The Inhomogenous Movement of the Left Ventricle Induced by Coronary Artery Occlusion

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Summary

The pattern of myocardial segment length change in an ischemic and nonischemic area of the left ventricle was studied acutely following ligation of the LADA in the dog. Results were interpreted in relation to other hemodynamic findings obtained in the same experiments. Paradoxical bulging during systole was found in the ischemic area, whereas increased shortening during systole was found in the nonischemic area. Since there was no increase in cardiac output or aortic pressure, it was assumed that the increased energy released by the nonischemic area of the ventricle was expended in stretching myocardial fiber in the ischemic area. Under the condition of ventricular afterloading, increased systolic bulging of the ischemic area and increased systolic shortening of the non ischemic area were noted. With maximum afterloading, systolic bulging of the ischemic area was diminished and ventricular systolic pressure increased. This suggested that a reduction in ventricular wall compliance might improve the function of the partially ischemic ventricle by diminishing the amount of energy expended during systole in stretching muscle fiber in the ischemic area.

Additional Indexing Words:
Paradoxical bulging  Myocardial segment length  Prongs  Ventricular function curve

By using the technique of angiography, several investigators1)-4) have studied the role of asynergy or dysynergy in the development of altered left ventricular function in patients with coronary heart disease. In 1965, Harrison5) postulated that different portions of the left ventricle would shorten differently due to regional myocardial ischemia.

The purpose of this study is to show not only that inhomogeneous movement occurs in the left ventricle following experimental occlusion of the left anterior descending coronary artery (LADA) in the dog, but that shortening in the nonischemic area increases in an effort to compensate for paradoxical movement in the ischemic area of the myocardium.

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METHOD

Experiments were performed on 15 mongrel dogs weighing between 9 and 14 Kg. After the intravenous administration of sodium pentobarbital in an initial dose of 25 mg./Kg., a thoracotomy was performed in the left 4th or 5th intercostal space. Respiration was maintained artificially. The left anterior descending coronary artery (LADA) was dissected 1 to 2 cm. below the bifurcation of the main segment for the passage of a ligature around the vessel. Aortic blood flow was measured with a squarewave electromagnetic flow meter. The flow transducer was placed on the root of the aorta. Left ventricular, ascending aortic blood pressure were measured through catheter. Left ventricular segment length was measured by the use of prongs which measured displacement through a strain gauge (Fig. 1). The characteristics of this measuring device were reported elsewhere by Tomoda, a member of our laboratory. In 5 cases, myocardial segmental length measurements were made adjacent to the LADA, in a direction perpendicular to the course of the vessel. In 4 cases, segment length measurements were made adjacent to the circumflex artery, in a direction parallel to the course of the vessel.

After obtaining control data, left ventricular function during afterloading was examined by constricting the descending aorta different amounts and relating stroke work to left ventricular enddiastolic pressure. Data were obtained after 3 min. for each degree of aortic constriction. During a period of continuous recording, the LADA was completely occluded. When a new steady state was reached, the descending aorta was constricted in the same manner as before to study the effects of afterloading the left ventricle.

RESULTS

Left Venticular Function During Afterloading—Before and After LADA Occlusion
The relationship between left ventricular enddiastolic pressure (LVEDP) and left ventricular stroke work (SW) during afterloading of the left ventricle is depicted in Fig. 2 for all animals in whom data were obtained before and after ligation of the LADA. The plot reveals two distinct zones, one for the control data and one for the data obtained following LADA ligation. One min. after ligation of the LADA aortic pressure and cardiac output were decreased, but heart rate and LVEDP were unchanged. Thereafter, no changes were rated in any of these hemodynamic parameters for observation periods up to 20 min.

The details of these experiments have been presented by one of the authors.7)

Changes in Myocardial Segment Length in the Ischemic and Nonischemic Areas of the Left Ventricle

Changes in myocardial segment length in the ischemic area are shown from the records of one experiment in Fig. 3. Five sec. after coronary ligation, shortening of the myocardial segment did not continue to the end of systole, as determined from left ventricular pressure, and 10 sec. after ligation, actual lengthening of the myocardial segment occurred in late systole. These late systolic changes were noted in all cases in which segment length was recorded
in the ischemic area, although their time of onset and duration varied among the animals. After 20 sec., paradoxical movement of the myocardial segment was noted in this case. Paradoxical movement was observed before 60 sec. in all cases. Progressive elongation of the myocardial segment at the end of diastole also was observed.

