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Abstract

The correlation between the systolic time interval and abnormal contraction in ischemic myocardium was studied in anesthetized open-chest dogs. A strain-gauge was sutured on the surface of the left ventricular wall perfused by the left anterior descending coronary artery (LAD) for measuring segment-length. The left ventricular stroke volume decreased progressively after occlusion of LAD. The left ventricular ejection time (LVET) was progressively shortened in close correlation with the elongation of segment-length at the onset of isometric relaxation in 20 seconds after LAD occlusion when early systolic myocardial contraction and isometric contraction time (ICT) were not affected. ICT was gradually prolonged and closely related with the lengthening of the early systolic segment-length, while LVET recovered toward the control level in spite of further decrease in stroke volume. A close relationship was observed between ICT/LVET and stroke volume (gamma = 0.76, P less than 0.01). The results suggested the possibility that LVET was normalized even when the left ventricular function was impaired, and ICT/LVET ratio was the most sensitive index of LV dysfunction.

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CORRELATION OF SYSTOLIC TIME INTERVAL WITH ABNORMAL MYOCARDIAL CONTRACTION BY CORONARY OCCLUSION IN ANESTHETIZED OPEN-CHEST DOGS

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Abstract. The correlation between the systolic time interval and abnormal contraction in ischemic myocardium was studied in anesthetized open-chest dogs. A strain-gauge was sutured on the surface of the left ventricular wall perfused by the left anterior descending coronary artery (LAD) for measuring segment-length. The left ventricular stroke volume decreased progressively after occlusion of LAD. The left ventricular ejection time (LVET) was progressively shortened in close correlation with the elongation of segment-length at the onset of isometric relaxation in 20 seconds after LAD occlusion when early systolic myocardial contraction and isometric contraction time (ICT) were not affected. ICT was gradually prolonged and closely related with the lengthening of the early systolic segment-length, while LVET recovered toward the control level in spite of further decrease in stroke volume. A close relationship was observed between ICT/LVET and stroke volume ($\gamma=0.76$, $P<0.01$). The results suggested the possibility that LVET was normalized even when the left ventricular function was impaired, and ICT/LVET ratio was the most sensitive index of LV dysfunction.

Key words: STI, segmental function, myocardial ischemia

Recent myocardial studies have stressed measurements of systolic time interval (STI). The greatest importance of STI measurements to clinical application is that they afford quantitation of left ventricular performance which can be obtained with invasive methods. This permits repeated observations with little or no discomfort to patients.

As noted previously (1), left ventricular dysfunction characteristically produces lengthening of isometric contraction time and preejection time and a reduction in the left ventricular ejection time. The basic STI may be modified by rapid changes in hemodynamics as induced by exercise or drugs. Moreover,

early in acute myocardial infarction, dyssynergy in the ischemic area results in rapid alteration of the hemodynamics and may modify STI.

The purpose of the present study is to investigate the relationship between STI and local abnormal contractions in the left ventricular wall.

MATERIALS AND METHODS

Experiments were conducted in ten mongrel dogs, weighing 13-20 kg, anesthetized with intravenous sodium pentobarbital (25-30 mg/kg) and respirated with an intermittent positive pressure pump. The heart was exposed through left thoracotomy in the fifth intercostal space and supported with a pericardial cradle. An electromagnetic flow transducer was placed around the aortic root for measuring aortic blood flow. Two catheters (#8 French) were inserted into the aortic root and the left ventricle by retrograde catheterization. The pressure was monitored with a Nihon-Koden model MP-24T strain gauges placed at the mid-chest level. Left ventricular dP/dt was determined from the left ventricular pressure tracing by a RC differentiation circuit. A segment length gauge (Nihon-Koden medel RP-5) was placed with deep suture in parallel with the superficial muscle fibers on the area perfused by the left anterior descending coronary artery. Recordings were made using a direct visual oscillographic recorder at a paper speed of 100 mm/sec.

