Does Intracoronary Infusion of Fluosol-DA 20% Prevent Left Ventricular Diastolic Dysfunction During Coronary Balloon Angioplasty?

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Distal intracoronary infusion of the perfluorochemical Fluosol-DA 20% has been shown to prevent systolic dysfunction during coronary artery balloon occlusion in coronary angioplasty. To assess its effect on global diastolic dysfunction, a randomized, single-blind, crossover protocol comparing intracoronary infusion of Fluosol or no infusion (control) was performed during 60 s balloon inflations in 10 patients (mean age 67 years) undergoing coronary angioplasty. Assessment of global systolic and diastolic function was obtained with high fidelity micromanometer measurements of left ventricular pressure. Eighteen pairs of balloon inflations (Fluosol versus control) were analyzed.

Patients reported significantly less severe chest pain during inflations accompanied by Fluosol compared with control. However, during coronary balloon occlusion, no significant differences in the changes from baseline values were observed between Fluosol and control with regard to ventricular relaxation, including the time constant of early ventricular relaxation (tau) and maximal rate of fall in left ventricular pressure (maximal negative dP/dt). No differences between Fluosol and control were observed in terms of the increase in end-diastolic pressure or minimal diastolic pressure during balloon inflation. Mean systolic pressure decrease from baseline values was greater during control than during Fluosol inflations (-9.0 ± 3.3 mm Hg, p = 0.013), but no significant difference was observed in the change in maximal rate of rise in left ventricular pressure (maximal positive dP/dt).

These results suggest that Fluosol does not preserve global left ventricular diastolic function during coronary balloon occlusion, possibly because of its limited oxygen delivery capability relative to arterial blood.

The consequences of balloon inflation during percutaneous transluminal coronary angioplasty on left ventricular function have been well described and include significant depression in both systolic and diastolic function (1-5). These changes are usually reversible after balloon deflation, although in patients with preexisting left ventricular dysfunction, prolonged hemodynamic deterioration may follow. If an effective means of myocardial protection could be employed during balloon inflation, prolonged balloon inflations might be possible, which may improve initial angiographic results (6) and aid in managing acute complications of the procedure such as coronary dissection. It could also enhance the overall safety and comfort of coronary angioplasty and permit its wider use in high risk patients.

One approach to myocardial protection during coronary angioplasty has been to infuse the oxygenated perfluorochemical Fluosol-DA 20% through the lumen of the dilating balloon catheter. Distal coronary infusion of Fluosol during angioplasty appears to reduce ischemia (7) and ameliorate left ventricular systolic dysfunction as assessed by echocardiography (8,9).

Global diastolic dysfunction is a sensitive marker of myocardial ischemia and occurs earlier than global systolic dysfunction (10-12). Because this observation has also been noted during coronary angioplasty (1), we hypothesized that Fluosol, if effective in preventing ischemia, should prevent...
diastolic dysfunction during balloon inflation. Therefore, we evaluated the effect of distal catheter infusion of Fluosol on the dynamic changes in systolic and diastolic left ventricular pressure variables, particularly those of ventricular relaxation, in patients during coronary angioplasty in a controlled crossover study.

Methods

Patient selection. Patients with angina pectoris having elective coronary angioplasty at the Mayo Clinic were considered for this study. The study was approved by the Mayo Clinic's Institutional Review Board on February 26, 1988 and informed written consent was obtained from each patient before study.

Selection criteria for the study included 1) planned coronary angioplasty of ≥70% stenosis (visually assessed) of the proximal or mid-left anterior descending coronary artery, left circumflex coronary artery or right coronary artery, with no visible collateral flow supplying the vessel to be dilated; and 2) normal regional wall motion in the territory of the coronary artery to be dilated as determined by left ventriculography or echocardiography. Patients were excluded from consideration if there was 1) recent acute myocardial infarction; 2) distal vessel disease in the vessel to be dilated; 3) valvular heart disease; 4) rhythm other than sinus; and 5) child-bearing potential.