Changes in the movement of the myocardium in the nonischemic area after coronary ligation are depicted in records obtained from one experiment and shown in Fig. 4. Ten sec. after coronary ligation, enddiastolic segment length increased. An increased amount of segment shortening in late systole
was seen 10, 20, and 30 sec. after ligation. This phenomenon was seen in all cases early in the first min. after coronary ligation. This suggests compensatory shortening of the intact myocardium for the late systolic bulging of the ischemic area. Thirty sec. after the ligation, enddiastolic segment length decreased, and muscle shortening during systole increased. This continued to the 60 sec. period after ligation. This phenomenon also suggests that the nonischemic area attempts to compensate for the decrease in ventricular function which results from bulging in the ischemic area.

Response of the Ischemic and Nonischemic Area of the Myocardium to Afterloading of the Left Ventricle

Fig. 5 shows the effects of afterloading on myocardial segment length in the intact heart and in the heart with a ligated LADA. In the intact heart (upper tracings) myocardial segment length changes during systole were not greatly affected by afterloading, but segment length during diastole was in-

![Graph showing response of ischemic area to afterloading]

Fig. 5. Response of the ischemic area of the myocardium to afterloading of the left ventricle.
creased. These findings were accompanied by an increase in left ventricular enddiastolic pressure, left ventricular systolic pressure, and systolic pressure. In the heart with a ligated LADA (lower tracings), afterloading resulted in greater paradoxical movement of the myocardial segment in the ischemic area without a rise in left ventricular systolic pressure. A stage of aortic constriction was reached, however, in which paradoxical bulging of the ischemic area was diminished and left ventricular pressure was increased.

In the nonischemic area (Fig. 6), afterloading at first caused a reduction in enddiastolic segment length and an accentuation of shortening during systole, although there was no rise in left ventricular pressure.

This suggests that at this time the increased shortening of the nonischemic area was expended in stretching the paradoxically bulging myocardium in the ischemic area.

With further aortic constriction a marked increase in enddiastolic seg-
ment length occurred in the nonischemic area, and in increase in left ventricular pressure was produced.

**DISCUSSION**

In 1935 Tennant and Wiggers8) reported the existence of paradoxical movement in an ischemic area of the left ventricle following ligation of a branch of the left coronary artery in dog. Systolic bulging of an ischemic area of the ventricle has been a well known phenomenon in coronary heart disease.

However, the relationship between this phenomenon and the hemodynamics of the heart has not been investigated until recent years. Most investigations have used the angiographic technique to study the effect of paradoxical bulging upon cardiac hemodynamics. Quantitative information on non-uniform myocardial movement is difficult to obtain by the angiographic technique. The direct method of measuring myocardial segment length changes would seem to afford an easy and accurate means of obtaining quantitative information. However, this method was found to have certain limitations: 1) the segment length changes were not necessarily representative of the entire area under study, and 2) the amount of myocardial segment movement could be varied by changing the position of the prongs on the left ventricle (Fig. 7). One of the factor which is probably involved is the orientation of the prongs with respect to the direction of the muscle fiber. Since the prongs penetrated the myocardium to a depth of only 2 mm., the movement recorded by them was greatly influenced by the course of muscle fibers on the surface of the heart.

![Fig. 7. Variation of the myocardial segment movement by changing the position of prongs on the left ventricle.](image-url)
heart. Because of these considerations, quantitative analysis of the data was not pursued, and only the pattern of the segment length changes is presented.

Prinzmetal et al.9) reported that at the onset of ballooning, 3 to 10 sec. after coronary ligation, the ischemic region was generally observed to contract early in systole and balloon late in systole. In our experiments, all cases showed the phase of late systolic bulging in the ischemic area, and this corresponded to the phase of late systolic increased shortening in the nonischemic area of myocardium. During this phase, neither aortic systolic pressure nor aortic blood flow increased, suggesting that the increased shortening of the nonischemic area was translated into late systolic bulging of the ischemic area.

In the phase of paradoxical bulging of the ischemic area, pansystolic accentuation of shortening was noted in the nonischemic area. Such hyperfunction of the uninvolved myocardium was demonstrated as long as 35 years ago by Tennant and Wiggers, but they did not show the precise relationship of this phenomenon to the hemodynamic state of the left ventricle.

During aortic constriction, the accentuated shortening of the nonischemic area was assumed to supply the energy for the systolic bulging of the ischemic area. This suggests that an increase in ventricular function in the partially ischemic heart may be induced by a method other than increasing contractility of the nonischemic area; i.e. reducing the compliance of the ventricular wall. Perhaps, increasing the preload might reduce ventricular compliance in a manner that would increase cardiac output, assuming LVEDP did not rise abnormally. The concept of recovery of cardiac function in the late stage of myocardial infarction due to a reduction in myocardial compliance10),11) is supported by our data.

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References