CEwebsource.com
Your Source for Continuing Education
developed exclusively for Medical Professionals

MYOCARDIAL PERFUSION STRESS TESTING IN NUCLEAR MEDICINE

Saabry Osmany, MD
Diplomate, American Board of Nuclear Medicine
Email: sosmany@hotmail.com

INTRODUCTION

Myocardial perfusion imaging (MPI) reflects myocardial flow in coronary arteries as well as the viability of the perfused myocardium. Stenosed arteries may have a reduced myocardial flow reserve compared with normal vessels. This difference is caused by physiological impairment due to atherosclerotic narrowing.[1]

Myocardial perfusion imaging is the only noninvasive, widely available test for assessing myocardial perfusion directly.[2] Radionuclides used in MPI[3] include single-photon emitters such as thallium-201 and technetium-99m and positron emitters such as nitrogen-13 and rubidium-82. The sensitivity of single-photon emission computed tomography (SPECT) can vary from 90% to 94% for multivessel coronary disease, to 60% to 76% for significant single-vessel disease.[4] PET radiotracers have been shown to have a sensitivity of 93% and a specificity of 92%.[3] MPI with single-photon emitters reflects relative differences in coronary blood flow at rest and relative coronary flow reserves during stress.[1] MPI with positron emitters can allow the quantification of coronary blood flow and coronary flow reserves.[3]

MPI can be used to detect coronary artery disease (CAD), determine the degree of CAD for treatment management planning, distinguish viable from nonviable myocardium, and evaluate patients before noncardiac surgery.[3] Additionally, SPECT or positron emission tomography acquisition with cardiac gating allows assessment of regional and global ventricular function.[3,4] A summary of indications for MPI is shown in Table 1.

*TABLE 1. Indications for Myocardial Perfusion Imaging**

Diagnosis and Prognosis of Coronary Artery Disease (CAD)
• Presence
• Location (coronary territory)
• Severity
Assessment and Risk Stratification for Selection of Therapy
• In known CAD, including postmyocardial infarction
• Distinguish viable ischemic myocardium from scar tissue
• Preoperative evaluation for noncardiac surgery
Evaluation of Therapy in Known CAD
• Coronary revascularization
• Medical therapy for congestive heart failure or CAD
• Lifestyle modification
Hemodynamic Significance of
• Coronary lesions
• Coronary aneurysms in Kawasaki disease
• Known or suspected anomalous coronary arteries and myocardial bridging

*Adapted from data from the guidelines of the Society of Nuclear Medicine (SNM),[4] the British Nuclear Cardiology Society (BNCS), the British Cardiac Society (BCS), and the British Nuclear Medicine Society (BNMS).[2]

Imaging can involve stress imaging, rest imaging, or stress and rest nuclear cardiac imaging.[3] At rest, if there is a drop in perfusion pressure across an atherosclerotic coronary artery stenosis, distal arteriolar resistance is decreased by autoregulatory mechanisms that maintain distal blood flow. This mechanism may maintain normal resting coronary blood flow by using flow reserve, thus maintaining perfusion in coronary stenosis of up to 85% to 90%.[1] If perfusion is not maintained at rest, the relative differences in coronary blood flow between stenosed and normal vessels may be visualized by differences in initial radiotracer uptake by the myocardium.

CEwebsource.com *is produced by Enterprises for Continuing Education Inc. (ECEI), 10381 Citation, Ste 200 Brighton, MI 48116,* **(810) 229-3354.** *The views expressed in the journal are the authors' and do not necessarily represent those of ECEI. Full and complete prescribing information should be reviewed regarding any product mentioned herein prior to use. ECEI hereby authorizes you to copy documents published by ECEI on the World Wide Web for non-commercial use only. In consideration of this authorization, you agree that any copy of these documents which you make shall retain all copyright and other proprietary notices contained herein.*

MODALITIES FOR MYOCARDIAL PERFUSION STRESS TESTING

Myocardial perfusion can be evaluated by the use of exercise or pharmaceuticals (vasodilators [e.g., adenosine] or inotropic agents [e.g., dobutamine]).[3] Dynamic exercise stress testing is preferred over other methods when the patient can achieve an adequate workload.[2,5] Stress testing with vasodilators is preferred when exercise is contraindicated (e.g., in patients with left bundle branch block [LBBB] or paced rhythms).[2] Inotropic agents are used when vasodilators are contraindicated. The indications and contraindications for stressing myocardial perfusion by exercise, adenosine, and dobutamine, as compiled from a number of guidelines, are summarized in Tables 2 and 3.

