Importance of the Location of Myocardial Infarction and Ventricular Aneurysm on Left Ventricular Function

Katsuo OZAWA, M.D.

SUMMARY

This experimental study was performed to evaluate the possibility that the site per se of infarction or ventricular aneurysm after acute myocardial infarction had a unique important role in left ventricular function.

Acute myocardial infarction and ventricular aneurysm were made in either the anterior or the inferior wall of the left ventricle using 24 dogs. Infarction was made by the multiple ligation method. The weight of the infarcted area was $23.1 \pm 3.9\%$ of the left ventricular free wall in the anterior wall group (12 dogs), and was $23.8 \pm 4.8\%$ in the inferior wall group (12 dogs). Ventricular aneurysm was made of right atrial homograft. The capacity of ventricular aneurysm, measured by casts, was $37.8 \pm 7.7\%$ of the left ventricular cavity in the anterior wall group and was $38.3 \pm 7.3\%$ in the inferior wall group. There was no significant difference in either the size of infarction or the capacity of ventricular aneurysm between 2 groups ($p=0.05$).

Ventricular function curves were obtained before infarction (control), after infarction, and after the creation of ventricular aneurysm, plotting left ventricular stroke work (LVSW) per body weight against left atrial mean pressure (LAMP). The change in ventricular function curves after infarction and the creation of ventricular aneurysm was not different significantly between 2 groups.

In the anterior wall group, LVSW/LAMP decreased to $47.8 \pm 9.8\%$ of the control after infarction, and further to $27.1 \pm 6.9\%$ after the creation of ventricular aneurysm. In the inferior wall group, it lowered to $47.6 \pm 12.5\%$ and again to $26.5 \pm 6.4\%$, respectively. There was no difference in these values between 2 groups statistically ($p=0.05$).

From the result of this experiment, it was concluded that the impairment of left ventricular function was equal between these 2 groups not only after infarction but also after the creation of ventricular aneurysm and that the anterior wall would not have more important contribution to the pumping ability of the left ventricle as compared with the inferior wall.

From the First Department of Surgery, University of Nagoya, School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466, Japan. (Director: Prof. Yohtaro Iyomasa, M.D.)

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AS a result of improved method of patient care, immediate diagnosis and treatment of life-threatening arrhythmias, death from arrhythmias complicating acute myocardial infarction has become less common. However, power failure of the heart and cardiogenic shock still remain the important mortality at present.

Although the degree of impaired function of the left ventricle is usually greater in anterior than inferior wall infarction, and cardiogenic shock develops more frequently in patients with acute infarction of the anterior wall than of the inferior one, the possibility that the anterior wall is unique in contribution to left ventricular performance is controversial. Major determinants of hemodynamic consequence of acute myocardial infarction such as extent, site and reserve of remaining viable mass, which mainly depend upon the state of coronary arteries, are so varied in degree that their isolated contribution to pump dysfunction is very difficult to evaluate in clinical cases.

The following experiment in dogs was performed to study the possibility that the location per se of infarction and ventricular aneurysm was related to the degree of left ventricular dysfunction in acute infarction and aneurysm of either the anterior or the inferior wall of the left ventricle.

METHODS

Twenty-four adult mongrel dogs of both sexes, weighing 10 to 30 Kg, were anesthetized with intravenously administered sodium thiopental of 25 mg/Kg and ventilated through an endotracheal tube with a respirator (MARK 7, Bird Co. Ltd.). A polyethylene catheter (6F) was introduced in a retrograde fashion from the femoral artery into the aorta. A Swan-Ganz Flow Directed Thermodilution Catheter (Model No. 93 A-118-7F, Edwards Lab.) was inserted into the femoral vein and advanced to the pulmonary artery under pressure monitoring. Electrocardiogram (ECG) was monitored in standard limb leads. Thoracotomy was made through the left 5th intercostal space on the right lateral position and the pericardial cradle was created to support the exposed heart. Another polyethylene catheter (6F) was placed into the left atrium through a small branch of the pulmonary vein. Aortic and left atrial mean pressures were recorded through transducers (TMI, BLH, MPU-0.5-290-0-III) on a 4-channel recorder (Multipurpose polygraph RH-150, Nihon Kohden). The zero reference point was at the level of the right atrium. Respiration was temporarily suspended by opening the airway to ambient pressure during recording. Cardiac output was determined by the thermodilution method with the use of a cardiac output computer (Model 9500, Edwards Lab.). Fig. 1 shows the schema of this preparation. Animals were divided into 2 groups, anterior

Fig. 1. Schema of the open chest preparation in this experiment.

and inferior wall group.

