THE QRS COMPLEX OF THE STANDARD 12-LEAD ELECTROCARDIOGRAM IN SEPTAL MYOCARDIAL INFARCTION

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Interventricular septal involvement in myocardial infarction is suggested by the findings of a QS deflection in lead V_1 and/or absence of the Q wave in leads I and V_6 , using the standard 12-lead electrocardiogram (ECG). However, these findings were not sufficiently established for the criteria of septal infarction, because several factors including anatomic position of the heart, changes of intraventricular conduction, and condition of the lung would affect the QRS complex in the leads. In this study, we analyzed the ECGs of anterior myocardial infarction with (group A) and without (group B) involvement of the first septal coronary artery. The R wave in V_1 was absent in 64.7% of group A and 60.0% of group B. The Q wave was absent in 35.3% in lead I and 64.7% in lead V_6 of group A, while in group B the Q wave in leads I and V_6 were not observed in 60.0% and 40.0%, respectively. The prevalence rates of the R wave and the Q wave in these leads were not significantly different between the two groups. Left ventriculography revealed that the prevalence rate of the R wave in V_1 , and the Q wave in I and V_6 was not significantly different, regardless of the presence or absence of impaired septal motion. Experimental study in anesthetized dogs confirmed the difficulty in clarifying acute septal ischemia by changes in the QRS complex. These results indicate that septal myocardial infarction is hardly detectable with changes in the QRS complex of the standard 12-lead electrocardiogram.

THEORETICAL considerations¹ of left ventricular function as well as laboratory experiments² support the contention that left ventricular failure is related to the amount of left ventricular damage. Swan et al¹ reported that when the infarcted segment is assumed to be totally noncontractile and non distensible, the resulting ejection fraction (EF) is a linear function of the remaining quantity of normally contracting myo-

cardium. With an infarct of 10%, EF would fall from 0.67 to 0.60 and with a 40% infarct to 0.40. Alonso et al³ observed a good correlation of cardiogenic shock in myocardial infarction with total left ventricular damage. The first septal branch of the left anterior descending coronary artery is the largest vessel supplying blood to the interventricular septum, and occlusion of the vessel causes myocardial cell damage in the relatively extensive area of the septum. Septal myocardial infarction usually associates with infarction of left ventricular anterior or posterior free wall. Association of septal infarction with infarction of left ventricular free wall results in

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more extensive loss of left ventricular musculature. Thus, presence or absence of the first septal branch involvement are important for hemodynamic impairment and prognosis of the patients.

Clinically, an electrocardiographic examination is most extensively and frequently used for detecting myocardial infarction, and the diagnostic criteria of the electrocardiogram (ECG) for septal involvement include a QS deflection in leads V_1 and V_2 , and absence of the q wave in leads V_5 and V_6 . However, there is relatively little evidence concerning ECG findings in septal infarction. In this study, we attempted to reveal characteristic ECG readings in septal infarction. For this purpose, the ECGs of myocardial infarction involving the first septal branch of the left anterior descending coronary artery (LAD) were analyzed and compared with the ECGs of myocardial infarction due to coronary obstruction distal to the first septal branch of the LAD.

METHODS

Clinical Study

The subjects were sequentially selected from our files of patients with recent or old myocardial infarction who had coronary arteriograms that fulfilled the criteria necessary for the study. Selective coronary arteriography was carried out by the Judkins technique. The extent of artery narrowing was estimated from the multiprojection views of the coronary arteries, and the narrowing was expressed as a percent reduction of luminal diameter. Left ventriculography (LVG) was performed and recorded in RAO-60° projections using biplane cineradiography. The left ventricular wall was divided into 7 segments, and abnormal wall motion was expressed according to the classification⁵ of the American Heart Association. Absent and dyskinetic contractility was used as impaired wall motion in this study. The criteria for the patient selection were as follows: 1, a history of typical chest pain; 2, electrocardiographic changes indicative of acute myocardial infarction within 2 years before study; 3, characteristic serial elevation of serum enzymes (CK, GOT and LDH) within 2 years before study; 4, a single-vessel disease of either the segment 6 or the segment 7 in the left anterior descending coronary artery according to AHA classification⁵ (without coronary obstruction equal to or greater than 50% reduction of luminal diameter in any other segments of coronary

