MSD MANUAL
Professional Version

Angina Pectoris

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Angina pectoris is a clinical syndrome of precordial discomfort or pressure due to transient myocardial ischemia without infarction. It is typically precipitated by exertion or psychologic stress and relieved by rest or sublingual nitroglycerin. Diagnosis is by symptoms, electrocardiography, and myocardial imaging. Treatment may include antiplatelet medications, nitrates, beta-blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, statins, and coronary angioplasty or coronary artery bypass graft surgery.

(See also Overview of Coronary Artery Disease.)

Etiology of Angina Pectoris

Angina pectoris occurs when

- Cardiac workload and resultant myocardial oxygen demand exceed the ability of coronary arteries to supply an adequate amount of oxygenated blood

Such imbalance between supply and demand can occur when the arteries are narrowed. Narrowing usually results from

- Coronary artery atherosclerosis

Narrowing of the coronary arteries can also result from

- Coronary artery spasm
- Coronary artery embolism (rare)

Acute coronary thrombosis can cause angina if obstruction is partial or transient, but it usually causes acute myocardial infarction (MI).

Because myocardial oxygen demand is determined mainly by heart rate, systolic wall tension, and contractility, narrowing of a coronary artery typically results in angina that occurs during exertion and is relieved by rest.

In addition to exertion, cardiac workload can be increased by disorders such as hypertension, aortic stenosis, aortic regurgitation, or hypertrophic cardiomyopathy. In such cases, angina can result whether atherosclerosis is present or not. These disorders can also decrease relative myocardial perfusion because myocardial mass is increased (causing decreased diastolic flow).

A decreased oxygen supply, as in severe anemia or hypoxia, can precipitate or aggravate angina.

Pathophysiology of Angina Pectoris

Angina may be

- Stable
- Unstable

In **stable angina,** the relationship between workload or demand and ischemia is usually relatively predictable.

Unstable angina is clinically worsening angina (eg, angina at rest or with increasing frequency and/or intensity of episodes).

Atherosclerotic arterial narrowing is not entirely fixed; it varies with the normal fluctuations in arterial tone that occur in all people. Thus, more people have angina in the morning, when arterial tone is relatively high. Also, abnormal endothelial function may contribute to variations in arterial tone; eg, in endothelium damaged by atheromas, stress of a catecholamine surge causes vasoconstriction rather than dilation (normal response).

As the myocardium becomes ischemic, coronary sinus blood pH falls, cellular potassium is lost, lactate accumulates, ECG abnormalities appear, and ventricular function (both systolic and diastolic) deteriorates. Left ventricular (LV) diastolic pressure usually increases during angina, sometimes inducing pulmonary congestion and dyspnea. The exact mechanism by which ischemia causes discomfort is unclear but may involve nerve stimulation by hypoxic metabolites.

Symptoms and Signs of Angina Pectoris

Angina may be a vague, barely troublesome ache or may rapidly become a severe, intense precordial crushing sensation. It is rarely described as "pain." Discomfort is most commonly felt beneath the sternum, although location varies. Discomfort may radiate to the left shoulder and down the inside of the left arm, even to the fingers; straight through to the back; into the throat, jaws, and teeth; and, occasionally, down the inside of the right arm. It may also be felt in the upper abdomen. The discomfort of angina is never above the ears or below the umbilicus.

Atypical angina, with bloating, gas, abdominal distress, or burning or tenderness in the back, shoulders, arms or jaw, may occur in some patients and is more common among females. These patients often ascribe symptoms to indigestion; belching may even relieve the symptoms. Other patients have dyspnea due to the sharp, reversible increase in LV filling pressure that often

accompanies ischemia. Frequently, the patient's description is imprecise, and whether the problem is angina, dyspnea, or both may be difficult to determine. Because ischemic symptoms require a minute or more to resolve, brief, fleeting sensations rarely represent angina.

Between and even during attacks of angina, physical findings may be normal. However, during the attack, heart rate may increase modestly, blood pressure (BP) is often elevated, heart sounds become more distant, and the apical impulse is more diffuse. The second heart sound (S2) may become paradoxical because LV ejection is more prolonged during an ischemic attack. A fourth heart sound (S4) is common, and a third heart sound (S3) may develop. A mid or late systolic apical murmur, shrill or blowing—but not especially loud—may occur if ischemia causes localized papillary muscle dysfunction, causing mitral regurgitation.

