Clinical Characteristics of Coronary Artery Spasm: Electrocardiographic, Hemodynamic and Arteriographic Assessment

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We studied the clinical characteristics of 153 patients with angina pectoris associated with coronary artery spasm (CAS). The study was designed to investigate the relationship of CAS to ST segment deviation and to the site of fixed stenosis, and hemodynamic alteration during a spastic event.

Analysis of coronary arteriograms and multilead electrocardiograms obtained simultaneously from 170 events of CAS by the use of radioluscent carbon-fiber electrodes resulted in 58 events with ST elevation which were related to total occlusion of major coronary arteries due to CAS; another 54 events with ST depression, in which the affected coronary arteries demonstrated severe but incomplete occlusion, or total occlusion but were visualized via collateral vessels; and remaining 58 events without ST deviation showing mild occlusion. The results indicate a close correlation between magnitude of CAS and ST segment deviation.

CAS occurred at the site of pre-existing fixed stenosis including minor plaque defect in 133 patients and at apparently normal site in 20 patients. In the former group, only four patients had triple vessel disease, while 95 had nonsignificant fixed lesion. In the latter group, 10 patients had minor lesion distant from the site of CAS. Thus, CAS is closely related to fixed stenosis, which may have but a limited role as a cause of CAS.

Hemodynamic measurements during spastic events were obtained from 49 patients including 41 events with spasm of the left anterior descending artery (LAD) and 21 events with spasm of the right coronary artery (RCA). The onset of an increase in left ventricular (LV) filling pressure and a reduction in LV dP/dt preceded ST segment deviation in all events. The first hemodynamic variable manifested in the spastic event was the reduction of LV contraction dP/dt in the majority of patients. The increase of LV filling pressure was greater in LAD spasm than RCA spasm (11 ± 6 mmHg vs 7 ± 4 mmHg, $P < 0.0125$) and in events with ST elevation than with ST depression (11 ± 5 mmHg vs 6 ± 5 mmHg, $p < 0.001$). Right ventricular functional impairment was mild in most patients during CAS. The study indicates that mechanical impairment precedes electrical impairment during CAS and that LAD spasm with ST elevation represents the most severe LV dysfunction.

Key Words:

Coronary artery spasm
ST segment deviation
Fixed stenosis
Hemodynamic alteration

IT is now well-known that coronary artery spasm (CAS) has a significant association with the pathogenesis of coronary artery disease, especially ischemic attacks in patients with

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TABLE 1 PATIENT'S BASELINE CHARACTERISTICS AND METHODS OF PROVOCATION

<i>Transmural MI</i>	<i>No. of pts</i>	<i>Fixed stenosis</i>	<i>No. of pts</i>	<i>Methods of provocation</i>	<i>No. of events</i>
<i>Present</i>	4	<i>Nonsignificant</i>	115	<i>Spontaneous</i>	24
<i>Absent</i>	149	<i>Single vessel disease</i>	28	<i>Hyperventilation</i>	38
		<i>Double vessel disease</i>	6	<i>Cold pressor</i>	2
		<i>Triple vessel disease</i>	4	<i>Atrial pacing</i>	2
				<i>Ergonovine</i>	104

MI = myocardial infarction; pts = patients

variant angina pectoris. However, the causative mechanism of CAS and the clinical characteristics of patients with CAS are not well understood. Various factors may be related to the genesis and clinical characteristics of CAS. Among them, following factors are subjects of controversy such as 1) relation between extent of CAS and ST segment deviation, 2) relation of CAS to the sites of fixed stenosis, 3) hemodynamic alteration during the events of CAS. Our study is designed to clarify these three problems.

SUBJECT AND METHODS

Patient Population

The study group consisted of 174 patients with angina pectoris, 141 men and 33 women with a mean age of 53 years (range 34 to 69) admitted to National Cardiovascular Center for diagnostic and/or therapeutic purposes between 1979 and 1983. All patients were tentatively diagnosed as having angina associated with CAS based on an episode of chest pain at rest or an attack with transient ST segment elevation. Patients who were administered digitalis were excluded from the study. Four patients with previous transmural myocardial infarction were included since the site of ST segment elevation was distant from transmural infarct zone.

