Journal of the American College of Cardiology Vol. 47, No. 11, 2006

Published by Elsevier Inc.
ISSN 0735-1097/06/$32.00
doi:10.1016/j.jacc.2005.11.088

# Effect of Caffeine on Ischemia Detection by Adenosine Single-Photon Emission Computed Tomography Perfusion Imaging

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**OBJECTIVES** The purpose of this research was to study the effect of one cup of coffee taken 1 h before adenosine stress on the results of myocardial perfusion imaging.

**BACKGROUND** Caffeine is believed to attenuate the coronary hyperemic response to adenosine by competitive blockade of the A2a receptor. Caffeine is commonly withheld before adenosine single-photon emission computed tomography (SPECT) perfusion imaging so as not to mask ischemia detection.

**METHODS** We studied the effect of one 8-oz cup of coffee taken 1 h before adenosine stress in patients who had demonstrable reversible defects on adenosine SPECT perfusion imaging performed while off caffeine.

**RESULTS** There were 22 men and 8 women, age 64 ± 9 years. The blood level of caffeine 1 h after intake was 3.1 ± 1.6 mg/l. There were two patients with ST-segment depression before and one after caffeine intake (p = NS). The summed stress score (SSS) based on 17 segments (scale of 0 to 3, 3 being normal) was 44 ± 5 before and 45 ± 5 after caffeine (p = NS). The summed difference score was 3.8 ± 1.9 before and. 3.9 ± 2.3 after caffeine (p = NS), reflecting that around 50% of the perfusion abnormality was reversible before and after caffeine. Using polar maps, the perfusion abnormality was 12 ± 10% at baseline and 12 ± 10% after caffeine (p = NS) in agreement with SSS. The left ventricular ejection fraction by gated SPECT was 50 ± 13% at baseline and 51 ± 13 % after caffeine (p = NS).

**CONCLUSIONS** A cup of coffee does not mask the presence or severity of reversible defects induced by adenosine SPECT imaging. (J Am Coll Cardiol 2006;47:2296–302) © 2006 by the American College of Cardiology Foundation

Approximately 50% of the 2.4 million pharmacological single-photon emission computed tomography (SPECT) perfusion tests performed each year in the U.S. are done with adenosine (1). It is generally accepted that caffeine lowers the sensitivity of adenosine SPECT by masking myocardial ischemia because it is a nonselective competitive inhibitor of A2 receptors (2). Activation of these receptors by adenosine and dipyridamole results in augmentation of myocardial blood flow (MBF). The guidelines of the American Society of Nuclear Cardiology consider the use of caffeine within 12 h of pharmacologic vasodilator perfusion imaging as an absolute contraindication to performing the test (3). Additionally, task force guidelines from the American Society of Nuclear Cardiology, American Heart Association, and American College of Cardiology recommend abstinence from caffeine for 24 h before vasodilator SPECT perfusion imaging (4). These recommendations stem from a few false-negative dipyridamole myocardial perfusion imaging results in the presence of caffeine (5,6). However, there are no data regarding the effects of caffeine on adenosine myocardial perfusion imaging. Based on the assumptions that: 1) the nature of the interaction between adenosine and caffeine is a competitive one; 2) most patients are likely to consume inadvertently one cup of coffee before the scheduled test; 3) the blood level of caffeine from such use is likely to be low; and 4) there is a higher interstitial level of adenosine after adenosine than dipyridamole use, we hypothesized that results from dipyridamole should not be extrapolated to adenosine use. This issue of interaction between caffeine and adenosine is clinically relevant because patients are denied vasodilator imaging and have their studies cancelled or switched to dobutamine stress testing if they had consumed coffee before testing. Therefore, we attempted to study the effect of caffeine on adenosine SPECT perfusion imaging.

## METHODS

**Study population.** The study included 30 patients with known or high likelihood of coronary artery disease who underwent adenosine gated SPECT perfusion imaging for clinical indications and who had evidence of reversible defects in one or more vascular territory. Patients with prior myocardial infarction (>3 months), prior percutaneous coronary intervention, prior coronary bypass grafting, or patients with an abnormal coronary angiogram showing ≥50% stenosis in one or more vessels were included in the study. Patients with unstable angina, known severe left main

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Manuscript received August 14, 2005; revised manuscript received November 2, 2005, accepted November 8, 2005.