arteries); and 5, the reduction of luminal diameter in the segment 6 or segment 7 was greater than 90%. Standard 12-lead electrocardiograms were recorded at a paper speed of 25 mm/sec or 50 mm/sec within a week before the coronary arteriographic study in the patients with old myocardial infarction. In the patients with recent myocardial infarction in which the onset of infarction ranged between 2 and 6 weeks before study, we used the electrocardiograms taken within 2 days before coronary arteriography. The electrocardiograms were analyzed by 2 or more observers, and records showing intraventricular conduction disturbance including right or left bundle branch block were excluded from analysis. Patients with complications of valvular heart disease, cardiomyopathy or congenital heart disease, and patients taking digitalis, β -blocking agents or antiarrhythmic agents except lidocaine were excluded.

Of 35 patients studied, 20 had narrowing of segment 6 (group A), and 15 had lesions of segment 7 (group B). Group A consisted of 14 males and 6 females, and age ranged from 31 to 73 years (mean 58 years), while group B was 14 males and 1 female, and mean age was 57 years with range of 41 to 69 years.

The left ventriculography was recorded in 30 out of 35 subjects. LVG of the other 5 was not recorded due to markedly elevated left ventricular end-diastolic pressure. The 30 patients who received LVG were rearranged into two groups according to involvement of the interventricular septum; with abnormal wall motion of the interventricular septum (group C), and with normal motion of the septum (group D).

Experimental Study

Eighteen dogs of either sex weighing 10–15 kg were anesthetized with pentobarbital sodium (25–30 mg/kg, iv), and were ventilated with a mixture of oxygen and room air at a rate that maintained arterial oxygen and carbon dioxide tensions (PO₂, PCO₂) and pH in the physiological ranges. The right femoral artery was cannulated for monitoring arterial blood pressure. The chest was opened in the left 5th intercostal space and a pericardial cradle was constructed. The first septal branch of the left coronary artery and the anterior descending coronary artery (LAD) just distal to the first diagonal branch were dissected free and 6G silk strings were placed around the vessels for occluding the arteries. Six unipolar electrodes were used for recording intramyocardial

Fig.1. Electrocardiograms of patients with occluded first septal coronary artery.

Left hand 2 columns (case 1) show the normal Q wave in leads I and V_6 , and the R wave in lead V_1 . In the right hand columns (case 2) the Q wave in leads I and V_6 and the R wave in lead V_1 are absent.

Fig.2. The standard 12-lead electrocardiograms of anterior myocardial infarction with the normal first septal coronary artery.

The ECG in the left hand columns (case 18) has the initial, small Q wave in leads I and V_6 , and the R wave in lead V_1 . The Q wave in leads I and V_6 and the R wave in lead V_1 are not observed in the right hand 2 columns (case 19).

cardial or cardiac surface electrocardiograms. Two needle type intramyocardial electrodes which can record electrogram on the tip were inserted into the interventricular septum to a depth of 1.5 cm via the surface of the anterior interventricular sulcus: one was applied in the basal third and the other was in the middle third of the septum. Two electrodes were sutured onto the anterior free wall of the left ventricle perfused by the LAD, and another 2 were sutured onto the right ventricular free wall to monitor cardiac surface electrograms. The chest was then closed with sutures and the 2 strings for coronary occlusion were led extracorporally through small polyethylene tubes separately. Thirty minutes after the chest was closed, either the first septal coronary branch or the LAD was occluded by pulling the silk string in order to produce acute myocardial ischemia of the interventricular septum or the anterior free wall of the left ventricle, respectively. Intramyocardial and cardiac surface electrograms were recorded with a standard 12-lead body surface electro-

cardiogram at a paper speed of 50 mm/sec before, 20 min, 60 min and 90 min after coronary occlusions.

After completion of the experiment, 0.1% Evans blue dye solution injected into the coronary artery just distal to the occlusion delineated the myocardium under study. The dog was then killed with an intracardiac injection of saturated KCl solution, and the dyestained myocardium was excised and weighed. Thirteen dogs were subjects for occlusion of the first septal branch and 5 dogs for LAD occlusion.

