Chest Pain
Donald L. Renfrew, MD

This chapter covers three main points designed to help you order the correct first test when evaluating patients with chest pain:

1. Virtually everyone with chest pain gets a chest x-ray.
2. Patients with suspected pulmonary embolism need emergent chest CT with contrast.
3. Imaging in suspected coronary artery syndrome depends on the clinical condition of the patient and risk assessment.

IMAGING OPTIONS IN CHEST PAIN

Primary care practitioners (PCPs) may order any of several exams in the evaluation of chest pain, including plain films, CT scans, nuclear medicine scans, magnetic resonance imaging scans, and ultrasound studies.

Plain films

See page 133 for a description of plain films of the chest. In addition to the chest x-ray (CXR), the PCP may also order rib detail films, which provide greater detail of ribs and demonstrate fractures that may be missed on plain films (Figure 1).

Computed Tomography

See page 131 for a description of computed tomography (CT) performed for cough and dyspnea. Typically, a standard, contrast-enhanced exam is obtained for these indications. As an alternative to the standard contrast-enhanced exam, CT may be timed to optimize enhancement of the pulmonary arterial tree. This is called a “chest computed tomographic angiogram” (Chest CTA), and the data from this technique is processed to create specific views of the pulmonary arterial tree, usually in an oblique plane optimized for visualization of the main pulmonary arteries with maximum intensity projections (MIPs) (Figure 2). CT may also be used to measure the coronary artery calcium content, called “coronary artery calcification scoring” (CACS). For this study, the patient is hooked up to an EKG and a noncontrast CT scan is obtained with the data acquisition coordinated with the heart beat to minimize motion. Finally, more advanced equipment (at least 64-slice, by current recommendations), can acquire a “coronary artery computed tomographic angiogram” (CCTA), which is a map of the coronary arterial tree obtained with EKG gating and intravenous contrast (Figure 3). For 64-slice scanners, the heart rate must be below 70; many patients will require beta-blockers to achieve this low rate. CT scanners with more slices and faster imaging times can obtain diagnostic images with higher heart rates. Note that, at present, a chest CTA and a CCTA (of the coronary arteries) cannot be performed simultaneously with the same bolus of contrast material, although advances in scanning.

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technology will probably soon allow the “rule-out triple” exam whereby a single study can evaluate for pulmonary embolism, aortic arch dissection, and coronary arterial disease. These techniques will be discussed in context below.

**Nuclear medicine studies**

Primary care providers may order ventilation-perfusion lung scans for patients suspected to have a pulmonary embolism who are allergic to contrast or have renal insufficiency. This study relies on the distribution of two different radioactively labeled substances: one is an aerosol used to evaluate ventilation, and the other an intravenous substance designed to be filtered at the smallest level of pulmonary vasculature and therefore to evaluate perfusion (Figure 4).

Nuclear medicine heart studies include infarct-avid imaging (now largely supplanted by serial enzyme evaluation) and myocardial perfusion studies, which may be performed at the time of the supposed cardiac event in equivocal cases, or, more typically, with a stress test in patients who have clinical features suspicious for coronary artery syndrome. For myocardial perfusion studies, a radioactively labeled material (typically tetrofosmin labeled with 99m-Tecnitium) is injected intravenously when the patient is at rest, and images of the heart are obtained. Later in the same day, or on a different day, additional radioactively labeled material is injected during cardiac loading (caused either by exercise or drugs). Images are obtained in both cases. Either planar or single photon emission computed tomographic (SPECT) images may be obtained; the latter are preferred as the test has higher sensitivity and the same specificity. Normal myocardium shows uniform uptake of radiotracer, infarcted areas show decreased uptake on both the rest and stress studies, whereas areas of reversible ischemia show normal activity at rest but decreased activity following exercise (see below).
Figure 2. Normal chest CT angiogram in a 28 year old woman with acute chest pain.  

A. Axial contrast-enhanced CT study at the level of the main pulmonary artery (arrow) shows a normal appearance of the pulmonary artery. Note dense contrast coming into the heart through the superior vena cava.  

B. Axial contrast-enhanced CT at a slightly lower level shows that the main pulmonary artery (arrow) is approximately the same size as the ascending aorta.  