**Abbreviations and Acronyms**

ATP = adenosine triphosphate
CFR = coronary flow velocity ratio
MBF = myocardial blood flow
SDS = summed difference score
SPECT = single-photon emission computed tomography
SRS = summed rest score
SSS = summed stress score

stenosis, active bronchospasm, severe heart failure, uncontrolled arrhythmias, symptomatic hypotension or severe hypertension (systolic blood pressure <90 or >200 mm Hg, respectively), high-degree atrioventricular block in the absence of a functioning pacemaker, and those on theophylline-containing medications were excluded from the study.

**Study design.** The second adenosine gated SPECT perfusion imaging (study test) was performed after the patients drank an 8-oz cup of brewed caffeinated coffee 1 h before adenosine stress. The stress modality in both studies was adenosine only, without augmentation with low level of exercise. There was no intervention between the first and second studies and no change in clinical status or medications. All patients drank the same kind and quantity of coffee, which was prepared by the cafeteria at the University of Alabama at Birmingham. Blood caffeine levels were drawn 1 h after coffee ingestion. Patients abstained from caffeine for 24 h before each test, and the same SPECT protocol (1-day rest/stress or stress/rest or 2-day protocol) and same tracer were used in both studies. A 6-min infusion of 140 μg/kg/min of adenosine was performed. Tc-99m sestamibi or Tc-99m tetrofosmin was injected at the third minute of infusion. Image acquisition was done approximately 60 min after injection using a dual-head gamma camera according to accepted standards (7). The institutional review board approved the study protocol, and informed consent was obtained from each patient before enrollment. There were no complications.

**SPECT interpretation.** The images were coded so that the interpreters were blinded regarding the sequence of studies. The two studies, without and with caffeine, were read blindly side by side. The interpretation addressed presence, severity, and extent of abnormality. The summed stress score (SSS) based on 17 segments (scale of 0 to 3, where 3 is normal and 0 is absent perfusion) and the summed rest score (SRS), which reflect the total perfusion defect at stress and rest respectively, were determined. The summed difference score (SDS), which reflects reversible defects, was determined as the difference between SSS and SRS. Quantitative analysis of total defect size was performed with the polar maps using Quantitative Perfusion SPECT (Cedars-Sinai Quantitative Software Package, Los Angeles, California). The left ventricular ejection fraction was measured from the gated images.

**Statistical analysis.** Statistical analysis was performed using SAS version 8.1 for Windows (SAS Institute Inc., Cary, North Carolina) and the SPSS software (version 10.05.5, SPSS Inc., Chicago, Illinois). Results are expressed as percent frequency or mean ± 1 SD where appropriate. Categorical variables were assessed by a chi-square test and continuous variables by a paired Student *t* test. Correlations were measured using a Pearson coefficient and the Bland-Altman plots. A p value <0.05 (two-tailed) was considered statistically significant. It was estimated that a sample size of 30 patients would provide 80% power to detect a total defect size reduction of approximately 1.2% using a one-sided test at the 5% significance level or two-sided test at the 2.5% significance level. A 95% t-distribution-based confidence interval for the mean within-patient difference is 0.23 ± 0.92%.

## RESULTS

The baseline characteristics are listed in Table 1. All patients had coronary artery disease by history of prior myocardial infarction, history of revascularization, or >1 coronary artery with ≥50 stenosis. None of the eight women aged 48 to 76 years was on hormone replacement therapy. None of the patients had a history of liver disease. The daily and weekly caffeine consumptions are listed in Table 2. Serum caffeine levels were available for 29 patients. The mean caffeine level was 3.1 ± 1.6 mg/l (range 1 to 7 mg/l), and 60% of patients had caffeine levels from 1 to 3 mg/l (Fig. 1).

**Table 1.** Baseline Characteristics (n = 30 Patients)

| | |
|---|---|
| Demographics | |
| White | 25 (83) |
| Male | 22 (73) |
| Age (yrs) | 64 ± 9 |
| Weight (lbs) | 209 ± 39 |
| Height (inches) | 69 ± 3 |
| Medical history | |
| Hypertension | 26 (87) |
| Diabetes mellitus | 19 (63) |
| Dyslipidemia | 26 (87) |
| Active smoker | 7 (23) |
| Peripheral vascular disease | 5 (17) |
| Prior myocardial infarction | 18 (60) |
| Prior coronary artery bypass grafting | 16 (53) |
| Prior percutaneous coronary intervention | 18 (60) |
| Pacemaker | 3 (10) |
| Left bundle branch block | 1 (3) |
| Referral diagnosis | |
| Chest pains | 20 (67) |
| Preoperative | 2 (7) |
| Congestive heart failure | 8 (27) |
| Medications | |
| Beta-blockers | 25 (83) |
| Calcium blockers | 5 (17) |
| Nitrates | 15 (50) |
| Angiotensin-converting enzyme inhibitor | 15 (50) |
| Angiotensin receptor blocker | 8 (27) |
| Lipid lowering | 24 (80) |
| Theophylline | 0 (0) |

Values are n (%) or mean ± SD.