Following control observations, the left anterior descending coronary artery was completely occluded for one minute near its origin, and all variables were recorded continuously. Left ventricular ejection time (LVET) and the rapid phase of left ventricular ejection (LVERT) were derived from the aortic pressure tracing, and isometric contraction time (ICT) was the duration from onset

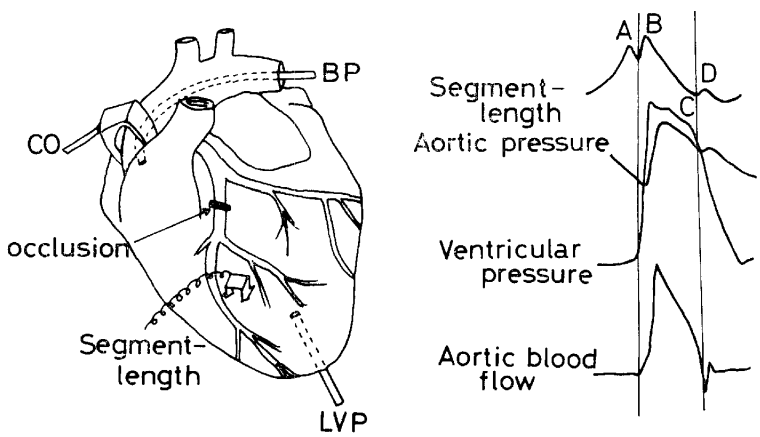

Fig. 1. Experimental model showing the attachment site of segment-length gauge (left) and the time relationship between segment-length tracing, aortic blood pressure, left ventricular pressure and cardiac output (right). CO, probe for measuring cardiac output; BP, catheter for measuring central aortic blood pressure; LVP, catheter for left ventricular pressure.

of rapid upstroke of the left ventricular pressure curve to the beginning of left ventricular ejection.

Segment-length curve was defined at four points according to Tawara (2), as illustrated in Fig. 1. Point A and point B were coincident with the end of diastole and the early phase of ejection, respectively. Point C was the concave point in the reduced ejection. Point D was a convex point at the end of the left ventricular ejection. The segment-length was represented as unit of weight (g) causing a length-change between the limbs of gauge.

The paired Student's t-test was used to compare the coronary preocclusion and after occlusion data.

RESULTS

The hemodynamic data in the control period and during coronary occlusion are presented in Table 1 and Table 2. A slight but significant fall in blood

TABLE 1. PREOCCLUSION DATA OF SEGMENT-LENGTH, STI AND HEMODYNAMICS

	Point B (g)	ICT (msec)	Point D (g)	LVET (msec)	LVERT (msec)	ICT/LVET	Aortic BP (mmHg) Systolic	Aortic BP (mmHg) Diastolic	SI (ml/min)	Min dP/dt (mmHg/sec)
Mean	0	53.8	2.5	207.9	57.2	0.268	112	70	1.16	1050
±SD	—	5.07	0.37	35.55	3.19	0.0623	7.2	5.6	0.206	430

ICT, isometric contraction time; LVET, left ventricular ejection time; LVERT, rapid phase of left ventricular ejection; BP, blood pressure; SI, strome index; SD, standard deviation.

TABLE 2. AORTIC BLOOD PRESSURE, STROKE INDEX AND MINdP/dt FOLLOWING CORONARY OCCLUSION

		Seconds 5	10	20	30	40	50	60
Aortic blood pressure (△mmHg)								
Systolic	Mean	−1.5	−2.3	−1.0	−1.5	−6.1[d]	−6.6[d]	−9.2[d]
	±SD	4.4	5.6	6.0	5.5	6.0	6.2	6.8
Diastolic	Mean	−2.3	−5.6[b]	−6.1[a]	−7.2[a]	−7.0[a]	−10.9[a]	−11.0[a]
	±SD	5.5	4.2	4.8	5.3	4.2	3.9	6.0
Stroke index (△%)	Mean	−8.1[c]	−18.0[a]	−22.7[a]	−24.0[a]	−24.8[a]	−25.9[a]	−26.7[a]
	±SD	6.8	5.1	5.8	9.2	9.1	9.9	11.4
Min dP/dt (△%)	Mean	−10.4[b]	−21.4[a]	−28.7[a]	−31.2[a]	−35.5[a]	−38.6[a]	−39.5[a]
	±SD	6.0	5.0	13.3	12.7	13.6	15.6	15.4

SD, standard deviation.

