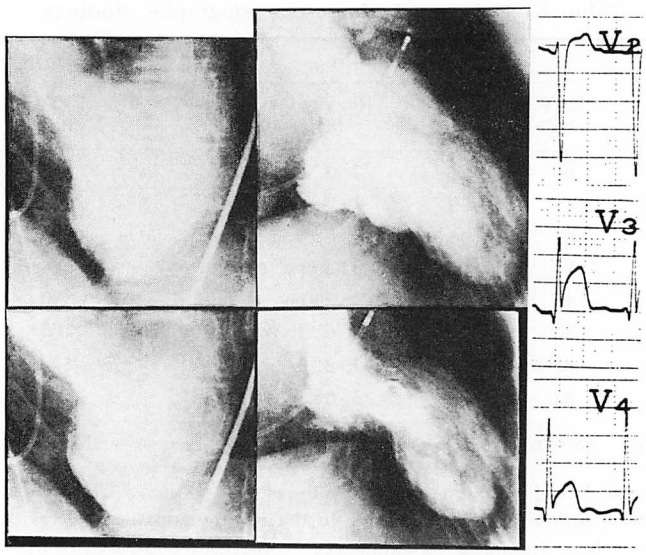

Fig. 2-A. The emergency biplane left ventricular cineangiograms and ECG of Patient 3 immediately before the angioplasty procedure (June 2, 1989).

Extensive akinesis (areas 2, 3, 6[12]) was evident. The ECG showed ST elevation with small q waves in V_3 and V_4.

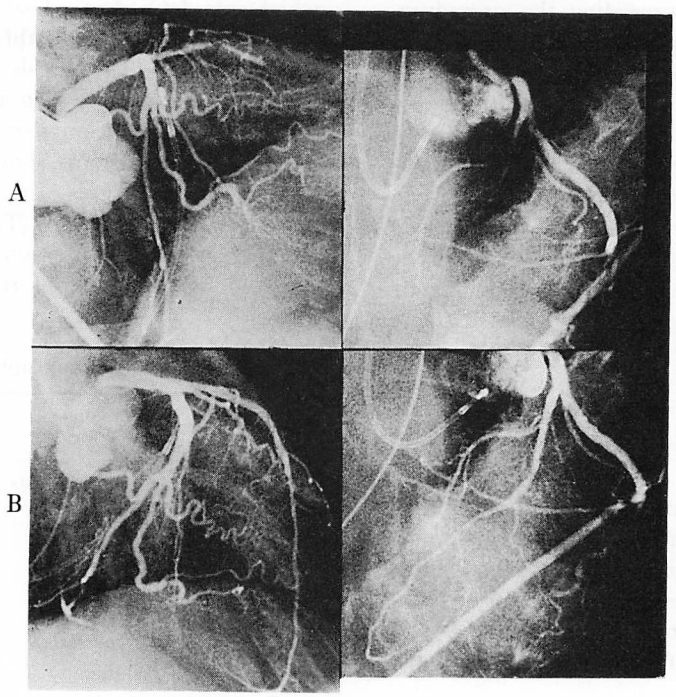

Fig. 2-B. Coronary cineangiograms of Patient 3 before (A) **and after** (B) **angioplasty** (June 2, 1989).

A: Total obstruction was present in the proximal left anterior descending artery. B: The obstruction was corrected by angioplasty without residual stenosis.

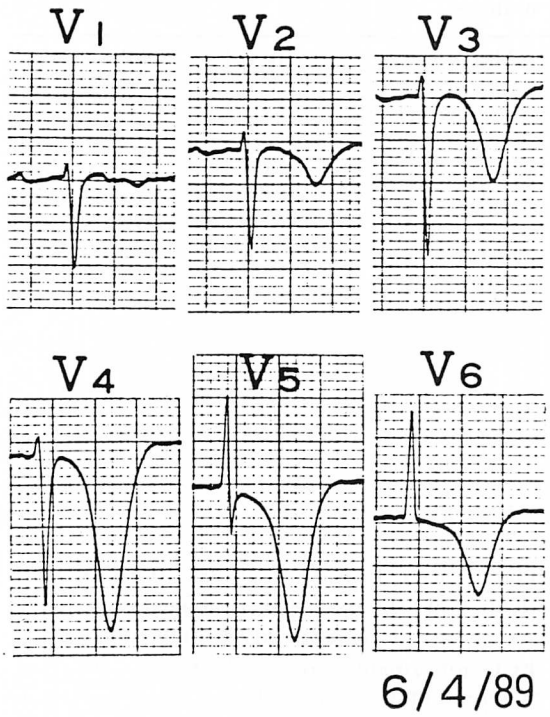

Fig. 2-C. ECG of Patient 3 2 days after emergency angioplasty (June 4, 1989).