C. Coronal oblique reformatted contrast-enhanced CT study shows the left pulmonary artery (arrow) and branches to be free of filling defects.  

D. Coronal oblique reformatted contrast-enhanced CT study shows the right pulmonary artery (arrow) and branches to be free of filling defects as well.
Figure 3. Computed tomographic coronary arteriography in a 52 year old with chest pain. This composite figure demonstrates a reconstructed view of the heart and coronary vessels, obtained following EKG-gated computed tomography. The left anterior descending (LAD) coronary artery is depicted along the surface of the left ventricle, with the long arrow connected to the associated cross-sectional image of the LAD. Images proximal to the level of the arrow are arrayed above the target location, and more distal locations below it. Along the right side of the image are two longitudinal reconstructions (at right angles) of the LAD. Note that in the right-sided image, there is a significant (>50%) stenosis of the proximal coronary artery (double arrow). Case courtesy of Dr. Marc Miller, Radiology Associates of the Fox Valley.
Magnetic resonance imaging

Despite years of intensive development and investigation, chest MRI remains a relatively infrequently performed method of cardiac imaging, with most uses restricted to patients who are allergic to iodine-containing IV contrast. However, this technique continues to evolve, and it is possible that cardiac MR may one day provide a single modality capable of evaluating the coronary arteries, cardiac valves, and myocardium in one exam. At this time, however, the technique is not routinely used in the evaluation of chest pain.

Echocardiography

Cardiologists interpret most echocardiograms. This examination is excellent for evaluation of the cardiac valves (including morphology, stenosis, and insufficiency), pericardium, chamber size, and wall motion, as well as obtaining some information about heart and great vessel pressures. It is the examination of choice for evaluating suspected acute valve insufficiency, for example, in a patient with chest pain and a new murmur. Since echocardiography is largely performed and controlled by cardiologists, it is not covered in this chapter.

CXR IN ALL PATIENTS WITH CHEST PAIN

Imaging of chest pain starts with a CXR. In most cases, a plain film does not reveal anything particularly helpful or diagnostic: Templeton et al\(^2\) found that 23% of plain films obtained in patients with chest pain in the emergency room had an abnormality that influenced therapy, and the likelihood of finding a significant abnormality in a patient undergoing evaluation in an outpatient clinic is likely even less. Perusal of Table 1, a list of causes of chest pain in 300 patients presenting to a clinic with chest pain reveals why most plain films are unremarkable: the commonly encountered causes of chest pain seldom have any plain film manifestations. Nonetheless, there are a few relatively uncommon diseases that may cause chest pain and which have specific abnormalities on chest radiography.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percent</th>
</tr>
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<tbody>
<tr>
<td>Musculoskeletal, including costochondritis</td>
<td>36%</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>19%</td>
</tr>
<tr>
<td>Cardiac</td>
<td>10.5%</td>
</tr>
<tr>
<td>- stable angina</td>
<td>16%</td>
</tr>
<tr>
<td>- unstable angina or MI</td>
<td>1.5%</td>
</tr>
<tr>
<td>- other cardiac</td>
<td>3.8%</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>8%</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>5%</td>
</tr>
<tr>
<td>Other</td>
<td>16%</td>
</tr>
</tbody>
</table>


Pneumothorax, typically from a ruptured bleb\(^3\), is one disease that plain films can diagnose without equivocation (Figure 5), although CT is better at demonstrating a small pneumothorax (Figure 1, Chapter 10, page 132). Patients with a pneumothorax will typically experience dyspnea in addition to chest pain, and are, of course, far more likely to present to an emergency room than an outpatient clinic. Pneumomediastinum (Figure 6) occurring with or without pneumothorax, and is an additional cause of chest pain which may be diagnosed on a CXR although, like a pneumothorax, the lesion is more easily seen on CT.

Rib fractures (Figure 1) may cause chest pain and while there is sometimes a history of trauma, such fractures may also occur.
Figure 4. Normal perfusion study in a 70 year old woman with chest pain. The patient had an allergy to intravenous contrast material. The perfusion study shows normal perfusion to both lobes of the lungs on all projections. The ventilation study (not shown) was also normal.