Study Protocol

After informed consent was obtained, cardiac catheterization was performed with patients in the fasting state. No routine premedication was administered. Following the left ventriculography, selective coronary arteriography were performed by the technique of either Judkins or Sones. For the purpose of simultaneous recording of 12-lead electrocardiogram (ECG) during the arteriography, all patients were monitored with

radioluscent carbon-fiber electrodes and connected cords. For an early detection of ST segment change, 12-lead ECG was recorded by a paper speed of 5 mm/sec using a 6 channel electrocardiograph throughout the procedure. After control arteriogram was obtained, provocation of CAS was tried by hyperventilation for 3 minutes, cold pressor test for 1 minute or atrial pacing at the rate of 150 beats/min for 3 minutes as previously reported¹⁻³. If CAS was not induced by these methods or spontaneous episode of CAS did not occur, intravenous ergonovine injection was given according to modified Schroeder's method⁴. A bolus of 0.05 mg of ergonovine was given as the first dose, followed by an additional 0.05 mg, 0.1 mg and 0.2 mg in the order when CAS was not documented. When ST segment deviation occurred, contrast medium was immediately injected to the target coronary artery. In 14 patients, more than two coronary arteriograms in one episode of CAS were obtained. After CAS was ascertained, nitroglycerin was administered sublingually or intravenously, then additional arteriograms with multiple projections were obtained.

In order to investigate hemodynamic alteration during the episodes of CAS, right and left ventricular pressures or pulmonary arterial and aortic pressures, or a combination of these were monitored in 64 patients. In the majority of patients, a catheter-tipped manometer was used to obtain the first derivatives of the both ventricular pressures. The data, together with plural ECG leads, were recorded continuously on a 14-channel magnetic tape recorder during the provocative testing.

Data Analysis

Standard 12-lead ECGs during the episodes of CAS were analyzed and ST segment deviation

TABLE II RELATIONSHIP BETWEEN MAGNITUDE OF CAS AND ST SEGMENT DEVIATION

ST changes	Involved vessels	No. of events	Maximal stenosis during CAS (%)					
			50	75	90	99	100 + GC	100 - GC
No deviation (n = 56)	RCA	31	18	8	4	1		
	LAD	18	7	7	4			
	LCX	7	1	3	2	1		
ST depression (n = 56)	RCA	20		2	10	7	1	
	LAD	31		7	8	16		
	LCX	1					1	
	LMT	4		2	1	1		
ST elevation (n = 58)	RCA	30						30
	LAD	27						27
	LCX	1						1
Total		170	26	29	29	26	2	58

CAS = coronary artery spasm; GC = good collaterals; + = present; - = absent; RCA = right coronary artery; LAD = left anterior descending artery; LCX = left circumflex artery; LMT = left main trunk.

was considered to be present if horizontal or downsloping ST depression of 0.05 mV or more, or ST elevation of 0.1 mV or more from the control tracing were documented. Coronary arteriograms were analyzed by at least three cardiologists. CAS was defined in this study according to following criteria: 1) appearance of more than 50% luminal narrowing from control in patients with apparently normal arteriograms, 2) an additional more than 25% narrowing from control in patients with 50% stenosis, 3) definite progression of luminal narrowing in patients with 75% or more stenosis, 4) disappearance of these luminal narrowing after nitroglycerin administration. When CAS appeared diffusely, the most severely narrowing site was assessed.