The abnormal q waves in V_3 and V_4 resolved, but poor R wave progression in V_1 to V_4 and giant negative T waves with QT prolongation were present.

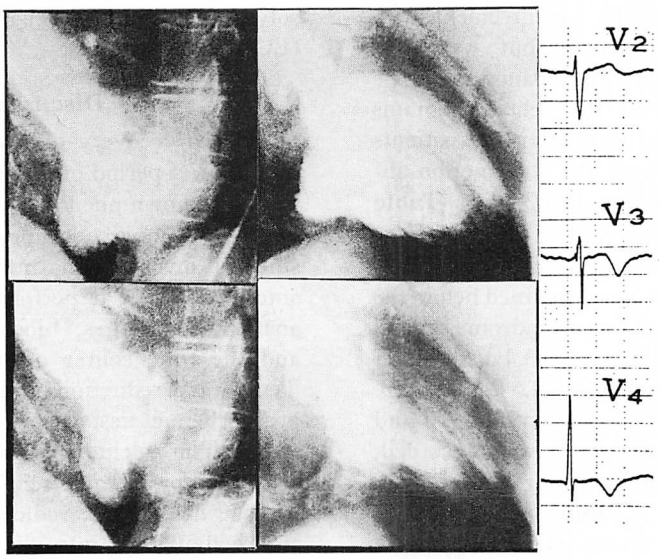

Fig. 2-D. Left ventriculograms with ECG repeated 5 weeks after the emergency procedure in Patient 3 (July 10, 1989).

Although the ventriculograms showed normal wall motion, the ECG showed residual T wave inversion of some degree.

Table 6. ECG findings

Patient No.	During or immediately after chest pain	Max negative T (mV)	Max QTc (sec)	Normalization (days)
1	ST ↓ in II, III, $_aV_F$, negative U in V_2-V_5	1.7 in V_3	0.64	45< <73
2	ST ↑ in V_3-V_6	2.6 in V_3	0.72	39< <54
3	qs in V_3, ST ↑ in V_1-V_5	1.7 in V_4	0.60	49< <95
4	ST ↑ in V_1-V_3, negative T in V_2-V_5	2.0 in V_3	0.60	36< <57
5	ST ↑ in III, $_aV_F$, V_2, V_3, ST ↓ in I, $_aV_L$, V_5, V_6	1.5 in V_4	0.60	18< <26
6	ST ↓ in V_3-V_6	1.0 in V_3	0.56	41< <75
7	qs in V_1, V_2, ST ↑ in V_1-V_5, ST ↓ in II, III, $_aV_F$, V_6	0.5 in V_4	0.44	25< <63
8	ST ↑ in III, $_aV_F$, V_1, V_2, ST ↓ in I, $_aV_L$, V_4-V_6	1.4 in V_4	0.48	3< <21
		1.6 ±0.6	0.56 ±0.08	32< <58 ±15 ±25

↑ =elevation; ↓ =depression.

parison of the emergency and the follow-up data showed that the left ventricular end-diastolic volume did not change, but the end-systolic volume decreased significantly ($p< 0.001$).

3. Echocardiographic observations

Echocardiographic regional wall motion abnormalities were generally in agreement with the cineangiography findings but were less marked (**Table 5**). This discrepancy might be related to the fact that the echocardiograms were recorded on the ward after the patients had stabilized. In all patients, wall motion abnormalities resolved within 4 to 28 days (**Table 5, Fig. 3**).

4. ECG changes

In all patients, ECGs were obtained before the onset of the acute ischemic syndrome. Atrial fibrillation was present in patient 3, but the other ECGs were considered normal. ECGs were recorded during attacks in patients 2, 3, 6 and 7, during which ST elevations were recorded, and within 30 min after the relief of pain in the remaining 4 (**Table 6**). Newly-developed abnormal q waves were observed in patients 3 (**Fig. 2**) and 7. Inverted T waves developed from 30 min to 5 hours after the spontaneous relief of pain or angioplasty and reached peaks one to 5 days later. The maximal negative T waves exceeded 1.0 mV in 7 patients (1.6±0.6 mV). QT prolongation was observed in all patients (0.56±0.08 sec). These ECG abnormalities resolved after 21 to 95 days. A representative series of sequential ECG findings is shown in **Fig. 4**. Mechanical abnormalities (regional wall motion abnormalities) resolved much earlier than did the electrical (ECG) abnormalities.