Figure 5. Pneumothorax in a 52 year old man with acute chest pain. A. CXR obtained in the ER shows a right sided pneumothorax (arrow). B. CXR following insertion of a chest tube shows re-expansion of the right lung, with the tube tip (arrow) in the superolateral thorax at about the 4th rib level.
secondary to prolonged or violent coughing, or sneezing. Note that, for the most part, rib detail films are more sensitive for the detection of rib fractures than is the routine CXR, but many rib fractures may escape detection because they are minimally displaced or occur at the costochondral junction. If a patient has focal reproducible tenderness of a rib, he probably has a rib fracture, and the important thing to exclude is any associated hemo-
Figure 7. Metastatic breast cancer in a 70 year old woman with chest pain. A. PA CXR shows superior retraction of the left breast shadow (arrow). The patient had *not* had a mastectomy or any left breast procedure: the breast was retracted because of a tumor. B. Lateral CXR shows subtle patchy sclerotic change of the vertebral bodies. C. Coronal reformatted CT study demonstrates diffuse metastatic disease of the spine. D. Sagittal reformatted CT demonstrates diffuse metastatic disease of the thoracic spine and sternum.

pneumothorax, which may be done with the CXR. Another cause of chest pain which may be seen on a CXR (but which is better seen on a CT exam) is metastatic deposit to the skeleton (Figure 7). Other infrequently encountered causes of chest pain include painful arteriovenous malformation (Figure 9, Chapter 10, page 141) painful pneumonia (Figure 8), aortic dissection, esophageal rupture, and pulmonary hemorrhage (Figure 9). Note that many times, these diseases may have abnormalities on chest radiographs which are not specific and therefore require further evaluation, typically with CT. Even CT will usually provide a specific diagnosis only when correlated with all relevant clinical data.
Figure 8. Pneumonia causing chest pain in a 47 year old man.  A.  PA CXR shows opacity in the mid left lung (arrow).  B. Lateral CXR shows the opacity along the posterior chest wall (arrows).  This is a so-called “round” pneumonia which mimics a mass.  C. Axial CT shows consolidation in the left lower lobe (arrow).  D. Sagittal reformatted CT shows consolidation along the posterior chest corresponding to the opacity seen on the plain film.  Follow-up CXR (not shown) following treatment demonstrated clearing of the pneumonia.
Figure 9. Pulmonary hemorrhage in a 75 year old man with chest pain. A. CXR taken three months before the onset of chest pain is normal. B. CXR done after onset of chest pain shows diffuse bilateral lung opacity. C. Axial CT shows extensive ground glass opacity (arrows). D. Coronal reformatted CT demonstrates ground glass opacity as well and demonstrates why the CXR had the appearance it did. Note that while the CXR and CT show striking abnormality, the findings are not specific and only correlation with the additional clinical features of anticoagulation and a sudden drop in hematocrit allowed the correct diagnosis.
SUSPECTED PULMONARY EMBOLISM

Pulmonary emboli frequently cause chest pain and remain a common cause of mortality, yet emboli often go undiagnosed. This follows from the fact that while the classic patient presents with an acute onset of severe chest pain and dyspnea, many patients have more subtle presentations. One approach calls for scoring on the basis of what are known as the Wells criteria (see Table 2) and performing chest CTA if the likelihood of pulmonary embolism is high but obtaining a D-dimer if the likelihood of pulmonary embolism is low, in which case a chest CTA should still be performed if the D-dimer is elevated.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Points</th>
</tr>
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<tbody>
<tr>
<td>Clinical signs and symptoms of leg DVT</td>
<td>3.0</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>3.0</td>
</tr>
<tr>
<td>Heart rate &gt; 100 BPM</td>
<td>1.5</td>
</tr>
<tr>
<td>Immobilization of more than three days or recent surgery</td>
<td>1.5</td>
</tr>
<tr>
<td>Previous PE or DVT</td>
<td>1.5</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>1.0</td>
</tr>
<tr>
<td>Malignancy</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Table 2. Clinical Decision Rule for DVT. Likelihood of pulmonary embolism is considered high if the point total is more than 4. From: Wells PS et al. Derivation of a simple clinical model to categorize patients’ probability of pulmonary embolism: increasing the model’s utility with the SimpliRED D-dimer. Thromb Haemost. 2000; 83:416-420.