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**Table 2.** Daily Caffeine Consumption

| | Mean Cups | Median (Range) |
|---|---|---|
| Coffee | 2.0 ± 2.2 | 1 (0–12) |
| Tea | 1.5 ± 1.6 | 1 (0–6) |
| Cola | 1.2 ± 1.4 | 1 (0–4) |

Results are mean ± SD.

Twenty patients underwent one-day stress/rest SPECT imaging, and 10 patients underwent two-day stress/rest SPECT imaging. The SPECT and stress results are listed in Table 3. The perfusion results by vascular territory are listed in Table 4. Ischemia alone was present in 14 patients, whereas 16 patients had a combination of ischemia and scar. Single-vessel ischemia was present in 20 patients, two-vessel ischemia in 9 patients, and three-vessel ischemia in 1 patient. The mean left ventricular ejection fraction was 50 ± 14% on baseline study and 51 ± 13% after caffeine (p = 0.7). There were no significant differences in the SSS, SDS, and quantitative defect size with and without caffeine (Table 5). Using SSS, the percent left ventricular abnormality (SSS divided by 51) was 13.7% at baseline and 11.8% after caffeine (p = NS). Reversible defects (SDS) accounted for 54% at baseline and 65% after caffeine of total abnormality (p = NS). The scatter plots of the SDS and percent total defect size with and without caffeine are shown in Figures 2 and 3. There was no difference in the defect size with and without caffeine stratified by caffeine blood levels (Fig. 4). There was no conversion of a positive to a negative study after intake of caffeine. A representative example of SPECT images is shown in Figure 5. Among the 14 patients who had reversible defects only, the quantitative total defect size was 7.1 ± 3.2% at baseline and 7.4 ± 4.1% after caffeine (p = NS). Appendix 1 shows the details of perfusion defect distribution by vascular territories for the 30 patients. The defect size by SSS agreed nicely with defect size measured by the polar maps, which was 12 ± 10% at baseline and 12 ± 10% after caffeine (p = NS).

## DISCUSSION

The main finding of our study is that one 8-oz cup of coffee 1 h before adenosine stress did not attenuate the extent and severity of reversible defects by adenosine SPECT myocardial perfusion imaging. This finding was consistent across the different caffeine blood levels, although 60% of patients had a caffeine level of ≤3 mg/l.

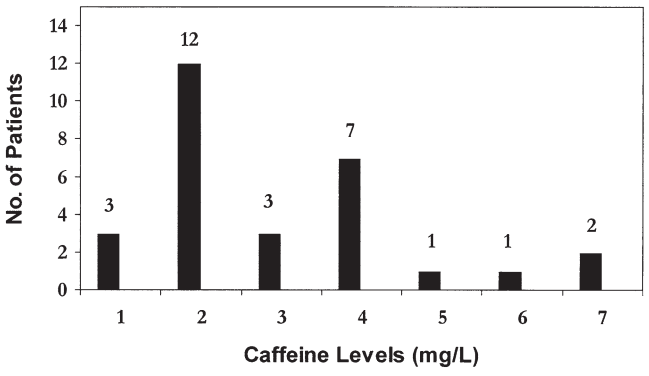


**Figure 1.** Distribution of caffeine levels among 29 patients.

**Table 3.** SPECT and Stress Characteristics Without and With Caffeine

| | Without Caffeine | With Caffeine | p Value |
|---|---|---|---|
| Tracer dosages | | | |
| 1-day rest dose (mCi) | 33.6 ± 3.0 | 32.0 ± 3.0 | 0.1 |
| 1-day stress dose (mCi) | 11.1 ± 1.3 | 11.2 ± 1.4 | 0.7 |
| 2-day rest dose (mCi) | 35.0 ± 3.6 | 36.9 ± 4.2 | 0.2 |
| 2-day stress dose (mCi) | 35.7 ± 3.6 | 34.3 ± 8.9 | 0.7 |
| Stress and hemodynamic results | | | |
| ECG positive | 2 (6.7) | 1 (3.3) | 0.4 |
| AV block (second degree) | 0 (0) | 1 (3.3) | 0.7 |
| Systolic BP decrease by >10 mm Hg | 21 (70.0) | 21 (70.0) | 0.8 |
| Decrease in systolic BP (mm Hg) | −20.7 ± 16.6 | −14.8 ± 15.6 | 0.1 |
| Increase in heart rate (beats/min) | 14.1 ± 9.1 | 13.4 ± 9.1 | 0.7 |

Values are n (%) or mean ± SD.
AV = atrioventricular; BP = blood pressure; ECG = electrocardiogram; SPECT = single-photon emission computed tomography.

