Dipyridamole Thallium Imaging

This is the second article in the series of "Nuclear Medicine Updates." Upon completion of this article, the technologist will: (1) have an awareness of an alternate method for evaluation of ischemic coronary artery disease (CAD) on patients who are unable to exercise on a treadmill and (2) have a guideline available to them for the use of dipyridamole.

Thallium (201Tl) myocardial perfusion imaging with exercise is widely accepted as a simple and safe method to diagnose coronary artery disease (CAD) or to evaluate the extent and significance of disease in patients with known CAD. A significant number of patients cannot achieve the necessary level of exercise with routinely performed 201Tl treadmill or bicycle exercise testing to properly allow for the accurate evaluation of ischemic CAD. Patients who are unable to exercise to a maximal effort may demonstrate a nonreproducible pattern and CAD may not be detected (1). Those patients who are impaired physically (amputation, stroke, joint and other musculoskeletal disorders), pharmacologically (beta blockers, calcium channel blockers), or psychologically (sedentary lifestyles, poor motivation) cannot be ignored by nuclear medicine departments when evaluation of anginal-type symptoms or clearance for a major surgical procedure is necessary. The careful use of a pharmacologic stress agent to assess coronary flow reserve is available to nuclear medicine departments in the form of dipyridamole (Persantine®). Examinations using orally administered dipyridamole are possible in most nuclear medicine laboratories, whereas intravenous dipyridamole use is currently performed only at institutions which are participants in the Phase III investigational new drug protocol. It is hoped that the Food and Drug Administration will soon approve intravenous dipyridamole for routine clinical use in the evaluation of patients for CAD. This article is intended to provide the non-user of dipyridamole with the necessary information to include this imaging procedure in his armamentarium of patient testing.

MECHANISMS OF ACTION

Dipyridamole is a complex, lipophilic pyridimine derivative, which is basic in pH and nearly water insoluble in other than an acidic solution (2). The principal effect of dipyridamole given intravenously or in large doses orally is to produce coronary artery and systemic vasodilation. Dipyridamole inhibits the uptake of adenosine across cell membranes, which results in an increase in interstitial adenosine levels (3). Adenosine is derived from the metabolism of adenosine tri-phosphate (ATP) and is a potent vasodilator in most vascular beds except the kidneys (4). Adenosine's specific action is at the receptor level located on the outer surface of smooth muscle (4). Therefore, the vasodilatory effect of dipyridamole is secondary to the increase in endogenous adenosine levels.

Experimentally, many different agents have been used as a satisfactory stimulus for coronary vasodilation. Administration of iodinated contrast material, papaverine, intracoronary nitroglycerine, and even temporary coronary artery occlusion have been attempted to assess the effects of coronary vasodilation (5). These techniques have received limited clinical use due to their inherent risks of morbidity and mortality, and more emphasis has been placed on use of routine, noninvasive techniques for the evaluation of CAD. Used correctly, dipyridamole has been shown to be both safe and effective in producing coronary artery vasodilation. An important characteristic of this action is that dipyridamole is readily counteracted by xanthine derivatives (i.e., aminophylline).

Physiologic requirements for assessing coronary flow reserve and CAD with radionuclide imaging techniques include (a) an increase in coronary flow to maximize the difference in regional perfusion abnormalities and, (b) uptake of the imaging agent in the myocardium proportionately to the relative flow, especially at high flow rates (5). An agent whose uptake is not related to flow will be insensitive for detecting mild stenosis. Concentration of ionic 201Tl into the myocardium is directly proportional to blood flow over a clinically important range (6).