The severity of fixed stenosis of the major coronary arteries was appraised from the arteriograms obtained after nitroglycerin was given, and was classified as follows: 1) normal: no pre-existing lesion including minor plaque defect, 2) minor stenosis: less than 50% stenosis, 3) 50% stenosis: 50% or more but less than 75%, 4) 75% stenosis: 75% or more but less than 90%, 5) 90% stenosis: 90% or more but less than 100%, 6) 99% stenosis: 90% or more but less than 100%

and with delayed filling of the contrast medium beyond the stenotic site, 7) total occlusion. This classification was also applied to the magnitude of CAS. A coronary artery with 75% or more fixed stenosis was regarded as a diseased vessel. The quality of collateral circulation was graded as good if the distal segment of a coronary artery beyond the stenotic or occluded site was visualized densely and promptly, and as poor if opacification of the distal segment was faint and delayed, or absent.

Hemodynamic measurements, together with ST segment level and heart rate were played on a photographic recorder as a trend recording at a paper speed of 5 cm/sec. We analyzed the relationship between the onset of ST segment deviation and that of hemodynamic changes, and whether the magnitude of hemodynamic alteration arose from difference in involved vessels or direction of ST deviation. Analysis was limited to a point when contrast medium was injected to the involved coronary artery.

Statistical analysis was made using the Student's t test, and a p-value of less than 0.05 was considered significant.

Fig.1. Ergonovine-induced spasm of the right coronary artery.

A = Control; B = Three minutes after a bolus injection of 0.05 mg of ergonovine; C = One minute later; D = More one minute later, chest pain occurred; E = After nitroglycerin was given, spasm, ST segment changes and chest pain disappeared

RESULTS

One hundred and seventy events of CAS were documented in 153 patients, including 81 events in 69 patients with right coronary artery (RCA) spasm, 76 events in 71 patients with left anterior descending artery (LAD) spasm, nine events of the left circumflex artery (LCX) spasm and four events of left main trunk (LMT) spasm. In three patients, CAS occurred in more than two coronary arteries, and in 15 patients, two or more sequential events with different magnitude of CAS were observed in the same coronary artery. Baseline characteristics and number of patients with positive provocative testing were shown in Table I.

1. Relation between the magnitude of CAS and ST segment deviation

Eighty-one events of RCA spasm included 30

with ST segment elevation and 20 with ST segment depression at the inferior leads (II, III, aVF and occasionally V6), and 31 without ST deviation (Table II). There were 27 episodes of LAD spasm with ST elevation at the anterior precordial leads, 31 with ST depression at the lateral leads (I, aVL, V5-6) and 18 without ST deviation. The number of precordial leads with ST elevation varied in each episode and depended upon the site of LAD spasm. Nine events of LCX spasm consisted of one with ST elevation and one with ST depression at the lateral leads, and seven without ST deviation. All four events of LMT spasm induced ST depression at the inferolateral leads (II, III, aVF, V4-6). The relationship between the maximum luminal narrowing of the involved coronary arteries during the episodes of CAS and ST segment deviation was shown in Table II. Fifty-six events without ST segment deviation included 26 events with 50% transient stenosis

Fig.2. Spontaneous spasm of the left anterior descending coronary artery. A = Control; B = Initial phase of ST elevation; C = Maximal phase of ST elevation. No arteriogram was obtained; D = Late phase of ST elevation; E = After nitroglycerin was administered

due to CAS of the affected coronary arteries, 18 of 75% stenosis, 10 of 90% stenosis and 2 of 99% stenosis. Fifty-six events with ST segment depression included 11 events of 75% stenosis, 19 of 90% stenosis, 24 of 99% stenosis and two of total occlusion accompanied by good collateral flow beyond the occluded site. All 58 events with ST segment elevation were induced by total occlusion of the affected arteries. No good collateral vessel was visualized in these events. In five patients, anginal attacks with both ST segment depression and elevation in the same leads were elicited, and coronary arteriograms were obtained during both types of attack. One example was shown in Fig. 1. In this patient, RCA spasm due to ergonovine injection was documented to gradually progress from 90% stenosis without ST deviation, 99% stenosis with ST depression, then to total occlusion with ST elevation. In nine patients, coronary arteriograms at the different

phase of ST elevation were obtained during one episode of CAS (Fig. 2). All arteriograms at the initial phase of ST elevation showed total occlusion of the affected arteries, whereas they were patent at the recovery but still elevated phase.