Data Analysis

Student's t-test was used for comparison of group data of RR interval, PR interval, QRS duration and voltage of R and S waves in leads I, aVF, and V_1 through V_6 between group A and group B. Comparison of rate of disappearance of normal small Q waves in leads I, V_5 and V_6 , and R wave in lead V_1 was performed using Fisher's exact test for comparison of 2 rates, and prevalence rate of impaired segment 6 (the interven-

TABLE I ELECTROCARDIOGRAPHIC DATA OF THE PATIENTS

Patient group	No of cases		PQ interval (sec)	QRS duration (sec)	Voltage (mV)						
					QI	QV ₆	RI	RaVF	RV ₁	RV ₂	
Group A (seg. 6)	17	mean	0.15	0.09	0.052	0.012	0.386	0.497	0.026	0.024	
		SD	0.01	0.01	0.066	0.020	0.321	0.366	0.036	0.022	
Group B (seg. 7)	15	mean	0.17	0.08	0.024	0.008	0.683	0.536	0.070	0.070	
		SD	0.02	0.01	0.035	0.013	0.358	0.347	0.147	0.151	

Voltage (mV)											
RV ₃	RV ₄	RV ₅	RV ₆	SI	SaVF	SV ₁	SV ₂	SV ₃	SV ₄	SV ₅	SV ₆
0.013	0.252	0.527	0.667	0.091	0.214	0.787	0.024	1.420	0.909	0.423	0.208
0.434	0.434	0.628	0.482	0.144	0.176	0.405	0.042	0.825	0.573	0.350	0.194
0.100	0.653	1.100	1.100	0.100	0.150	1.280	1.980	1.670	1.160	0.513	0.163
0.169	0.808	1.000	0.722	0.119	0.179	0.599	0.946	1.040	0.699	0.376	0.206

Significantly different between 2 groups at the level of $**p < 0.01$ and $*p < 0.05$.

tricular septum) in LVG was also compared with Fisher's exact test. The difference was considered significant when $p < 0.05$.

RESULTS

Clinical Study

Of 35 patients, 3 were excluded from analysis because their electrocardiograms showed complete right bundle branch block; all 3 patients were of group A. The remaining 32 patients (17 of group A and 15 of group B) were used for following analysis. With coronary arteriograms the first diagonal branch (segment 9) of the anterior descending coronary artery was shown to branch distal to the coronary lesion in 16 out of 17 patients in group A, while group B 14 patients had an almost normal branch of the artery. All patients studied LVG demonstrated abnormal wall motion at least 1 segment of the left ventricular wall. The interventricular septum (segment 6) showed abnormal contraction in 12 patients of group A and 3 of group B. The abnormal septal motion appeared more frequently in group A than in group B ($p < 0.01$).

Figure 1 shows representative electrocardiographic records of patients in group A, and electrocardiograms of patients in group B are presented in Fig. 2. The two left hand columns of both Fig. 1 (case 1) and Fig. 2 (case 18) showed a normal Q wave in leads I and V₆, and the right

hand columns of the figures (case 2 and case 19) had no Q waves in the leads. Small R waves are present in the left hand columns of 2 figures, and are absent in the right.

The mean values of electrocardiographic data from the patients are summarized in Table I. All of 32 patients studied had abnormal Q wave (QR or QS pattern) in at least 1 lead of V₂ through V₄, suggesting myocardial infarction of the left ventricular anterior wall. The PR interval was slightly shorter in group A, while the QRS duration of group A patients was slightly longer compared to that of group B. No patients had PR interval longer than 0.2 seconds, 2nd degree A-V block or complete A-V block. Mean magnitude of the R wave in the leads aVF and V₁ through V₆ and S wave in leads I, aVF and V₂ through V₆ was not significantly different between 2 groups. However, the magnitude of R wave in lead I and of S wave in lead V₁ was significantly small in group A compared to the wave in group B ($p < 0.05$). The abnormal Q wave in lead I appeared in 4 cases of group A, whereas in group B no patients showed the Q wave in this lead, suggesting more extensive infarct area to the left in group A. Six patients of group A and 7 patients of group B had the apparent R wave in lead V₁. There was no significant difference in prevalence rate of the R wave in lead V₁ between 2 groups (Table II). A small initial Q wave in leads I and V₆ dis-