In the normal resting state, coronary blood flow is relatively low with a high degree of peripheral vascular and coronary circulatory resistance. Dipyridamole acts to reverse this environment and produces a high flow, low resistance system (7). In this state, the hemodynamic effect of fixed coronary lesions
becomes much more significant. Investigators have demonstrated that dipyridamole alone or in conjunction with isometric handgrip exercise can increase coronary flow rates 2.5–3.3 times the resting coronary flow levels (7,8). The severity of coronary stenosis can best be defined in terms of pressure gradients across a stenotic lesion and coronary flow-velocity relationships. For a fixed coronary lesion, the pressure gradient increases as the flow velocity increases in response to some type of stimulus (exercise or dipyridamole). Coronary vasodilation in the presence of coronary stenosis will result in an increased pressure gradient and a decrease in distal coronary pressure as the coronary flow increases. This increase in pressure loss is associated with reduced subendocardial perfusion despite an increase in epicardial flow (5). To explain the effect of relative regional myocardial ischemia under these circumstances, it has been proposed that there is a relative maldistribution of myocardial blood flow in areas supplied by stenotic arteries with preferential blood flow to epicardial vessels with a consequent decrease in perfusion to the endocardium (Figs. 1 and 2).

In addition to the initial differences of $^{201}$Tl distribution related to flow, there are also differences in the washout rates from normal and stenosed territories (5). As flow increases, normal vessels have increased $^{201}$Tl activity due to the increased endocardial and epicardial flow and also have a more rapid intrinsic washout. Territories supplied by stenotic vessels have less $^{201}$Tl uptake than the normal zone and a slower intrinsic washout. In general, dipyridamole $^{201}$Tl images have equal or better quality than treadmill $^{201}$Tl images. During both dipyridamole and treadmill exercise stress, the myocardial-to-background ratio of $^{201}$Tl increases, but due to two different mechanisms. Treadmill exercise stress decreases background activity in the lungs due to competition of tracer localization in other working muscle masses, and dipyridamole stress increases myocardial $^{201}$Tl uptake in the myocardium in relation to pulmonary background.

The hemodynamic effects of the relatively high doses of dipyridamole used in conjunction with $^{201}$Tl scintigraphy include: (a) at least a three-fold increase in coronary flow over the resting state without increasing myocardial oxygen demand; (b) a slight decrease in systemic blood pressure; and (c) a modest increase in heart rate, a reflex response to the drop in systemic blood pressure.

**INDICATIONS FOR USE**

The following list indicates those patient populations which might benefit from the use of dipyridamole-thallium imaging due to their inability to adequately undergo routine exercise testing.

**NORMAL CORONARY ARtery**

**PERSANTINE**

**CORONARY ARtery WITH STENOSIS**

**FIG. 1.** Diagram representing the effects of Persantine® on the normal coronary artery. Persantine produces a vasodilatory effect in both the epicardial and endocardial vessels, thereby increasing coronary flow and $^{201}$Tl uptake (3–4X) over the resting value. (Shading represents relative $^{201}$Tl uptake in the myocardium.)

**FIG. 2.** Diagram representing the effect of Persantine® on the coronary artery with a single fixed lesion. There is a preferential flow to the epicardium with an increased flow rate over the resting state, while the endocardium may demonstrate no increase in flow or only a slight increase in flow and $^{201}$Tl concentration. Therefore, comparing the myocardial uptake of $^{201}$Tl between the territory supplied by the normal and abnormal vessels, it is easy to demonstrate the difference in $^{201}$Tl distribution.
I. Patients with physical limitations:
   A. Lower limb amputation.
   B. Acute or chronic joint disease.
   C. Prosthetic joint implants.
   D. Obstructive lung disease.
   E. Orthopedic deformity.
   F. Peripheral vascular disease.
   G. Previous cardiac transplantation.

II. Neurologic impairment:
   A. Previous stroke with residual neurologic deficit.
   B. Debilitating neuromuscular disorders.

III. Patients undergoing medical therapy which precludes achieving adequate stress levels:
   A. Beta blockers.
   B. Calcium channel blockers.

IV. Previous non-diagnostic $^{201}$TI treadmill test:
   A. Patient did not reach 70% MPHR due to fatigue or inadequate effort.
   B. Preexisting electrocardiogram (ECG) abnormalities.

