Factors Contributing to Altered Left Ventricular Diastolic Properties During Angina Pectoris

TIFT MANN, M.D., SHELDON GOLDBERG, M.D., GILBERT H. MUDGE, JR., M.D., AND WILLIAM GROSSMAN, M.D.

SUMMARY  Mechanisms involved in the altered left ventricular (LV) diastolic properties during angina were studied in 26 patients with coronary artery disease. Angina was induced by rapid atrial pacing and measurements were made at rest and during angina in the immediate post-pacing period. No changes occurred in heart rate (71 ± 3 to 73 ± 3 beats/min, NS) or right ventricular (RV) end-diastolic pressure (10 ± 1 to 11 ± 1 mm Hg, NS), while significant increases occurred in LV end-diastolic pressure (17 ± 1 to 30 ± 1 mm Hg, p < 0.01), aortic diastolic pressure (74 ± 3 to 80 ± 3 mm Hg, p < 0.01), coronary sinus blood flow (133 ± 15 to 212 ± 32 ml/min, p < 0.01), and the time constant (T) of LV pressure fall in early diastole (43 ± 2 to 58 ± 4 msec, p < 0.01). Despite the rise in arterial pressure, a significant fall was observed in peak negative dP/dt (1961 ± 106 to 1751 ± 80 mm Hg/sec, p < 0.01). Changes in RV end-diastolic pressure do not explain the increased LV end-diastolic pressure during angina. Increased aortic pressure and coronary blood flow may contribute, but the simultaneous fall in peak negative dP/dt and rise in T suggest that impaired ventricular relaxation is an important factor contributing to the previously demonstrated alteration in LV diastolic properties during angina pectoris.

ABNORMAL LEFT VENTRICULAR diastolic pressures transiently occurring during angina pectoris are the result of both impaired left ventricular systolic performance and altered left ventricular diastolic properties.1-4 It is clear that ischemia-induced regional contraction abnormalities may be of sufficient degree to impair overall left ventricular function, and the resulting increased diastolic volumes contribute to the increased left ventricular end-diastolic pressure.1-3, 9, 10 However, previous studies by us1 and from other laboratories4, 11-13 have clearly shown that diastolic pressure is higher at any given volume throughout diastolic filling, indicating some change in left ventricular distensibility during ischemia.

Many factors influence the left ventricle in diastole, but the completeness of ventricular relaxation and external diastolic constraints of the ventricle are among the most important.5, 12-20 With regard to external constraints, attention has focused on the influence of the pericardium, right ventricular loading, and coronary perfusion pressure5, 18-20 on left ventricular diastolic compliance.

Glantz and Parmley have suggested that during ischemia induced by atrial pacing, increased right-sided volume and pressure cause the left ventricular diastolic pressure-volume curve to shift upward as a result of “direct mechanical coupling between the two ventricles,” enhanced by the intact pericardium.20 They cite data of Weiss et al.20 as being inconsistent with the concept that impaired relaxation is playing a major role in causing the altered left ventricular diastolic pressures, as had been suggested by several investigators.4, 6, 12, 13, 15, 16

The present study was designed to examine the role of altered right ventricular loading and pericardial influences on the increased left ventricular diastolic pressure during angina. We reasoned that if such external constraints were sufficient to cause large increases in left ventricular diastolic pressure, they should cause detectable increases in right ventricular diastolic pressure as well.21 In addition, we measured coronary blood flow and perfusion pressure to assess the possible importance of these factors and the so-called “erectile properties” of the left ventricle.20

Methods

Patient Population

The study population included 26 patients who had undergone elective cardiac catheterization for evaluation of their chronic stable angina pectoris. All patients had coronary artery disease, as defined by the presence of lesions resulting in 75% or greater narrowing of the intraluminal diameter of one or more coronary vessels at coronary angiography. Patients who had an unstable clinical course, significant left ventricular systolic dysfunction, hypertension or valvular heart disease were excluded from the study. Nitrate preparations were withheld before the procedure in all patients, but other medications, including propranolol, were continued at dosages prescribed before the catheterization. The experimental protocol was approved by the Committee on the Rights of Human Subjects, and informed consent was obtained from each patient. No complications occurred as a result of the study.
### Table 1. Hemodynamic Variables Before and During Angina Pectoris