2. Relation of CAS to the site of fixed stenosis

The relationship of CAS to the site of fixed stenosis can be easily assessed when the coronary artery narrowing associated with CAS was incomplete. However, it is often difficult to assess in the spastic events with total occlusion, because CAS may extend for considerable distance proximally from the site of fixed stenosis. Thus, a fixed stenosis, which existed at the proximal or mid portion of the affected artery, was considered to be related to CAS in the present study.

As shown in Table I, 38 patients had at least one diseased vessel. In 32 patients of them, CAS occurred in accord with the site of pre-existing

TABLE III RELATIONSHIP BETWEEN CAS AND SITE OF FIXED STENOSIS

Fixed stenosis	No. of pts	Site of CAS			Distance from site of CAS		
		SL	ML	NL	SL	ML	NL
Present							
SL ($\geq 75\%$)	38	32	6		16	10	12
ML ($< 75\%$)	105		95	10		65	40
Absent	10			10			10

CAS = coronary artery spasm; pts = patients; SL = significant lesion; ML = mild lesion; NL = normal

Fig.3. Hyperventilation-induced spasm of the right coronary artery.
A = Control; B = Five minutes after hyperventilation; C = More two minutes later, chest pain occurred; D = After nitroglycerin was given

fixed stenosis. In the remaining six patients, CAS appeared distant from the significant fixed stenosis, but at a site of another mild stenoses

less than 50% (Table III). In 95 patients of 115 who had no diseased vessel, CAS was documented at the pre-existing mild fixed stenosis.

Fig.4. Representative tracing from a patient with spontaneous spasm of the left anterior descending coronary artery.

Time from the onset of hemodynamic changes to the onset of ST elevation was expressed as T_1 to T_6 . In this patient, no conspicuous change of RV dp/dt was documented.

LV = left ventricular; P = pressure; RV = right ventricular; HR = heart rate

While in the remaining 20 patients, CAS appeared at an angiographically normal site, although control arteriogram showed mild narrowing in the majority of patients. In 55 patients of 95 who had mild lesions at the site of CAS, another mild lesion were found to be present in the same coronary artery or other one which was free from CAS. Moreover, in 10 patients of 20 whose

CAS occurred at the apparently normal site, mild organic lesions pre-existed in the same vessel or other one. Additionally, double CAS with different magnitude or extent was documented in some patients, as in one instance which is shown in Fig. 3. In this patient, the first CAS was not related to organic lesion, but the second one was related to the lesion.

Fig.5. Increase in left ventricular filling pressure.

RCA = spasm of the right coronary artery; LAD = spasm of the left anterior descending coronary artery

TABLE IV HEMODYNAMIC MEASUREMENT

Variables	Onset time (sec)	Maximal change			
		LAD spasm	RCA spasm	ST elevation	ST depression
Heart rate (beat/min)	$+49 \pm 13$	$+7 \pm 7$ (29) ^{oo}	-7 ± 9 (21)	$+3 \pm 12$ (43)	$+1 \pm 6$ (17)
LV pressure (mmHg)					
Systolic	-31 ± 23	-18 ± 13 (30) ^{oo}	-12 ± 14 (20)	-19 ± 14 (37) ^{**}	-6 ± 5 (13)
Filling	-40 ± 23	$+11 \pm 6$ (35) ^o	$+7 \pm 4$ (17)	$+11 \pm 5$ (37) ^{**}	$+6 \pm 3$ (15)
LV dp/dt (%)					
Contraction	-45 ± 34	-20 ± 12 (28)	-16 ± 20 (7)	-25 ± 11 (23) ^{**}	-7 ± 10 (12)
Relaxation	-35 ± 33	-23 ± 17 (28)	-23 ± 29 (7)	-31 ± 18 (23) ^{**}	-7 ± 11 (12)
RV pressure (mmHg)					
Systolic	-62 ± 65	$+7 \pm 5$ (30)	$+6 \pm 4$ (20)	$+7 \pm 5$ (39) [*]	$+4 \pm 4$ (11)
Diastolic	-71 ± 79	$+3 \pm 2$ (26)	$+4 \pm 1$ (10) ^o	$+4 \pm 2$ (28) [*]	$+2 \pm 2$ (8)
RV dp/dt (%)					
Contraction	-27 ± 19	-5 ± 13 (21)	-12 ± 26 (5)	-8 ± 18 (20)	-3 ± 5 (6)
Relaxation	—	-7 ± 11 (20)	-22 ± 26 (5)	-11 ± 17 (19)	-6 ± 10 (6)