Discussion

Delayed recovery of myocardial contraction after a brief period of coronary artery occlusion is a well-known phenomenon[13,14]. Since Braunwald & Kloner[5] proposed the concept of stunned myocardium, numerous experimental studies have been performed to elucidate its anatomical features, biochemical mechanisms, and the time course of mechanical recovery. Temporary reduction in blood flow into the post-ischemic tissue (no-reflow phenomenon), intracellular calcium overload (calcium paradox), accumulation of fatty acids, myocardial edema, damage due to free radicals (oxygen paradox), and hemorrhage into the necrotic tissue are recognized as the causes of reperfusion injury, which can result in stunned myocardium and/or exacerbation of the infarct size[1,15].

Jennings et al[16] have proposed the definition

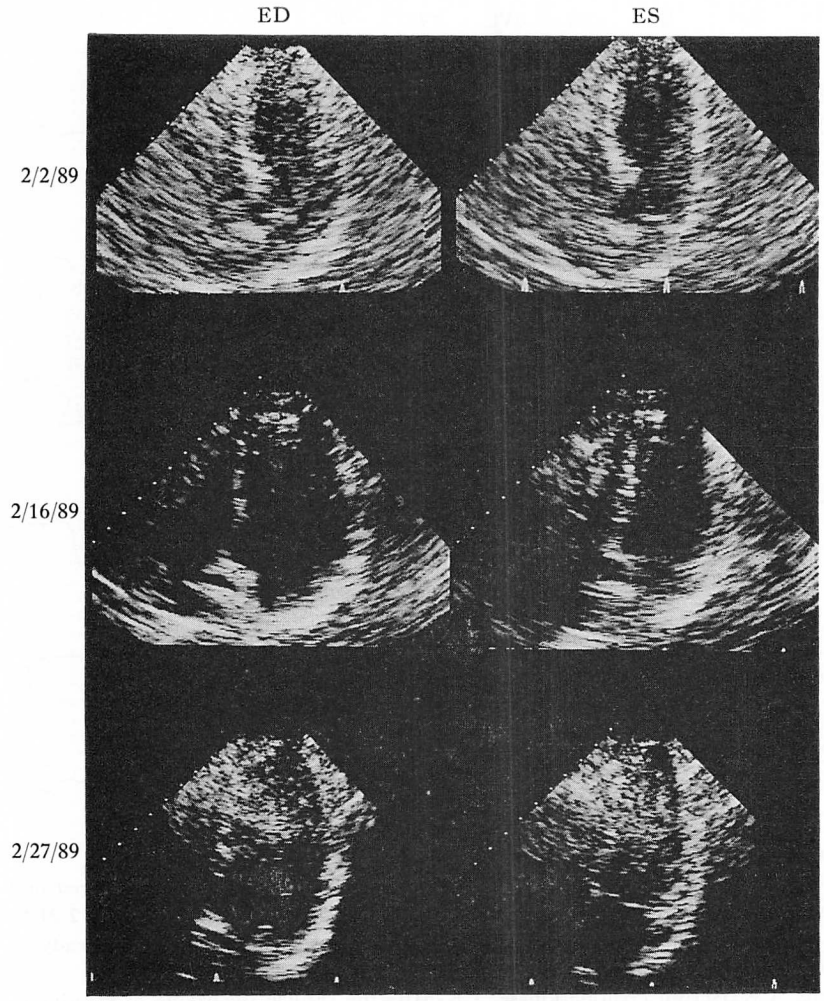

Fig. 3. Serial two-dimensional echocardiograms of Patient 1.

The apical 4-chamber view showed a large area of hypokinesis around the septum on 2/2 and 2/16, which resolved by 2/27/89.

Abbreviations: ED = end-diastole; ES = end-systole.

of reperfusion injury as accelerated myocardial cell death with reperfusion, but this term is generally used to convey a broader meaning, including reperfusion arrhythmia and myocardial stunning[15]. In contrast to the hazards of reperfusion injury, benefits of the restoration of blood flow into totally occluded arteries have been well established in terms of the reduction in mortality and the preservation of ventricular function[2-4]. In experimental animals, it has been demonstrated that functional recovery of the post-ischemic myocardium occurs days to weeks after occlusion of 2 to 3 hours[17]. Although case reports have been sporadically published[6-8], prospective analysis of recovery from myocardial stunning after reperfusion has not yet been reported in patients with the acute ischemic syndrome.