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*SEM* = +3 ± 3 = +3 ± 3 = ±1 ± 1 = ±106 ± 80 = ±2 ± 4 = +1 ± 1 = ±2 ± 2 = ±3 ± 3 = ±15 ± 32

| *p* | NS | *p* < 0.01 | *p* < 0.01 | *p* < 0.01 | NS | *p* < 0.01 | *p* < 0.01 | *p* < 0.01 |

Abbreviations: HR = heart rate; MAP = mean arterial pressure; LVEDP = left ventricular end-diastolic pressure; T = time constant of left ventricular pressure fall in diastole (see text); RVEDP = right ventricular end-diastolic pressure; RVSP = right ventricular systolic pressure; ADP = aortic diastolic pressure; Control = control; a = angina.

### Methods and Experimental Protocol

Cardiac catheterization was performed using the brachial or femoral approach in the fasting state after premedication with diazepam (10 mg orally). Systemic arterial pressure was measured from a femoral artery monitor line. Left ventricular pressure was recorded in each patient using a high fidelity micromanometer-tipped catheter (Mikro-Tip, Millar Instruments, Houston, Texas). A pacing catheter was positioned either in the right atrium or coronary sinus. Coronary arteriography was performed using either the Sones or Judkins technique.

The study was performed either before coronary angiography or after pressures had returned to baseline following coronary angiography. After collection of control data, the heart rate was incrementally increased until typical angina pectoris of moderate severity developed, at which time the pacing was abruptly discontinued. Repeat data were then obtained in the immediate post-pacing period during the first 10–15 beats, when left ventricular end-diastolic pressure typically is maximally elevated. In all patients, heart rate, high fidelity left ventricular diastolic pressure and systemic arterial pressure were recorded. In addition, data reflecting either left ventricular relaxation or external ventricular constraints were recorded in subgroups of patients.

Left ventriculography in the control state and during angina in the immediate post-pacing period was not carried out in the present study simultaneous with measures of coronary blood flow and right ventricular hemodynamics. We felt that the added time required to place the coronary sinus thermocatheter and the right ventricular balloon-tipped catheter and to make appropriate calibrations and baseline recordings while left ventricular and arterial catheters were already in position added sufficient risk to each study that the added risk of two ventriculograms was unjustified. Instead, left ventriculography in the control state and during angina in the immediate post-pacing period was performed by us in a separate group of patients using an identical protocol. These data, which showed a definite upward shift in the left ventricular diastolic pressure-volume curve during angina, have recently been reported by us.1

When mean arterial pressure, left and right ventricular end-diastolic pressures, aortic diastolic pressure, right ventricular systolic pressure, ECG, and coronary blood flow were all recorded simultaneously (patients 17–26, table 1), the time constant (T) could not be calculated. This problem was related to the limited

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1 No study was attempted until it was demonstrated that a micromanometer tipped catheter could be successfully placed in the left ventricle.
number of amplifiers which could be simultaneously used with our recording system (Electronics for Medicine DR8) and the fact that both high and low gain left ventricular pressure tracings are required for accurate measurement of both T and left ventricular end-diastolic pressure. Accordingly, measurements were made in subsets of patients.

**Left Ventricular Relaxation**

Previous studies have demonstrated that the rate of pressure fall with respect to time during isovolumic relaxation is exponential, and log pressure vs time plots from peak $-\frac{dP}{dt}$ to mitral valve opening are linear. Thus, isovolumic relaxation can be characterized by time constant $T$ which is the negative reciprocal of the slope of the above line. This time constant was used as one index of left ventricular relaxation. A previous study has shown that $T$ is unchanged in the immediate post-pacing period in normal patients. Peak $- \frac{dP}{dt}$ was used as a second index of left ventricular relaxation.