When assessing a patient with unstable angina for contraindications, if one of the following conditions are present, the patient has high-risk angina (i.e., a high short-term risk of death or myocardial infarction)[6]:

- Prolonged, ongoing (>20 min) chest pain at rest
- Pulmonary edema, most likely related to ischemia
- New or worsening mitral regurgitation murmur
- S_3 or new/worsening rales
- Hypotension, bradycardia, tachycardia
- Age older than 75 years
- Angina at rest with transient ST changes <0.05 millivolt
- Bundle branch block–new or presumed new/sustained
- Ventricular tachycardia
- Elevated biochemical cardiac markers (e.g., troponin T or I greater than 0.1 mg per mL)

*TABLE 2. Indications for Stress Myocardial Perfusion Imaging**

Exercise	Adenosine	Dobutamine
Detection of CAD Assessing prognosis in patients with symptoms suggestive of CAD or in those with risk factors for CAD Risk stratification of postmyocardial infarction patients and in patients with unstable angina or chronic CAD into low-risk category who can be managed medically, or into high-risk category who should be considered for revascularization procedures Cardiac risk stratification before noncardiac surgery in patients with known CAD or those with risk factors for CAD Evaluation of the efficacy of therapeutic interventions (anti-ischemic drug therapy/revascularization) in patients with known CAD	Patients unable to exercise optimally: extracardiac factors (pulmonary, peripheral vascular, musculoskeletal conditions) Treatment with medications that blunt the heart-rate response to exercise (ß-blockers, calcium channel blockers) Poor motivation Evaluation of patients very early after acute myocardial infarction (< 2 days) or very early after angioplasty/stenting (< 2 weeks) Left bundle branch block (LBBB)—exercise stress leads to perfusion abnormalities in the septum and adjacent walls in the absence of obstructive coronary disease.	Patients who cannot undergo exercise stress and have contraindications to pharmacologic vasodilator stressors

*Data adapted from ASNC[5,7,8] and BNCS/BCS/BNMS[2] guidelines

*TABLE 3. Contraindications for Stressing Myocardial Perfusion**

	Exercise	Adenosine	Dobutamine
ABSOLUTE	Acute myocardial infarction (within 2 days) High-risk unstable angina Uncontrolled cardiac arrhythmias causing symptoms or hemodynamic compromise Symptomatic severe aortic stenosis (possible syncope and cardiac arrest) Uncontrolled symptomatic heart failure Acute pulmonary embolus or pulmonary infarction Acute myocarditis or pericarditis Acute aortic dissection	Asthmatic patients with ongoing wheezing or requiring methylxanthine medication for control Pulmonary hypertension A history of severe bronchospasm Greater than first-degree heart block without a pacemaker Sick sinus syndrome SBP < 90 mm Hg Medications: Dipyridamole in the last 24 hours, Xanthines (aminophylline, caffeine) in the last 12 hours Prior hypersensitivity to adenosine History of prior intubation for severe pulmonary disease Severe mitral valve disease Severe aortic stenosis Severe obstructive hypertrophic cardiomyopathy Severe orthostatic hypotension Pregnant or lactating patients	Recent (< 1 week) myocardial infarction Unstable angina Hemodynamically significant left ventricular (LV) outflow tract obstruction Critical aortic stenosis Atrial tachyarrhythmias with uncontrolled ventricular response Prior history of ventricular tachycardia Uncontrolled hypertension Patients with aortic dissection or large aortic aneurysm Patients who are on ß-blockers may not show adequate heart rate response to dobutamine. It is preferable to use adenosine or dipyridamole in these patients. Known hypokalemia
RELATIVE	Left main coronary stenosis Moderate stenotic valvular heart disease Electrolyte abnormalities Severe arterial hypertension (systolic blood pressure (SBP) of > 200 mm Hg and/or diastolic blood pressure (DBP) of > 110 mm Hg) Tachyarrhythmias or bradyarrhythmias Hypertrophic cardiomyopathy and other forms of outflow tract obstruction (sudden death due to arrhythmias) Mental or physical impairment leading to inability to exercise adequately High-degree atrioventricular block	Severe sinus bradycardia (heart rate < 40/min) Recent cerebrovascular accident	Left bundle branch block Bifascicular block Paced rhythm

*Adapted from AHA/ACC,[6] ASNC,[5] SNM[4] and BNCS/BCS/BNMS[2] guidelines.