**Caffeine properties.** Caffeine, a methylxanthine, is present in coffee, tea, soda, and chocolate (1). The caffeine content per 8-oz cup of coffee varies from 25 mg to 240 mg depending on the type of coffee and brewing method. Caffeine is completely absorbed from the gastrointestinal tract and is metabolized in the liver via the cytochrome P450 system. Its half-life is 2.5 to 4.5 h but can be as long as 12 h, although its metabolism is fairly uniform in the same individual (8,9). Cytochrome P450-inducing drugs and smoking tend to decrease the half-life of caffeine, whereas pregnancy, oral contraceptive drugs, cimetidine, and alcohol-induced liver disease tend to increase its half-life (1,8). The peak caffeine levels after 100 mg of oral caffeine range from 0.5 to 3.0 mg/l and are achieved within 15 to 120 min after consumption (8–10). In a study of 70 patients who were scheduled for pharmacologic myocardial perfusion imaging and who abstained from caffeine for 12 h, 74% of patients had caffeine levels from 0.1 to 8.8 mg/l and half had levels >1 mg/l (11). In another study in which 86

**Table 4.** Site of Ischemia in Relationship to Major Vessels

| Ischemia | |
|---|---|
| LAD only | 3 (10.0) |
| LCX only | 6 (20.0) |
| RCA only | 11 (36.7) |
| LAD/LCX | 2 (6.7) |
| LAD/RCA | 1 (3.3) |
| LCX/RCA | 6 (20.0) |
| LAD/LCX/RCA | 1 (3.3) |

Values are n (%). Sixteen of 30 patients also had fixed defects in the same or other vascular territories. The anterior wall and septum were considered to represent left anterior descending artery (LAD) territory, the inferior wall to represent right coronary artery (RCA) territory, and the lateral wall to represent the left circumflex artery (LCX) territory. The apex was considered in relation to adjacent territory, which was abnormal.

**Table 5.** Perfusion Defect Extent and Size Without and With Caffeine

| | Without Caffeine | With Caffeine | p Value |
|---|---|---|---|
| SSS | 44.0 ± 5.0 | 45.0 ± 5.0 | 0.2 |
| SDS | 3.8 ± 1.9 | 3.9 ± 2.3 | 0.8 |
| Total defect size (%)* | 12.4 ± 10.4 | 12.6 ± 10.1 | 0.6 |

*Using quantitative polar maps. Values are mean ± SD.
SDS = summed difference score; SSS = summed stress score.

patients abstained from caffeine for 24 h before pharmacologic myocardial perfusion imaging, 40% of patients had caffeine levels from 0.1 to 5.0 mg/l (12). Zheng et al. (13) reported that 66% of 36 patients who abstained from caffeine for 24 h before dipyridamole myocardial perfusion imaging had detectable plasma caffeine within the range of 0.1 to 0.8 mg/l.

**The effects of caffeine on the cardiovascular system.** Caffeine is a weak nonselective competitive adenosine A2 receptor antagonist compared with theophylline (14). Caffeine results in a modest increase in the mean, diastolic, and systolic blood pressures and a decrease in heart rate (2,9,15). It is hypothesized that caffeine affects the coronary vasomotor tone directly by blocking the A2 receptors and indirectly by stimulating catecholamine release, which stimulates the alpha-2 receptors resulting in vasoconstriction (15). In human studies, caffeine attenuated the dipyridamole and adenosine-induced decrease in blood pressure and the increase in heart rate (2,5,6,9,13,16–18). Smits et al. (17) showed that caffeine at a mean concentration of 5.2 ± 0.6 mg/l significantly attenuated the blood pressure and heart rate responses of adenosine, although other investigators showed these effects at caffeine levels as low as 1 mg/l (16).

**The effects of caffeine on MBF.** Bottcher et al. (15) studied the effect of caffeine on MBF at rest and during dipyridamole-induced hyperemia in 12 healthy volunteers after one to two cups of coffee using dynamic positron emission tomography. Caffeine levels achieved in this study ranged from 0 to 8 mg/l. Caffeine did not alter the resting MBF, but caused a dose-dependant attenuation of the dipyridamole-induced hyperemic flow velocity and the coronary flow velocity reserve ratio (CFR) (3.4 ± 0.8 without caffeine vs. 2.3 ± 0.7 with caffeine). These effects of caffeine were attributed to blunting of the rate-pressure product, but especially the heart rate response, which was inversely related to the plasma caffeine concentration. However, 75% of the subjects had a flow reserve >2 despite caffeine, and the investigators suggested that the diagnostic accuracy of pharmacologic stress testing might not be altered in the majority of patients who drank coffee.