**External Ventricular Constraints**

Right ventricular pressure was evaluated in 10 patients. A Swan-Ganz catheter was inserted into a peripheral vein and advanced to the right ventricle where right ventricular pressures were simultaneously recorded with left ventricular pressures during the experimental protocol. Both right and left ventricular end-diastolic and peak systolic pressure were measured. All tracings were judged of adequate quality to permit clear identification of ventricular end-diastolic pressure.

Myocardial blood flow was evaluated in 11 patients using a thermodilution pacing catheter which was placed in the coronary sinus via an antecubital vein. Room temperature indicator solution was injected at 40 ml/min through the catheter, and coronary blood flow calculated using the thermodilution technique previously described. Heart rate, mean and phasic arterial pressure, and coronary sinus blood flow were measured before and during angina in the immediate post-pacing period.

Measurements of right ventricular pressures and coronary sinus blood flow permit only an indirect examination of external constraints. More direct assessment requires measuring right ventricular volumes, radii of curvature and wall stress, as well as coronary blood volume and its regional distribution.

Data before and during angina pectoris were compared using the $t$ test for paired data.

**Results**

Table 1 summarizes hemodynamic data obtained during control compared to that obtained during angina pectoris in the immediate post-pacing period. Heart rate was unchanged during angina (71 $\pm$ 3 beats/min, control, vs 73 $\pm$ 3 beats/min, angina, NS).

Mean arterial pressure increased significantly (101 $\pm$ 3 mm Hg, control, to 113 $\pm$ 3 mm Hg, angina, $p < 0.01$). Left ventricular end-diastolic pressure increased from 17 $\pm$ 1 mm Hg during control to 30 $\pm$ 1 mm Hg during angina. During angina, peak $-\frac{dP}{dt}$ decreased significantly (1961 $\pm$ 106 mm Hg/sec, control, vs 1751 $\pm$ 80 mm Hg/sec, angina, $p < 0.01$), while $T$ increased significantly (43 $\pm$ 2 msec, control, to 58 $\pm$ 4 msec, angina, $p < 0.01$). Analysis of responses in subjects taking propranolol before study showed no consistent difference in response compared to those not taking propranolol.

In contrast to the substantial increase in left ventricular end-diastolic pressure, right ventricular end-diastolic pressure remained unchanged during angina and was not statistically different from control (10 $\pm$ 1 mm Hg, control, vs 11 $\pm$ 1 mm Hg, angina, NS). This is further illustrated in figure 1. There is a clear separation of right and left ventricular diastolic pressures during angina. Right ventricular systolic pressure increased significantly from 31 $\pm$ 2 mm Hg, control, vs 39 $\pm$ 2 during angina ($p < 0.01$).

Aortic diastolic pressure increased modestly but significantly (74 $\pm$ 3 mm Hg, control, vs 80 $\pm$ 3 mm Hg, angina, $p < 0.01$). Coronary sinus blood flow increased substantially, from 133 $\pm$ 15 ml/min at rest to 212 $\pm$ 32 ml/min during angina ($p < 0.01$).

**Discussion**

The left ventricular end-diastolic pressure is transiently elevated to a striking degree during angina pectoris. Previous studies have demonstrated that this is the result of both impaired left ventricular systolic performance and, perhaps more importantly, altered diastolic properties, since the elevated pressures are out of proportion to the changes in volume. Thus, the pressure-volume curve of the left ventricle is transiently shifted upward during angina.

The mechanism responsible for this alteration in the diastolic properties of the ventricle during angina remains unclear, and the purpose of the present study was to investigate potential mechanisms. It is customary to subdivide the principal factors influencing diastolic properties into those intrinsic and extrinsic to the ventricle. During myocardial ischemia, both intrinsic properties such as completeness of ventricular relaxation and passive elastic properties of the ventricle may be altered. Similarly, external factors such as coronary artery pressure and flow and right ventricular loading properties may also be affected by myocardial ischemia. Finally, an important role for the pericardium in enhancing “mechanical coupling” of the ventricles has been suggested.