EXERCISE MYOCARDIAL PERFUSION STRESS TESTING

Exercise stressing of myocardial perfusion can be submaximal, symptom limited, or maximal.[4] It may be conducted through a number of methods using treadmills or bicycle ergometers. Rhythmic exercise stressing gradually increases myocardial oxygen consumption, which leads to a 2.0- to 2.5-fold increase of blood flow in normal coronary arteries.[9]

Coronary vessels with stenosis do not have as great an increase in blood flow[9] because part of the flow reserve has already been used to maintain flow at rest.[1] This leads to a relative decrease in myocardial perfusion in the regions supplied by the stenosed vessels.[9] If the discrepancy between increased demand and relatively decreased supply is great enough, myocardial ischemia may be elicited.[9] A finding of adequate exercise stress not causing ischemia suggests that the degree of arterial narrowing is insufficient to cause ischemia during exercise, although ischemic events through spasm, plaque rupture, and thrombosis are still possible.[6] Induced ischemia is not necessary to visualize blood flow differences.[1]

For maximal exercise the preferred endpoint is one wherein an adequate workload has been reached, one indicator of which is achieving ≥85% of the patient's heart rate as predicted by age and gender, which can be calculated as follows:

0.85 x 0.85 x (220 for men or 210 for women) – patient's age[10]

However, this calculation is not a very reliable marker[9] of maximal effort due to statistical variation in the normal maximal values[1] as well as variation due to other factors, including medication and heart disease.

Patient effort can also be assessed using the patient exhaustion/Borg scale.[11] The Borg scale is a subjective scale that measures perceived exertion. A Borg scale rating of 9 to 10 on a scale of 0 to 10[11] suggests adequate exercise.

Peak stress can also be considered to have been reached when the heart-rate–blood-pressure product (obtained by multiplying the systolic blood pressure by the heart rate) is greater than 25,000. Other indicators include the onset of chest pain or significant electrocardiographic (ECG) changes.[12] There are several indications for early termination of an exercise stress test. These premature endpoints for exercise stressing of myocardial perfusion are shown in Table 4.

When an endpoint is reached, the radiotracer is injected and stressing is continued for an additional 1 minute after the injection of thallium-201[2] or 2 minutes after ^{99m}TC perfusion tracer injection.[2,5] Positron emitters are not usually used in conjunction with exercise myocardial perfusion stressing.[3] The continuation of exercise gives time for the clearance of the radiotracer from the blood pool while the myocardial perfusion achieved during maximal stress is maintained.[9] If the same level of exercise cannot be maintained, a lower level can be used.[9] Failure to reach an adequate workload reduces the sensitivity of the test.[4] In pharmacologically induced stress, the achievement of an adequate workload cannot be assessed.[2]

*TABLE 4. Premature Endpoints for Exercise Stress of Myocardial Perfusion**

Absolute Indications for Early Termination
Drop in SBP of > 10 mm Hg from baseline blood pressure despite an increase in workload, when accompanied by other evidence of ischemia
Moderate-to-severe angina
Increasing nervous system symptoms (e.g., ataxia, dizziness, or near-syncope)
Signs of poor perfusion (e.g., cyanosis or pallor)
Technical difficulties in monitoring ECG or SBP
Subject's desire to stop
Sustained ventricular tachycardia
ST elevation (≥1.0 mm) in leads without diagnostic Q-waves (other than V_1 or aVR)
Relative Indications for Early Termination
Drop in SBP of (≥10 mm Hg) from baseline blood pressure, despite an increase in workload and in the absence of other evidence of ischemia
ST or QRS changes such as excessive ST depression (>2 mm of horizontal or downsloping ST-segment depression) or marked axis shift
Arrhythmias other than sustained ventricular tachycardia, including multifocal premature ventricular contractions (PVC), triplets of PVCs, supraventricular tachycardia, heart block, or bradyarrhythmias
Fatigue, shortness of breath, wheezing, leg cramps, or claudication
Development of bundle-branch block or intraventricular conduction that cannot be distinguished from ventricular tachycardia
Increasing chest pain
Hypertensive response (SBP of >250 mm Hg and/or a DBP of > 115 mm Hg)

*Adapted from 2002 ANA/ACC Guidelines[6]

VASODILATOR MYOCARDIAL PERFUSION STRESS TESTING

Pharmaceutical stress increases blood flow in normal coronary arteries 3- to 5-fold.[1] Vasodilators (i.e., adenosine and dipyridamole) cause coronary artery dilatation. Adenosine acts directly, whereas dipyridamole increases the availability of endogenous adenosine. Adenosine increases the heart rate and decreases both systolic and diastolic blood pressure because. Coronary blood flow is increased 3.5- to 4.0-fold. This increase may be limited in stenosed arteries. Depending on coronary flow reserves and the degree of stenosis, this may cause a relatively low distribution of radiotracer in the regions supplied by the stenosed coronary arteries. Additionally, in severe CAD, coronary steal phenomenon may cause ischemia.[5] *Coronary steal* is defined as a fall in blood flow toward a certain vascular region in favor of another area during arteriolar vasodilation. Adenosine can be administered as a continuous infusion at a rate of 140 µg/kg/min over 4.5 or 6 minutes. In higher risk patients (e.g., those patients who have had a recent ischemic event), the infusion can be started at 70 µg/kg/min. If this is tolerated for 1 minute, the dose can be increased to 140 µg/kg/min for 4 minutes. The radiotracer is administered 3 minutes before the end of adenosine infusion.[5] Positron emitters may be injected after completing the infusion.[13] Indications for stopping adenosine are given in Table 5.