Regarding imaging, patients suspected to have a pulmonary embolism (like all patients with chest pain) will have a CXR. The CXR is therefore always obtained, and it is frequently abnormal, but, unfortunately, seldom helpful. Stein et al found that while 84% of CXRs in patients with pulmonary embolism showed a variety of abnormalities, patients without pulmonary embolism had similar abnormalities at about the same rate. For example, 69% of patients with PE had pulmonary parenchymal opacity versus 58% without PE; 47% of patients with PE had a pleural effusion versus 39% without. Of course, a wide variety of diseases, many of which cause chest pain, may result in nonspecific CXR abnormalities such as pulmonary opacity and pleural effusion, making the CXR unhelpful in rendering the specific diagnosis of pulmonary embolism. As noted above, CXR is helpful in excluding certain other causes.

The mainstay of imaging diagnosis for suspected pulmonary embolism is now chest CTA (Chapter 10 Figure 2, page 134 and Figure 7, page 138). While pulmonary angiography was the reference standard in the diagnosis for years, CT is far more readily available, faster, cheaper, and less prone to iatrogenic mishap than is pulmonary angiography. CT allows quantification of the pulmonary emboli size, demonstrates their location, and diagnoses associated pulmonary infarction. In addition, CT allows an assessment of how severe the disease is: relative increases in right heart size (with a right ventricle: left ventricle ratio of more than one) or distention of the pulmonary artery (to greater than 30 mm) indicate severe disease with a worse prognosis (Figure 10). These findings are associated with right heart failure, which is typically the cause of death in patients with large pulmonary emboli, and may indicate the need for emergent embolectomy. Finally, as when CT scanning finds alternative causes of flank pain in patients suspected of renal stone disease, CT scanning for chest pain may find such alternative diagnoses as pneumonia, cardiovascular disease, pulmonary fibrosis, and malignancy in patients with suspected pulmonary embolism.
For those who cannot undergo CT of the chest for evaluation of pulmonary embolism because of contrast allergy or renal failure, nuclear medicine ventilation-perfusion imaging (long a mainstay in the diagnosis of pulmonary embolism) is probably the best alternative. A normal perfusion study (Figure 4) essentially excludes pulmonary embolism, whereas gross mismatches of ventilation and perfusion (Figure 11) are diagnostic of pulmonary embolism. Unfortunately, many scans are indeterminate.

Figure 10. Pulmonary embolism in a 61 year old man with acute shortness of breath following recent abdominal surgery. A. Axial contrast-enhanced CT months before the patient’s PE shows a normal sized main pulmonary artery (arrow). B. Axial contrast-enhanced CT shows a filling defect in the right pulmonary artery (pulmonary embolism) (black arrow) along with a dilated main pulmonary artery (white arrow). C. Axial contrast-enhanced CT at the level of the right ventricle months before the patient’s PE shows a normal sized right ventricle (between arrows) D. Axial contrast-enhanced CT following the patient’s pulmonary embolism shows a dilated right ventricle. An enlarged right ventricle is a poor prognostic sign in patients with pulmonary embolism.
Figure 11. Pulmonary embolism in an 86 year old man with acute onset of shortness of breath and renal failure. 
A. Anterior perfusion study shows a focal area of decreased blood flow to the right upper lobe (arrow). B. Matched anterior ventilation study shows normal ventilation in the same region. C. Posterior perfusion study shows a focal area of diminished perfusion in the left lower lobe (arrows). D. Matched posterior ventilation study shows normal perfusion in the same region. Multiple mismatched ventilation-perfusion defects indicate a high probability of pulmonary embolism.

Another alternative to CT scanning is to perform bilateral lower extremity deep venous ultrasound examination (see page 179 for a discussion of this study), with the notion that since nearly all pulmonary emboli originate from the lower extremities, and since it is unlikely that all of the clot will break off to embolise to the lungs at one time, residual clot is likely to be found in the lower extremities. However, Turkstra et al\(^9\) found a 3% false-
positive rate of diagnosis of DVT and a 70% false-negative rate. Note that technical aspects of lower extremity venous ultrasonography have evolved considerably since the publication of Turkstra et al (in 1997), however, and most departments would probably do better than these figures indicate. Subsequent studies by other authors have found much lower false negative rates\textsuperscript{10, 11}.