Anterior wall group: Twelve dogs comprised this group. The anterior descending artery distal to the first marginal branch was ligated, together with interconnecting coronary arteries, while a fresh homologous right atrial graft obtained from a little smaller donor dog was sutured on the anterior wall of the left ventricle (Fig. 2A, Fig. 3A). Then, anterior wall infarction was made.

Inferior wall group: Twelve dogs were included in this group. Inferior wall infarction was made by ligating the posterior descending branch of the left circumflex artery 1 to 1.5 cm distal to its origin together with interconnecting coronary arteries, while the same kind of graft was sutured on the inferior wall of the left ventricle (Fig. 2B).

In each animal, ventricular aneurysm was created by removing a core of the myocardium, about 1 cm in diameter, from the area within the confines of the sutured graft with a puncher, which was introduced from the vena cava of the grafted right atrium (Figs. 3, 4). Cardiac output, heart rate, aortic, and left atrial mean pressures were measured. The measurement was done before and after volume loading in 3 stages: prior to infarction (control), after infarction and after the creation of ventricular aneurysm. The volume loading was made by a rapid intraatrial injection of 100 to 200 ml of lactated Ringer's solution in the initial stage. Then, an adequate amount of blood was extracted to return the left atrial mean

Fig. 2. The location of infarction following the ligation of the anterior descending branch (AD) in the anterior wall group (A) and the posterior descending branch (PD) in the inferior wall group (B), in the course of grafting the homologous right atrium (HRA) as a sac of ventricular aneurysm. VC, the vena cava of the grafted right atrium.

Fig. 3. Technique employed for the creation of ventricular aneurysm. A, the right atrium of a donor dog was grafted on the left ventricular wall as a sac of ventricular aneurysm. B, a puncher (metal cylinder with a circular blade) was inserted through the vena cava (VC) of the graft (HRA). C, the myocardium was punched out and a communication to the left ventricular cavity was made. D, a homologous right atrial left ventricular aneurysm was created.

Fig. 4.

Fig. 4. A specimen of ventricular aneurysm. This dog survived 10 days after the creation of ventricular aneurysm.

Fig. 5.

Fig. 5. Casts of the left ventricular cavity and ventricular aneurysm of the inferior wall group.

pressure as close to the initial level as possible, and was stored in a reservoir after heparinization. This blood was reserved for the subsequent volume loading.

Left ventricular function curves were composed by plotting left ventricular stroke work (LVSW) per body weight in kilogram against left atrial mean pressure in 3 stages. Left ventricular stroke work was calculated as follows,

$$\text{LVSW} = (\text{AMP} - \text{LAMP}) \times \text{SV} \times 1.36 \times 0.01$$

where LVSW=left ventricular stroke work in gram-meter/sec

AMP=aortic mean pressure in mmHg

LAMP=left atrial mean pressure in mmHg

SV=stroke volume or cardiac output (ml/min)/heart rate.

LVSW/LAMP was calculated after infarction and after the creation of ventricular aneurysm in each animal and expressed in per cent of the control.

After all hemodynamic measurements were done, the animal was sacrificed by injecting 20 ml of KCl solution into the left atrium. The left ventricle with the aneurysm was casted. Their volumes were measured from these casts (Fig. 5). Then, the infarcted area of the left ventricle was resected and weighed.

RESULTS

Production of acute myocardial infarction resulted in ventricular fibrillation in 2 cases of each group. All of them were successfully reversed with electroversion allowing continuation of the experiment. The weight of the in-

farcted area was $23.1 \pm 3.9\%$ of the left ventricular free wall in the anterior wall group, and was $23.8 \pm 4.8\%$ in the inferior wall group. The difference between 2 groups was not significant statistically ($p=0.05$). Ventricular aneurysm had the capacity of $37.8 \pm 7.7\%$ of the left ventricular cavity in the anterior wall group, and $38.3 \pm 7.3\%$ in the inferior wall group. There was no difference in relative size of ventricular aneurysm between 2 groups statistically ($p=0.05$).