In another study, Kubo et al. (19) studied the effect of caffeine intake on hyperemic MBF induced by adenosine triphosphate (ATP) and dipyridamole using positron emission tomography in 10 healthy volunteers with and without 3 mg/kg of oral caffeine (approximately two to three cups of coffee). The caffeine serum levels achieved were 3.1 ± 1.6 mg/l in the ATP arm and 3.3 ± 1.3 mg/l in the dipyridamole arm of the study. The MBF induced by ATP decreased from 3.7 ± 0.67 ml/min/g before caffeine to

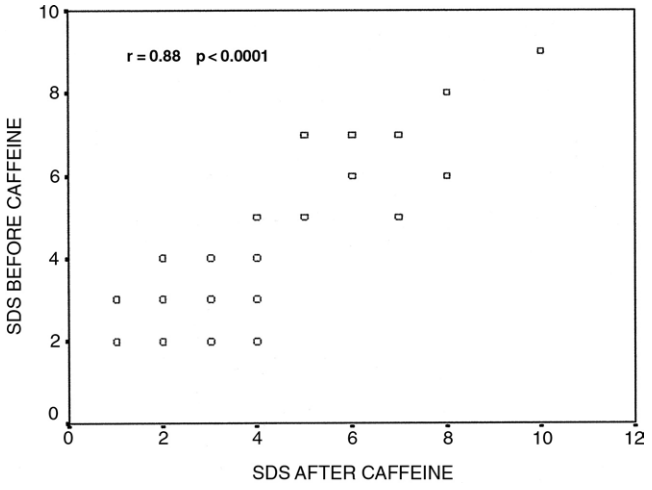


**Figure 2.** Scatter plot of the summed difference score (SDS) before and after caffeine.

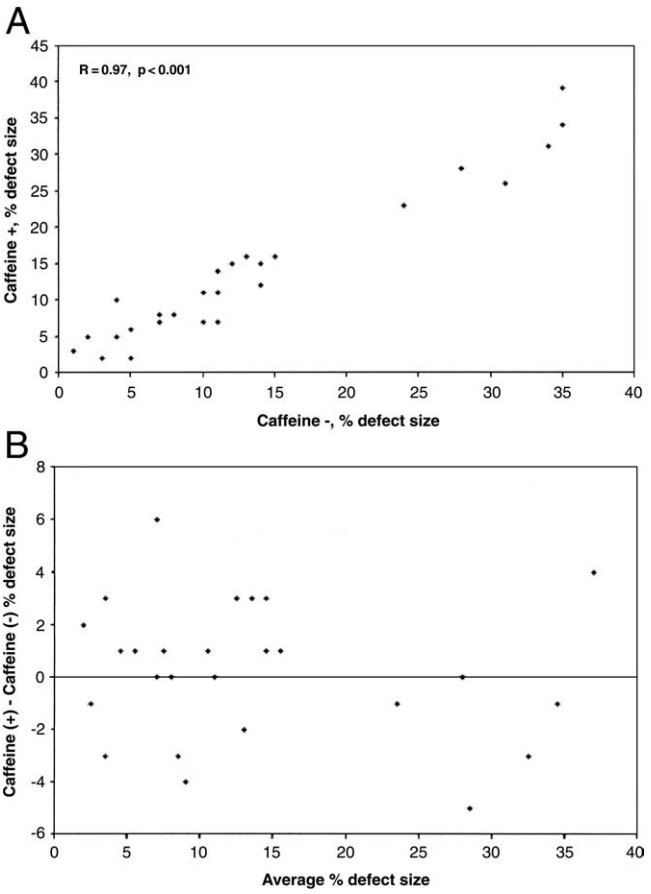


**Figure 3.** **(A)** Scatter plot of percent total defect size with (Caffeine +) and without (Caffeine −) caffeine. **(B)** Bland-Altman plot of the difference of the percent total defect sizes between the study with caffeine (Caffeine +) and the study without caffeine (Caffeine −) versus average percent total defect size. The data are compatible with the hypothesis that the mean Caffeine (+) and Caffeine (−) percent defect size difference is 0 ($t$ test p value = 0.61). For percent defect size, the empirical variance of within-patient differences is 2.46%, which for n = 30 yields a 95% confidence interval for the mean difference with half-width <1% (0.92%).

