Effect of Intra-aortic Balloon Counterpulsation on the Motion and Perfusion of Acutely Ischemic Myocardium

An Experimental Echocardiographic Study

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SUMMARY The effect of intra-aortic balloon counterpulsation (IABC) on the motion and perfusion of ischemic left ventricular posterior myocardium was studied in 30 open-chest dogs, using ultrasound to register motion and 7-10 μ radioactive microspheres to determine perfusion. Circumflex coronary artery ligation produced acute aneurysmal bulging during isovolumic contraction and diminished ischemic wall velocity during systolic ejection. Myocardial perfusion was determined in five dogs; perfusion of the area supplied by the ligated coronary artery fell from a control value of 72.9 ± 13.8 (SE) to 30.0 ± 2.3 cc/100 g/min (P < 0.05) at 5 minutes after coronary occlusion. IABC was then administered for one hour, with a fall in aortic systolic pressure (112 ± 6 to 105 ± 7 mm Hg, P < 0.05) and rise in peak aortic diastolic pressure (94 ± 6 to 102 ± 7 mm Hg, P < 0.05). Despite this the ischemic area showed no change in perfusion (measured at the same time): 30.0 ± 2.3 to 28.0 ± 2.4 cc/100 g/min. Little change in wall motion occurred: aneurysmal bulging decreased modestly (4.5 ± 0.3 to 3.6 ± 0.3 mm, P < 0.05), but ischemic wall velocity did not increase. After cessation of counterpulsation and one hour of coronary reperfusion aneurysmal bulging disappeared and wall velocity improved. The addition of norepinephrine (eight dogs) or nitroprusside (seven dogs) to intra-aortic balloon counterpulsation did not cause a significant further improvement in the response of the dyskinesis during the period of ischemia. We conclude that IABC has little effect on ischemic dyskinesis, probably due to its failure to improve perfusion of the acutely ischemic myocardium.

INTRA-AORTIC BALLOON COUNTERPULSATION (IABC) is an effective clinical method for the treatment of cardiogenic shock and refractory left ventricular failure.1 2 It has been shown to reduce chest pain and electrocardiographic changes in patients with preinfarction angina3 and to diminish the size of an experimental myocardial infarction.4 Interventions which alter infarct size have been shown experimentally to affect segmental cardiac wall motion.5 6 No information is currently available, however, on the effect of IABC on the segmental dyskinesis of ischemic myocardium. This is important since the known beneficial effects of IABC on overall ventricular performance may be due to reduction of segmental dyskinesis, or to reduced afterload and improved coronary perfusion, or both. The purposes of this study were 1) to determine if IABC has a beneficial effect on ischemic dyskinesis; 2) to establish the relationship of changes in ischemic dyskinesis, if any, to changes in perfusion of the ischemic areas; 3) to ascertain the effects on dyskinesis during IABC of further manipulation of arterial pressure by administering norepinephrine or nitroprusside in addition to IABC; and 4) to ascertain if the improvement in dyskinetic motion known to occur following coronary reperfusion is enhanced by IABC, with or without additional drugs.

Methods

Thirty adult mongrel dogs weighing 15 to 25 kg were anesthetized with sodium pentobarbital, 25 mg/kg i.v. Respiration was maintained by a Harvard respirator and a cuffed endotracheal tube. Periodic hyperinflation and supplemental low flow oxygen to inspired air allowed us to maintain arterial Po2 and pH within a physiologic range. A midsternal thoracotomy was performed, the pericardium incised, the heart exposed. The circumflex coronary artery was dissected free from the epicardial fat, and either an inflatable balloon cuff or a loose ligature was placed around it. Heparin (250 units/kg) was administered intravenously, and cannulae were placed directly in the right brachial and femoral arteries and the left atrial appendage. Polyurethane catheters (#8 French) were inserted into the ascending aorta and left ventricle by retrograde catheterization; arterial and left ventricular pressures were measured with Statham P23 strain gauges. Left ventricular dp/dt was determined from the left ventricular pressure tracing by an RC differentiation circuit. Cardiac output minus coronary artery flow was determined by an electromagnetic flow probe placed around the ascending aorta. All recordings were made utilizing an Electronics-for-Medicine DR12 multichannel photographic recorder. Aortic balloon counterpulsation was accomplished by use of a DataScope-80 counterpulsation device, using a dual-chamber balloon catheter containing 30 cc of air, placed in the descending thoracic aorta. Peak aortic diastolic pressure during counterpulsation was determined; the time between this point and the preceding incisura on the aortic pulse wave was measured. The aortic pressure at the same interval from the preceding incisura was measured.