Fifteen patients were initially enrolled in the study. Two patients were withdrawn when it was discovered that the vessel to be dilated had become occluded since the diagnostic angiogram. A further two patients were withdrawn because of inability to cross the lesion in one and the finding of a previously unrecognized significant left main coronary stenosis in the other. Another patient completed the protocol, but the data were not analyzed because of baseline drift of the micromanometer signal during the study. Therefore, the study group consisted of 10 patients whose characteristics appear in Table 1; their mean age was 67 years. The dilated vessels were the left anterior descending coronary artery in eight patients, the circumflex artery in one and the right coronary artery in one.

Study design. The study was designed as a controlled, single-blind, randomized crossover trial with each patient undergoing four consecutive 60 s balloon inflations. During the first inflation, each patient was randomly assigned to receive either distal catheter infusion of Fluosol-DA 20% (Alpha Therapeutic) or undergo routine inflation without infusion. After balloon deflation, 3 min were allowed before the second inflation, at which time the patient crossed over to receive the alternative treatment. This sequence was repeated for the third and fourth inflations, maintaining the same order of randomization. Thus, each patient served as his or her own control and comparisons were made between the inflations within each pair.

| Table 1. Clinical and Angiographic Characteristics of 10 Patients |
|-----------------------------|------------------|
| Characteristic               |                   |
| Mean age ± SD (yr)          | 67 ± 9           |
| Men/women                   | 5/5              |
| Angina (CHA class)          |                   |
| I                           |                  |
| II                          | 7                |
| III                         | 3                |
| IV                          |                  |
| Site of angioplasty (coronary artery) |           |
| Left anterior descending    |                   |
| Proximal                    | 6                |
| Mid                         | 2                |
| Left circumflex             | 1                |
| Right                       |                  |
| Proximal                    | 1                |
| Mid                         |                  |
| Ejection fraction [%]*      | 73 ± 12          |

*Obtained in 6 patients. CHA = Canadian Heart Association.

Technique and medication. Coronary angioplasty was performed using Profile-Plus balloon catheters (USCI Division, C. R. Bard) inserted through the right femoral artery using standard techniques. Attempts to use a Mini-Profile balloon catheter (USCI) in one patient and The Skinny (SciMed) in another failed as a result of catheter rupture during Fluosol infusion. Before the procedure, all patients received 325 mg of aspirin. All but two patients were receiving a calcium channel antagonist at the time of angioplasty. two patients were treated with an oral long-acting nitrate, one with a beta-adrenergic blocking agent and two patients were receiving no regular medication. All patients were given sublingual nitroglycerin in the catheterization laboratory before angioplasty and received 10,000 to 15,000 U of intravenous heparin.

The Fluosol was thawed before the study, warmed to 37°C in a water bath and bubble-oxygenated for 30 to 60 min. The mean (±SD) of the partial pressure of oxygen (P'O2) of the Fluosol measured within 30 min of initiation of the infusion was 685 ± 57 mm Hg (range 613 to 776). Before the procedure, each patient received a 0.5 ml intravenous test dose of Fluosol. The infusate was maintained at body temperature until immediately before use and infused through the distal port of the balloon catheter at 60 ml/min by means of an angiographic power injector equipped with a warming sleeve (Angiomat 6000, Liebel-Flarsheim).

Hemodynamic measurements. Left ventricular pressure was measured using a 3F micromanometer catheter (Millar Instruments) inserted through a pigtail catheter positioned in the left ventricle by means of the left femoral artery. The tip of the pigtail catheter was situated approximately half-way between the mid-left ventricular cavity and apical levels and its position was checked periodically throughout the study to ensure that it had not changed. The catheter pressure signal was calibrated and digitized at 5 ms intervals by means of an
analogue to digital converter and stored for later analysis. During each inflation sequence, left ventricular pressure was recorded at end-expiration for 5 s immediately before balloon inflation, at 30 and 60 s during the inflation and at 60 s after balloon deflation.