V. Prognosis of patients with acute infarcts in the early post-infarction period (9).

VI. Risk stratification of patients requiring medical clearance for a major surgical procedure (Studies have shown that patients with ischemic perfusion abnormalities are six to twenty times more likely to have postoperative events [death and nonfatal myocardial infarction] when compared to patients without transient perfusion abnormalities (5):
   A. Abdominal aortic aneurysm repair.
   B. Ilio-femoral, femoral-popliteal bypass.
   C. Renal transplantation.
   D. Orthopedic prosthesis implantation.

CAUTIONS AND WARNINGS

The use of dipyridamole is relatively contraindicated in the evaluation of patients with known bronchospastic lung disease (asthma), systemic hypotension with systolic blood pressure of 90 mm Hg or less, unstable angina, or acute myocardial infarction (<48 hr). Side effects associated with the intravenous use of dipyridamole include headache, nausea, syncope, and facial flushing. Only those persons appropriately qualified to administer medications with significant pharmacologic effects should be authorized to administer intravenous dipyridamole. As with any stress test, the infusion of dipyridamole must be monitored by an appropriately qualified individual. The same type of cardiac complications (ECG changes, chest pain, infarction) experienced with standard exercise testing can occur with the "pharmacologic stress test."

Aminophylline is an antagonist to the effects of the elevated adenosine level produced by the infusion of dipyridamole. It is believed that aminophylline acts to block specific adenosine receptors and therefore the systemic and coronary effects of increased adenosine levels (1). The effects of aminophylline are shorter than the effects of dipyridamole. Therefore, in those patients who have received aminophylline, the patient must be monitored for at least 30 min following the intravenous administration of dipyridamole. If aminophylline is requested by the monitoring physician to be administered to counteract the patient’s symptoms, the administration of aminophylline should be delayed at least 1 min after the $^{201}$TI dose has been infused. If it is necessary to give the aminophylline dose prior to the $^{201}$TI, then the $^{201}$TI should not be administered and the procedure aborted.

OUR EXPERIENCE

Over the last 5 yr our department has performed over 600 intravenous dipyridamole studies in conjunction with $^{201}$TI scintigraphy using the protocol described below. Indications for these studies have included those discussed above, but approximately 80% have been performed on patients who are unable to reach an acceptable level of cardiac stress with routine treadmill exercise due to either drug therapy (especially beta blockers) or generally poor exercise tolerance due to chronic lack of exercise and/or low cardiac output.

No patients were studied within 30 days after myocardial infarction, while patients with unstable angina were also excluded. Patients with COPD not requiring theophylline compounds were studied, but patients with active or significant past history of reactive airways disease and any patients using inhalant bronchodilators were excluded. No pregnant patients were studied. A limited number of children were included under a separate investigational protocol.

Symptomatic side effects of intravenous dipyridamole infusion were common, but generally minor (Table 1). Nausea, flushing, lightheadedness, and headache were the most common side effects, at least one of the signs occurring in approximately 30% of patients. Vomiting, severe dizziness (while supine), and hypotension were less common occurrences. Muscle spasms associated with the handgrip phase occur in <1% of patients, but hand/arm aching was common. Even though the dipyridamole was injected with little or no dilution in over 500 patients, local pain was not a significant problem. Minor burning at the injection site occurred in approximately 8% of patients, but was of such severity as to require discon-

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**TABLE 1. Adverse Reactions to Intravenous Dipyridamole**

<table>
<thead>
<tr>
<th>Event</th>
<th>% Occurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>28</td>
</tr>
<tr>
<td>Angina</td>
<td>27</td>
</tr>
<tr>
<td>ECG changes</td>
<td>14</td>
</tr>
<tr>
<td>Nausea</td>
<td>11</td>
</tr>
<tr>
<td>Flushing</td>
<td>9</td>
</tr>
<tr>
<td>Dizziness</td>
<td>7</td>
</tr>
<tr>
<td>Hypotension</td>
<td>4</td>
</tr>
<tr>
<td>Vomiting</td>
<td>1</td>
</tr>
</tbody>
</table>
tinuation of infusion in only two cases. (In both of these patients, dipyridamole was injected through an existing i.v. line which had recently been used for infusion of potassium chloride (KCl) or a hypermolar agent. In each event, changing to another venous access site solved the problem.)