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during these recording periods when the counterpulsation device was not being used, and this pressure was then considered peak diastolic pressure for comparison with the peak diastolic pressure achieved during counterpulsation.

In some animals either no substantial increase in diastolic pressure could be achieved by counterpulsation, or an increase was achieved only by concomitantly raising systolic pressure as well. Such results may have been due to very rapid heart rates with incomplete balloon relaxation before the onset of systole, and/or a small aorta so that the balloon largely occluded the aorta in systole as well as diastole. Data from such animals were not included in the results; ten additional animals were thereby excluded.

Ultrasound recordings of the motion of the interventricular septum and left ventricular posterior wall were obtained using a 2.25 MHz unfocussed transducer and ultrasonoscope (Smith-Kline Ekoline 20); the ultrasound signal was displayed on the photographic recorder simultaneously with the pressure, dp/dt and electrocardiographic signals. The method involved in obtaining the recordings has been described in detail in a previous report from our laboratory. Briefly, the ultrasound transducer was placed lightly on the exposed anterior surface of the heart and directed inferiorly to the mitral leaflet echoes so as to record the characteristic motion of the left ventricular posterior wall. The sensitivity of the ultrasonoscope was manipulated so as to best define the ultrasonic signal from the left ventricular endocardium. The transducer was fixed in place by a stationary arm and strips of tape to avoid transmitted motion from the heart in order to provide a fixed reference point. Once fixed in place, the transducer was neither lifted nor moved throughout the study. Verification of the ultrasound identification was achieved by rapid injection of 5 cc of normal saline or cardiogreen dye through the left ventricular or left atrial catheters; this maneuver produces ultrasonic contrast reflections which fill the chamber and outline the endocardial-blood interface bordering the ventricular cavity. Standard designations were used for the labeling and description of left ventricular posterior endocardial motion4 (fig. 1). Point B, the posterior wall position at end-diastole, is approximately simultaneous with the R wave of the ECG. During isometric contraction the wall moves posteriorly from B to C. The anterior motion from C to D is coincident with ventricular ejection. The mean posterior endocardial wall velocity (WV) was obtained by calculating the slope of the line drawn from the onset (C) to the end (D) of ventricular ejection, in mm/sec. Posterior endocardial wall excursion (WE) was obtained by measuring the amplitude of posterior wall motion as the vertical distance from C to D, in millimeters. The left ventricular end-diastolic diameter was measured, at the R wave of the ECG, as the distance between the left side of the interventricular septum and the left ventricular posterior endocardium, in mm.

Left ventricular myocardial perfusion was determined by use of 7-10 microspheres labeled with 141Ce, 88Sr, 51Cr and 44Sc. The average number of microspheres in each injection was 6.6 ± 1.9 × 106. The microspheres were suspended in 0.1 to 3.0 ml of saline and injected over a 10 second period into the left atrium, and the cannula was then flushed with 5 ml of saline at 37oC. Left ventricular and aortic pressures were recorded during this period to assure that no hemodynamic alteration occurred; previous investigators9 have demonstrated that injection of as much as 8 × 106 microspheres directly into the coronary circulation does not alter coronary vascular resistance. Prior to injection, the vial containing the microspheres and one drop of Tween-80 was vigorously agitated mechanically for at least four minutes. Microscopic examination of microspheres prepared in this manner showed dispersion of at least 98% of the spheres. Occasional small groups of three to five spheres were observed. Starting one minute before injection and continuing until three minutes after injection, blood for reference flow determinations was withdrawn simultaneously from the right brachial and right femoral arteries at 2.06 ml/min with a Harvard pump.

