Influence of Dobutamine on Regional Myocardial Blood Flow and Ventricular Performance During Acute and Chronic Myocardial Ischemia in Dogs

JAMES T. WILLERSON, M.D., IAN HUTTON, M.D., JOHN T. WATSON, PH.D., MELVIN R. PLATT, M.D., AND GORDON H. TEMPLETON, PH.D.

SUMMARY The data from this study document that dobutamine is a powerful inotropic agent in anesthetized dogs with acute myocardial ischemia and in awake, unsedated ones with chronic myocardial infarction. Dobutamine significantly increases heart rate at relatively small doses in anesthetized dogs with acute myocardial ischemia but considerably larger amounts of dobutamine are required to significantly increase heart rate in awake, unsedated dogs with myocardial infarction. Dobutamine also significantly increases regional myocardial blood flow to all areas of the heart at 20μg/kg/min in both anesthetized dogs with acute myocardial ischemia and awake, unsedated ones with myocardial infarction. However, in anesthetized dogs 20μg/kg/min of dobutamine significantly increases epicardial ST-segment elevation during acute myocardial ischemia. Propranolol prevents the inotropic and chronotropic effects of dobutamine in both anesthetized and awake, unsedated dogs. This study suggests that during experimental acute myocardial ischemia dobutamine given at doses that significantly increase heart rate and contractility may increase the extent of myocardial damage. The data also suggest that this agent should be of value in the setting of severe myocardial depression without associated severe coronary artery disease to increase cardiac contractility at doses that do not markedly alter heart rate. The hemodynamic and coronary blood flow effects of dobutamine in patients with and without severe coronary artery disease should be evaluated.

DOBUTAMINE HAS RECENTLY been shown to be a powerful inotropic agent in experimental animals, and its chronotropic effect seems minimal.1,2 This drug might be more useful in treating “low output” states in patients with ischemic heart disease than certain other currently existing medications that are known to adversely effect the extent of myocardial damage.3 The crucial factor is its relative influence on several different parameters including contractile state, left ventricular volume, heart rate, and regional myocardial blood flow. Thus if dobutamine’s major influence is to increase contractility and/or heart rate, then one would expect it to have an adverse effect on the extent of myocardial damage during myocardial ischemia. On the other hand, if dobutamine produces a greater effect on coronary blood flow and especially flow to the ischemic area than on contractility then it will either not extend, or might even reduce the extent of myocardial damage during myocardial ischemia. The present study was designed to test the influence of dobutamine on contractility, regional myocardial blood flow, and heart rate in anesthetized dogs with acute myocardial ischemia and in awake, unsedated dogs with chronic myocardial infarction.

Methods

Anesthetized Dog Studies

Mongrel dogs were anesthetized with Nembutal (30 mg/kg), intubated and ventilated with a Harvard respirator, using a gas mixture of 95% oxygen and 5% carbon dioxide. The chest was opened through a median sternotomy, the proximal left anterior descending coronary artery (LAD) was freed and a reversible ligature placed around it. Left ventricular systolic pressure and the maximal rate of pressure rise (LV dp/dt) were measured through a short, wide-bore Y-shaped metal cannula inserted through the apex of the left ventricle. LV dp/dt was measured by continuously differentiating left ventricular systolic pressure. LV dp/dt/P was computed from the maximal rate of left ventricular pressure rise divided by developed pressure during isovolumic systole.4 Proximal aortic pressure was measured through a short, wide-bore polyethylene catheter inserted into the aortic arch through the left carotid artery. Epicardial ST-segments were measured in the anesthetized animals using a rounded, smooth tip electrode attached to...
the V lead of the electrocardiogram; epicardial ST-segment changes were measured from 14 sites adjacent to the LAD occlusion at the end of each period of coronary insufficiency. Pressures were measured with Statham P23Db pressure transducers and recorded on a multichannel Sanborn or an Electronics for Medicine direct writing recorder.