Significantly different from preocclusion values: *a* $P<0.001$ *b* $P<0.01$ *c* $P<0.02$ *d* $P<0.05$

pressure was seen within 10 seconds after coronary occlusion. Stroke volume rapidly decreased during the initial 20 seconds and then slowly decreased until 40 seconds to 73% of the control level without a change in heart rate. The maximal rate of pressure fall decreased rapidly and progressively immediately after occlusion.

LVET was progressively shortened from onset of total coronary artery occlusion and reached the lowest level at approximately 10 seconds after occlusion. From 40 seconds after occlusion, LVET gradually increased toward the control level despite the occlusion. The rapid phase of left ventricular ejection (LVERT) was slowly prolonged from 20 seconds after coronary occlusion. Therefore, shortening in LVET resulted from a decrease in the reducing phase of ejection, and recovery followed from prolongation of LVERT. Isometric contraction time (ICT) was progressively prolonged from 30 seconds and reached 114.8% of the control level at 60 seconds after occlusion. The ratio of ICT to LVET (ICT/LVET) increased in association with a fall in stroke volume.

The coronary occlusion increased the length of point D immediately. Paradoxical expansion at the end of systole was observed at approximately 20 seconds after occlusion and progressively increased in the following 40 seconds. Lengthening of point B occurred at 20 seconds after occlusion and increased with myo-

TABLE 3. CHANGES IN SEGMENT-LENGTH AND SYSTOLIC TIME INTERVAL AFTER OCCLUSION

		Seconds						
		5	10	20	30	40	50	60
Point B	Mean	+0.06	+0.01	−0.24	−0.48^{b}	−0.77^{a}	−1.22^{a}	−1.65^{a}
(△g)	±SD	0.09	0.15	0.20	0.37	0.47	0.60	0.62
ICT	Mean	−0.1	−1.4	−0.5	+4.5^{a}	+7.8^{a}	+10.4^{a}	+14.8^{a}
(△%)	±SD	1.4	2.2	1.8	2.9	3.4	4.1	3.7
Point D	Mean	−1.03^{a}	−1.89^{a}	−2.84^{a}	−3.35^{a}	−3.77^{a}	−4.40^{a}	−4.60^{a}
(△g)	±SD	0.48	0.67	0.71	0.79	0.90	1.16	1.21
LVET	Mean	−7.2^{a}	−18.4^{a}	−19.5^{a}	−18.5^{a}	−14.6^{a}	−10.1^{a}	−2.5
(△g)	±SD	3.0	2.0	2.2	4.0	4.2	4.4	4.7
LVERT	Mean	−0.6	+1.4	+3.9	+5.7^{c}	+10.0^{b}	+13.2^{a}	+16.2^{a}
(△g)	±SD	1.3	2.3	4.1	5.9	6.3	4.7	3.6
ICT/LVET	Mean	+8.3^{b}	+21.1^{a}	+27.4^{a}	+32.0^{a}	+29.4^{a}	+28.8^{a}	+30.0^{a}
(△%)	±SD	3.7	4.8	5.0	8.6	6.7	8.1	7.2

ICT, isometric contraction time; LVET, left ventricular ejection time; LVERT, rapid phase of left ventricular ejection; SD, standard deviation.
Significantly different from preocclusion values: *a* $P<0.001$, *b* $P<0.01$ *c* $P<0.02$ *d* $P<0.05$

cardial ischemia. Therefore, at 20–30 seconds after occlusion point B and point D formed two peaks of systolic bulge and tended to fuse with a lapse of time (Table 3).