One patient with a history of recent cerebrovascular accident experienced a transient ischemic attack (TIA) 25 min after dipyridamole infusion. This patient experienced additional TIAs on several occasions over the next 3 mo without any recognizable precipitating factor. The etiologic relationship of a TIA to dipyridamole infusion is unproven in this case, but the temporal relationship raises the possibility of an intracerebral steal phenomenon as has been postulated in the heart.

Approximately one-third of patients undergoing dipyridamole handgrip stress tests were asymptomatic, including many with CAD and ischemia documented by scintigraphy. An additional one third of patients experienced one or more cardiac related effects such as chest pain or pressure, ischemic ECG changes, or shortness of breath. Five patients had chest pain of such severity that transfer to the CCU was warranted for observation. One additional patient developed acute pulmonary edema without chest pain. None of these six patients with severe cardiac symptoms experienced myocardial infarction as evidenced by lack of continued chest pain, lack of permanent ECG changes, and lack of abnormal cardiac enzyme activity.

Aminophylline has been shown to be a competitive antagonist of dipyridamole vasodilation as described above, and some investigators routinely administer aminophylline after 201TI injection in conjunction with oral or i.v. dipyridamole tests. We administered aminophylline only in cases of severe, increasing, or persistent symptoms (usually cardiac). Such injection was felt to be necessary in only 4% of patients. When administered in a dose of 75–250 mg to patients with chest pain, ECG changes resolved within 3 to 5 min in all cases though chest pain was slower to resolve generally requiring 10–15 min. In the six patients admitted to the CCU, ECG changes resolved quickly in all but chest pain was not significantly affected early and the patients received supplemental treatment with nitroglycerin (sublingual or i.v.), oxygen, aminophylline infusion, and other standard therapy. No patient experienced side effects of aminophylline administration.

As mentioned above, cardiopulmonary effects occurred in approximately one third of patients in our series. The incidence of CAD in patients with angina or ECG changes was higher than that of patients without these symptoms, but the degree of chest pain and the severity of ST changes correlated only generally with the severity of CAD as evidenced by either coronary angiography or concomitant 201TI scintigraphy. Lack of such signs or symptoms was not a reliable predictor of the absence of CAD. Likewise, the presence or absence of non-cardiopulmonary symptoms had no correlation with CAD.

Though the patient population undergoing dipyridamole testing in our laboratory was different in some respects from the population of patients undergoing treadmill testing, the sensitivity for detection of significant CAD (>50% diameter narrowing) was similar for patients receiving dipyridamole (85%) compared to several studies of patient subgroups undergoing 201TI treadmill testing (86%–94%). Specificity was difficult to accurately assess because few patients with negative dipyridamole tests underwent cardiac catheterization.

DIPYRIDAMOLE PROTOCOLS

Intravenous

Protocol: Thallium myocardial imaging following intravenous dipyridamole administration (Fig. 3).

Indications:
1. To diagnose ischemic heart disease.
2. To evaluate the extent of coronary artery disease.
3. To evaluate therapeutic procedures such as bypass surgery or angioplasty.

Instrumentation: LFOV scintillation camera with SPECT acquisition and processing (This procedure may be performed with SFOV instrumentation and utilizing planar imaging techniques.)

Collimator: Low-energy all-purpose.

Radiopharmaceutical: Thallium-201-chloride 3.0 mCi (111 MBq).

Patient preparation:
1. NPO 2 hr prior to testing.
2. Restrict intake of xanthine derivatives. Patients should be instructed not to consume coffee, tea, carbonated drinks containing caffeine, or chocolate at least 8 hr prior to the study. Patients taking theophylline should discontinue the medication 24–36 hr prior to the study.