TABLE 5. Indications for Early Termination of Adenosine Stress Testing[5]

Severe hypotension (SBP <80 mm Hg)
Development of symptomatic, persistent second-degree or complete heart block
Wheezing
Severe chest pain associated with ≥2 mm ST depression

DOBUTAMINE MYOCARDIAL PERFUSION STRESS TESTING

The inotropic agents dobutamine and arbutamine are also used in stress testing. Dobutamine causes a direct, dose-related stimulation of β1- and β2-adrenergic receptors that increases heart rate, blood pressure, and myocardial contractility. These changes increase myocardial oxygen demand and thus increase coronary blood flow. Dobutamine may be administered by intravenous infusion using an infusion or syringe pump. The administered dose is usually started at 5 to 10µg/kg/min and increased incrementally every three to five minutes to 10, 15, 20, 30, and 40 µg/kg/min.[2,5] The radiopharmaceutical should be injected when >85% of the age- and sex-adjusted maximum predicted heart rate is reached, 1 minute into the maximum tolerated rate,[5] or at 40 µg/kg/min.[2] The infusion should be continued for 1 minute after injection of thallium–201 or 1 to 2 minutes after injection of ^{99m} Tc-labeled tracers and then stopped.[14] Positron emitters should be injected after the end of the infusion.[13]

Although atropine can be used in conjunction with dobutamine to increase the heart rate response, an increase in heart rate due to atropine administration does not cause the increase in coronary blood flow that exercise would have.[4] It has been stated[2] that for myocardial perfusion imaging, atropine may not be necessary. It has been shown that at doses as low as 20 µg/kg/min, dobutamine can cause a degree of vasodilation close to that induced by adenosine if the patient is pretreated with molsidomine.[15]

The indications for early termination of dobutamine are similar to those for exercise stress (see Table 4).[2] Termination for ventricular tachycardia or ST segment elevation is more likely with dobutamine than with other stressors.

SIDE EFFECTS OF EXERCISE AND PHARMACEUTICAL STRESS

Common side effects with dobutamine and adenosine are shown in Table 6. With adenosine, 80% of patients may experience minor side effects.[5] Due to a short half-life of 10 seconds, most side effects from adenosine resolve rapidly on stopping the infusion, although aminophylline may be used if needed. Low-level, upright treadmill exercise during infusion may reduce the side effects experienced with adenosine.[5]

Common side effects may be experienced by 75% of patients stressed with dobutamine.[5] Severe side effects from dobutamine may require intravenous administration of a short-acting β-blocker such as esmolol,[5] although stopping administration may be sufficient.[13]

Fatal myocardial infarction or cardiac death may occur in 0.01% of all patients stressed using exercise. Nonfatal myocardial infarction or major cardiac complications may occur in 0.02% of patients stressed by exercise, 0.3% of patients stressed with dobutamine, and 0.01% of patients undergoing myocardial perfusion stress with adenosine.[2]

TABLE 6. Commonly Reported Side Effects During Pharmacological Stress Testing[2]

Side Effects	Adenosine	Dobutamine
	(% of patients affected)	
Chest pain (nonspecific and not indicative of CAD[5])	35	31
≥1 mm ST depression (indicates significant CAD)	15-20	33
Dyspnea	35	14
Flushing	37	14
Headache	14	14
Palpitations	1	29
Dizziness	9	–
Hypotension (symptomatic)	2-5	15
High-degree atrioventricular block	5-7	0
Supraventricular tachycardia/ventricular arrhythmias	3	4
Bronchospasm	0.1	0

PROGNOSTIC MARKERS

Exercise stress testing provides additional prognostic information that is unavailable through pharmaceutical stress testing.[1]

BLOOD PRESSURE

In healthy individuals, the SBP increases linearly with increasing workloads (approximately 40–80 mm Hg over the test), whereas the DBP remains at baseline or even decreases. Exercise-induced hypotension is a drop in SBP of 20 mm Hg or more. It suggests a poor prognosis or severe coronary disease[16] and has a positive predictive value of 50% for left main or triple vessel disease. Other causes of exercise-induced hypotension include valvular heart disease, cardiomyopathy, dehydration, antihypertensive therapy, or prolonged strenuous exercise.[8]

HEART RATE

Reaching 85% of the maximum predicted heart rate is a good prognostic indicator. If 85% of the maximum predicted heart rate is achieved along with a physiological blood pressure response and no ST depression, the patient has a low probability of CAD. Conversely a high probability of CAD is likely with ST depression at low work rates, a drop in blood pressure, and typical angina.[10]

RATE PRESSURE PRODUCT (HR x SBP)

This product, obtained by multiplying the SBP by the heart rate (HR), is an estimation of the myocardial oxygen uptake and can indicate the severity of the exercise stress.[9] The amount of myocardium in jeopardy is inversely related to the double product at the onset of ischemia.[8] Patients who achieve a product greater than 25,000 have a better prognosis.