Angina pectoris is typically triggered by exertion or strong emotion, usually persists no more than a few minutes, and subsides with rest. Response to exertion is usually predictable, but in some patients, exercise that is tolerated one day may precipitate angina the next because of variations in arterial tone. Symptoms are exaggerated when exertion follows a meal or occurs in cold weather; walking into the wind or first contact with cold air after leaving a warm room may precipitate an attack. Symptom severity is often classified by the degree of exertion resulting in angina (see table Canadian Cardiovascular Classification System for Angina Pectoris).

TABLE

Canadian Cardiovascular Society Classification System for Angina Pectoris

Class	Activities Triggering Chest Pain
1	Strenuous, rapid, or prolonged exertion
	Not usual physical activities (eg, walking, climbing stairs)
2	Walking rapidly

Adapted from 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). *Circulation* 102(10):1193–209, 2000. doi: 10.1161/01.cir.102.10.1193

Class	Activities Triggering Chest Pain
	Walking uphill
	Climbing stairs rapidly
	Walking or climbing stairs after meals
	Cold
	Wind
	Emotional stress
3	Walking, even 1 or 2 blocks at usual pace and on level ground
	Climbing stairs, even 1 flight
4	Any physical activity
	Sometimes occurring at rest

Adapted from 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). *Circulation* 102(10):1193–209, 2000. doi: 10.1161/01.cir.102.10.1193

Attacks may vary from several a day to symptom-free intervals of weeks, months, or years. Attacks may increase in frequency (called crescendo angina), leading to an MI or death. Conversely, attacks may gradually decrease or disappear if adequate collateral coronary circulation develops, the ischemic area infarcts, or heart failure or intermittent claudication supervenes and limits activity.

Nocturnal angina may occur if a dream causes striking changes in respiration, pulse rate, and BP. Nocturnal angina may also be a sign of recurrent LV failure, an equivalent of nocturnal dyspnea. The recumbent position increases venous return, stretching the myocardium and increasing wall stress, which increases oxygen demand.

Angina decubitus is angina that occurs spontaneously when a person is lying down but not necessarily at night. It is usually accompanied by a modestly increased heart rate and a sometimes markedly higher BP, which increase oxygen demand. These increases may be the cause of rest angina or the result of ischemia induced by plaque rupture and thrombus formation. If angina is not relieved, unmet myocardial oxygen demand increases further, making MI more likely.

Unstable angina

Because angina characteristics are usually predictable for a given patient, any changes (ie, angina at rest, new-onset angina, increasing angina, new nocturnal angina, or new angina decubitus) should be considered serious, especially when the angina is severe (ie, Canadian Cardiovascular Society class 3 or 4). Such changes are termed unstable angina and require prompt evaluation and treatment.

Diagnosis of Angina Pectoris

- Typical symptoms
- Electrocardiography (ECG)
- Stress testing with ECG or imaging (using echocardiography, radionuclide imaging, positron emission tomography [PET], or MRI)
- CT angiography or CT fractional flow reserve (CT FFR)
- Coronary angiography for significant symptoms, positive stress test, or significant lesions noted on CT FFR.

Diagnosis of angina is suspected if chest discomfort is typical and is precipitated by exertion and relieved by rest. Presence in the history of significant risk factors for coronary artery disease (CAD) adds weight to reported symptoms. Patients whose chest discomfort lasts > 20 minutes or occurs during rest or who have syncope or heart failure are evaluated for an acute coronary syndrome.

Chest discomfort may also be caused by gastrointestinal disorders (eg, gastroesophageal reflux, esophageal spasm, indigestion, cholelithiasis), costochondritis, anxiety, panic attacks, hyperventilation, and other cardiac disorders (eg, aortic dissection, pericarditis, mitral valve prolapse, supraventricular tachycardia, atrial fibrillation), even when coronary blood flow is not compromised.

ECG is always done. More specific tests include stress testing with ECG or with myocardial imaging (eg, echocardiography, radionuclide imaging, PET, MRI) and coronary angiography. Noninvasive tests are considered first.

ECG

If typical exertional symptoms are present, ECG is indicated. Because angina resolves quickly with rest, ECG rarely can be done during an attack except during stress testing.