Relationship of segment-length to STI and hemodynamics. No significant relationship was observed between LVET and point D during occlusion. However, as illustrated in Fig. 2, LVET decreased in close relationship to lengthening of

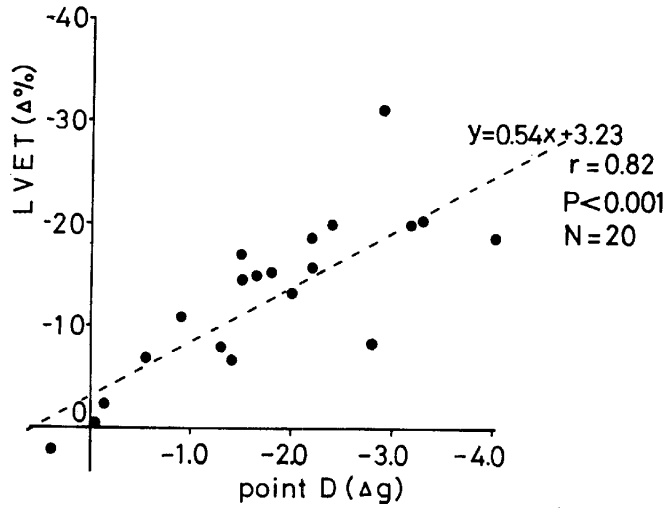

Fig. 2. Relationship between LVET and point D at five to ten seconds after occlusion.

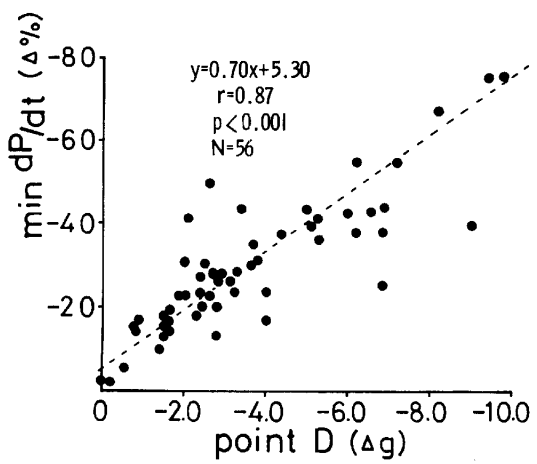

Fig. 3. Relationship between the maximal rate of pressure fall (min dp/dt) and point D after coronary occlusion.

point D in the initial 10 seconds when point B was still constant ($\gamma = 0.82$, $P < 0.001$). The decrease in maximal rate of pressure fall (min dP/dt) was also correlated closely with lengthening of point D (Fig. 3) and resulted in a correlation with reduction of LVET (Fig. 4).

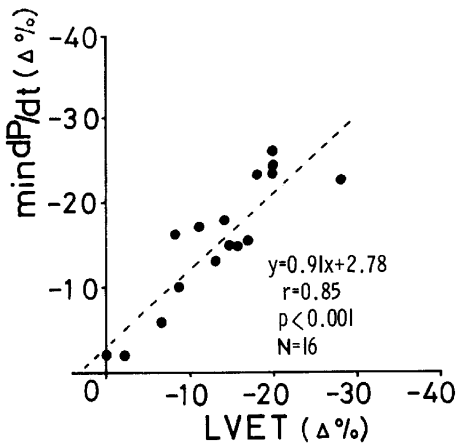

Fig. 4. Relationship between maximal rate of pressure fall (min dp/dt) and LVET after coronary occlusion.

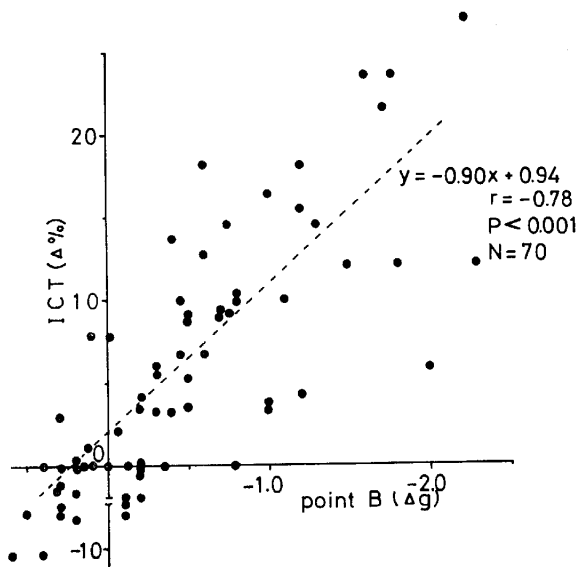

Fig. 5. Relationship between ICT and point B.