METABOLIC EQUIVALENT (MET)

MET is the equivalent of the resting metabolic oxygen requirement. One metabolic equivalent equals 3.5 mL/kg per minute.[11] Knowing the MET allows assessment of the workload.[10] Table 7 shows the correlation of MET with several exercise protocols. Table 8 shows clinically significant MET levels.

Five METs are required to carry out the activities of daily living.[10] Failure to achieve 5 METs during treadmill exercise is associated with a worse prognosis[6] in patients under 65 years of age[8]; there is a 16-fold increase in cardiac deaths if a patient cannot achieve a 4-MET workload.[1] Reaching 13 METS indicates a good prognosis.

ELECTROCARDIOGRAPHY

ECG provides essential data during myocardial perfusion stress. This modality helps identify contraindications, monitor the patient, and identify indications for early termination. For the purposes of this article, one small vertical square (1mm in height) = 0.1 mV, and one small horizontal square (1mm in width) = 1 small square. Normal ECG changes during exercise are shown in Table 9. Findings suggesting CAD are shown in Table 10. For easier visualization of selected segments of the ECG, please refer to Figure 1, which shows the J point, ST segment, and TP segment.

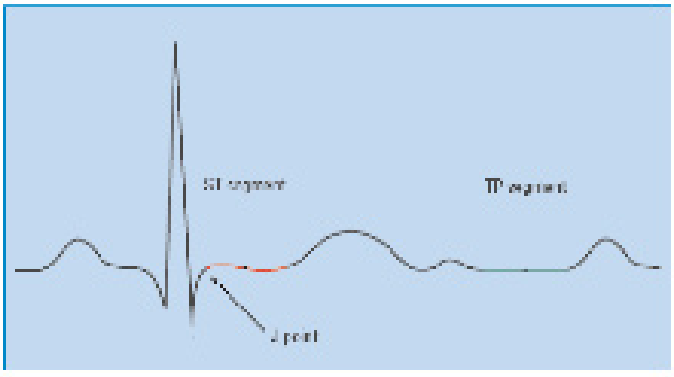
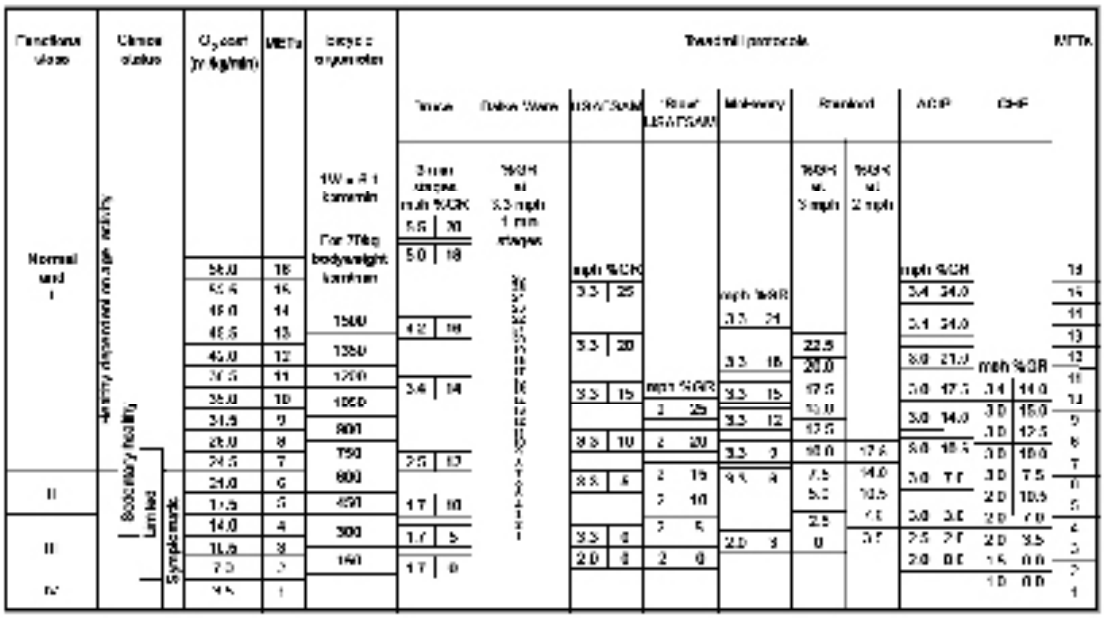


FIGURE 1. ECG—J point, ST segment, and TP segment. (Reproduced with permission from Meek S, Morris F. ABC of clinical electrocardiography Introduction II—basic terminology. BMJ. 2002;324:472.)