After all recordings were completed, two metal probes were lined up on either side of the ultrasound transducer and passed through the heart in parallel to mark the path of the ultrasound beam. To minimize deformation of the left ventricular wall (and consequent errors in beam localization) we used thin, #20 needles, with sharp points. The maneuver was done on the beating heart so as to avoid any deformity of the ventricle which might occur after death. The points of in-

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**FIGURE 1. Echocardiographic and pressure recordings in a typical dog.** EKG = electrocardiogram, IV = interventricular, Ao = aortic, LV = left ventricular, WV = wall velocity. See text for explanation of points B, C, D and E.
tersection of the probes with the left ventricular posterior endocardium were noted and the myocardial segments between these points subsequently identified, in order to verify that the specific segments traversed by the ultrasound beam were in fact hypoperfused segments from the area supplied by the ligated coronary artery. The animals were then killed with an injection of potassium chloride. The heart was excised and the free walls of the right ventricle, the right and left atrium, great vessels, valves, surface vessels and epicardial fat were removed. Utilizing the posterior descending coronary as a starting point, the left ventricle was divided into four equal levels of eight segments each, and each segment was divided into three layers — endocardium, mid-wall and epicardium. Thus the left ventricle was divided into 96 segments of about 1.6 x 1.6 x 0.3 cm in size, with an average weight of 0.8 g. Since the size of the probes was very small compared to the size of the segments, errors in beam localization were minimized. The relative geometric position of each segment was constant from animal to animal.

Using techniques previously described in detail we determined the perfusion of each of the 96 small myocardial segments as well as the size of the ischemic area and the endocardium-epicardium perfusion ratio. Ischemic segments were identified utilizing a statistical method which in effect estimates the heterogeneity of perfusion to normally perfused segments and then uses this information to establish the level below which perfusion of normal segments does not fall. Segments found to have such abnormally low perfusion (seen only following coronary ligation) were classified as ischemic. All segments classified as ischemic were then divided into three subgroups on the basis of the severity of perfusion deficit. In the present study the average perfusions of severely ischemic, moderately ischemic and borderline ischemic segments expressed as a percent of normal perfusion were 14.9 ± 2.1%, 38.0 ± 2% and 61.1 ± 1.5%, respectively.

Using these techniques, the following experimental protocol was employed. The animals were divided into three groups. In Group I, 15 animals, control hemodynamic and ultrasound recordings were made. In order to assess the effect of IABC on normal myocardium, counterpulsation was begun in the first ten animals in this group at this point (i.e., precoronary occlusion) and continued for 5 min, when a second set of hemodynamic and ultrasound recordings was obtained. IABC was then discontinued, the animals allowed to stabilize for 5 min, and then acute ischemia was created by occlusion (with a snare device) of the posterior descending coronary artery. Recordings were obtained 5 min later, and then IABC was begun again and continued for one hour. After this IABC was stopped, the coronary occlusion released, and coronary reperfusion allowed to occur for one hour. Recordings during this period of ischemia and IABC were obtained at 15 min, 30 min and one hour; similarly recordings during coronary reperfusion were obtained at 15 min, 30 min and one hour. In Group II, eight animals, counterpulsation was administered during the one hour of ischemia and these dogs also received norepinephrine intravenously at rates of 1–8 μg/min in order to raise mean aortic pressure to approximately 133% of the level noted after coronary ligation (before counterpulsation and norepinephrine infusion was begun). In Group III, six animals, counterpulsation was administered during the one hour of ischemia and these dogs also received nitroprusside intravenously at rates of 0.1 to 0.2 mg/min in an attempt to lower mean aortic pressure to approximately 67% of the level noted after coronary ligation (just before counterpulsation and nitroprusside infusion was begun). Recordings in these groups were also obtained at 15 min, 30 min and one hour of ischemia. Counterpulsation and drugs were discontinued after recordings were made at one hour of ischemia plus interventions; the coronary ligature was released and reperfusion allowed to occur for an additional hour, during which recordings were made at 15 min, 30 min and one hour.