Regional myocardial blood flow was measured using radioactive microspheres $9 \mu$ in diameter* ($^{141}$Ce, $^{85}$Sr, and $^{42}$Sc) (3M Company). At specified times 600,000-1,000,000 microspheres were injected into the left atrium from a small injection vial with 10 ml of warm saline over a 20 sec period. Immediately before the injection blood was withdrawn at a rate of 15-20 ml/min using a small Holter pump into counting vials from a catheter tied in the femoral arteries. During the microsphere injection four 30-sec reference blood samples were collected to insure that all the microspheres had cleared the dead space of collecting catheter and none were recirculating.

At the end of each experiment the heart was removed and a small catheter inserted into the LAD just distal to the ligature around the proximal LAD. India ink (0.1 ml) was injected to outline the area of ischemia. This small volume of India ink was used so as to be certain that only the center of the ischemic area was removed and labeled as “ischemic area.” The atria, epicardial fat, and coronary vessels were removed and the ventricles separated into the right and left ventricular free walls and the ventricular septum. The right and left ventricular free walls were then further subdivided into subendocardial, subepicardial, and middle layers of approximately equal thickness. The septum was subdivided into right and left portions. In the left ventricular free wall the ischemic and nonischemic portions were kept separate. The 1 cm strip of myocardial tissue immediately adjacent to the “ischemic area” was identified in the studies done in awake, unseparated dogs and removed and counted separately as the “peri-ischemic area.” The heart and reference blood samples were placed in glass counting vials and counted in a well scintillation counter (Nuclear Chicago).

The total activity of each nuclide was computed using the method of Rudolph and Heymann. Total and regional myocardial blood flows were computed using the following equation: regional myocardial blood flow equals flow in reference sample (timed volumetric collection from the femoral artery) multiplied by nuclide counts in the myocardial region of interest divided by nuclide counts in the reference sample. Regional myocardial blood flows were expressed as flows in ml/min/g of tissue. Absolute flows and subendocardial/subepicardial wall flow ratios were determined for each ventricle in the ischemic and nonischemic areas.

The experimental format for the anesthetized studies was as follows. The dog was allowed to stabilize immediately following the instrumentation surgery and then control pressures were measured. A control epicardial ECG map was obtained from the 14 sites adjacent to the LAD occlusion. Next the proximal LAD was occluded for a 12 min period and isotonic saline was infused intravenously beginning the infusion immediately after LAD occlusion. The infusion speed of the saline was identical to that subsequently used for the dobutamine studies. Pressures, epicardial ST-segment changes, and regional myocardial blood flow were measured at the end of the LAD occlusion. The ligature was then released and a 30 min recovery period provided.

Next, in one group of nine dogs, dobutamine was infused at 4ug/kg/min throughout a second period of coronary occlusion lasting 12 min. In another group of ten dogs dobutamine was infused at 20ug/kg/min for the 12 min period of acute coronary occlusion. Pressures, epicardial ST-segment changes, and regional myocardial blood flow were again measured at the end of the LAD occlusion. In these anesthetized dog studies the ligature was released at the end of the second period of acute myocardial ischemia associated with the administration of dobutamine and another 30 min recovery period allowed. Finally, a third 12 min period of acute coronary insufficiency was provided in three dogs in which 20ug/kg/min of dobutamine was infused again, but this time the dogs had received 2 mg/kg of propranolol intravenously 30 minutes prior to the infusion of dobutamine. In eight additional dogs a final period of coronary insufficiency was provided in which isotonic saline was infused in exactly the same manner as during the initial period of LAD occlusion. The same pressures, epicardial ST-segments, and regional myocardial blood flow measurements were made at the end of this period of coronary insufficiency.

In the studies described above at the conclusion of the last period of coronary insufficiency the dog was sacrificed and the heart removed and sectioned for microsphere counting as described earlier.