Procedure:
1. Check the patient’s chart or request slip for the order for the study.
2. Ascertain the clinical indications for performing the study.
3. Properly identify the patient.
4. Obtain a drug allergy history:
   A. Dipyridamole (Persantine®).
   B. FD&C yellow dye (Tartrazine).
   C. Aspirin.
   D. Aminophylline.
   E. Ethylenediamine.
5. Give the patient a verbal explanation of the procedure and ask him to read and sign the appropriate consent form. The patient is then placed on the imaging table in the supine position.
6. Determine the patient's maximum isometric handgrip effort (for dominant hand and opposite to the indwelling catheter). If the patient cannot maintain 25% of the maximal effort for 1 min, use the walking-in-place or table elevation protocols.

7. A 12-lead ECG is to be taken at rest and at 1-min intervals throughout the dipyridamole infusion.

8. An indwelling catheter is introduced via a 20-gauge Jelco needle in an antecubital vein. A 500-cc or 1000-cc bag of D5W is attached to the catheter via standard i.v. tubing delivery systems to maintain i.v. patency.

9. A blood pressure cuff is placed on the patient's arm opposite the catheter. Heart rate and blood pressure are recorded every minute throughout drug infusion and during subsequent physiologic maneuvers (upright position, walking, or handgrip) and for 6 min after infusion.

10. Calculate the total dose for dipyridamole infusion. (See Fig. 4.) Verify the dose calculation with the physician.

11. Dipyridamole is administered at the rate of 0.142 mg/kg/min for 4 min by the supervising physician (Fig. 5).

Note: Our experience has shown that the dose can be successfully administered by manually pushing the dose via a syringe during the predefined 4-min period. The dose is injected at the i.v. port closest to the i.v. catheter with the i.v. flowing at the maximum rate. This tends to moderate the dipyridamole infusion and reduce the incidence of irritation at the injection site.

12. Following the dipyridamole infusion, the patient is asked to perform isometric handgrip exercise holding 25% of his previously determined maximum effort for 5 min using the hand opposite the catheter. If handgrip exercise cannot be performed, the patient will be asked to walk in place or be placed in the sitting position for 3 min.

13. Thallium-201 is then injected through the catheter at 4 min following initiation of the handgrip exercise, or 2 min following initiation of the walking exercise or sitting position (Fig. 6). Handgrip, walking, or sitting is continued for an additional 1 min following 201 Tl administration.

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**DIPYRIDAMOLE THALLIUM WORKSHEET**

**DATE:**

**PATIENT NAME:**

**PATIENT WEIGHT:** kgms.

1. Pt. wt. (kgms) X 0.142 mg/kg/min = concentration mg/min.
   
   
   
   

2. Concentration mg/min X 4 min. = total mgs.

3. Total mgs. ÷ 5 mgs/cc = total volume (cc).

**FIG. 4.** Intravenous dipyridamole dose calculation worksheet.
14. Immediate and 4-hr delayed imaging are performed using the routine stress-thallium protocol.

15. The patient should be instructed to return to the laboratory 3-4 hr after the time of $^{201}$Tl dose administration for redistribution imaging. The patient may be allowed to eat a light meal during the waiting interval.

**Precautions**

Intravenous dipyridamole has been shown to induce angina. For any patient who experiences chest pain during dipyridamole infusion, the following steps should be taken:

1. Assess the severity of chest pain (0 = none, 1+ = mild, 2+ = moderate, 3+ = moderately severe, 4+ = severe).
2. At the point of 4+ chest pain:
   A. Stop dipyridamole.
   B. Administer $^{201}$Tl dose.
   C. Administer i.v. aminophylline (100-125 mg; rate = 50 mg/min) 1 min following $^{201}$Tl administration if the pain persists.
3. For 2+-3+ chest pain or substantial ST segment depression without pain that persists 5 min after the intervention is completed, administer i.v. aminophylline as above.
4. If symptoms continue at 3 min following the aminophylline administration, give sublingual nitroglycerin (0.4-0.8 mg).
5. If the symptoms persist at 4 min following the nitroglycerin, repeat the initial aminophylline dose.