In the last five animals of Group I radioactive microspheres were also injected during the hemodynamic and ultrasound recordings made at control, 5 min after coronary occlusion, after 60 min of counterpulsation and after 60 min of coronary reperfusion, to determine the effect of counterpulsation on myocardial perfusion. These five animals were not selected for any hemodynamic criteria; subsequent analysis confirmed that hemodynamic and echocardiographic data from these five dogs were similar to the other ten dogs in Group I.

Statistical analysis of the data was performed using Student’s paired t-test. All results are expressed as the mean ± 1 standard error.

Results

Effects of IABC on Normal Myocardium

IABC produced diastolic pressure augmentation so that peak aortic diastolic pressure exceeded peak aortic systolic pressure (table 1). No other significant changes in hemodynamics or cardiac wall motion occurred when IABC was administered before coronary occlusion.

Effects of Acute Circumflex Coronary Artery Occlusion

The changes in cardiac wall motion and hemodynamics produced by acute occlusion of the circumflex coronary

<table>
<thead>
<tr>
<th>Table 1. Effect of Aortic Counterpulsation on the Motion of Nonischemic Myocardium in Ten Dogs (Percoronary Occlusion)</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td>PWV (mm/sec)</td>
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<tr>
<td>B-C (mm)</td>
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<tr>
<td>PWE (mm)</td>
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<tr>
<td>EDD (mm)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
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<td>Aortic diastolic pressure (mm Hg)</td>
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<tr>
<td>Aortic mean pressure (mm Hg)</td>
</tr>
<tr>
<td>Peak aortic diastolic pressure (mm Hg)</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
</tr>
<tr>
<td>Cardiac output (ml/min)</td>
</tr>
</tbody>
</table>

* = P < 0.05 control vs counterpulsation.

All values are mean ± s.e.

Abbreviations: PWV = posterior wall velocity; PWE = posterior wall excursion; B-C = B-C amplitude (see text); EDD = end-diastolic diameter; LV = left ventricle.
artery were similar to those previously described by us. Posterior wall velocity and excursion decreased and aneurysmal bulging (increased B-C amplitude during isometric contraction) developed (tables 2-4). These changes reflect localized dyskinesis of acutely ischemic myocardium, and do not necessarily indicate alterations of generalized left ventricular function, nor are such marked motion abnormalities seen in areas remote from the site of occlusion. Left ventricular end-diastolic diameter increased; aortic pressures and cardiac output and left ventricular dp/dt fell.

Myocardial perfusion of the areas not supplied by the ligated coronary artery did not change significantly (table 2). In contrast, there was a sharp fall in the perfusion of the acutely dyskinetic area struck by the ultrasound beam, from 72.9 ± 13.8 to 30.0 ± 2.3 ml/100 g/min, P < 0.05 (fig. 2). The endocardium-epicardium perfusion ratio in this area declined significantly, indicating the endocardial layer was more severely ischemic. The number of ischemic segments caused by coronary occlusion was 15.0 ± 2.6 (out of 96 total segments) representing 15.2 ± 2.7% of total left ventricular weight.