Conscious Dog Studies

Adult mongrel dogs of either sex were anesthetized with Nembutal (30 mg/kg) and artificial ventilation provided with a Harvard respirator after endotracheal intubation. The heart was exposed through a left thoracotomy under sterile conditions. The left anterior descending coronary artery was mobilized between 2 and 3 cm from its origin and an inflatable silicone rubber balloon with a silastic backing sutured around the artery at that level. The tube from this balloon was brought through the skin behind the animal's neck. Preliminary studies were performed in each dog at the time of insertion of the balloon device to determine what volume of saline should be added to the balloon to fully inflate it and totally occlude the underlying coronary artery. This same balloon occlusion cuff has previously been used by us and others to provide acute and chronic coronary insufficiency in the dog.*

At least one and usually two small diagonal branches of the LAD near the apex of the left ventricle appearing to provide collateral blood flow between the circumflex coronary artery and distal left anterior descending coronary artery were permanently suture-ligated after the reversible balloon occluder was placed around the LAD. A Königsberg pressure transducer (model P21) was positioned in the left ventricle and its external connection brought to the surface. The pericardium was loosely resutured. A left atrial catheter was also positioned, stabilized, and brought to the surface. All of these catheters were periodically filled with small amounts of heparin. The catheters were covered with a sleeve and tape to protect them.

*Standard deviation for the diameter of these microspheres was 1.0 $\mu$ or less.
These dogs were studied no sooner than two weeks postoperatively at a time when they appeared to have fully recovered from the previous instrumentation. They were returned to the study area and placed in support slings. If necessary the dogs were trained prior to the actual experiment to lie quietly in the sling. Any animal that was uncooperative or not apparently relaxed during the study period was removed and studied at a later date.

Two days prior to the actual study each animal was anesthetized and catheters were positioned in the carotid artery and jugular veins. The balloon occlusive device around the LAD was also inflated. The inflated balloon was checked daily for the next 48 hours to be certain it had remained fully inflated.

The dog was then returned to the support sling where the following study protocol was utilized. Control measurements of pressures were made. Then saline was infused for a 12 min period intravenously at the same speeds subsequently utilized for dobutamine infusions using a constant speed Harvard infusion pump. At the end of the 12 min period of time pressures and regional myocardial blood flow were measured; the latter was measured utilizing a batch of radioactive microspheres injected into the left atrium just as described previously.

A 30 min rest period was provided and then dobutamine was infused intravenously for 6 min at 8 and then 6 minutes at 20μg/kg/min (11 dogs).* Pressures and heart rate were measured prior to beginning dobutamine and at the conclusion of each different infusion of dobutamine; regional myocardial blood flow was measured at the end of the 20μg/kg/min dobutamine infusion in six of the animals.

The dobutamine infusion was then discontinued and another 30 min rest period provided. During the rest period 2 mg/kg of propranolol was given intravenously in eight of the dogs and 20 min later 20μg/kg/min of dobutamine was infused for 12 min and pressures and regional myocardial blood flow were again measured at the end of the dobutamine infusion.

The dog was then sacrificed and the heart removed and sectioned for microsphere counting as described earlier. In each of the dogs in this study the balloon occlusive device was fully inflated and there was a gross area of myocardial infarction visible on the anterior surface of the left ventricle at the time of the postmortem examination. Histological confirmation that myocardial infarction was present was obtained in each animal by obtaining a small sample of tissue from this damaged area for microscopic examination. The area of the infarction on the anterior surface of the left ventricle was removed when the heart was sectioned, and counted separately.

Statistical comparisons between results obtained prior to coronary artery occlusion and those obtained at the end of coronary occlusion associated with the administration of saline, dobutamine, and dobutamine after propranolol were made in each individual animal using a paired r-test. Statistical comparisons for hemodynamics, regional myocardial blood flow and epicardial ST-segment elevation were made between values present at the end of the periods of LAD occlusion associated with the administration of saline and dobutamine. Differences were considered significant when P < 0.05.

Results

Dobutamine Administration in Anesthetized Dogs

Dobutamine given at 4μg/kg/min to anesthetized dogs tended to increase heart rate during acute myocardial ischemia (5 ± 1.7% [SE], P < 0.05) (table 1). Similarly mean systemic arterial pressure also tended to increase after dobutamine (table 1). Dobutamine also increased maximal LV dp/dt during acute coronary insufficiency (27 ± 9.4%, P < 0.01) (table 1). There was no change in left ventricular end-diastolic pressure after dobutamine. Dobutamine at this concentration did not produce a significant change in epicardial ST-segment elevation during acute myocardial ischemia. Neither did dobutamine at this dose significantly increase myocardial blood flow to any region of the heart. Saline did not significantly change any aspect of left ventricular hemodynamics during acute myocardial ischemia.