**Possible Dipyridamole Side Effects**

Angina, nausea, headache, and hypotension.

**Possible Aminophylline Side Effects**

Nausea, headache, hypotension, tachycardia, flushing, and ventricular arrhythmias.

**Contraindications**

1. Allergy to aminophylline, ethylenediamine, dipyridamole, FD&C yellow (tartrazine) dye, aspirin.
2. Patients with resting angina should not be given dipyridamole.

**ORAL ADMINISTRATION**

**Protocol:** Thallium myocardial imaging following oral dipyridamole administration.

**Indications:**
1. To diagnose ischemic heart disease.
2. To evaluate the extent of coronary artery disease.
3. To evaluate therapeutic procedures such as bypass surgery and angioplasty.

**Instrumentation:** LFOV scintillation camera with SPECT acquisition and processing (This procedure may be performed with SFOV instrumentation and utilizing planar imaging techniques).

**Collimator:** Low-energy all-purpose.

**Radiopharmaceutical:** Thallium-201-chloride 3.0 mCi (111 MBq).

**Patient preparation:**
1. NPO 2 hr prior to testing.
2. Restrict intake of xanthine derivatives. Patients should be instructed not to consume coffee, tea, carbonated drinks containing caffeine, or chocolate at least 8 hr prior to the study. Patients taking theophylline should discontinue the medication 24-36 hr prior to the study.

**Procedure:**
1. Check the patient's chart or request slip for the order for the study.
2. Ascertain the clinical indications for performing the study.
3. Properly identify the patient.
4. Obtain a drug allergy history:
   A. Dipyridamole (Persantine®).
   B. FD&C yellow dye (Tartrazine).
   C. Aspirin.
   D. Aminophylline.
   E. Ethylenediamine.
5. The patient is given a verbal explanation of the procedure and is asked to read and sign the appropriate consent form.
6. A 12-lead ECG is to be taken at rest and at 15-min intervals following the administration of the dipyridamole solution.
7. An indwelling catheter is introduced via a 20-gauge Jelco needle in an antecubital vein. A 500-cc or 1000-cc bag of D5W is attached to the catheter via standard i.v. tubing delivery systems to maintain i.v. patency.
8. A blood pressure cuff is placed on the patient's arm opposite the catheter. Heart rate and blood pressure are recorded every 15 min following administration of the oral dipyridamole solution.
9. An oral dipyridamole suspension can be prepared by crushing tablets and suspending the powder in solution (water or corn syrup). A dose of 300 mg of oral dipyridamole provides serum levels
FIG. 5. Manual intravenous administration of dipyridamole dose.

FIG. 6. Administration of \(^{201}\)TI dose.

FIG. 7. Case 1. Rows 1 and 3 are the short-axis slices immediately following dipyridamole infusion. Rows 2 and 4 are the same slices 4 hr later. The slices are contiguous and represent the myocardial activity from the apex to the base of the heart.


10. The patient is requested to drink the prepared dipyridamole solution. The dose solution may be followed with 100–200 cc of water.

11. Thirty-five to 40 min after the patient has taken the dipyridamole dose, or earlier if symptoms are present, the patient is asked to walk in place for 5 min.

12. Thallium-201 is injected through the catheter at 4 min following initiation of the walking exercise, or sitting position. Walking or sitting is continued for an additional 1 min following $^{201}$Tl administration.

13. Immediate and 4-hr delayed imaging are performed using the routine stress thallium protocol.

14. The patient should be instructed to return to the laboratory 3–5 hr after the time of $^{201}$Tl dose administration for redistribution imaging. The patient may be allowed to eat a light meal during the waiting interval.