Effects of Interventions during One Hour of Ischemia Counterpulsation (table 2)

Intra-aortic balloon counterpulsation produced diastolic aortic pressure augmentation; the difference between aortic peak and end-diastolic pressures rose from 6 mm Hg in the early postischemic period (before counterpulsation was initiated) to 23 mm Hg with one hour of counterpulsation. Systolic unloading also occurred as peak aortic systolic pressure declined from 112 to 105 mm Hg. These changes were accompanied by a decline in B-C amplitude (aneurysmal bulging) and left ventricular end-diastolic diameter. Ischemic wall velocity and excursion did not change. Neither myocardial perfusion nor the endocardium-epicardium perfusion ratio of the entire ischemic area improved during counterpulsation (fig. 2); nor was there any selective improvement in the perfusions of the severely, moderately or borderline ischemic areas when these are examined separately (table 2).

The hemodynamic and echocardiographic changes produced by counterpulsation (without or with drugs) were virtually identical on the recordings obtained at 15 minutes, 30 minutes and 60 minutes of counterpulsation. In the in-

### Table 2. Effect of Aortic Counterpulsation and Coronary Reperfusion on Ischemic Dyskinesis in Fifteen Dogs

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Ischemia (5 min)</th>
<th>Ischemia + counterpulsation (60 min)</th>
<th>Reperfusion (60 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PWV (mm/sec)</td>
<td>27 ± 3</td>
<td>12 ± 1*</td>
<td>12 ± 2</td>
<td>17 ± 1†</td>
</tr>
<tr>
<td>B-C (mm)</td>
<td>2.6 ± 0.2</td>
<td>4.5 ± 0.3*</td>
<td>3.6 ± 0.3†</td>
<td>2.3 ± 0.2</td>
</tr>
<tr>
<td>PWE (mm)</td>
<td>2.4 ± 0.3</td>
<td>1.9 ± 0.2*</td>
<td>1.8 ± 0.2†</td>
<td>2.4 ± 0.1†</td>
</tr>
<tr>
<td>EDD (mm)</td>
<td>24.9 ± 1.7</td>
<td>27.7 ± 1.7*</td>
<td>27.5 ± 1.7†</td>
<td>21.8 ± 1.9†</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>103 ± 5</td>
<td>157 ± 5</td>
<td>158 ± 7</td>
<td>150 ± 7</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
<td>118 ± 8</td>
<td>112 ± 6</td>
<td>105 ± 7†</td>
<td>103 ± 6†</td>
</tr>
<tr>
<td>Aortic diastolic pressure (mm Hg)</td>
<td>91 ± 6</td>
<td>88 ± 5</td>
<td>80 ± 6†</td>
<td>78 ± 6†</td>
</tr>
<tr>
<td>Aortic mean pressure (mm Hg)</td>
<td>100 ± 7</td>
<td>96 ± 5</td>
<td>93 ± 7</td>
<td>87 ± 6†</td>
</tr>
<tr>
<td>Peak aortic diastolic pressure (mm Hg)</td>
<td>104 ± 7</td>
<td>94 ± 6*</td>
<td>103 ± 7†</td>
<td>88 ± 6†</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
<td>2169 ± 157</td>
<td>1511 ± 156*</td>
<td>1482 ± 152</td>
<td>1447 ± 162†</td>
</tr>
<tr>
<td>Cardiac output (ml/min)</td>
<td>2578 ± 277</td>
<td>2294 ± 272*</td>
<td>2047 ± 265†</td>
<td>1891 ± 342†</td>
</tr>
</tbody>
</table>

Myocardial perfusion - nonischemic area (ml/100 g/min) 85.3 ± 9.5 75.5 ± 7.8 63.7 ± 5.3 78.0 ± 1.5
Myocardial perfusion - entire ischemic area (ml/100 g/min) 72.9 ± 13.8 30.0 ± 2.3* 28.0 ± 2.4 76.4 ± 36.9
Myocardial perfusion - severely ischemic area (ml/100 g/min) 72.3 ± 21.7 12.1 ± 1.8* 10.8 ± 2.8 63.1 ± 54.0
Myocardial perfusion - moderately ischemic area (ml/100 g/min) 82.3 ± 18.1 28.1 ± 1.6* 27.8 ± 1.7 86.5 ± 53.8
Myocardial perfusion - borderline ischemic area (ml/100 g/min) 63.1 ± 8.9 45.9 ± 4.5* 41.6 ± 3.3 63.2 ± 14.3
Endocardium - epicardium - perfusion ratio 1.17 ± 0.23 0.55 ± 0.04* 0.54 ± 0.11 0.83 ± 0.22