Dobutamine given at 20μg/kg/min increased LV dp/dt by 97 ± 10.13% (P < 0.001) (table 1), and LV dp/dt/P from 21 ± 1.77 sec⁻¹ to 42 ± 3.4 (P < 0.001). At this infusion rate of dobutamine mean systemic arterial pressure and left ventricular end-diastolic pressure did not significantly change. Heart rate, however, was significantly increased by 24 ± 2.48% (P < 0.01). Dobutamine at this infusion rate also generally increased regional myocardial blood flow during acute myocardial ischemia (fig. 1). Thus, flow to the left

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*LV dp/dt was measured in seven of these dogs.

Figure 1. The influence of dobutamine (20μg/kg/min) on regional myocardial blood flow in anesthetized dogs (N = 9) during acute myocardial ischemia is shown. The bars represent mean values; the brackets the standard errors.
Table 1. Experimental Data

<table>
<thead>
<tr>
<th>Protocol</th>
<th>HR (beats/min)</th>
<th>Systolic AP (mm Hg)</th>
<th>Systemic AP (mm Hg)</th>
<th>LV dp/dt (mm Hg/sec)</th>
<th>EST (mm)</th>
<th>LVAT (ml/min/gm)</th>
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<tbody>
<tr>
<td><strong>Anesthetized Dogs with Acute Myocardial Ischemia</strong></td>
<td></td>
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<tr>
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<tr>
<td>Control</td>
<td>125 ± 8.8</td>
<td>123 ± 12.5</td>
<td>96 ± 10.6</td>
<td>1611 ± 209.1</td>
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<tr>
<td>Coronary occlusion with saline</td>
<td>123 ± 8.0</td>
<td>116 ± 13.6</td>
<td>90 ± 11.6</td>
<td>1476 ± 186.7</td>
<td>53 ± 25.8</td>
<td>0.5 ± 0.2</td>
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<tr>
<td>Control</td>
<td>120 ± 8.7</td>
<td>120 ± 10.4</td>
<td>93 ± 10.1</td>
<td>1589 ± 222.9</td>
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<td>Coronary occlusion with dobutamine (4 μg/kg/min)</td>
<td>130 ± 9.0*</td>
<td>128 ± 12.3</td>
<td>101 ± 10.8</td>
<td>2005 ± 266.7*</td>
<td>52 ± 21.0</td>
<td>0.6 ± 0.2</td>
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<td>2. N = 10</td>
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<tr>
<td>Control</td>
<td>152 ± 6.06</td>
<td>130 ± 8.04</td>
<td>112 ± 5.8</td>
<td>2123 ± 225.2</td>
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<tr>
<td>Coronary occlusion with saline</td>
<td>152 ± 5.7</td>
<td>139 ± 6.1</td>
<td>114 ± 5.3</td>
<td>2051 ± 194.8</td>
<td>32 ± 4.5</td>
<td>0.6 ± 0.16</td>
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<tr>
<td>Control</td>
<td>155 ± 5.6</td>
<td>140 ± 6.9</td>
<td>111 ± 6.0</td>
<td>2010 ± 199.6</td>
<td></td>
<td>N.M.</td>
</tr>
<tr>
<td>Coronary occlusion with dobutamine (20 μg/kg/min)</td>
<td>192 ± 7.21*</td>
<td>139 ± 5.7</td>
<td>105 ± 5.1</td>
<td>3928 ± 393.61*</td>
<td>64 ± 12.7†</td>
<td>0.9 ± 0.2†</td>
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<td><strong>Awake, Unsedated Dogs with Myocardial Infarction</strong></td>
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<tr>
<td>1. N = 11</td>
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</tr>
<tr>
<td>Control</td>
<td>122 ± 9.5</td>
<td>138 ± 8.8</td>
<td>105 ± 7.3</td>
<td>2345 ± 343.9</td>
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<td>N.M.</td>
</tr>
<tr>
<td>Saline</td>
<td>118 ± 8.5</td>
<td>135 ± 7.4</td>
<td>102 ± 6.9</td>
<td>2391 ± 403.3</td>
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<td>N.M.</td>
</tr>
<tr>
<td>Control</td>
<td>117 ± 7.8</td>
<td>133 ± 7.8</td>
<td>100 ± 6.5</td>
<td>2425 ± 414.6</td>
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<td>N.M.</td>
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<tr>
<td>Dobutamine</td>
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<tr>
<td>8 μg/kg/min</td>
<td>113 ± 8.7</td>
<td>135 ± 8.0</td>
<td>100 ± 6.5</td>
<td>2539 ± 447.8†</td>
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<tr>
<td>Dobutamine</td>
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</tr>
<tr>
<td>20 μg/kg/min</td>
<td>121 ± 9.0</td>
<td>143 ± 8.4†</td>
<td>103 ± 6.4</td>
<td>3413 ± 607.5†</td>
<td></td>
<td>N.M.</td>
</tr>
</tbody>
</table>