LAD = left anterior descending artery; RCA = right coronary artery; LV = left ventricular; RV = right ventricular;

Values are mean $\pm$ SD. Time is referred to the onset of ST segment deviation, and it is negative when the hemodynamic change occurs before the onset of ST deviation. ^{oo}, ^o Significant difference (^{oo} = $p < 0.001$, ^o = $p < 0.05$) between LAD spasm and RCA spasm. ^{**}, ^{*} Significant difference (^{**} = $p < 0.001$, ^{*} = $p < 0.05$) between ST elevation and ST depression.

Maximal change of each variable is expressed as positive when it increases from control, and negative when it decreases.

Numbers in parenthesis represent available data.

3. Hemodynamic measurement during the event of CAS

Among 65 patients whose hemodynamic data were obtained, CAS with ST segment deviation occurred in 51. Hemodynamic measurements were analyzed in 29 patients (41 events) with LAD spasm and 20 (21 events) with RCA spasm. Fourteen patients who had neither ST segment deviation nor significant CAS during provocative testing were designated as a control group for hemodynamic study. Forty-one events of LAD spasm consisted of 29 with ST elevation and 12 with ST depression, and 21 events of RCA spasm included 16 with ST elevation and 5 with ST depression. Figure 4 illustrates a typical hemodynamic response to spontaneous LAD spasm.

Heart rate increased 7 ± 7 (mean $\pm$ SD) beats/min from control value in the episodes of LAD spasm, in contrast, it decreased 7 ± 9 beats/min in the episodes of RCA spasm ($p < 0.001$) after the onset of ST segment deviation (Table IV). The left ventricular (LV) filling pressure elevated 10 ± 5 mmHg from control in all spastic events. There was significant difference in an elevation of LV filling pressure between the episodes of LAD spasm and those of RCA spasm (11 ± 6 mmHg vs 7 ± 4 mmHg, $p < 0.01$), and between the event with ST segment elevation and depression (11 ± 5 mmHg vs 6 ± 3 mmHg, $p < 0.001$). The increase of LV filling pressure was greater in the episodes of LAD spasm with ST segment elevation than in those with ST depression, while no difference was shown between RCA spasm with ST elevation and depression (Fig. 5). The onset of the increase of LV filling pressure began with an interval of 40 ± 23 seconds (range 12 to 103) preceding to the onset of ST deviation in all episodes (Table IV). An early decrease of LV or aortic systolic pressure before the onset of ST deviation was observed in 40 events. The first derivatives (dP/dt) of LV pressure obtained from 36 events decreased in 28 during the spastic episodes, and 19 of them had LAD spasm with ST segment elevation. LV contraction dP/dt began to decrease before the onset of ST deviation with a interval of 45 ± 34 seconds and maximal decrease accounted for $19 \pm 14\%$ from control level. The onset of decrease in LV relaxation dP/dt preceded the onset of ST deviation with a interval of 39 ± 33 seconds, and the maximal decrease was $30 \pm 19\%$ from control. The magnitude of decrease in both LV dP/dt was greater in the episodes with ST elevation than in those with ST depression. Among LV hemo-

dynamic variables, the onset of decrease of contraction dP/dt was the first event in the majority of patients.