Off-line data analysis was performed on an IBM-AT clone. Analysis of hemodynamic data was performed without knowledge of the randomization sequence. Three to four cardiac cycles were analyzed individually and a mean value obtained for each measurement. The following diastolic variables were measured: left ventricular end-diastolic pressure measured at the peak of the accompanying R wave of the electrocardiogram, minimal diastolic pressure, maximal rate of fall in left ventricular pressure (maximal negative dP/dt) and the time constant of ventricular relaxation (tau). Tau was described as the negative inverse of the slope of the natural logarithm of pressure versus time, as originally described by Weiss et al. (13), between the points of maximal negative dP/dt and 5 mm Hg above the previous end-diastolic pressure. A minimum of eight sample points was chosen for this measurement, but in one patient with a heart rate up to 102 beats/min, such sampling could be done only by measuring the second point at a pressure equal to the previous end-diastolic pressure. This latter definition of tau provides an estimation that is not significantly different from that obtained by measuring it up to 10 mm Hg above the previous end-diastolic pressure (1). A least squares analysis was used to determine the best fit ($r > 0.95$) of the data to this monoexponential function. Estimates of global systolic function were peak left ventricular pressure and maximal rate of rise in left ventricular pressure (maximal positive dP/dt).

Angina grading. Before the study, patients were instructed to grade their angina on a scale of increasing severity from 0 to 9 during the angioplasty procedure. The time of onset and duration of any pain were recorded throughout the procedure, as were the location, quality and relation to previous anginal symptoms. Patients were not aware of the order of inflations. The highest grade reported by the patient during the period of balloon inflation was used as an index of severity. The duration of any pain was also recorded.

Statistical analysis. All data are expressed as mean values ± SEM unless otherwise stated. The changes from baseline study observed at 30 and 60 s after balloon inflation and at 60 s after balloon deflation were recorded for each inflation. To reduce the statistical problem of multiple measurements during each inflation, individual changes from baseline values observed at 30 and 60 s were averaged. It should be emphasized, however, that apart from peak systolic pressure, no significant differences between Fluosol and control inflations at the individual time points of 30 or 60 s were found for any measured variable. For each patient, these changes from baseline values were calculated for each inflation and a comparison was made within each pair of inflations between Fluosol and control using paired t tests, with a significance level of alpha = 0.05.

On the basis of results of studies (8,9) showing complete preservation by Fluosol of systolic function during coronary occlusion, we estimated the statistical power to detect 100% preservation of diastolic function. Using the observed standard error of the difference between Fluosol and control, we computed the power, around the noncentral $t$ distribution, of rejection at the 0.05 level if the true reduction was equal to 100% of the observed change from the baseline value under control conditions. With this approach, the estimated statistical power of the measured diastolic variables was end-diastolic pressure 85%, minimal pressure 99%, maximal negative dP/dt 92% and tau 100%.

**Results**

**Results of balloon inflation.** Ten patients had hemodynamic data suitable for analysis. A mean (±SD) diameter stenosis, visually assessed, of the studied vessels of 84 ± 12% before angioplasty was reduced to 34 ± 22% after angioplasty. No adverse effects were noted in any patient after administration of the test dose of Fluosol nor during its intracoronary infusion. The procedure was successful in all except one patient, who had a severe coronary dissection during the third inflation and required emergency coronary artery bypass surgery with subsequent evidence of a non-Q wave infarction. In this patient, the data from only the first pair of inflations were used for analysis. In one other patient, the final inflation during Fluosol infusion was aborted at commencement of inflation after rupture of the balloon catheter without any clinical sequelae. Thus, there were 18 pairs of Fluosol versus control inflations available for comparison.

The duration of balloon inflation was 60 s for every inflation except the final noninflation inflation in one patient when the balloon was deflated after 35 s because of severe chest pain. The rest heart rate before inflations accompanied by Fluosol infusion was 68.4 ± 3.3 beats/min compared with 70.2 ± 3.4 beats/min before control inflations ($p = NS$). The heart rate after 60 s of balloon inflation during Fluosol infusion decreased to 66.1 ± 2.8 beats/min ($p = NS$) and decreased to 68.5 ± 3.3 beats/min during control inflations ($p = NS$).

**Hemodynamic variables (Table 2).** There were no significant baseline differences before inflations with or without Fluosol in any left ventricular diastolic or systolic variable. During balloon inflation, there were significant increases from preinflation values in all variables except maximal negative dP/dt and peak systolic pressure when inflations were accompanied by Fluosol infusion.