TABLE 7. Oxygen Cost per Stage of Some Commonly Used Protocols



%GR = percentage gradient; ACIP = asymptomatic cardiac ischemia plot; CHF = congestive heart failure (modified Naughton); kpm/min = kilopond meters per minute; METs = metabolic equivalents; mph = miles/h; USAFSAM = United States Air Force School of Aerospace Medicine.

TABLE 8. Key Metabolic Equivalents for Maximum Exercise

1 MET	Resting
2 METs	Level walking at 2 mph
4 METs	Level walking at 4 mph
<5 METs	Poor prognosis; usual limit immediately after myocardial infarction; peak cost of basic activities of daily living
10 METs	Prognosis with medical therapy as good as coronary artery bypass surgery
13 METs	Excellent prognosis regardless of other exercise responses
18 METs	Elite endurance athletes
20 METs	World class athletes

*TABLE 9. Normal ECG Changes During Exercise**

P wave increases in height (normally upright in leads I and II and < 0.25 mV or 2.5 small squares)
R wave decreases in height
J point (the point of inflection at the junction of the S wave and ST segment becomes depressed during exercise, with maximum depression at peak exercise)
ST segment becomes sharply upsloping (lies between the J point and the beginning of the T wave, upsloping due to a depressed J point)
Q–T interval shortens (measured from the beginning of the QRS complex to the end of the T wave)
T wave decreases in height (T wave orientation usually corresponds to the preceding QRS complex)

*Data adapted from Hill J, Timmis A. ABC of clinical electrocardiography: exercise tolerance testing. *BMJ*. 2002:324;1084-7; Meek S, Morris F. ABC of clinical electrocardiography introduction. I—leads, rate, rhythm, and cardiac axis. *BMJ*. 2002;324:415-8; Meek S, Morris F. ABC of clinical electrocardiography Introduction. II—basic terminology. *BMJ*. 2002;324:470-3.

TABLE 10. ECG Findings Suggesting a High Probability of CAD[10-18]

Horizontal ST-segment depression of ≤ 2 mm (normally should be level with the subsequent TP segment), measured 0.6–0.8 s after the J point. If the baseline ECG shows ST-segment depression, an additional 2.0-mm ST-segment depression may indicate ischemia.[16] Resting ST depression in itself is a negative prognostic marker.[5] If the patient has a left bundle branch block or Wolfe-Parkinson-White syndrome, ST-segment changes are not significantly related to ischemia.[5,16] When there is a coexisting right bundle branch block, only changes occurring in lead V5 and V6 are associated with ischemia.[16]
Downsloping ST-segment depression
Early positive response within 6 minutes
Persistence of ST depression for more than 6 minutes into recovery
ST-segment depression in five or more leads

CONCLUSION

Nuclear medicine studies of myocardial perfusion provide heathcare professionals with a range of useful information. Stressing the patient adds to the utility of the study when used in an appropriate setting; this process allows for assessment of the coronary perfusion reserve. Exercise stress provides the opportunity for gathering additional prognostic information. Adenosine testing has the most rapid protocol. Dobutamine allows for the evaluation of patients who cannot be otherwise stressed. Identifying the endpoint of the stress is important, not only for the safety of the patient, but for optimal administration of the radiotracer. Knowledge used in identifying the endpoints can help gather further prognostic and diagnostic information. A wealth of information is available before image acquisition begins.

REFERENCES

1. Travain MI, Wexler JP. Pharmacological stress testing. *Semin Nucl Med.* 1999;29:298-318.
2. Anagnostopoulos C, Harbinson M, Kelion A, et al. Procedure guidelines for radionuclide myocardial perfusion imaging. *Heart*. 2004;90(suppl I):i1-i10.
3. Strauss HW, Miller DD, Wittry MD, et al. Society of Nuclear Medicine procedure guideline for myocardial perfusion imaging 3.0. *Society of Nuclear Medicine Procedure Guideline Manual*, 2002. Reston, Va: Society of Nuclear Medicine; 2002.
4. Machac J. Cardiac positron emission tomography imaging. *Semin Nucl Med*. 2005;35:17-36.
5. American Society of Nuclear Cardiology. Updated imaging guidelines for nuclear cardiology procedures, part 1[comment]. *J Nucl Cardiol*. 2001;8:G5-G58.
6. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). *J Am Coll Cardiol*. 2002;40:1531-40.
7. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients with Unstable Angina). J *Am Coll Cardiol*. 2000;36:970-1062.
8. Fletcher GF, Balady G, Froelicher VF, et al. Exercise standards: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1995;91:580-615.
9. Sandler MP, Coleman RE, Patton JA, et al. *Diagnostic Nuclear Medicine*. 4th ed. Philadelphia, Pa: Lippincott, Williams & Wilkins; 2003.
10. Hill J, Timmis A. ABC of clinical electrocardiography: exercise tolerance testing. *BMJ*. 2002:324;1084-7.
11. American Thoracic Society; American College of Chest Physicians. ATS/ACCP statement on cardiopulmonary exercise testing. *Am J Respir Crit Care Med*. 2003;167:211-277.
12. Mettler FA, Guiberteau MJ. *Essentials of Nuclear Medicine Imaging*. 4th ed. Philadelphia, Pa: WB Saunders;1998.
13. Crawford ES, Husain SS. Nuclear cardiac imaging terminology and technical aspects. Reston, Va: *Society of Nuclear Medicine*; 2003.
14. Schinkel AF, Elhendy A, Biagini E, et al. Prognostic stratification using dobutamine stress ^{99m}Tc-tetrofosmin myocardial perfusion SPECT in elderly patients unable to perform exercise testing. *J Nucl Med.* 2005;46:12-8.