An early rise in right ventricular (RV) systolic pressure before the onset of ST segment deviation was observed in 40 events among 50 obtained from 38 patients, and maximal rise showed 7 ± 5 mmHg from control level in the events of ST elevation and 4 ± 4 mmHg in ST depression ($p < 0.015$). There was no difference in the rise of RV systolic pressure between the events with LAD spasm and RCA spasm. RV diastolic pressure also increased in those events which the increase of the systolic pressure was documented. The elevation of RV diastolic pressure was greater in the events with ST elevation and RCA spasm than ST depression and LAD spasm, respectively. In most events, the onset of increase in RV systolic pressure coincided with that of diastolic pressure. The onset of RV pressure rise preceded the beginning of reduction in LV contraction dP/dt in approximately half of the events. Appreciable trend recordings of RV dP/dt was gained from 22 events with LAD spasm and 6 events with RCA spasm, and reduction of RV dP/dt was documented in only four events and one event, respectively. No conspicuous hemodynamic changes were proven in 14 patients with no or mild CAS.

DISCUSSION

1. Correlation between magnitude of CAS and ST segment deviation

For precise appraisal of the relationship between the magnitude of CAS and ST segment deviation which induced by CAS, it is necessary to gain simultaneous multilead ECG and coronary arteriogram during a episode of ischemic attack, because evanescence of CAS is often rapid. As MacAlpin mentioned⁵, it was difficult to obtain these data simultaneously during coronary arteriography, because presetted electrodes often obstructed an image intensifier and caused inconvenience. The time lag was unavoidable between coronary arteriogram and ECG recording obtained by the usual manner. However, the use of carbon-fiber electrodes made it possible to obtain these two diagnostic tests simultaneously. As illustrated in Fig. 3, coronary arteriogram showed only 75% stenosis of the affected artery when ST elevation still remained. The observation that ST elevation may be induced even by incomplete occlusion of a coronary artery is

misleading unless coronary arteriogram during an early phase of anginal episode is available.

Our study demonstrated a definite correlation between the magnitude of CAS and the manifestation of ST segment deviation. In the first place, ST segment depression began to be manifest when acute luminal narrowing of the affected coronary artery approached over 75% stenosis. Secondly, 54 events with incomplete spastic occlusion of coronary artery, or 2 events of total occlusion of the artery but opacified by good collateral circulation induced ST depression at those leads where the vessel distributed. Thirdly, all 58 events with complete occlusion without good collateral circulation caused ST elevation. Thus, ST elevation indicates total occlusion of the affected artery.

Ekmekci et al. stated in their canine heart study that ligation of LAD caused ST elevation of the epicardial leads and ST depression by release from ligation.⁶ A clinical study using radioactive thallium scintigram suggested that anginal attack with ST elevation was caused by transmural myocardial ischemia, while attack with ST depression arose from nontransmural one.⁷ Our study indicates that ST segment deviation (depression or elevation) is closely associated with a reduction of coronary blood flow estimated by coronary arteriogram. However, some questions are still left regarding the relationship. First of all, a time delay is likely to be present between the onset of CAS and development of myocardial ischemia as a result of CAS.

As a clinical implication, it is evident that ST segment elevation, which disappears after nitroglycerin is given, is induced by total occlusion of the affected coronary artery. Then, if an anginal attack with ST elevation either at rest or on effort at a distance from transmural infarct zone is documented, CAS is considered to be associated with the attack, as transient total occlusion of the coronary artery could not have occurred but for CAS.