All values in mean ± standard error.

*P < 0.05 by t-test for difference between values at end of pre-dobutamine control and occlusion associated with dobutamine administration.

†P < 0.05 by t-test for difference between values at end of saline and dobutamine occlusions.

N.M. = not measured.

Abbreviations: HR = heart rate; AP = arterial pressure; LV = left ventricle; EST = sum of epicardial ST elevation during acute myocardial ischemia; LVAT = flow to ischemic or infarcted region of LV.

ventricular ischemic area increased by 37 ± 8.66% (P < 0.01) (fig. 1). Dobutamine administration was also associated with an increase in flow to the nonischemic portion of the left ventricle by 66 ± 17.12% (P < 0.01) and to the ventricular septum of 60 ± 13.28% (P < 0.01) (fig. 1). However, there was also a significant increase in epicardial ST-segment elevation during the dobutamine infusion as epicardial ST-segment elevation increased from 32 ± 4.5 mm at the end of the period of acute coronary insufficiency associated with the administration of saline to 64 ± 12.7 mm at the end of the period of coronary insufficiency associated with the administration of dobutamine (P < 0.05). Dobutamine did not alter the inner-outer wall flow ratio in any of the different regions as compared to that present during the control period of acute myocardial ischemia.

Propranolol in three dogs prevented dobutamine (20 μg/kg/min) from significantly increasing mean systemic arterial pressure, maximum LV dp/dt, LV dp/dt/P and heart rate during the final period of myocardial ischemia. Propranolol also prevented dobutamine from significantly altering regional myocardial blood flow. Saline administration in eight dogs during the final period of coronary insufficiency did not significantly increase any parameter of hemodynamic performance nor did it alter coronary blood flow.

Dobutamine 8 μg/kg/min did not change either mean systemic arterial pressure or left ventricular end-diastolic pressure. Maximum LV dp/dt tended to increase (4 ± 0.95%, P < 0.05), but LV dp/dt/P did not change after this dose of dobutamine. Heart rate also did not increase significantly (table 1).

Dobutamine 20 μg/kg/min did not significantly alter mean systemic arterial pressure or left ventricular end-diastolic pressure. This dose of dobutamine did significantly increase maximum LV dp/dt, however (40 ± 5.66%, P < 0.001) and LV dp/dt/P (32 ± 5.4 sec·1 to 43 ± 8.08 sec·1, P < 0.05). Dobutamine at this dose did not significantly increase heart rate (table 1). Dobutamine significantly increased regional myocardial blood flow as flow increased: to the LV infarced area by 48 ± 16.18%, P < 0.05; to the peri-infarced region by 98 ± 17.74%, P < 0.01; to the nonischemic portion of the LV by 61 ± 12.24%, P < 0.01; and to the ventricular septum by 46 ± 13.48%, P < 0.01 (fig. 2).

As in the experiments with anesthetized dogs propranolol prevented the hemodynamically significant alterations and the increases in regional myocardial blood flow produced by dobutamine.