TABLE II PREVALENCE OF THE ELECTROCARDIOGRAPHIC FINDINGS

	No. of patients		P
	group A (n = 17)	group B (n = 15)	
QI absent	6	9	ns
QV ₆ absent	11	6	ns
RV ₁ absent	11	8	ns
RV ₂ absent	11	9	ns
RV ₅ < 0.1 mV	8	2	< 0.05
RV ₆ < 0.1 mV	1	0	ns

Group A consists of patients with coronary lesion proximal to the first septal branch, and group B is a patient group with the lesion distal to the vessel.
ns = not significant.

TABLE III ELECTROCARDIOGRAPHIC FINDINGS IN PATIENTS WITH AND WITHOUT SEPTAL INVOLVEMENT

No of patients			P
group C (n = 15)	group D (n = 15)		
Lead I			
normal Q wave	7	5	ns
abnormal Q wave	2	1	ns
Q wave absent	6	9	ns
Lead V ₁			
normal R wave	6	7	ns
QS or QR pattern	9	8	ns
Lead V ₆			
normal Q wave	6	6	ns
abnormal Q wave	0	3	ns
Q wave absent	9	6	ns

Group C consists of patients having abnormal motion, and group D shows normal motion of the septum.
ns = not significant.

appeared in 6 and 11 cases in group A and B respectively, and, in group B, 9 patients in lead I and 6 in lead V₆ did not show the small Q wave. No significant difference in the prevalence rate of the Q wave in lead I was observed between the two groups, as was also the case in lead V₆ (Table II). A small Q wave in lead aVL behaved in a similar way to those in lead I and V₆: no significant prevalence rate of the Q wave was observed between two groups.

As described in the paragraph of "methods", 30 patients out of all 35 subjects used in this study received LVG, and were rearranged into two groups, group C and group D, according to

findings of the ventriculograms. Group C consisted of 15 patients showing abnormal wall motion of the interventricular septum, and group D consisted of the remaining 15 patients with normal motion of the septum. There was no significant prevalence rate of the normal, small Q waves in leads I and V₆ regardless of involvement of the interventricular septum. Six patients in group C and 7 in group D had the normal R wave in lead V₁, while a QS or QR pattern in this lead was observed in 9 patients in group C and 8 in group D (Table III).

Experimental Study

The first septal branch was occluded in 13 dogs and the LAD in 5 dogs. Advanced AV block developed in 5 dogs and intraventricular conduction disturbance in 4 dogs 70–90 min after the coronary occlusion. Thus, the data of the 9 dogs obtained 90 min after the occlusion were excluded from analysis. Twenty minutes after the occlusion of the septal branch, the electrogram of the interventricular septum demonstrated marked elevation of the ST segment and abnormal Q waves, confirming the acute septal ischemia in the interventricular septum. Concomitant slight elevation of ST segment was observed in the electrograms derived from the right and the left ventricular free walls. The ST segment of body surface 12-lead electrocardiogram was depressed in leads II, III and aVF from -0.24 ± 0.16 mV, -0.22 ± 0.11 mV and -0.24 ± 0.11 mV to -0.35 ± 0.25 mV, -0.30 ± 0.19 mV and -0.36 ± 0.20 mV, respectively. The ST depression continued for a further 70 min. However, neither abnormal Q waves nor ST elevation appeared in any leads of the standard 12-lead electrocardiogram throughout the experiment: the R wave in lead V_1 and the Q wave in leads I and V_6 remained unchanged.

The occlusion of the LAD caused a ST segment elevation in leads V_1 – V_5 and a decrease in R wave magnitude in lead V_2 to V_5 after 20 min. Ninety minutes after the occlusion abnormal Q waves were observed in V_2 through V_4 . The R wave in lead V_1 at 90 min after the occlusion slightly decreased from 0.52 ± 0.18 mV to 0.46 ± 0.20 mV on average, while abnormal Q waves were not observed in this lead.