If done **during an angina attack,** ECG is likely to show reversible ischemic changes:

- T wave discordant to the QRS vector
- ST-segment depression (typically)
- ST-segment elevation
- Decreased R-wave height
- Intraventricular or bundle branch conduction disturbances
- Arrhythmia (usually ventricular extrasystoles)

Between angina attacks, the ECG (and usually LV function) at rest is normal in about 30% of patients with a typical history of angina pectoris, even those with extensive 3-vessel disease. In the remaining 70%, the ECG shows evidence of previous infarction, hypertrophy, or nonspecific ST-segment and T-wave (ST-T) abnormalities. An abnormal resting ECG alone does not establish or refute the diagnosis.

Stress testing

Stress testing is needed to

- Confirm the diagnosis
- Evaluate disease severity
- Determine appropriate exercise levels for the patient
- Help predict prognosis

If the clinical or working diagnosis is unstable angina, early stress testing is contraindicated.

Exercise stress testing with ECG is done if a patient has a normal resting ECG and can exercise. In males with chest discomfort suggesting angina, stress ECG testing has a sensitivity of approximately 70% and a specificity of approximately 80% (1). These estimates are slightly lower in females. However, females are more likely than males to have an abnormal resting ECG when CAD is present (32% versus 23%). Although sensitivity is reasonably high, exercise ECG can miss severe CAD (even left main or 3-vessel disease). In patients with atypical symptoms, a negative stress ECG usually rules out angina pectoris and CAD; a positive result may or may not represent coronary ischemia and indicates the need for further testing.

Stress testing with myocardial imaging is done when the resting ECG is abnormal because false-positive ST-segment shifts are common on the stress ECG. Exercise or pharmacologic stress (eg, with dobutamine or dipyridamole infusion) may be used. Imaging options include stress echocardiography, myocardial perfusion imaging with single-photon emission CT (SPECT) or PET, and stress MRI. The choice of imaging technique depends on institutional availability and expertise. Imaging tests can help assess LV function and response to stress; identify areas of ischemia, infarction, and viable tissue; and determine the site and extent of myocardium at risk. Stress echocardiography can also detect ischemia-induced mitral regurgitation.

Coronary angiography

Coronary angiography is the gold standard for diagnosing coronary artery disease but is not always necessary to confirm the diagnosis. It is indicated primarily to locate and assess severity of coronary artery lesions when revascularization (percutaneous coronary intervention [PCI] or coronary artery bypass grafting [CABG]) is being considered. Angiography may also be indicated when knowledge of coronary anatomy is necessary to advise about work or lifestyle needs (eg, discontinuing job or sports activities). Although angiographic findings do not directly show hemodynamic significance of coronary lesions, obstruction is assumed to be physiologically significant when the luminal diameter is reduced > 70%. Angina does not usually develop when the diameter reduction is < 70% unless spasm or thrombosis is present.

Intravascular ultrasonography **(IVUS)** provides images of coronary artery structure. An ultrasound probe on the tip of a catheter is inserted in the coronary arteries during angiography. This test can provide more information about coronary anatomy than other tests; it is indicated when the nature of lesions is unclear or when apparent disease severity does not match symptom severity. Used with PCI, it can help ensure optimal placement of stents.

Guidewires with pressure or flow sensors can be used to estimate blood flow across stenoses. Blood flow is expressed as fractional flow reserve (FFR), which is the ratio of maximal flow through the stenotic area to normal maximal flow. These flow measurements are most useful when evaluating the need for angioplasty or CABG in patients with lesions of questionable severity (40 to 70% stenosis). An FFR of 1.0 is considered normal, while an FFR < 0.75 to 0.8 is associated with myocardial ischemia. Lesions with an FFR > 0.8 are less likely to benefit from stent placement.

Optical coherence tomography **(OCT)** is another imaging modality that can be used during coronary angiography that uses near-infrared light to provide high-resolution cross-sectional images of the coronary arteries, which are higher in resolution than with IVUS (2, 3). OCT has been used to help optimize placement and sizing of stents during PCI. However, its role in routine clinical care for patients with CAD who are undergoing angiography has not been fully established.

Imaging the coronary arteries

Noninvasive imaging studies done at rest can evaluate the coronary arteries.