**Diastolic variables: Fluosol versus control (Table 3).** When paired Fluosol and control inflations were analyzed, no significant differences were observed for the changes from
Table 2. Hemodynamic Changes From Baseline (before inflation) During 60 s Balloon Inflations (n = 18)

<table>
<thead>
<tr>
<th>Hemodynamic Measurement</th>
<th>Fluosol or Control</th>
<th>Baseline (mean of 30 and 60 s)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastole</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>F</td>
<td>26.0 ± 2.6</td>
<td>31.1 ± 2.2</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>27.4 ± 2.0*</td>
<td>32.2 ± 2.1</td>
</tr>
<tr>
<td>Minimal pressure (mm Hg)</td>
<td>F</td>
<td>15.3 ± 1.9</td>
<td>20.8 ± 1.8</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>16.0 ± 1.6*</td>
<td>21.2 ± 1.8</td>
</tr>
<tr>
<td>Maximal negative dP/dt (mm Hg/s)</td>
<td>F</td>
<td>1.446 ± 79</td>
<td>1.334 ± 69</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>1.501 ± 80*</td>
<td>1.250 ± 68</td>
</tr>
<tr>
<td>Tau (ms)</td>
<td>F</td>
<td>51.2 ± 2.7</td>
<td>61.7 ± 4.0</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>51.3 ± 2.6*</td>
<td>64.8 ± 4.2</td>
</tr>
<tr>
<td>Systole</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak pressure (mm Hg)</td>
<td>F</td>
<td>144.4 ± 7.1</td>
<td>143.0 ± 6.7</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>148.2 ± 8.2*</td>
<td>137.7 ± 6.3</td>
</tr>
<tr>
<td>Maximal positive dP/dt (mm Hg/s)</td>
<td>F</td>
<td>1.548 ± 68</td>
<td>1.380 ± 69</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>1.586 ± 62*</td>
<td>1.301 ± 67</td>
</tr>
</tbody>
</table>

*p = not significant (Fluosol versus control). C = control; F = Fluosol; LVEDP = left ventricular end-diastolic pressure; Maximal negative dP/dt = maximal rate of fall in pressure; Maximal positive dP/dt = maximal rate of rise in pressure; Tau = time constant of ventricular relaxation.

Systolic variables: Fluosol versus control (Table 3). Paired comparison between inflations with or without accompanying Fluosol infusion revealed a small but significant difference in the change from baseline peak systolic pressure (Fig. 3A). During control inflations, systolic pressure decreased more compared with Fluosol inflations (-9.0 ± 3.3 mm Hg, p = 0.013). However, no significant difference was observed in the changes in maximal positive dP/dt (Fig. 3B). No significant differences were noted between the two types of inflations in either of these two variables at 60 s after deflation (Fig. 4).

Angina during coronary angioplasty. Anginal pain reported by patients was significantly less during balloon inflations accompanied by Fluosol infusion than inflations without. The reported peak intensity in patients during Fluosol inflations was 2.7 ± 0.6 versus 4.4 ± 0.8 during control inflations (p < 0.001). The average duration of pain was similar (41 ± 16 s during Fluosol inflations and 46 ± 13 s during control inflations, p = NS).

Discussion

Previous studies (1,2,10–12) have demonstrated that global diastolic dysfunction is usually an earlier and more sensitive marker for myocardial ischemia than changes in global systolic function. Hemodynamic studies (1,3) during coronary artery balloon occlusion have demonstrated that the time constant of ventricular relaxation (tau), left ventricular end-diastolic pressure and minimal pressure all increase and that peak left ventricular pressure and maximal positive and
Figure 1. Paired comparison between Fluosol and control inflations in the change from baseline measurements to inflation of the diastolic variables. A, Left ventricular end-diastolic pressure. B, Minimal diastolic pressure. C, Maximal rate of fall in pressure (maximal negative dP/dt). D, Time constant of ventricular relaxation (tau).

Figure 2. Paired comparison between Fluosol and control inflations in the change from baseline measurements to recovery (60 s after deflation) of the diastolic variables. A, Left ventricular end-diastolic pressure. B, Minimal diastolic pressure. C, Maximal rate of fall in pressure (maximal negative dP/dt). D, Time constant of ventricular relaxation (tau).
negative dP/dt decrease. In addition, Serruys et al. (1) have shown that the relaxation variables during early diastole, tau and maximal negative dP/dt precede changes in left ventricular end-diastolic pressure and the hemodynamic variables of systolic function. Therefore, these diastolic variables may more accurately reflect the efficacy of myocardial protection techniques during coronary angioplasty than do other more frequently used measurements.