Precautions

Oral dipyridamole may induce angina. For any patient who experiences chest pain during dipyridamole administration, the following steps should be taken:

1. Assess the severity of chest pain (0 = none, 1+ = mild, 2+ = moderate, 3+ = moderately severe, 4+ = severe).

2. At the point of 4+ chest pain:
   A. Administer $^{201}$Tl dose.
   B. Administer i.v. aminophylline (100–125 mg; rate 50 mg/min) 1 min following $^{201}$Tl administration if the pain persists.
3. For 2+-3+ chest pain or substantial ST segment depression without pain that persists 5 min, administer i.v. aminophylline as above.
4. If symptoms continue at 3 min following the aminophylline administration, give sublingual nitroglycerin (0.4-0.8 mg).
5. If the symptoms persist at 4 min following the nitroglycerin, repeat the initial aminophylline dose.

**Possible Dipyridamole Side Effects**

Angina, nausea, headache, and hypotension.

**Possible Aminophylline Side Effects**

Nausea, headache, hypotension, tachycardia, flushing, and ventricular arrhythmias.

**Contraindications**

1. Allergy to aminophylline, ethylendiamine, dipyridamole, FD&C yellow (tartrazine) dye and, aspirin.
2. Patients with resting angina should not be given dipyridamole.

**CASE EXAMPLES**

**Case 1**

A 45-yr-old female with a past history of CAD and previous coronary angioplasty was referred for $^{201}$TI-dipyridamole imaging. Pharmacologic stress testing was preferred to routine treadmill testing, due to the patient’s exercise intolerance. Intravenous dipyridamole thallium imaging was performed according to the above protocol.

The patient’s resting heart rate (HR) and blood pressure (BP) were 65 and 126/78, respectively. The baseline ECG showed poor R-wave progression with T-wave inversion in V4-V6. Twenty-six milligrams of dipyridamole were infused, and the patient performed 3 min of handgrip exercise. The handgrip exercise was terminated earlier than the normal protocol due to chest pain and the $^{201}$TI dose was administered. At the time of $^{201}$TI administration, the HR = 90, and BP = 126/76. Six minutes after the completion of dipyridamole infusion the ECG demonstrated 1-mm ST depression. Following the $^{201}$TI injection, the patient continued to complain of moderately severe chest pain. Two 125-mg doses of i.v. aminophylline were administered and resolved the patient’s complaint of chest pain. Figures 7 and 8 are the short-axis slices and the polar coordinate maps of the $^{201}$TI distribution. This study shows normal tracer distribution.

**Case 2**

A 64-yr-old male with a history of previous aorto-coronary bypass (1977) was referred for $^{201}$TI-dipyridamole testing to evaluate his current complaint of “chest burning.” Pharmacologic stress testing was preferred to standard treadmill exercise due to the patient’s peripheral vascular disease.

The patient’s resting HR and BP were 59 and 160/97, respectively. The baseline ECG demonstrated a previous anterior wall MI. Forty-nine milligrams of dipyridamole were administered intravenously followed by 5 min of handgrip exercise. The $^{201}$TI dose was administered 1 min prior to the predetermined endpoint of the handgrip exercise. At the time of $^{201}$TI administration the HR was 80 and BP was 160/80. During the dipyridamole infusion and the period of handgrip exercise, the ECG demonstrated occasional premature atrial contractions without evidence of ischemic changes. During the procedure, the patient complained of mild chest pain and throat tightness.

Tomographic imaging was performed immediately after dipyridamole infusion and 4 hr later. Figures 9 and 10 represent the short-axis slices and the polar coordinate maps of the $^{201}$TI distribution. These images show inferior and apical infarction with ischemia in the anterior, lateral, and inferior walls.

**CONCLUSIONS**

The use of intravenous dipyridamole in concert with $^{201}$TI imaging has been proven to be a safe and effective method to evaluate patients suspected of CAD. The availability of this test to your patients and referring medical staff provides a method for noninvasive assessment of CAD in patient groups that previously could not be reliably studied using other clinically accepted procedures. An additional benefit derived from providing this diagnostic technique will be an enhancement of your department's esteem and prestige.

**ACKNOWLEDGMENTS**

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**REFERENCES**