15. Bartunek J, Wijns W, Heyndrickx GR, de Bruyne B. Effects of dobutamine on coronary stenosis physiology and morphology:comparison with intracoronary adenosine. *Circulation*. 1999;100:243-9.
16. Lear SA, Brozic A, Myers JN, Ignaszewski A. Exercise stress testing. An overview of current guidelines. *Sports Med*. 1999;27:285-312.
17. Meek S, Morris F. ABC of clinical electrocardiography introduction. I—leads, rate, rhythm, and cardiac axis. *BMJ*. 2002;324:415-8.
18. Meek S, Morris F. ABC of clinical electrocardiography introduction II—basic terminology. *BMJ*. 2002;324:470-3.

MYOCARDIAL PERFUSION STRESS TESTING IN NUCLEAR MEDICINE POST TEST

Expires: August 15, 2011 Approved for 1 ARRT Category A Credit

1. The sensitivity of SPECT for multivessel coronary disease varies from
a. 50%–55%.
b. 72%–80%.
c. 90%–94%.
d. 96%–100%.

2. Which of the following are positron emitters that are used in MPI?
a. Thallium 201 and technetium 99m
b. Strontium 89 and indium 111
c. Fibrinogen I 125 and sodium iodide I 125
d. Nitrogen 13 and rubidium 82

3. Quantitative evaluation of coronary blood flow in MPI is possible using which radionuclide?
a. technetium-99m
b. thallium-201
c. fluorine-18
d. rubidium-82

4. Assuming there are no contraindications, which of the following modalities is preferred for myocardial perfusion stressing?
a. Adenosine
b. Exercise
c. Dobutamine
d. Dipyridamole

5. Adenosine might be used for myocardial perfusion stressing when a patient
a. is unable to exercise optimally.
b. is very athletic and therefore has a low resting heart rate.
c. has contraindications to pharmacologic vasodilator stressors.
d. has pulmonary hypertension.

6. Exercise stress leads to perfusion abnormalities in the septum and adjacent wall in the absence of obstructive coronary disease in patients with
a. unstable angina.
b. chronic CAD.
c. left bundle branch block.
d. a history of myocardial infarction.

7. All of the following are contraindications for using dobutamine in myocardial perfusion stressing EXCEPT for patients who have
a. had a recent (< 1 week) myocardial infarction.
b. taken xanthine derivatives in the last 12 hours.
c. uncontrolled hypertension.
d. known hypokalemia.

8. Which of the following agents is contraindicated in asthmatic patients who have ongoing wheezing or require methylxanthine medication for control?
a. Treadmill exercise
b. Adenosine
c. Dobutamine
d. Stationary bicycle exercise

9. High-risk angina is associated with a
a. high short-term risk of death or myocardial infarction.
b. low short-term risk of death or myocardial infarction, but a moderate long-term risk.
c. history of angina in a patient who tends to be active, exercise, and only has ECG changes late in a stress test.
d. history of chest pain on exertion that abates when the patient is at rest but has a normal ECG.

10. In patients with unstable angina, all of the following are characteristic of high-risk angina EXCEPT
a. pulmonary edema.
b. new or worsening mitral regurgitation murmur.
c. hypertension.
d. tachycardia.

11. Rhythmic exercise stressing gradually increases myocardial oxygen consumption, which leads to a _____ fold increase of blood flow in normal coronary arteries.
a. 1.5- to 2.0-
b. 2.0- to 2.5-
c. 2.5- to 3.0-
d. 3.0- to 3.5-

12. One formula for calculating a target heart rate that may be used to determine when an adequate workload has been reached is
a. 2 x patient's resting heart rate – the patient's age.
b. 3 x patient's resting heart rate + the patient's age.
c. 0.85 x [(220 for men or 210 for women) – the patient's age].
d. (220 for men or 210 for women) – the patient's age.