Present study. In this study, we found that, as expected, there were significant changes during the control coronary balloon inflations in all of the measured variables. Distal catheter infusion of Fluosol during coronary angioplasty reduced the degree of angina experienced by patients and prevented any significant decrease in peak systolic pressure, but the benefit over control inflations appeared small. Compared with control inflations, the changes observed during balloon inflation with Fluosol in end-diastolic pressure, minimal diastolic pressure, tau and maximal negative dP/dt were not significantly different. Similarly, there were no differences between the two types of inflation with respect to the recovery of these variables at 1 min after balloon deflation.

Clinical relevance. If any method of myocardial protection is to be clinically useful (for example, in allowing prolonged balloon inflations or performing coronary angioplasty in high risk patients with limited ventricular contractile reserve), it should be capable of completely or almost completely preventing ischemia and left ventricular dysfunction. Global responses (for example, in left ventricular end-diastolic pressure) are relevant to the acute management of the patient during angioplasty. Prolongation of ventricular relaxation and abnormally elevated diastolic pressures will adversely affect coronary perfusion, wall stress and pulmonary venous emptying. Therefore, because our results suggest that the effect of Fluosol during 60 s of coronary occlusion with balloon inflation appears minimal in patients with relatively normal left ventricular function, its efficacy in patients with preexisting ventricular dysfunction could be limited. However, this needs to be studied carefully using both echocardiographic and hemodynamic measurements.

Previous studies. Animal studies. Virmani et al. (14) showed no significant difference in left ventricular pressure, heart rate or maximal positive dP/dt in dogs during eight 90 s balloon inflations with or without Fluosol infusion. They also found ultrastructural changes on electron microscopy consistent with myocardial ischemia in animals treated with Fluosol, suggesting that prevention of ischemia was not complete. In a canine study of 2 min coronary balloon

![Figure 3](image1.png)  
**Figure 3.** Paired comparison between Fluosol and control inflations in the change from baseline measurements to inflation of the systolic variables. A, Peak left ventricular systolic pressure. B, Maximal rate of rise in pressure (positive dP/dt).

![Figure 4](image2.png)  
**Figure 4.** Paired comparison between Fluosol and control inflations in the change from baseline measurements to recovery (60 s after deflation) of the systolic variables. A, Peak left ventricular systolic pressure. B, Maximal rate of rise in pressure (positive dP/dt).
infections with or without distal Fluosol infusion. Tokioka et al. (15) demonstrated only partial improvement of regional and global myocardial function with Fluosol compared with control. Fluosol did not prevent the production of lactate or the development of ST segment elevation on the electrocardiogram and only catheter perfusion of arterial blood offered complete myocardial protection in this same study.

In another canine study comparing Fluosol and autologous blood as coronary perfusion agents during ischemia, Christensen et al. (16) found that Fluosol induced a significant decrease in diastolic coronary pressure and resistance. They also observed that there was a relative decrease in subendocardial blood flow, with a significant reduction in the amount of oxygen delivery to this region. In contrast, autologous blood was able to maintain normal flow and oxygen delivery.

**Human studies.** Clinical studies examining the efficacy of Fluosol infusion during coronary angioplasty with respect to left ventricular performance have generally employed quantitative echocardiographic techniques. These studies indicated that Fluosol completely prevented regional wall motion abnormalities (8) and preserved global ejection fraction (9). However, because hemodynamic variables were not measured in either study, direct comparison with the present study is not possible.

Despite the results of these studies, possible methodologic problems should be considered. The method of measuring chord shortening to quantify regional wall motion used in the first study (8) was originally described for analysis of contrast ventriculograms; in addition to its own limitations (17), it may not necessarily be applicable to conventional echocardiography with its inherent limitations of suboptimal spatial resolution and noise, patient motion and the requirement that the imaging plane remain constant throughout the 60 to 90 s of balloon inflation. Problems in quantifying echocardiographic images have also been described previously (18).