The recovery of LVET observed after 40 seconds of occlusion correlated well with lengthening of point B in the ischemic area. These findings suggested that the shortening of LVET and a decrease in min dP/dt were derived from end-systolic myocardial lengthening (point D) in mild ischemia, and the development of ischemia caused early systolic myocardial expansion, resulting in restoration of LVET. ICT changed with close relationship to changes in point B, as represented in Fig. 5.

Fig. 6 schematically illustrates the relationship between the systolic time interval and segmental myocardial length in the ischemic area. When point D on the segment-length curve was lengthened in mild ischemia, left ventricular pressure decreased at the end of systole, and the pattern of the left ventricular pressure was deformed. This resulted in a reduction of LVET (middle panel). Following this, when the abnormal contraction in the ischemic area reached the early phase of systole due to increased ischemia, left ventricular pressure was lower throughout systole with a restoration of pattern in left ventricular pressure tracing, and the resultant recovery of LVET and increase in ICT.

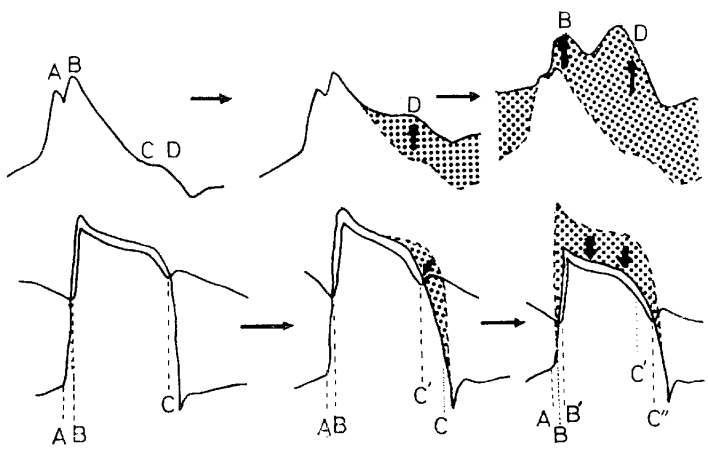

Fig. 6. Schematic illustration of relationship between systolic time interval, left ventricular pressure and segment-length in ischemic area.

In STI measured, changes in ICT/LVET had the closest relationship with changes in the left ventricular stroke volume, as illustrated in Fig. 7.

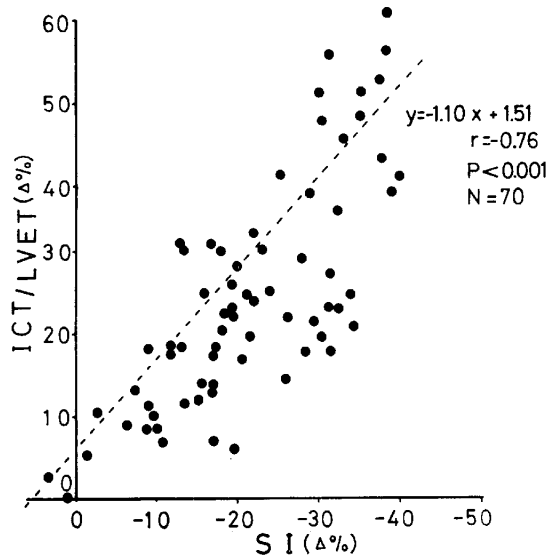

Fig. 7. Linear regression analysis between ICT/LVET and stroke index after coronary occlusion, demonstrating a significant correlation.

DISCUSSION

Since the investigation of Wiggers (3, 4) and Katz (5) in the 1920's, many studies have been reported on the left ventricular systolic time interval. Previous studies attempting to validate LVET and ICT with non-invasive methods in man have demonstrated the accuracy of STI measurement by the non-invasive technique in comparison with recordings obtained simultaneously from catheters in proximal aorta (6–8). The clinical significance of STI, however, could not be accepted in cases of rapidly changing abnormal contraction in local myocardium. Some investigators (9) reported in a study of myocardial infarction that STI was of little use for estimating myocardial function in individual cases, though a group of myocardial infarction patients could be statistically discriminated from a normal group. This is in part due to the fact few studies are available on the relationship between changes in hemodynamics, regional myocardial contraction and STI.