13. What action should be taken if a patient undergoing an exercise stress test begins to appear cyanotic?
a. The patient should be given oxygen and encouraged to continue the examination.
b. Exercise should continue for no longer than 5 more minutes.
c. The examination must be terminated.
d. The patient should be asked if he or she wishes to continue with the study.

14. What must be done to allow the radiotracer to be cleared from the blood pool while maintaining the myocardial perfusion achieved during maximal stress?
a. When the endpoint is reached, the patient should rest for 2 minutes before the injection of the radiotracer.
b. When the endpoint is reached, the patient should lie down and then immediately be injected with the radiotracer.
c. The patient must be injected with the radiotracer well before the endpoint is reached.
d. This varies depending on the stress modality and the type of tracer used.

15. Pharmaceutical stress increases blood flow in normal coronary arteries
a. 2- to 3-fold.
b. 3- to 5-fold.
c. 4- to 6-fold.
d. 7- to 10-fold.

16. When using adenosine for stressing, when should the radiotracer be administered?
a. Three minutes before the end of the adenosine infusion
b. At the completion of the adenosine infusion
c. Three minutes after the adenosine infusion is completed
d. As soon as the target heart rate is achieved.

17. What may help reduce the side effects associated with adenosine?
a. Use of atropine
b. Rapid infusion
c. Injection while patient recumbent
d. Low-level upright treadmill exercise during infusion

18. What is the most common side effect associated with adenosine during pharmacologic stress?
a. Ventricular arrhythmias
b. Symptomatic hypotension
c. Flushing
d. Headache

19. Exercise-induced hypotension is defined as a drop in systolic blood pressure of
a. 5 mm Hg or more.
b. 20 mm Hg or more.
c. 40 mm Hg or more
d. 50 mm Hg or more.

20. The rate pressure product is
a. the equivalent of the resting metabolic oxygen requirement.
b. calculated as 85% of the maximum predicted heart rate.
c. an estimation of the myocardial oxygen uptake.
d. the ventilatory response to exercise.

Enterprises for Continuing Education, Inc.
10381 Citation Dr, Ste 200
Brighton, MI 48116
Phone: 810-229-3354 Fax: 810-229-3235
E-mail: info@cewebsource.com

CEwebsource.com ANSWER KEY
MYOCARDIAL PERFUSION STRESS TESTING IN NUCLEAR MEDICINE
EXPIRES AUGUST 15, 2011

CEwebsource.com ANSWER SHEET

Approved by the AHRA for 1 Category A CE Credit
Please Note: *Approved for ARRT and NMTCB Direct Credit*

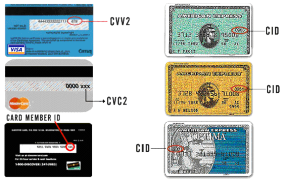
- Circle the letter corresponding to the correct answer for each question.
- Mail or fax this completed answer sheet along with payment to the address or fax number on the top of this page. If faxing, credit card information must be included.
- You must receive a score of 75% or better to receive credit in any section. Allow up to 4 weeks to process. A Record of Continuing Education will be sent to you.
- Include payment. Answer keys must be accompanied by a $10 processing fee.
- In a hurry? RUSH SERVICE is available for an additional $10 for CREDIT CARD ORDERS. Fax this answer key along with your credit card information to (810) 229-3235 for a 48-hour (M-F) turn-around! Whether faxing one answer key or several, only one $10 charge is added to the total of your order when faxing multiple sheets at once!

❑ **YES**, in addition to the standard processing fee of $10, please charge my credit card account **$10 EXTRA for RUSH SERVICE**. FAX my expedited record of Continuing Education to me at: (_____) _____-_____.
(If a fax number is not provided, a copy will be sent to the address indicated below within 48 hours)

Method of Payment: (checks payable to **ECEI**)
❑ Check ❑ Money Order ❑ Visa ❑ MasterCard ❑ Discover ❑ AmEx

☐☐☐☐ ☐☐☐☐ ☐☐☐☐ ☐☐☐☐ V-Code _____
Account Number

Expires _____ / _____



Identification Section (Please **print legibly** in blue or black ink)

Name _____ Email _____

Address _____ Birth Month _____

_____ City _____ State _____ Zip _____

Daytime Phone: (__ __ __) __ __ __ – __ __ __ __

California Nuc Med Techs: RHN __ __ __ __

Please check ONE:
❑ MAIL my Record of Continuing Education
❑ E-MAIL my Record of Continuing Education

Article Title: Myocardial Perfusion Stress Testing in Nuclear Medicine

Office Use: NM65

1. a b c d	6. a b c d	11. a b c d	16. a b c d
2. a b c d	7. a b c d	12. a b c d	17. a b c d
3. a b c d	8. a b c d	13. a b c d	18. a b c d
4. a b c d	9. a b c d	14. a b c d	19. a b c d
5. a b c d	10. a b c d	15. a b c d	20. a b c d