Apparent improvement in diastolic function, using Doppler-derived indexes of mitral flow velocity, during angioplasty accompanied by Fluosol infusion was described in preliminary form from the same group (19). However, recent studies (20–22) concerned with the hemodynamic validation of current Doppler indexes of diastolic function have stressed important limitations in the interpretation of some of these indexes.

**Diastolic versus systolic dysfunction.** Although coronary perfusion with Fluosol may preserve regional (8) as well as global (9) systolic function, our results suggest that it does not preserve global diastolic function. It is possible that systolic function was also preserved in our study as indicated by the effect on peak systolic pressure, although no beneficial effect on maximal positive dP/dt was observed. One possible explanation for these disparate findings may be that Fluosol is a relative volume expander. Although this property of Fluosol could favorably influence both regional and global systolic function, it may adversely influence ventricular relaxation. However, it should be realized that sudden volume expansion is unlikely to be well tolerated by patients with severe left ventricular dysfunction.

*An alternative explanation* is that despite the potential to preserve regional and global systolic function, Fluosol is unable to preserve global diastolic function. Dissociation between systolic and diastolic measurements of ventricular dysfunction has been documented in various cardiac diseases including ischemia (10–12). This could be explained by the greater vulnerability of diastolic function to ischemia compared with systolic function (10, 23) and the inability of Fluosol to completely prevent myocardial ischemia (15, 16).

**Oxygen delivery.** The inability of Fluosol to completely prevent myocardial ischemia may be related to its inferior oxygen delivery to the myocardium relative to that of arterial blood. Normal coronary blood flow at rest (for example, in the left anterior descending artery) varies among individuals, but an average flow of 80 ml/min is assumed. Normal myocardial oxygen extraction in the left ventricle is much higher than for most other organs and is approximately 12 vol% (24), which is extracted from arterial blood with an oxygen content of 17 to 18 vol%. The oxygen content of 6 vol% (25) for Fluosol, coupled with its delivery rate of 60 ml/min indicate that it is capable of delivering only about one third the volume of oxygen to the left ventricle compared with normal arterial blood. One method to overcome this would be to infuse Fluosol at a higher rate, but this could present some technical problems.

Furthermore, during manipulation of the balloon catheter, severe ischemia may result because of subtotal occlusion by the deflated balloon at the site of a severe stenosis (we actually observed an increase in left ventricular end-diastolic pressure in most patients during the positioning of the balloon). The normal response of the coronary circulation after relief of this obstruction is to increase coronary flow briefly to about four to six times that of the flow at rest, the so-called hyperemic response. “Relief” of this occlusion in this study was obtained at the time of balloon inflation with perfusion of Fluosol, but as discussed, the relatively inefficient delivery of oxygen would, at least initially, be unable to meet the demands of the ischemic myocardium.

**Limitations of study.** When interpreting the results of this study, there are potentially important limitations to consider. A relatively small number of patients were studied. Also, although there were no differences among diastolic variables to favor Fluosol, a small possibility of a type II error is acknowledged. However, in our study there was sufficient power (85% to 100%) to detect complete preservation of these variables, which was the same magnitude of effect on systolic function observed in previous studies (8, 9).

Regional ventricular function was not assessed in this study and our measurements were probably insensitive to...
changes in regional diastolic function. In comparing infusion of Fluosol with "no infusion," it is possible that despite the low viscosity of Fluosol the hydraulic effect of infusion through the coronary arteries altered the diastolic properties of the ventricular wall, although the effects of this would appear to be the same as ischemia alone. However, because patients did experience chest pain of similar duration during Fluosol infusion, although less severe, these findings are most likely secondary to ischemia.

Finally, there are also some possible limitations to consider in the measurements used in our study. Tau can be computed for a number of mathematical models of pressure decay, but may not always reflect true ventricular relaxation, although the mathematical model for describing tau used in this study appears to more closely reflect the known physiology (26).

Conclusion. Distal catheter infusion of Fluosol during coronary angioplasty does not preserve global diastolic function and specifically does not prevent abnormal ventricular relaxation. This raises the possibility that Fluosol may be sufficient to lessen systolic but not global diastolic deterioration of function during coronary occlusion. Future studies should address this possibility.

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References