Discussion

The data obtained in this study document that dobutamine exerts a significant inotropic effect in both anesthetized dogs with acute myocardial ischemia and in awake, unsedated dogs with chronic myocardial infarction. In some of the experimental circumstances in which
dobutamine was administered, there were also slight changes in heart rate and systemic arterial pressure in association with the increase in LV dp/dt produced by dobutamine. Thus, dobutamine in relatively small doses tended to increase mean systemic arterial pressure in anesthetized dogs with acute myocardial ischemia but in larger doses did not significantly increase mean systemic arterial pressure in either the anesthetized or awake, unsedated dogs. However, the overall increases in LV dp/dt after dobutamine in the different models that were studied cannot be attributed to changes in heart rate, arterial pressure or LV filling pressure and thus do reflect actual increases in contractility.

Earlier studies have reported that dobutamine does not alter heart rate significantly in awake, unsedated animals. In the present study dobutamine had a different effect on heart rate in anesthetized dogs with acute myocardial ischemia than in awake, unsedated dogs with chronic myocardial infarction. In anesthetized dogs with acute myocardial ischemia relatively small doses of dobutamine increased heart rate significantly but in awake, unsedated dogs relatively large doses of dobutamine failed to significantly increase heart rate.

The data from this study also demonstrate that dobutamine significantly increases regional myocardial blood flow to all portions of the heart including the ischemic and the infarcted area during acute myocardial ischemia in anesthetized dogs and in the presence of myocardial infarction in awake, unsedated dogs. The increases in regional myocardial blood flow in both preparations are of approximately the same magnitude in nonischemic and in ischemic LV.

In the anesthetized dogs with acute myocardial ischemia dobutamine at 20μg/kg/min appeared to alter oxygen demand more than oxygen availability since it significantly increased epicardial ST-segment elevation. In the anesthetized dogs both heart rate and contractility did increase significantly at this dose of dobutamine. A smaller dose of dobutamine (4μg/kg/min) produced no significant increase in epicardial ST-segment elevation during acute myocardial ischemia in anesthetized dogs but did significantly increase LV dp/dt. This dose of dobutamine did not significantly increase regional myocardial blood flow, however.

Propranolol prevented the significant increase in contractility, mean arterial blood pressure, heart rate, and regional myocardial blood flow after dobutamine in both the anesthetized dogs with acute myocardial ischemia and in awake, unsedated dogs with chronic myocardial infarction. The mode of action for propranolol could be explained in two ways. The drug could block the effect of dobutamine on improving cardiac contractility; in this case blood flow would remain the same. Blood flow would also remain unchanged if dobutamine had a coronary vasodilatation property which propranolol blocked.

Previously Vatner and his colleagues have shown that dobutamine is a potent inotropic agent in awake, unsedated dogs without myocardial ischemia and seems to have little effect on heart rate in these animals. Previous investigations have also suggested that dobutamine exerts a much less significant inotropic effect in dogs with "moderate acute global ischemia" produced by partial constriction of the left main coronary artery than in dogs without myocardial ischemia. The present studies differ from those done by Vatner and his colleagues in that the LAD was completely occluded and at least one diagonal branch of the LAD and usually two were suture ligated; a large area of infarction was verified in our studies in awake, unsedated dogs. In addition the agent was evaluated in both anesthetized and awake, unsedated dogs.

In summary, dobutamine seems to be a powerful inotropic agent at doses that do not dramatically alter heart rate. It also seems to increase regional coronary blood flow during acute myocardial ischemia and chronic myocardial infarction. However, in anesthetized dogs, the dosage that was found high enough to increase both LV dp/dt and heart rate significantly during acute myocardial ischemia was associated with an increase in epicardial ST elevation, suggesting extension of myocardial damage. When administered in doses small enough not to increase heart rate during acute myocardial ischemia it does not increase epicardial ST-segment elevation but it also does not significantly increase regional coronary blood flow. Dobutamine at comparable doses seems to have less influence on heart rate in awake, unsedated dogs than in anesthetized ones. These findings underline the need to use dobutamine cautiously in the setting of severe coronary artery disease and the importance of further evaluation in various clinical settings. The relatively small influence on heart rate and significant inotropic potential of dobutamine would be valuable in the setting of myocardial depression without associated severe coronary artery disease.