Changes reflecting other hemodynamic alterations in duration of systolic phase were clearly described by Frank (10). Recent studies have been designed to more completely separate the independent contributions of heart rate, stroke volume and aortic pressure to duration of STI. Remington *et al.* (11) revealed that changes in LVET were accompanied by consistent changes in the left ventricular stroke volume, and inverse changes in peripheral resistance and aortic

pressure. Braunwald *et al.* (12) reported that there was a close quantitative relationship between left ventricular stroke volume and LVET in isolated dog heart preparations. In the study on isolated dog hearts by Wallace *et al.* (13) changes in left ventricular preload were associated by consistent changes in LVET and inverse changes in ICT, when heart rate and aortic pressure were held constant. When heart rate and stroke volume were constant, increases in aortic pressure were accompanied by an increase in ICT, and LVET decreased. In addition, according to Spann and his coworkers (14), reduction in ventricular contractility was accompanied quantitatively by prolongation of ICT under constant aortic pressure, stroke volume and heart rate, because longer time was required to elevate the left ventricular pressure to a diastolic aortic pressure. When heart rate was increased while mean aortic pressure and stroke volume were maintained at constant levels, there was a concomitant diminution in ICT, LVET and mechanical systolic duration (13).

In the present study, however, STI results were represented as actual measurements without correction by heart rate, since heart rate did not change significantly during the course of the experiments.

Our findings confirmed notches on the segment-length curve of the left ventricular wall just after onset of left ventricular ejection (point B) and at onset of isometric relaxation (point D). These findings agree with the observations of Tawara (2) and Hisada *et al.* (15, 16). The obstruction of the coronary artery immediately caused a progressive decrease in min dP/dt associated with elongation of the isometric relaxation segment-length in the ischemic myocardium. This resulted in the initial shortening of LVET. LVET rose toward the preocclusion level in spite of progression of myocardial ischemia which caused lengthening of point B. Therefore, as described above, the mechanisms of LVET and ICT change in acutely-induced myocardial ischemia could be understood from abnormal motions in the ischemic myocardial segment. While Barrere *et al.* (17) suggested the relationship between the release of catecholamines from the ischemic myocardium and shortening LVET, catecholamines would not play an important part in shortened LVET in our previous experiment on dogs pretreated with propranolol (18).

Abnormal contraction was produced or increased by exercise (19, 20) and decreased by nitroglycerin (21) in the ischemic myocardium. According to Pouget *et al.* (22) and Murayama *at al.* (23) LVET was significantly prolonged by exercise in ischemic heart disease contrary to normal control subjects. In addition, nitroglycerin inhibited an increase in LVET induced by exercise. These results agree with our present findings if LVET was decreased by end-systolic abnormal contraction at rest and was increased by exercise with progression of myocardial ischemia which caused abnormal contraction in early systole.

Peak negative dP/dt (min dP/dt) always occurred at the onset of isovolumic relaxation (24) and changed at first after coronary occlusion (25). Our results also agree with these findings. The decrease in mindP/dt seen after coronary occlusion resulted from a lengthening of point D, and therefore had a close concomitant relationship with shortened LVET.

Point B was recorded concomitant with LVmaxdP/dt (2). ICT change in relation to alteration of point B was consistent with the findings of Beck and Schrire (26), who confirmed the reciprocal relationship between ICT and LVmaxdP/dt. While it had usually been believed that ICT was more sensitive than LVET for detecting myocardial ischemia (27, 28), the present study suggested that if the myocardial ischemia was mild as only to cause abnormal contraction in the end-systole, ICT showed no change but LVET was shortened. Furthermore, the possibility is present that the prolonged ICT would indicate a more severe ischemia than normal ICT with shortened LVET.

In the present study the ICT/LVET ratio was the most suitable index for detecting abnormal performance of the left ventricle in acute myocardial ischemia. The index increased with inverse relationship to changes in stroke volume. Mild ischemia in the intial 20 seconds after coronary occlusion increased ICT/LVET ratio due to reduction in LVET, and moderate or severe ischemia rose the ratio due to marked increase in ICT in spite of a restoration in LVET.

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