This investigation was initially performed to evaluate the clinical outcome of stunned myo-

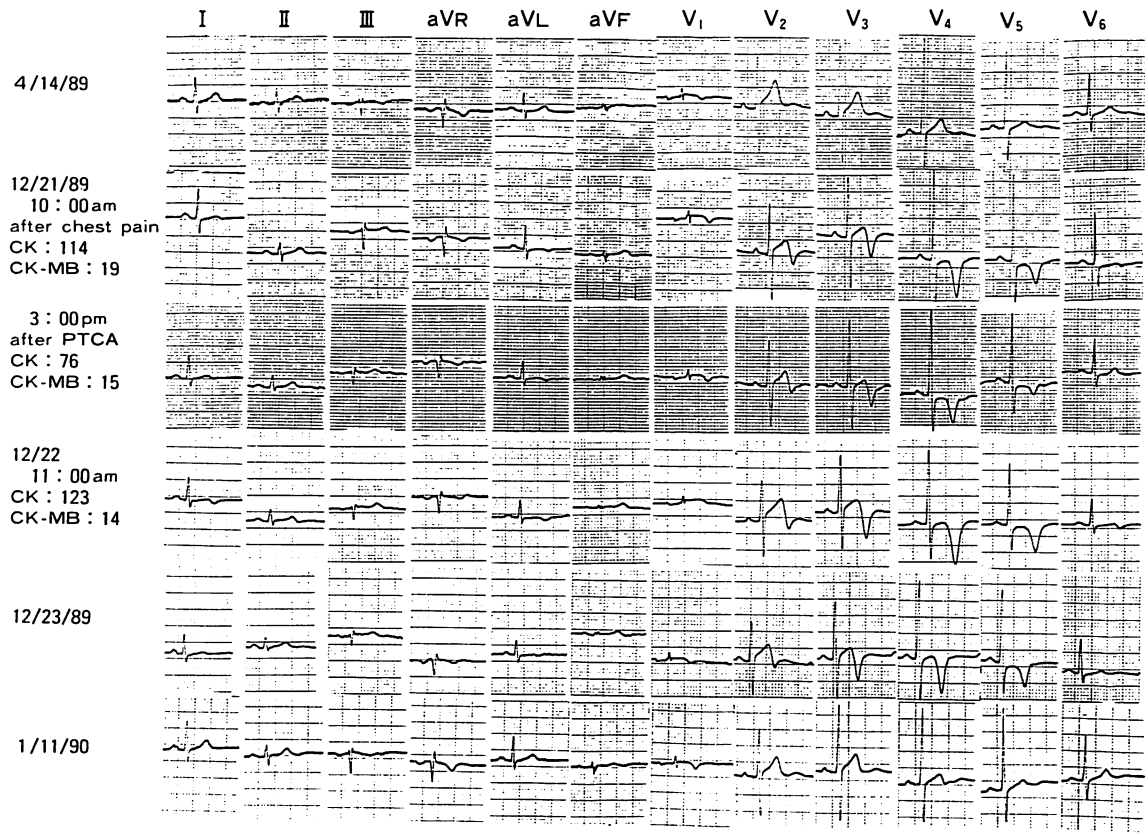

Fig. 4. Sequential ECG changes in Patient 8.

The ECG of 4/14/89, which was made by a private practice physician, was considered probably normal with a small s in V_1 and flat T waves in V_5 and V_6. The ECG at 10: 00 am on 12/21/89 was taken 30 min after the termination of severe chest pain in the outpatients' clinic, and already showed deep symmetrical negative T waves. These abnormalities disappeared on 1/11/90.

Abbreviations: CK=serum creatine kinase; CK-MB=the cardiac isoenzyme of creatine kinase; PTCA=percutaneous transluminal coronary angioplasty.

cardium after successful reperfusion by angioplasty in patients who had left ventricular wall motion abnormalities. Serial echocardiographic observations indicated that the wall motion abnormalities resolved 4 to 28 days after the acute episode. During the present investigation, we obtained very interesting and characteristic ECG findings, i.e., the development of prominent negative T waves in the left precordial leads with QT prolongation in patients with critical lesions in the left anterior descending artery. Previously, this unique ECG findings were interpreted as a "slight coronary attack"[18], "intramural myocardial infarction"[9], "myocardial injury"[10], or "subendocardial or non-Q wave infarction"[10,11]. The ECG of patient 1 (**Fig. 1**) is nearly identical with that published in a textbook[11].