All values are mean ± SE. * = P < 0.05 control vs ischemia. † = P < 0.05 control vs reperfusion. ‡ = P < 0.05 ischemia vs ischemia + counterpulsation.
terest of brevity we have therefore presented only the data obtained at 60 minutes in tables 2-4. The same is true of the coronary reperfusion data obtained after 15, 30 and 60 minutes, and only the 60 minutes data are presented.

Using the same techniques and methods as in the present investigation, we have previously studied the changes which occurred in myocardial perfusion and ischemic dyskinesis during periods ranging from one to four hours after circumflex coronary occlusion.\textsuperscript{6, 12, 13} Thus the effect of time only on these parameters has been evaluated. Using radioactive labeled microspheres,\textsuperscript{12} we previously showed that no significant changes occurred in myocardial perfusion or endocardial-epicardiac ratio during the period between five minutes and 60 minutes after coronary occlusion. Thus, in this study, the presence or absence of counterpulsation during the ischemic period made no additional difference to perfusion of the ischemic areas. Similarly, in two previous studies of ischemic wall motion using echocardiography we showed no change in either B-C amplitude (aneurysmal bulging) or ischemic wall velocity during a one hour period\textsuperscript{6} or a four hour period\textsuperscript{13} after coronary occlusion. In figures 3-4 we have plotted data from one of these earlier studies\textsuperscript{6} against data from this investigation. For both B-C amplitude (aneurysmal bulging) and ischemic wall velocity the changes in wall motion affected by counterpulsation are little different from those which occurred without any intervention during ischemia, although the small improvement in B-C amplitude during counterpulsation is statistically significant.

Counterpulsation Plus Norepinephrine (table 3)

The addition of norepinephrine to counterpulsation produced an elevation of aortic pressures and left ventricular dp/dt. Ischemic wall velocity tended to increase and aneurysmal bulging to decrease (figs. 5 and 6). A larger decline in ventricular end-diastolic diameter was noted when norepinephrine was added to IABC.

Counterpulsation Plus Nitroprusside (table 4)

The addition of nitroprusside to counterpulsation caused aortic pressures to decline. Left ventricular diameter and aneurysmal bulging declined, and ischemic wall velocity increased (figs. 5 and 6).

![Table 3. Effect of Aortic Counterpulsation with Norepinephrine and Coronary Reperfusion on Ischemic Dyskinesis in Eight Dogs](image)

![Figure 3. Aneurysmal bulging: Effect of aortic counterpulsation during ischemia compared with no intervention during ischemia. The reduction in aneurysmal bulging by counterpulsation was larger and statistically significant compared to the insignificant decline in the no-intervention group.](image)

![Figure 4. Ischemic posterior wall velocity: Effect of aortic counterpulsation during ischemia compared with no intervention during ischemia. No significant change in wall velocity during ischemia occurred in either group; subsequent response to reperfusion was also similar.](image)
Effects of Coronary Reperfusion

Release of the coronary occlusion resulted in a restoration of myocardial perfusion to control levels (fig. 2). Other studies of coronary reperfusion using large groups of animals have yielded similar results. Aortic pressures, LV dp/dt and cardiac output remained lower than control. In all three groups of animals the aneurysmal bulging which had been induced by coronary ligation was abolished by reperfusion (fig. 3), but the ischemic wall velocity and excursion, although increased compared to the post occlusion levels, remained significantly below control (fig. 4).