2. Relation of CAS to the site of fixed stenosis

Previous studies have indicated that the majority of western patients with angina associated with CAS have a significant fixed stenosis of a coronary artery⁷⁻⁹ when compared with Japanese,¹⁰⁻¹¹ and that CAS frequently appeared at and was superimposed upon a site of a pre-existing lesion.⁵ However, other literatures have indicated CAS which occurred on an apparently normal coronary artery.^{5,12} This disparity may

be based upon the fact that CAS documented in patients with a normal coronary artery are over-emphasized because such CAS is quite easy to demonstrate. In fact, patients with a normal coronary artery represent an unusual group.⁵ On the other hand, it is obvious that angina associated with CAS have a higher incidence in middle or older-aged people even in Japan. Thus, it is likely that CAS is closely related to development of atherosclerotic changes.

Another question may arise whether provoked CAS is compatible with the site of spontaneous CAS. In our study, most of CAS was provoked and ergonovine-induced CAS accounted for approximately 60% of total spastic events. However, ECG changes during provoked CAS were equated with those of spontaneous angina documented before cardiac catheterization. Therefore, the site of provoked CAS is likely to be equated with spontaneous one.

Our study shows that CAS is common in patients with a fixed stenosis including minor luminal irregularities, and suggests that CAS is closely related to sites of the fixed stenosis. However, it is also evident that CAS can occur in an angiographically normal coronary artery, and that CAS has low incidence in patients with severe triple vessel disease. Thus, atherosclerotic changes have a limited role as a causative mechanism of CAS.

3. Hemodynamic alteration during the episodes of CAS

In the episode of an anginal attack elicited by CAS, it has been demonstrated that there was an absence of hemodynamic alteration indicative of an increased myocardial oxygen demand such as increase of heart rate, systolic blood pressure or LV contraction dP/dt preceding the onset of ST segment deviation.^{1,7} Also it is well-known that LV dysfunction was manifested during the anginal attack, which was represented as impaired LV wall motion¹³⁻¹⁴ or increased LV diastolic pressure.⁷⁻¹⁵ However, few previous studies described the sequential process at the beginning of vasospastic myocardial ischemia in relation to the onset of LV dysfunction and ST segment deviation. According to Guazzi's study, the onset of ST elevation preceded that of an elevation of LV filling pressure.¹⁵ Maseri and his colleagues indicated that a reduction of LV dP/dt preceded the onset of ST segment changes immediately followed by an increase in LV diastolic pressure and a decrease of LV systolic pressure.⁷

Mizushige pointed out that LV asynergy documented by two-dimensional echocardiography together with an elevation of pulmonary artery diastolic pressure preceded the onset of ST elevation.¹⁴ Experimental animal studies have shown that regional contractile abnormalities or hemodynamic changes appeared within approximately 20 seconds after complete coronary occlusion.¹⁶⁻¹⁸ According to Battler et al., ST segment changes occurred 30 seconds after complete occlusion.¹⁸

Our study indicated that the onset of the elevation of LV filling pressure preceded that of ST deviation in all episodes of CAS with an average interval of 40.4 ± 23.0 seconds. The first altered hemodynamic variable was different between patients, but LV contraction dP/dt was the first variable in the majority of patients, and the onset of the reduction of contraction dP/dt was mostly in accord with that of the decrease of LV systolic pressure. Thus, it is evident that mechanical impairment precedes the electrical one in an episode of CAS. In addition, the magnitude of LV dysfunction is more severe in attacks with ST elevation and with LAD spasm than those with ST depression and RCA spasm. Clinically, anginal attack with ST elevation in the precordial leads represents the most severe LV dysfunction.

The presence of RV dysfunction, indicated by an elevation of RV diastolic pressure as in our study, is considered to reflect an extension of ischemia to the right ventricle. However, our study showed that RV or pulmonary artery systolic pressure began to increase before the onset of LV dysfunction in half of the patients, and RV contraction dP/dt did not change in most patients. Mechanism of this early elevation of RV or pulmonary artery systolic pressure is likely to be related to increased LV end-diastolic pressure,¹⁹ but may suggest that vasoconstriction of pulmonary vessels together with coronary artery occurs.

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