**SUSPECTED CORONARY ARTERY SYNDROME**

With coronary artery syndrome, the critical issues are:

1) Is a cardiac event (infarction) presently occurring?

2) Is the patient’s chest pain secondary to coronary artery narrowing?

3) How likely is the patient to have a cardiac event in the future?

**Acute myocardial infarction from ruptured/hemorrhagic plaque**

Imaging of patients with suspected coronary artery syndrome, like imaging any patient with chest pain, starts with obtaining a chest radiograph. Even before the patient has the radiograph done, however, it is necessary to evaluate whether the patient is actively infarcting or not. Most patients with an active infarct will present to the emergency room rather than a clinic, and many of these patients are severely ill with, for example, hypotension or tachycardia. If you suspect acute myocardial infarction, the best course of action is to start an IV and draw blood for cardiac enzymes, obtain an immediate 12-lead EKG, have the patient chew a 325 mg aspirin, and arrange immediate transport to an emergency room, preferably in an ambulance equipped with a defibrillator\textsuperscript{12}.

The CXR can wait until the patient is in the emergency room. The clock is running in these patients: there are only 60 to 90 minutes or so to save ischemic myocardium undergoing infarction, so it is imperative that these patients be transported to an emergency room (or straight to the cath lab) as rapidly as possible.

What is happening in most of these patients is that plaque, which may have been years accumulating, has ruptured, resulting in acute occlusion of a coronary artery\textsuperscript{13}. For these patients, while a chest radiograph will be obtained to evaluate for changes of acute congestive heart failure (Figure 12), the diagnosis will rely on EKG changes and/or cardiac enzymes, typically done sequentially until the diagnosis is secured. For those patients who proceed to the catheterization lab, imaging will be performed during catheterization, usually followed by stent placement or, if stents are not an option, emergency cardiac bypass surgery.

**Angina from coronary artery narrowing**

The drama of a patient presenting with an acute myocardial infarction is the exception, not the rule, for the patient seeing a primary care practitioner for evaluation of chest pain. Most patients with coronary artery syndrome have angina because of stenosis of the coronary arteries. To decide which study to order for evaluation of patients suspected to have chest pain on the basis of coronary artery stenosis, it is first necessary to evaluate the patient’s risk. You must calculate the pretest probability of coronary artery disease by assessing their chest pain pattern and correlating the pain pattern with their age and sex (Table 3). Low risk patients (pretest probability of $<$5\%) are unlikely to benefit from stress-EKG or perfusion imaging testing because a positive test is much more likely to represent a false positive result than to
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Figure 12. Florid congestive heart failure in 92 year old woman with chest pain from myocardial infarction. A. The baseline CXR from ten years prior to the patient's acute onset of chest pain shows a normal sized heart and normal pulmonary vessels. B. A CXR performed shortly after the onset of acute chest pain shows cardiomegaly, bilateral pleural effusions, and prominent pulmonary vasculature from congestive heart failure. The patient had acute abnormality on her ECG and abnormal cardiac enzymes.