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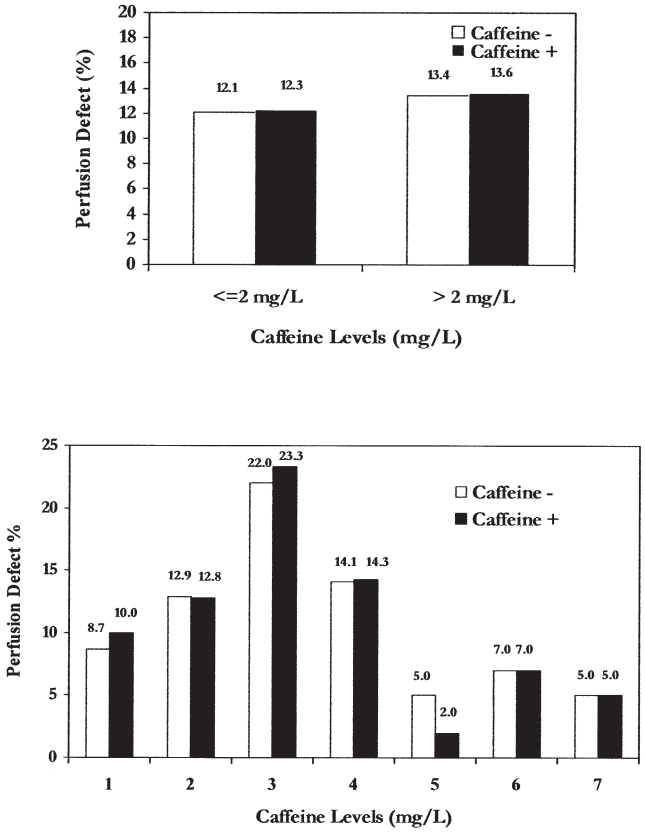


**Figure 4.** Perfusion defect size before **(top panel)** and after **(bottom panel)** caffeine stratified by caffeine levels.

1.68 ± 0.37 ml/min/g after caffeine ($p < 0.0001$), whereas that attributable to dipyridamole decreased from 3.0 ± 0.79 ml/min/g before caffeine to 1.52 ± 0.40 ml/min/g after caffeine ($p < 0.0001$).

In an animal study, the effect of caffeine on the selective adenosine receptor agonist ATL-146e was studied before and after 5 mg/kg of intravenous caffeine was administered to 19 anesthetized open-chest dogs. In this study, the ATL-146e–induced CFR decreased from 3.5 ± 0.4 without caffeine to 2.0 ± 0.3 with caffeine ($p < 0.05$) (20).

In a study of 10 patients, the fractional flow reserve induced by intracoronary adenosine administration was determined before and after 5 min of 4 mg/kg intravenous caffeine. At a serum caffeine level of 3.7 ± 1.8 mg/l, there was no difference in the fractional flow reserve compared with no caffeine (0.75 ± 0.14 vs. 76 ± 0.13, respectively, $p = 0.7$) (21).

**The effects of caffeine on myocardial perfusion imaging.** A case report by Smits et al. (5) showed that 4 mg/kg of caffeine infused 30 min before a dipyridamole thallium planar study (caffeine level of 8.8 mg/l) attenuated the ischemic electrocardiographic and the myocardial perfusion defects that were seen when the study was performed after 36 h of caffeine abstinence. In a subsequent study of eight patients, Smits et al. (6) showed that caffeine at an intravenous dose of 4 mg/kg with an achieved plasma concentration of 9.7 ± 3.7 mg/l reversed ST-segment changes and chest pain in four patients and attenuated perfusion defects in 75% of the patients. In a study of 86 patients who underwent vasodilator myocardial perfusion imaging after 24 h of abstinence from caffeine, the frequency of thallium redistribution was similar among patients with caffeine levels of 0 mg/l, 0.1 to 0.9 mg/l, and ≥1 mg/l (12). Heller et al. (22) studied the effects of intravenous theophylline on adenosine thallium-201 imaging in 16 patients. Theophylline significantly attenuated the adenosine-induced increase in heart rate, side effects, and ischemic electrocardiographic changes. However, despite the fact that theophylline is a stronger A2 receptor antagonist than caffeine (23), the size of the perfusion abnormality was unchanged with and without theophylline using segmental analysis or a computerized score (22).

It has been suggested that a caffeine level of 2 to 2.9 mg/l should be the lower limit for a false-negative dipyridamole myocardial perfusion imaging result based on the attenuation effect of caffeine on dipyridamole or adenosine humoral or side effects (11,13).