Left ventricular function curves shifted downward and to the right after

Fig. 6. Changes in left ventricular function curves of the anterior and the inferior wall group.

Fig. 7. Changes in left ventricular stroke work / left atrial mean pressure (LVSU/LAMP) of 2 groups.

infarction, and further displacement to the same direction was observed after the creation of ventricular aneurysm. There was no significant difference in the degree of shifting between 2 groups, either after infarction or after the creation of ventricular aneurysm (Fig. 6).

LVS_W/LAMP, expressing the average slope of each ventricular function curve, decreased to $47.8 \pm 9.8\%$ of the control in the anterior wall group and to $47.6 \pm 12.5\%$ in the inferior wall group, after infarction. After the creation of ventricular aneurysm, it lowered to $27.1 \pm 6.9\%$ of the control in the anterior wall group and to $26.5 \pm 6.4\%$ in the inferior wall group. The change in LVS_W/LAMP from the control was not different statistically between 2 groups either after infarction or after the creation of ventricular aneurysm ($p=0.05$) (Fig. 7).

DISCUSSION

Several aspects concerning the model for infarction and ventricular aneurysm in this experiment require comments. There are many terms specifying the location of infarction such as anterior, anteroseptal, anterolateral, apical, inferior, posteroinferior, diaphragmatic, posterior, posterobasal, and so on. The accuracy of these distinctions still remains unsettled. This experiment was performed in open chest preparation and exposed heart was supported by the pericardial cradle. Because the heart would be somewhat displaced from the natural position, the ECG diagnosis of the site and the extent of infarction seemed questionable in this type of experiment. Therefore, "anterior" and "inferior" were not used in the strict criterion of ECG interpretation in this experimental model. The location described here by "inferior" would be otherwise diaphragmatic or posteroinferior portion of the left ventricle (Fig. 2B).

The multiple ligation method is considered most reliable and reproducible in making experimental infarction in canine heart. In dogs, 75% of the ventricular septum is supplied by the anterior septal artery and the remaining 25% is nourished by deep perforators from the anterior and posterior descending artery.^{1),2)} The level of the most proximal ligation on the anterior descending artery was distal to the origin of the anterior septal artery. Therefore, the extent of septal involvement after the multiple ligation in these 2 groups was considered equal.

The shape of ventricular aneurysm was sac-like (Fig. 4). Though the caliber of the communication to the left ventricle was small, the paradoxical expansion of ventricular aneurysm with each systolic contraction was observed in all cases. The capacity of ventricular aneurysm was large enough as com-

pared with that of the left ventricle, but it did not impair ventricular performance so much that animals could not survive the experiment. This was proved with a fact that other 5 dogs which had not been sacrificed after this series of experiment survived more than 1 week.

Orias was the first who reported the dynamic change in the canine heart following the ligation of the anterior descending artery.³⁾ Tennant observed a marked expansion of the ischemic region after coronary occlusion, and suggested that a considerable fraction of the total pressure developed was lost in producing such an expansion.⁴⁾ Murray and Heimbecker connected paradoxical expansion of acutely infarcted myocardium with decreased cardiac output and low blood pressure.^{5),6)} Herman described in a group of patients that ventricular asynergy, local disturbance in ventricular wall motion which disrupted the normal pattern of left ventricular contraction, represented an important cause of cardiac failure.⁷⁾ Pairolero reported the hemodynamic effect of experimental cardiac akinesis in canine heart and showed that the impairment of left ventricular function was related to the size of the scar.⁸⁾ Experimental cardiac aneurysm was studied by Austen for the first time, who observed fall of left ventricular function following the creation of ventricular aneurysm and return of it after clamping of the aneurysm at its base.⁹⁾ Tyson made a study of hemodynamic effect of artificially constructed left ventricular aneurysm on left ventricular function in dogs.¹⁰⁾ In that study, left ventricular performance of animals which had expansile aortic homologous ventricular aneurysm was impaired more markedly than that of dogs which had relatively non-expansile Teflon ventricular aneurysm. Vayo and Klein made a theoretical analysis of left ventricular aneurysm and showed that if approximately 20 to 25% of the left ventricular area was inactivated by any pathological process, the degree of shortening distance required of myofibrils to maintain stroke volume exceeded physiological limits.^{11),12)} Ventricular aneurysm produces mechanical disadvantage not only by loss of contractile tissue but also by paradoxical expansion which further reduces forward stroke volume and ejection fraction.^{13),14)} The dilatation of the heart to maintain adequate stroke volume adversely affects ventricular performance because increased size produces an increase in wall stress and oxygen demand by the law of Laplace.¹⁵⁾ Hemodynamic importance of the size and compliance of ventricular aneurysm was also suggested by the remarkable postoperative improvement of left ventricular function in patients undergoing aneurysmectomy.¹⁶⁾⁻¹⁸⁾