In the present study, right ventricular end-diastolic pressure did not increase during angina in the immediate post-pacing period, although left ventricular end-diastolic pressure nearly doubled. If altered right ventricular afterload (right ventricular systolic pressure rose from 31 $\pm$ 2 mm Hg to 39 $\pm$ 2 mm Hg)
**Figure 1.** Hemodynamic variables in the control period (top) and during angina pectoris immediately post-pacing (bottom) in a patient with coronary artery disease. In the top panel, aortic pressure (AP) is switched from mean to phasic in mid-tracing. Right ventricular (RV), left ventricular (LV), and aortic (AP) pressures are recorded together with coronary sinus blood flow (CSF), ECG, and LV dP/dt. LV end-diastolic pressure rises from 10 mm Hg in control to 20 mm Hg during angina, while RV end-diastolic pressure remains virtually constant at 8 mm Hg. See text for discussion of other variables.
had resulted in a sufficient increase in right ventricular residual volume to raise left ventricular diastolic pressure, a detectable increase in right ventricular diastolic pressure should have occurred. Similarly, the constraint of the pericardium cannot adequately explain the isolated increase in left ventricular diastolic pressure, since this mechanism should equally affect pressures in both ventricles in diastole.

Increased coronary artery pressure and flow have also received attention as possible factors in the alteration of left ventricular diastolic properties during angina. Early studies in which coronary artery pressure and flow were increased in dogs yielded variable results on left ventricular chamber stiffness.20, 24-33 Ahn et al.30 recently demonstrated in isolated rabbit hearts that increases in coronary perfusion pressure resulted in increases in ventricular "effective stiffness," and that these changes in ventricular stiffness were parallel to acute substantial changes in wall thickness. It has thus been suggested that an increased aortic diastolic pressure may result in upward shift of the left ventricular diastolic pressure-volume curve due to changes in "erectile properties" of the left ventricle mediated through aortic diastolic pressure and coronary blood flow. In the present study, both diastolic pressure and coronary blood flow were substantially increased over control during angina. Although these observations are consistent with some contribution of an erectile effect, other considerations conflict with this interpretation.

First, coronary blood flow increases markedly in normal subjects with rapid atrial pacing,23, 36-39 although it has been repeatedly shown that left ventricular diastolic pressure actually falls in such subjects, with no shift in diastolic compliance.1, 4, 6, 11, 13 Second, although coronary sinus blood flow measures predominantly left ventricular myocardial flow, it can be assumed that right ventricular myocardial flow also increased in our subjects in response to the pacing tachycardia, increased aortic diastolic pressure (driving pressure for right coronary flow), and increased right ventricular systolic pressure. Thus, one would also expect an "erectile effect" for the right ventricle; yet, no increase in right ventricular diastolic pressure occurred.

Several previous studies both in man and animals have suggested that ischemia may impair left ventricular relaxation.4, 6, 8, 12-16 Peak -dP/dt has been the major index of left ventricular relaxation previously used and a decline in peak -dP/dt has been demonstrated in dogs after coronary occlusion and in man during atrial pacing-induced angina.6, 8, 13, 16 However, in patients with angina in the immediate post-pacing period or with spontaneous angina, there has been an inconsistent fall in peak -dP/dt.1 This inconsistent decline has been attributed to the increased systemic pressure that occurs during angina, since peak -dP/dt is substantially influenced by left ventricular systolic pressure.27-29 In the present study, the incremental increase in mean arterial pressure was less than that which occurred in a previous study, and peak -dP/dt declined significantly.

The time constant of left ventricular pressure fall in early diastole was also used as an index of left ventricular relaxation. Weiss et al.4 have demonstrated that the exponential fall of pressure during isovolumic relaxation between peak -dP/dt and mitral valve opening can be characterized by a time constant, T.26 They further demonstrated that T was a sensitive index of isovolumic relaxation in the dog independent of left ventricular systolic pressure. Taw et al.8 had similar conclusions in a group of patients and suggested that T was prolonged during myocardial ischemia.8 In the present study, T was significantly increased during angina in the immediate post-pacing period compared with control.