Although methylxanthines attenuate the humoral responses of adenosine and dipyridamole, the coronary vasodilatory effects are independent of its systemic hemodynamic effects. Additionally, dipyridamole and adenosine have different mechanisms of action and thus would interact differently with caffeine. Dipyridamole acts as an indirect A2 agonist by inhibiting the facilitated adenosine reuptake across the cellular membranes of vascular endothelial cells, thus increasing the concentration of endogenous adenosine at the receptor sites (24,25). Infused adenosine, on the other hand, acts directly with the various adenosine receptors. Thus adenosine and dipyridamole have different pharmacodynamic and bioavailability properties, and subsequently the interstitial concentration of adenosine is much higher after adenosine administration than after dipyridamole administration. This is an important difference because caffeine is a competitive inhibitor, which means that the very high adenosine interstitial concentration may overwhelm the receptors and continue to produce coronary hyperemia. This difference between adenosine and dipyridamole is seen as a more robust blood pressure decrease and heart rate increase and more defect severity and extent with adenosine compared with dipyridamole (26–28). Further, even if the degree of hyperemia is slightly reduced, it may have no important impact on the results of perfusion imaging because the tracer extraction levels off at flow rates above 2.5-fold of the baseline flow. In the studies in which caffeine attenuated the dipyridamole-induced CFR, the majority of patients had a MFR in excess of two despite caffeine inhibition.

The results of our study are different from those of Smits et al. (6) because adenosine was used instead of dipyridamole. Additionally, lower caffeine levels were achieved in our study, and were one-third of those achieved in the study by Smits et al. Thus, it is reasonable to conclude that one cup of coffee at a mean caffeine level of 3.1 ± 1.6 mg/l does not