The ECG findings in the acute ischemic syndrome vary, from ST elevation to ST depression, T wave inversion, and the development of abnormal q waves. Prognosis of patients with marked ST depression is reportedly very poor[9,19-21]. Multivessel disease with poor left

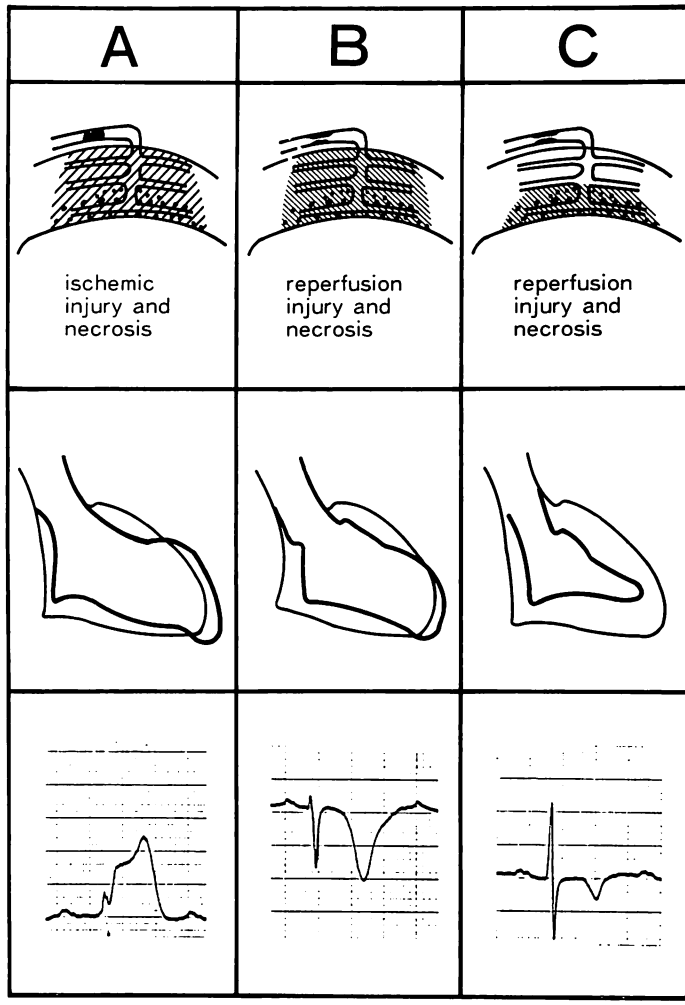

Fig. 5. Diagrams of coronary and myocardial anatomy (upper panels), **left ventricular wall motion abnormalities** (middle panels), **and ECG changes** (lower panels).

A: Acute coronary occlusion results in transmural ischemic injury (shown by the coarsely shaded area) and patchy myocardial necrosis (dark spots) which starts from the subendocardial area. Marked left ventricular wall motion abnormality is present, as is ST elevation with or without abnormal Q waves.

B: With the restoration of coronary perfusion, reperfusion injury can be expected when the reperfusion occurs after a prolonged period of ischemia, and is associated with wall motion abnormalities which are called myocardial stunning. We suspect that prominent negative T waves with QT prolongation might be the ECG manifestation of this phenomenon.

C: While some residual reperfusion injury is still present, the ventricle is restored to normal wall motion despite persistent ECG abnormalities.

ventricular function is frequently associated with this type of the acute ischemic syndrome[19-21]. Conversely, the prognosis of newly-developed T wave inversion is controversial. The classical papers of Papp & Smith[18] and Lown et al[9], as well as recent reports[19-22] have suggested a good prognosis, but other investigators[23-25] have reported unfavorable outcomes for these patients. They found that critical stenosis was present in the proximal portion of the left anterior descending artery in such cases[23-25].