represent true disease, and high risk patients (pretest probability >90%) are unlikely to benefit because a negative result is likely to be a false negative and these high risk patients should probably proceed to catheterization (or at least evaluation by a cardiologist) anyway. The intermediate risk patients (pretest probability of between 25% and 75%; note that recommendations are less clear for patients with a pretest probability of heart disease between 5% and 25%, and for those with risk between 75% and 90%) should proceed to stress-EKG testing, or stress-EKG testing combined with myocardial perfusion imaging or echocardiography. The imaging component of the examination is typically added when there are baseline ECG abnormalities making interpretation of stress-induced changes problematic, when the patient is on digitalis, when there has been previous revascularization, or when a stress-EKG done without imaging produces equivocal results. Note also that addition of imaging improves sensitivity in intermediate risk patients by about 20%. SPECT myocardial perfusion images are interpreted by comparing the rest and stress images, usually with the assistance of a computer which will make a map of abnormally perfused areas (Figure 13). These abnormally perfused areas may be either fixed, indicating chronic ischemic change and scarring, or reversible, indicating myocardium that is at some risk for infarction. Either a positive EKG, or changes on a myocardial perfusion study, similar to a high pretest probability of coronary artery stenosis, usually indicates the need for coronary angiography or at least consultation with a cardiologist. With regard to imaging of the coronary arterial tree, CACS and CCTA are presently undergoing rapid evolution, and the inclusion of these tests in the algorithm for the work-up of suspected coronary artery syndrome depends on the availability of appropriate equipment, expertise, and
physician acceptance. For patients with nonanginal chest pain, which is unlikely to represent coronary artery stenosis, coronary artery calcification scoring (CACS) offers an alternative to the stress test, or an additional test if the stress test is negative or equivocal. For the CACS to be helpful in this regard, it should be zero. In explanation: the score is based on the amount of calcium in the coronary arteries, and a lower score (less calcium) is better, with a score of 0 being ideal. Georgiou et al found that if the CACS is zero, the annual cardiac event rate was 0.6% per year over the next five years, and McLaughlin et al found only one cardiac event (in a cocaine user) within one month among 48 chest pain patients with a CACS of zero, whereas the 30 day event rate was 8% in those with a CACS of greater than zero. On the basis of their results, McLaughlin et al felt that CACS excluded patients with nonanginal chest pain from further costly evaluation. If the CACS is not zero, or if direct anatomic visualization of the coronary arterial tree is desired, CCTA may be performed. The ordering, interpreting, and control of this modality continues to be controversial, in part because of the turf war it provokes between cardiologists (who have traditionally controlled most cardiac imaging modalities such as echocardiography, cardiac catheter angiography, and nuclear medicine myocardial perfusion studies) and radiologists (who have traditionally read all studies obtained on the CT scanner). From the perspective of the primary care provider, what matters is that this test is performed correctly and interpreted accurately, and at present the ability to perform the test is not widespread. If it is available, which patients should be sent for CCTA? Present recommendations (which are evolving with changes in technology) are: patients at intermediate risk for coronary artery disease (including those with equivocal stress tests), patients with known or suspected congenital or acquired coronary artery anomalies, and patients with coronary artery bypass grafts in whom it is not possible to engage the grafts during angiography.

<table>
<thead>
<tr>
<th>Pretest Probability of coronary artery disease</th>
<th>Description</th>
<th>Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (&lt;5%)</td>
<td>Asymptomatic or Women &lt; 50 and Men &lt; 40 with nonanginal pain</td>
<td>Typically none</td>
</tr>
<tr>
<td>Intermediate (25% - 75%)</td>
<td>Women &gt; 50 with atypical angina or &gt;30 and &lt; 60 with typical angina Men &gt; 60 with nonanginal pain, or &gt; 30 with atypical angina</td>
<td>Stress EKG or stress EKG with MPI</td>
</tr>
<tr>
<td>High (&gt;90%)</td>
<td>Men &gt; 50 with typical angina</td>
<td>Cardiac catheterization</td>
</tr>
</tbody>
</table>

Table 3. Risk categories and test recommendations for coronary heart disease. Typical angina = Chest pain 1) with a typical quality and duration, 2) which is provoked by exertion or emotional stress, and 3) which is relieved by rest or nitroglycerine (all three); Atypical angina = Chest pain with two of the three characteristics; Nonanginal pain = chest pain with none or one of the three characteristics. Table modified from Garber AM, Hlatky MA. Stress testing for the diagnosis of coronary artery disease. UpToDate, accessed 10/7/09.
Figure 13. Positive myocardial perfusion study in a 64 year old man with exertional chest pain. A. Select vertical long axis views from a myocardial perfusion study show a defect along the septum extending into the apex (arrows) on two sequential images obtained during stress, with a normal appearance on the corresponding resting images. B. The computer generated map shows significant difference between the stress and resting radiotracer distribution (higher numbers imply lower perfusion), compatible with reversible ischemic changes. The patient underwent cardiac catheterization, with diagnosis (and stenting) of a left anterior descending coronary artery stenosis, with subsequent relief of symptoms.