Discussion

This study establishes that the improvement in segmental ischemic dyskinesis produced by aortic balloon counterpulsation was minimal. A small but significant reduction in aneurysmal bulging during isometric contraction occurred, but there was no increase in ischemic wall velocity or excursion during ventricular ejection. In a previous study from this laboratory using a similar model directionally similar changes in both these parameters occurred during sixty minutes of ischemia without intervention.

Acute alterations in myocardial perfusion correlate well with changes in cardiac wall motion following acute coronary ligation. In the present study, the failure of IABC to produce large changes in wall motion may in part be due to lack of an increase in myocardial perfusion of the involved area during IABC. Two previous studies of the effect of counterpulsation on myocardial perfusion, by Shaw et al. and Watson et al. also showed that IABC, when begun one hour or 20 minutes after coronary occlusion, did not increase myocardial perfusion in the ischemic region. These studies did not study the effect of IABC on dyskinesis.

We have previously established in a similar experimental model that norepinephrine and nitroprusside alter ischemic dyskinesis, and wondered if these agents would increase the

![Figure 5. Effect of aortic counterpulsation alone and with norepinephrine or nitroprusside on aneurysmal bulging during isometric contraction (B-C amplitude). Decreased bulging occurred with counterpulsation with or without drugs, and further improvement was seen with reperfusion.](image1)

**TABLE 4. Effect of Aortic Counterpulsation with Nitroprusside and Coronary Reperfusion on Ischemic Dyskinesis in Seven Dogs**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Ischemia (5 min)</th>
<th>Ischemia + counterpulsation (60 min) + norepinephrine</th>
<th>Reperfusion (60 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PWV (mm/sec)</td>
<td>30 ± 5</td>
<td>14 ± 4*</td>
<td>19 ± 3</td>
<td>19 ± 2*</td>
</tr>
<tr>
<td>B-C (mm)</td>
<td>2.3 ± 0.4</td>
<td>4.5 ± 1.1*</td>
<td>3.1 ± 0.4</td>
<td>2.6 ± 0.4</td>
</tr>
<tr>
<td>PWE (mm)</td>
<td>3.9 ± 0.4</td>
<td>1.8 ± 0.4*</td>
<td>2.0 ± 0.2</td>
<td>2.6 ± 0.3</td>
</tr>
<tr>
<td>EDD (mm)</td>
<td>20.5 ± 3.7</td>
<td>23.8 ± 2.4</td>
<td>20.0 ± 3.2†</td>
<td>18.3 ± 2.4†</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>159 ± 7</td>
<td>156 ± 9</td>
<td>155 ± 6</td>
<td>154 ± 8</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
<td>121 ± 8</td>
<td>102 ± 8*</td>
<td>79 ± 7†</td>
<td>101 ± 9†</td>
</tr>
<tr>
<td>Aortic diastolic pressure (mm Hg)</td>
<td>88 ± 7</td>
<td>77 ± 7</td>
<td>52 ± 7†</td>
<td>77 ± 8</td>
</tr>
<tr>
<td>Aortic mean pressure (mm Hg)</td>
<td>98 ± 7</td>
<td>86 ± 7*</td>
<td>63 ± 7†</td>
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<tr>
<td>Peak aortic diastolic pressure (mm Hg)</td>
<td>91 ± 8</td>
<td>79 ± 8</td>
<td>73 ± 7</td>
<td>83 ± 9</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>6 ± 2</td>
<td>7 ± 1</td>
<td>5 ± 1</td>
<td>3 ± 1</td>
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<td>LV dp/dt (mm Hg)</td>
<td>1531 ± 147</td>
<td>1177 ± 107*</td>
<td>1245 ± 149</td>
<td>1320 ± 125</td>
</tr>
<tr>
<td>Cardiac output (ml/min)</td>
<td>2622 ± 213</td>
<td>2316 ± 209</td>
<td>2099 ± 225†</td>
<td>1911 ± 157†</td>
</tr>
</tbody>
</table>

All values are mean ± se.
* = $P < 0.05$ control vs ischemia; † = $P < 0.05$ ischemia vs ischemia + counterpulsation + norepinephrine.
‡ = $P < 0.05$ control vs reperfusion.