The decline in peak -dP/dt and increase in time constant T in the present study suggests that left ventricular relaxation is impaired during angina and is at least in part responsible for the altered diastolic properties. Since ventricular relaxation involves the active uptake of calcium by the sarcoplasmic reticulum, it is not surprising that hypoxia or ischemia substantially may impair this process.40-42 Indeed, Palacios and Powell43 and Frist et al.44 have presented strong evidence that hypoxia impairs the relaxation process of cardiac muscle both in the intact heart and the isolated papillary muscle preparation. Their studies did not permit assessment of the persistence of incomplete relaxation to end-diastole.

Although myocardial ischemia in the intact human ventricle is generally a regional process, this study and others4, 6, 8, 13, 16 have found impairment in indices of relaxation of the ventricle as a whole. One possible explanation is that the demonstrated impairment of overall ventricular relaxation may be the net expression of brisk relaxation of normal and delayed relaxation (or increased diastolic tone4) of ischemic areas. As proposed by Waters et al.,45 the impaired relaxation may be the result of dysynchronous wall motion in the ischemic zone during isometric relaxation. According to this hypothesis, peak -dP/dt decreases because of persistent contraction in the ischemic zone at the time of isovolumic relaxation in the normal zone.

Even if it is accepted that left ventricular ischemia is associated with impaired myocardial relaxation, the question arises as to whether the impaired relaxation is related to the increased left ventricular end-diastolic pressure. Could the impaired relaxation be influencing only early diastolic pressure-volume relations, while the late diastolic shift in pressure-volume relations1 is due to a different cause? Our study cannot answer this question. We have directed our attention to other proposed mechanisms46 for the pressure-volume shift, and can only support impaired relaxation as an explanation for this shift by exclusion. Weisfeldt has reported preliminary data46 that in the non-ischemic dog heart relaxation is essentially complete by 3.5 T after peak -dP/dt occurs. Accordingly,
even in our patients with the longest T during ischemia (83 msec), relaxation should have become complete by 290 msec, which occurred before end-diastole. Whether the relationship of 3.5 T to compete relaxation will hold during ischemia of the type associated with angina pectoris (i.e., increased coronary flow, regional ischemia) remains to be determined.

An important experimental study that bears on our findings has recently been reported by Henry et al.47 These investigators found that left ventricular ischemia in the rabbit was associated with a progressive rise in diastolic pressure at constant volume (isovolumic preparation) and that this decreased distensibility could be completely prevented by pretreatment with nifedipine, an antagonist of myocardial calcium uptake. The fact that such decreases in left ventricular distensibility with ischemia are not seen in the dog48 emphasizes the importance of species-determined differences in myocardial relaxation and diastolic tone. For example, Green and Weisfeldt49 reported that under nearly identical experimental situations, hypoxia produced a substantial rise in resting tension of isolated rat trabecular cornae (+25% to +90%), while an actual decline in resting tension of cat papillary muscle occurred with the same degree and duration of hypoxia. Also, Nayler and coworkers48* reported that considerable species determined differences exist in the calcium accumulating activity of myocardial sarcoplasmic reticulum fractions. Whether the human heart is more like that of the rabbit, rat, dog or cat remains to be determined. With regard to the protective effect of nifedipine reported by Henry et al.,47 this is consistent with the hypothesis that increased diastolic tone during myocardial ischemia in man is related to increased intracellular Ca++ relative to the ability of sarcoplasmic reticulum to sequester Ca++. In summary, the present study demonstrates that factors both intrinsic and extrinsic to the left ventricle are altered during angina pectoris, and may play a role in the altered diastolic pressures that occur during angina pectoris. Coronary artery pressure and flow are increased during angina and may remain contributing factors. Peak -dP/dt and time constant T of left ventricular pressure fall during early diastole were both significantly affected by the development of angina. These findings suggest that impaired left ventricular relaxation is an important contributing factor in the previously demonstrated altered diastolic properties during angina pectoris.

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