**Risk of future cardiac events**

In evaluation of patients with suspected coronary artery disease, two separate risks are important: 1) the risk that a patient with chest pain has significant coronary artery stenosis (discussed above) and 2) the risk that a given patient will suffer a cardiac event (defined as death, infarction, or surgical/percutaneous intervention) during some specified time period. In the two prior sections, I discussed patients who are suspected of having a cardiac event (whose “risk” is 100%, right now!), and patients who are suspected to have coronary artery stenosis. Patients with stenosis have a high risk of future events, and are generally under the care of a cardiologist. In these two sets of patients, there is either plaque which has ruptured, or there has been demonstration of plaque which may rupture. Absent a current infarction or known stenosis, in patients with no chest pain, what is the risk of a future event?

Cardiac risk may be calculated using the age and sex of the patient, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure, whether or not the patient is on antihypertensive medication, and serum cholesterol. This risk is generally reported in 10 year increments, with *low risk* defined as a less than 10% chance of cardiac event in the next 10 years, *intermediate risk* defined as between a 10% and 20% chance of cardiac event in the next 10 years, and *high risk* defined as a greater than 20% chance of cardiac event in the next 10 years. Note that those with diabetes and previous episodes of coronary artery disease are all at high risk.

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1 See on line calculator at http://hp2010.nhlbihin.net/atpiii/calculator.asp
What is the role of imaging in evaluating risk? Coronary artery calcification scoring (CACS) measures the calcium load within the coronary arterial tree. Such calcifications denote plaque and show a correspondence with risk for plaque rupture: the more calcification, the higher the risk of plaque rupture. As noted above, an Agatston score of zero is associated with a less than 1% rate of coronary artery stenosis and a low zero risk of a cardiac event in the next 10 years. In fact, a study of over 25,000 patients demonstrated that the CACS predicted all-cause mortality independent of and more accurately than standard coronary artery disease risk factors.

Regardless of the additional and independent value of CACS in risk evaluation, the official recommendation of the American College of Cardiology Foundation and the American Heart Association is to use the CACS in patients with intermediate risk, to determine whether to treat such patients more aggressively (which generally means adding or changing drugs to lower the serum cholesterol level). Patients with a low Agatston score (typically zero) remain at intermediate risk, whereas those with a non-zero Agatston score are upgraded to the high risk category.

While the recommendation to order CACS for intermediate risk patients is certainly reasonable for the primary care practitioner to follow, note that many imaging facilities will perform CACS on a self-referral basis. Since many insurance companies do not cover the cost of this exam, and the patient pays out of pocket, this is an unusual example of the free market at work in medicine within the U.S. It is instructive to note that this has resulted in aggressive pricing (some would argue below cost) and direct patient marketing of this exam. For this reason, patients may choose to obtain a CACS without referral and then come to a primary care practitioner with a request to explain the results.

Probably the most reasonable thing to do in this situation is to explain to the patient the AHA’s recommendation and then calculate the patient’s risk. If the patient is at an intermediate risk by the calculator, great: the test was indicated anyway and you can advise/treat the patient as noted above. If the patient is at low risk and has a score of zero, or is at high risk and has a non-0 score, then the CACS has only confirmed what the risk calculator told you anyway and no change in treatment or prognosis is forthcoming. The CACS was a waste of money and needless radiation exposure. The other situations that may arise are when the CACS conflicts with the risk calculation: either a low risk patient has a non-0 CACS, or a high risk patient has a 0 score. Such findings may be reassuring to the high risk patient or motivating to the low risk one, but at present recommendations are to not change treatment of such patients. Note that in regard to motivating patients to change behavior, O’Malley et al found that knowledge and even visual presentation of CACS information did not provide patients any additional motivation (or improve their compliance with treatment).
SUMMARY

When imaging patients with chest pain, nearly all patients will first have a plain film. For patients with acute severe shortness of breath or who have other features strongly suggesting pulmonary embolism, an emergent chest CT should be obtained; nuclear medicine ventilation-perfusion imaging may be performed if the patient has renal failure or contrast allergy. Imaging of suspected coronary artery syndrome depends on the clinical condition of the patient and risk assessment.

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