![Figure 6. Effect of aortic counterpulsation alone and with norepinephrine or nitroprusside on ischemic posterior wall velocity. Improvement occurred during counterpulsation with the drugs, but reperfusion did not restore velocity to control levels.](image2)
minimal changes seen with counterpulsation alone — nitroprusside by enhancing systolic unloading and norepinephrine by further raising coronary perfusion pressure. But neither agent when administered in combination with counterpulsation was significantly more effective than counterpulsation alone in altering ischemic wall motion. In another investigation from our laboratory we have studied the effect of norepinephrine and nitroprusside alone on the perfusion of acutely ischemic myocardium. Norepinephrine caused an increase in ischemic area perfusion: 19.6 ± 5.9 to 36.0 ± 7.5 ml/100 g/min (P < 0.05), while nitroprusside decreased perfusion: 12.7 ± 3.3 to 6.5 ± 1.7 ml/100 g/min (P < 0.05). With both drugs perfusion of the ischemic myocardium remained far below (P < 0.05) the pre-counterpulsation occlusion perfusion levels: 69.2 ± 5.8 ml/100 g/min. The doses of the two drugs administered, and the hemodynamic changes produced by them, are similar in the present and our earlier study. Thus, the failure of these drugs to effect further changes in ischemic dyskinesia in the present study is probably due at least in part to the presence of a substantial and persisting perfusion deficit in the ischemic area during the combined drug/counterpulsation intervention.

Coronary reperfusion after counterpulsation produced improvement similar to that occurring with reperfusion after no intervention during a one hour ischemic period: abolition of aneurysmal bulging and improvement, but not full restoration, of ischemic wall velocity. The persistent dyskinesia seen following reperfusion may result from metabolic alteration and/or structural myocardial damage during the ischemic period; counterpulsation either alone or in combination with norepinephrine and nitroprusside did not enhance the reperfusion-induced improvement in dyskinesia during the limited time range of this study.

Are the findings in this experimental model applicable to patients undergoing balloon counterpulsation? None of our experimental animals were in cardiogenic shock or severe congestive heart failure, the two most common clinical hemodynamic settings in which IABC is employed. Important differences also exist in the timing of IABC use: we began IABC five minutes after coronary occlusion and continued it for one hour, whereas patients do not receive IABC for at least several hours after the onset of ischemia, and it may be continued for as long as several days. Finally, Shaw et al. found that animals with the highest rate of flow to ischemic areas after coronary ligation had the greatest increases in myocardial perfusion with counterpulsation, and suggested that improvement in blood flow to the involved myocardium was dependent on the presence of pre-existing collateral vessels. Such collaterals are probably variable in both animals and man, and were not assessed in this study. For all these reasons, the findings of this investigation should be applied clinically with caution.

The echocardiographic technique only evaluates the motion of one small area of the cardiac wall. We cannot entirely exclude the possibility that areas of wall not struck by the beam in any given animal might have shown improvement with IABC which would not have been recognized. Evidence against this possibility, however, includes the failure of both normally perfused as well as ischemic myocardial segments to show improvement in wall motion on the ultrasound recordings during IABC, and also the lack of increase of perfusion of ischemic segments at each level of perfusion deficit.

We conclude that in these experiments aortic balloon counterpulsation produces little improvement in the segmental dyskinesia of acutely ischemic myocardium. The modest changes seen may be attributable to alteration of afterload and reduction in myocardial oxygen requirements; they cannot be attributed to improvement in regional perfusion, since no increase in perfusion could be demonstrated.

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