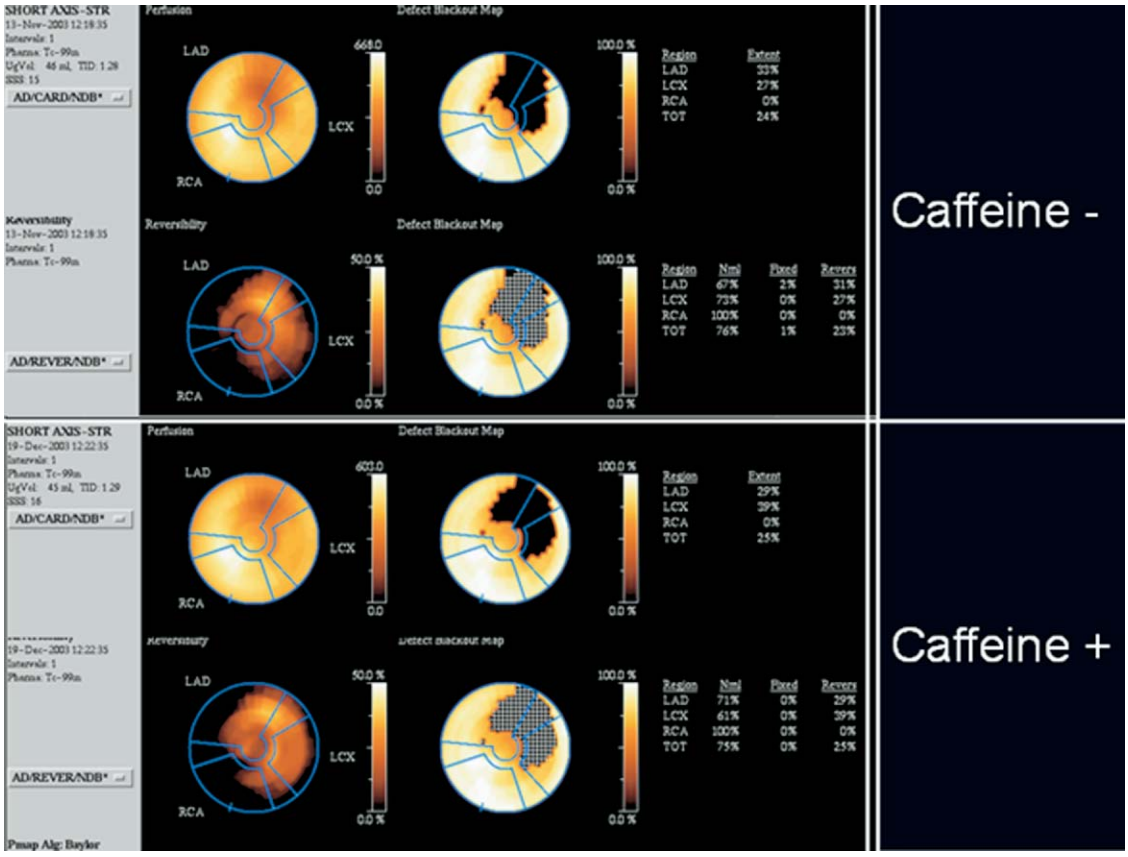


**Figure 5.** Polar map showing a perfusion defect before (Caffeine −) and after caffeine (Caffeine +). A 53-year-old white woman with diabetes mellitus, hypertension, dyslipidemia, and a history of moderate coronary artery disease underwent adenosine single-photon emission computed tomography for evaluation of chest pain. The baseline and caffeine study showed two-vessel ischemia with no significant difference in extent and severity of the defect between the two studies (caffeine blood level 2 mg/l). LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery; TOT = total.

attenuate the results of adenosine myocardial perfusion imaging, and therefore these patients could undergo adenosine SPECT imaging and the results should be clinically meaningful.

**Study limitations.** The first limitation of this study is that it did not have a randomized crossover design. It is true that because all patients underwent a no-caffeine study followed by a prior-caffeine study, the period effect, if any, is confounded with the effects of prior caffeine. A crossover design would address this concern but might raise ethical concerns because results of the no-caffeine study would be delayed for patients randomized to receive the prior caffeine study first. There was no change in clinical condition and medications between the two studies. Additionally, both studies were performed similarly with respect to adenosine dose, SPECT protocol, tracer, tracer dose, and image acquisition. The caffeine levels were not measured in the first or baseline study or before ingestion of caffeine in the second or test study. It could be that some patients had a measurable caffeine level despite abstinence for 24 h. The sample size did not allow further determination of a dose-response effect. However, our study is the largest study thus far on the effects of caffeine on adenosine myocardial perfusion imaging. Many (approximately 50%) of our patients had fixed in addition to reversible defects. Although it would have been ideal to select only those with reversible defects, we were more interested in testing the hypothesis on a representative group of patients undergoing pharmacological stress testing. Further, even in patients with fixed defects, approximately 50% of the perfusion abnormality was still reversible. Although other medications, such as nitrates, calcium channel blockers, and beta-blockers, could have attenuated the degree of ischemia, the patients were on the same cardiac medications in the two studies. Thus any medication-related attenuation in perfusion defects would be expected to occur in both studies and would not alter the effects of caffeine on adenosine SPECT perfusion imaging. Finally, although the interpretation was blinded and sequential, the readers were aware that one study was with and one was without caffeine. Mixing the images with images from other patients would have been preferred. The results, however, were consistent with automated measurements using polar maps.

**Conclusions.** One cup of coffee did not attenuate the results of adenosine myocardial perfusion imaging. Until further studies become available, we believe that one cup of coffee should not be a reason to cancel a scheduled adenosine study or to change to dobutamine. We continue to ask

our patients to refrain from taking coffee or similar products the morning of the test. We believe the current guidelines need to be revised to improve laboratory throughput and patient comfort.

### Acknowledgment

The authors thank Ann Olmsted, Associate Director, Biostatistics (CV Therapeutics, Palo Alto, California) for her statistical support.

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## APPENDIX

Perfusion Defect Distribution by Vascular Territories for the Individual Patients

| Patient | Ischemia | Scar |
|---|---|---|
| 1 | LCX/RCA | LCX |
| 2 | RCA | |
| 3 | RCA | |
| 4 | RCA | RCA |
| 5 | LCX | |
| 6 | LCX | RCA |
| 7 | RCA | |
| 8 | LCX | |
| 9 | LCX | LCX/RCA |
| 10 | RCA | LAD |
| 11 | RCA | |
| 12 | LCX/RCA | LCX |
| 13 | LCX | RCA |
| 14 | RCA | |
| 15 | LCX/RCA | |
| 16 | RCA | RCA |
| 17 | LAD | |
| 18 | LAD | |
| 19 | LAD/RCA | |
| 20 | LCX/RCA | RCA |
| 21 | RCA | |
| 22 | LAD/LCX | RCA |
| 23 | LAD/LCX | LAD |
| 24 | LCX | RCA |
| 25 | LAD/LCX/RCA | LCX/RCA |
| 26 | RCA | |
| 27 | LCX/RCA | LAD/LCX/RCA |
| 28 | LAD | LCX/RCA |
| 29 | RCA | |
| 30 | LCX/RCA | RCA |

LAD = left anterior descending; LCX = left circumflex; RCA = right coronary artery.