Acknowledgment

The authors wish to express their appreciation to Miss Judy Ober, Mr. Curtis Garner and Mr. Julius Lamar for expert technical assistance. Appreciation is also expressed to Mrs. Donna Place and Mrs. Belinda Lambert for secretarial assistance. The authors are grateful to the Eli Lilly Pharmaceutical Co. for helpful advice and for making dobutamine available for these studies.

Figure 2. The influence of dobutamine (20μg/kg/min) on regional myocardial blood flow in six awake, unsedated dogs with chronic myocardial infarction is shown. The format of the figure is the same as in figure 1.
Technetium 99m Stannous Pyrophosphate Myocardial Imaging in Patients with and without Left Ventricular Aneurysm

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SUMMARY To further explore the usefulness of technetium 99m pyrophosphate (99mTc-PYP) myocardial imaging and test its validity in the diagnosis of acute myocardial infarction, 99mTc-PYP myocardial scintigrams were performed in 50 patients. Out of 28 patients with acute myocardial infarction, myocardial scintigrams demonstrated localized activity in the 15 patients with transmural, and diffuse activity in the 13 patients with subendocardial myocardial infarction. Twenty-two patients with significant coronary artery disease documented by coronary angiography but without acute myocardial infarction were also studied. Nine of ten patients with clinical evidence of left ventricular aneurysm from previous myocardial infarction and definite left ventricular dyskinesia had positive scintigrams with activity localized to the site of the wall motion abnormality. Two of five patients without definite aneurysm but with left ventricular akinesis also had localized uptake in the involved area of the left ventricle. Seven patients with normal left ventricular wall motion had negative scintigrams. These findings suggest caution in interpreting positive 99mTc-PYP scintigrams as being indicative of acute myocardial infarction when evidence of a left ventricular aneurysm is also present.

MYOCARDIAL IMAGING with 99mTc-PYP has been recently reported to be a reliable noninvasive technique in the diagnosis of acute myocardial infarction.1-3 Experimental infarcts are known to display scintigraphic activity approximately 12 to 16 hours after infarction with progressively decreasing activity over the next one to two weeks.2 Positive myocardial scintigrams with localized activity are associated with appropriate cardiac enzyme and/or electrocardiographic findings of acute transmural myocardial infarction.1 Some patients with unstable angina pectoris and occasional patients with other types of chest pain have been reported to demonstrate positive scintigrams in the absence of conclusive electrocardiographic or enzymatic evidence of acute myocardial infarction.4

This communication reports our experience with 99mTc-PYP myocardial scintigrams in patients with acute myocardial infarction and in patients with previous myocardial infarction and documented ventricular aneurysm or other wall motion abnormalities without evidence of acute myocardial infarction.

Methods

99mTc-PYP myocardial scintigrams were performed in 50 patients. Twenty-eight patients had acute myocardial infarction, and 22 had significant coronary artery disease documented by coronary angiography without acute myocardial infarction. After informed consent had been obtained, myocardial scintigrams were performed in the Nuclear Medicine Laboratory. Patients with acute myocardial infarction were studied under constant electrocardiographic monitoring with emergency drugs and a defibrillator immediately available. Myocardial imaging was performed in anterior, lateral, and left anterior oblique positions 45-60 minutes after the intravenous injection of 15 mCi of 99mTc-PYP tagged to 5 mg of stannous pyrophosphate. Average duration of imaging in each patient was 20 minutes. Repeat scintigrams were obtained two hours following the 99mTc-PYP injection in four patients. Sequential Polaroid scintigrams were obtained with a Pho/Gamma Camera H.P. (Searle Radiographics) utilizing a 15,000 hole

References


From the Division of Cardiology, Department of Medicine, University of Missouri Medical Center, Columbia, Missouri. Supported in part by NIH Training Grant 3 T01 HL05787. Presented in part at the national meeting of the American Federation for Clinical Research, Atlantic City, May 1975. Address for reprints: Masood Ahmad, M.D., Division of Cardiology, University of Missouri Medical Center, Columbia, Missouri 65201. Received July 8, 1975; revision accepted for publication December 18, 1975.
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