DISCUSSION

The present study on electrocardiograms (ECG) showed no difference in Q waves in leads I and V_6 , and R waves in lead V_1 in the patients with myocardial infarction either with or without involvement of the interventricular septum. Smaller magnitude of R wave in lead I and S wave in lead V_1 was only but small difference between 2 groups. The results suggest that myocardial infarction of the septum was hard to detect from changes of depolarization in the standard 12-lead ECG. The experimental study performed in this investigation confirmed that acute ischemia of the interventricular septum did not cause significant changes of the QRS complex in the standard 12-lead ECG.

In the majority of cases the initial forces of

septal depolarization are oriented anteriorly and to the right, producing the small “septal r wave” usually seen in the right precordial leads. A QS complex in lead V_1 in the absence of septal infarction could theoretically occur (1) if these interseptal forces were oriented at right angles to electrode, or (2) if the initial septal forces were oriented away from the lead V_1 . These normal variations in the direction of septal depolarization vector could result from either an actual anatomic shift in the position of the septum relative to the chest lead or from an intrinsic change in the balance of early depolarization forces. Thus, while it may be true that the most common cause of a QS in the right precordial leads is anterior (anteroseptal) infarction, cardiac position is one of the important causes of a QS pattern in lead V_1 as are left ventricular hypertrophy, left bundle branch block, and chronic cor pulmonale⁶. However, it is unlikely that in the present study a difference of the cardiac position masked effects of the interseptal involvement on the ECG pattern of lead V_1 between two patient groups with and without obstruction of the septal coronary branch. There was no reason to believe the position of the interventricular septum of group B oriented at right angles or abnormal direction to the electrode of the lead.

From pathological studies, it is well known that infarction of the interventricular septum usually accompanies infarction of the free anterior wall of the left ventricle. It often occurs in association with infarction of the free posterior wall, and rarely develops as a primary lesion. Nevertheless, little has been known of the electrocardiographic characteristics of septal infarction. Meyers and associates⁷ classified the electrocardiographic patterns encountered in a series of 89 cases of septal infarction into the following 5 categories: (1) atrioventricular conduction block (A-V block); (2) right ventricular conduction defects; (3) left ventricular conduction defects; (4) Q waves and/or RS-T segment displacement in right ventricular leads directly referable to, or at least suggestive of, septal infarction; and (5) absence of direct evidence of septal infarction. A-V block is a well-documented complication of septal infarction. In a series of 375 cases of coronary occlusion, Master and coworkers⁸ found prolongation of the PR interval beyond 0.20 sec in 10%, second degree A-V block in 2.4%, and complete block in 1.6%. Prolongation of the QRS duration is also a recognized manifestation of septal infarction.

Wilson and his associates^{9,10} demonstrated that right bundle branch block (RBBB) due to septal infarction was differentiated from that due to other causes by the direction of the initial phase of the QRS complex in leads from the right precordium. Uncomplicated RBBB was manifested by an initial R wave, a subsequent downstroke or coarse slurring, and a second late upstroke, whereas RBBB associated with septal infarction was characterized by an abnormal Q wave and a late R wave in these leads. However, in our series no patients showed the signs of A-V block, and only 3 patients showed RBBB. We excluded data from patients with RBBB from analysis, because intraventricular conduction disturbance would complicate statistical analysis of the QRS duration and magnitude of QRS complex. The experimental study exhibited A-V block in 5 and intraventricular conduction block in 4 out of 13 dogs with an isolated acute septal ischemia. All 4 dogs presenting the intraventricular conduction block had an initial small R wave in leads V_1 and V_2 . These results suggest that an initial R wave in the right precordial lead does not necessarily disappear in RBBB due to septal infarction. Nevertheless, we agree with the view that A-V conduction block and/or intraventricular conduction block are diagnostic or suggestive for septal infarction when the findings are associated with myocardial infarction of anterior or inferior free wall of the left ventricle.