The location itself of infarction is obviously important in relation to involvement of specific structures such as the mitral valve apparatus, the interventricular septum and the conduction system.¹⁹⁾ When those important structures are intact and the state of coronary arteries is equal, whether there

is any difference in left ventricular dysfunction between anterior and inferior wall infarction of the same size, is a question. In clinical studies, hemodynamic abnormality associated with anterior myocardial infarction has been reported to be severer than that of inferior myocardial infarction.^{20)–23)} But, in clinical cases, the extent of infarction and associated coronary artery diseases varied so much in degree.²⁴⁾ Generally, the septal involvement of acute myocardial infarction is severer in anterior infarction than in inferior one. This is explained by the blood supply of the human interventricular septum. Seventy-five to 90% of blood supply of the human interventricular septum is derived from branches of the anterior descending artery.²⁵⁾ Anterior myocardial infarction develops in the occlusive disease of the anterior descending artery and usually is complicated with the lesion of septal arteries.

Herman and Kong showed in the human heart that there was greater segmental shortening during systole of the anterior wall as compared with the inferior wall.^{7),26)} Then, Zaret deduced that anterior wall contraction should have a more important contribution to left ventricular ejection, and that infarction of the anterior wall might therefore produce greater impairment than would result from the equivalent loss of inferior wall infarction.²⁸⁾ However, if the shape of the left ventricle is considered, a difference in curvature between the anterior and the inferior wall is noted. The anterior wall is more convex than the inferior wall, so it may take more concentric movement during systole.²⁹⁾ The magnitude of excursion of any region of the left ventricle may not necessarily relate to the role played in ventricular ejection. In contrast to Zaret's speculation, Miller demonstrated in patients carefully matched for similar extent and pattern of dyssynergy confined to either the anterior or the inferior ventricular wall that the location of dyssynergy was unimportant as an isolated determinant of left ventricular performance in coronary artery diseases.³⁰⁾ The result of this experiment also showed that the contribution of the anterior wall to left ventricular function was equal to that of the inferior wall. The location itself of left ventricular aneurysm did not play an important role either.

As for the mechanism of developing of ventricular aneurysm in the infarcted myocardium, Lowe made an analysis of the distortion expected to occur after infarction of various types and sizes.³⁰⁾ The study showed that 2 important factors determining the distortion produced in the wall of the left ventricle were the proportion of the thickness of the wall infarcted and the size of the area destroyed. From the law of La-Place and the hypothesis of Woods, the more convex anterior wall of the left ventricle would be thinner than the inferior wall which is more flat.^{15),31)} On the view point of blood supply of the human heart, the inferior wall of the left ventricle has the ad-

vantage of dual blood supply, from the circumflex branch of the left coronary artery and from the posterior descending branch of the right coronary artery, in contrast to the anterior wall which is nourished only by the anterior descending branch of the left coronary artery. Therefore, massive transmural necrosis is unlikely to occur in the inferior wall infarction. The speed of thinning and bulging of the infarcted area may be slow in the inferior wall because it may be supported by less compliant diaphragmatic portion of the pericardium, though this was not proved in this acute experimental model. These factors may be probable causes of relatively rare occurrence of inferior ventricular aneurysm.³²⁾⁻³⁴⁾

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