The new development of T wave inversion is known to be associated with prolonged episodes of chest pain in patients with vasospastic angina and normal coronary arteries[26,27]. Hamashige et al[22] reported that about 50% (11/24) of their patients with unstable angina and T wave inversion had insignificant coronary artery disease. They also reported that this type of T wave inversion was observed after treadmill exercise testing in patients with left main trunk disease or triple vessel disease[28]. Although the etiology of the T wave inversion might be spasm or organic stenosis, this phenomenon seems to be related to reperfusion of the left anterior descending artery after severe and prolonged myocardial ischemia. Therefore, differences in the reported prognosis of these patients seem to be related to those in the underlying pathogenesis and in therapeutic approaches.

Pathologic, angiographic, and angioscopic studies have shown that endothelial injuries and subsequent coronary artery thrombus formation are the major causes of unstable angina and acute myocardial infarction[2,29]. An intracoronary thrombus may occlude the lumen completely at one moment, then be resolved spontaneously at the next moment by the function of intrinsic thrombolysis, apparently with fixed complete occlusion being the one extreme of this syndrome[29]. The development of prominent negative T waves with QT prolongation spontaneously or after percutaneous transluminal coronary angioplasty, as demonstrated in the present study, seems to represent reperfusion injury of the anterior wall. Marked QT prolongation might be related to intramyocardial electrolyte imbalance. The ST-T wave abnormalities persisted much longer than did regional wall motion abnormalities. Although the exact mechanisms of these electrical and mechanical abnormalities are unknown, a diagram presenting our hypothesis is shown in **Fig. 5**. It is well-known that regional wall contraction abnormalities occur 15 to 30 sec after total occlusion of a large epicardial artery[20,31] (**Fig. 5**, A). Reperfusion injury is spread throughout the myocardium with the restoration of perfusion after a prolonged period of total obstruction, and wall motion abnormalities would present with T wave inversion (**Fig. 5**, B). Restoration of contraction then occurs while injury is still present in the subendocardial region with some residual ECG abnormalities (**Fig. 5**, C), as myocardial necrosis begins from the subendocardial region and the healing process starts from the epicardial region[32,33].

The only method currently available for detecting successful reperfusion is coronary cineangiography, and there is no reliable bedside procedure for estimating the extent of reperfusion. If such means were to become available, they would be extremely helpful, especially for thrombolytic agents to be administered intravenously in hospitals where coronary cineangiography cannot currently be performed. Although 100% obstruction of the left anterior descending artery was demonstrated angiographically in only one patient in the present study, temporary obstruction was anticipated for the remaining patients from evidence of severe stenosis with or without thrombus formation, and the indicative ECG findings and clinical histories were obtained for all the patients. Therefore, we suspect that this characteristic ECG change is a clinical manifestation of early reperfusion of the occluded anterior descending artery.

要　約

急性虚血性症候群における巨大陰性 T 波の意義

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急性冠動脈閉塞に対する血行再建(reperfusion)および遷延する局所心筋収縮異常 (stunned myocardium) の臨床像を明らかにすべく，1989年1年間に，急性期に左前下降枝 (LAD) に冠動脈修復術 (PTCA) を施行し，左室造影にて局所壁運動異常を認め，1-2ヵ月後の再造影にて正常壁運動を示した LAD 一枝病変急性虚血性症候群患者8例の臨床像を検討した．

全例に CK または CK-MB の軽度上昇を認めたが，正常上限の2倍以上の上昇は2例のみであった．慢性期の左室造影では，拡張末期容量は急性期と変わらず，収縮末期容量の減少 ($68\pm16 \rightarrow 39\pm13$ m*l*, $p<0.001$)，駆出分画の正常化 ($51\pm6 \rightarrow 71\pm6\%$, $p<0.001$) を認め，心エコー図では，壁運動は1-3日目より改善し，4-28日で正常化した．ECG では全例に再灌流直後より V_4 を中心に巨大陰性 T 波 (1.6 ± 0.6 mV)，QT 延長 (0.56 ± 0.08 sec) を記録し，1-5日で最高に達し，T 波の逆転は，約1ないし3ヵ月間認められた．

重症虚血発作に伴う巨大陰性 T 波の出現は，一般には心内膜下，または心筋内梗塞と考えられている．この8例中有意(正常の2倍以上)の CK-MB の上昇を認めたのは2例のみであった．V_4 を中心とした巨大陰性 T 波と QT 延長の出現は，心筋梗塞の有無にかかわらず，左室前壁の再灌流による心筋障害 (reperfusion injury) と，それに伴う一過性収縮異常 (stunned myocardium) の存在を示し，この心電図変化は LAD 再灌流の非観血的指標と考えられる．

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