Meyers and associates¹¹ reported from findings at autopsy that, in anterior myocardial infarction, extension of the infarction to the septum may be revealed by an abnormal initial downward deflection in right ventricular leads, V_1 and V_2 , or may occur without diagnostic signs in these leads. They classified that QRS complex in the right precordial leads of patients with septal infarction into 3 main groups: (1) triphasic QRS complex, consisting of a small Q, small R and deep S wave; (2) monophasic QS deflection; and (3) normal RS complex. The abnormal Q waves in leads V_1 and V_2 for septal infarction were also emphasized by Niitani,¹² Ishihara,¹³ and Kossmann and associates.¹⁴ Niitani¹² observed an abnormal Q wave in leads V_1 and V_2 in 10 out of 12 cases of septal infarction. Vectorcardiographic analysis by Hugenholtz and coworkers¹⁵ and Howitt and Lawrie¹⁶ demonstrated that the average direction of the 0.02 second vector in the horizontal plane shifted to the posterior compared to the normal vector. This posterior shift of the 0.02 second vector was also consistent with an abnormal Q

wave in lead V_1 . Sodi-Pallares and associates^{17,18} investigated septal infarction from an anatomic and electrical viewpoint, using 42 necropsy materials. Of the 42 cases, only 8 were not complicated with bundle branch block, and of the 8 cases they observed the persistent R wave in the right precordial leads in cases of massive septal infarction and infarction of the lower third of the septum in association with the QR or QS complexes in leads V_3 and V_4 . The presence of QS complex in lead V_1 did not permit a diagnosis of septal infarction in our study, similar to the results of Sodi-Pallares, because the prevalence rate of the QS complex in this lead was not significantly different regardless of involvement of the first septal coronary branch. Ventriculographic study of patients and experimental studies again demonstrated the difficulty of detecting septal infarction from the initial R wave in lead V_1 . Computer simulation methods¹⁹ suggested the difficulty of diagnosis of septal infarction from the viewpoint of R wave disappearance in this lead: a normal initial rightward vector disappeared and an initial vector directed to the left only when the small central segment of the septum which was divided into 7 areas was removed. Durrer et al²⁰ demonstrated that activation starts on the left septal surface in the middle third part, somewhat anteriorly, and at the lower third part at the junction of the septum and posterior wall. Right ventricular activation starts near the insertion of the anterior papillary muscle, and the rapid invasion of the septum and the adjoining free right ventricular wall occurs. Thus, activation of the septum and activation of the anterior wall of the right ventricle are jointly responsible for the initial R wave normally found in the right precordial leads. Therefore, when activation of the septum diminishes due to septal infarction, the R wave in lead V_1 may remain unchanged or reduced only slightly because an intact activation of the right ventricular anterior wall participates in the R wave in lead V_1 .

In 1955, Burch²¹ described an electrocardiographic syndrome characterized by absence of the Q wave in leads I, aVL, V_5 and V_6 due to septal fibrosis. This description was followed by a necropsy study of 1,184 hearts, 142 patients had septal fibrosis, and of 127 whose ECG was studied, 101 patients had the syndrome of septal fibrosis.²² Remanelli and colleagues²³ reported a positive correlation between the severity of obstructive lesions in the proximal left anterior

descending coronary artery (LAD) and the ECG syndrome of septal fibrosis. Approximately 60% of patients with the ECG syndrome of septal fibrosis having abnormal septal myocardial scintigrams also represented a positive correlation between the proximal LAD lesion and reduced blood flow to the ventricular septum. Conversely, in the present study, there was no significant difference of the Q wave in leads I and V₆ between with and without involvement of the first septal coronary artery. Activation of the right ventricular anterior wall may also contribute to exerting the small Q wave in leads I and V₆ in septal myocardial infarction. Coronary stenosis in the report of Romanelli²³ was approximately 74% in the septal fibrosis group, while all of our patients had LAD narrowing greater than 90%. In the present study, we used the patients with single vessel disease of the LAD without no significant lesions both in the right coronary artery (RCA) and the left circumflex coronary artery, while it was supposed that some patients of Romanelli et al might have obstructions of the RCA in association with LAD lesion. If that is the case, combination of LAD obstruction and RCA lesions would cause the ECG syndrome of septal fibrosis. Therefore, though we did not clearly explained the difference of our results from findings of Romanelli et al, it may account for the severity of LAD narrowing and/or for the extent of coronary stenosis of the RCA. The present investigation could not propose definitive ECG criteria for septal myocardial infarction. We have to remember, with regret, the statement of Levy and Hymann²⁴; "There are certain regions of the myocardium in which the absence of activation, due to dead muscle, does not cause appreciable change in the standard or in the six precordial leads; one of the sites is the interventricular septum".

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