Electron beam CT can detect the amount of calcium present in coronary artery plaque. The calcium score is roughly proportional to the risk of subsequent coronary events. However, because calcium may be present in the absence of significant stenosis, the score does not correlate well with the need for PCI or CABG. Thus, the American Heart Association recommends that screening with electron beam CT should be done only for select groups of patients and is most valuable when combined with historical and clinical data to estimate risk of death or nonfatal myocardial infarction (4). These groups may include asymptomatic patients with an intermediate 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimate (10 to 20%) and symptomatic patients with equivocal stress test results. Electron beam CT is particularly useful in ruling out significant CAD in patients presenting to the emergency department with atypical symptoms, normal troponin levels, and a low probability of hemodynamically significant coronary artery disease. These patients may have noninvasive testing as outpatients.

Multidetector row CT (MDRCT) coronary angiography can accurately identify coronary stenosis and has a number of advantages. The test is noninvasive, can exclude coronary stenosis with high accuracy, can establish stent or bypass graft patency, can show cardiac and coronary venous anatomy, and can assess calcified and noncalcified plaque burden. Estimation of the fractional flow reserve (FFR) across significant lesions and estimation of lesion-specific ischemia are also possible (5). However, radiation exposure is significant, and MDRCT must be used judiciously in patients with a heart rate of > 65 beats/minute, those with irregular heart beats, those with impaired kidney function, and pregnant females. Patients must also be able to hold their breath for 15 to 20 seconds, 3 to 4 times during the study.

Other indications for MDRCT coronary angiography include

- Asymptomatic high-risk patients
- Patients with atypical or typical angina who have inconclusive exercise stress test results, cannot undergo exercise stress testing, or need to undergo major noncardiac surgery
- Patients in whom invasive coronary angiography was unable to locate a major coronary artery or graft

Cardiac magnetic resonance imaging (MRI) is invaluable in evaluating many cardiac and great vessel abnormalities. It may be used to evaluate CAD by several techniques, which enable direct visualization of coronary stenosis, assessment of flow in the coronary arteries, evaluation of myocardial perfusion and metabolism, evaluation of wall motion abnormalities during stress, and assessment of infarcted myocardium vs viable myocardium.

Indications for cardiac MRI include evaluation of cardiac structure and function and assessment of myocardial viability. Cardiac MRI, specifically stress perfusion MRI and quantitative myocardial blood flow analysis, may also be indicated for diagnosis and risk assessment in patients with either known or suspected CAD.

Diagnosis references

1. Fletcher GF, Ades PA, Kligfield P, et al: Exercise standards for testing and training: a scientific statement from the American Heart Association. *Circulation* 128(8):873–934, 2013. doi:10.1161/CIR.0b013e31829b5b44
2. Araki M, Park SJ, Dauerman HL, et al: Optical coherence tomography in coronary atherosclerosis assessment and intervention. *Nat Rev Cardiol* 19(10):684–703, 2022. doi:10.1038/s41569-022-00687-9
3. Wulandari AP, Pintaningrum Y, Adheriyani R. Outcomes of optical coherence tomography guided percutaneous coronary intervention against angiography guided in patients with coronary artery disease: A systematic reviews and meta-analyses. *Indian Heart J* 2023;75(2):108-114. doi:10.1016/j.ihj.2023.01.009
4. Taylor AJ, Cerqueira M, Hodgson J McB, et al: ACCF/SCCT/ACR/AHA/ASE/ASNC/SCAI/SCMR 2010 Appropriate Use Criteria for Cardiac Computed Tomography. *J Am Coll Cardiol* 56: 1864–1894, 2010. doi: 10.1016/j.jacc.2010.07.005
5. Gulati M, Levy PD, Mukherjee D, et al: 2021 AHA/ACC Guideline for Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation* 144:e368–e454, 2021. https://www.ahajournals.org/doi/epdf/10.1161/CIR.0000000000001029

Treatment of Angina Pectoris

- Modification of risk factors (smoking, blood pressure, lipids)
- Antiplatelet agents (aspirin and sometimes clopidogrel, prasugrel, or ticagrelor)
- Beta-blockers
- Nitroglycerin and calcium channel blockers for symptom control
- Angiotensin-converting enzyme (ACE) inhibitors and statins

- Revascularization if symptoms persist despite medical therapy

Reversible risk factors are modified as much as possible. People who smoke should stop smoking; 2 or more years after stopping smoking, risk of MI is reduced to that of people who never smoked. Hypertension (BP > 130/80 for patients with CAD) is treated diligently because even mild hypertension increases cardiac workload. Weight loss alone often reduces the severity of angina. Sometimes treatment of mild LV failure markedly lessens angina. However, digitalis occasionally intensifies angina, presumably because increased myocardial contractility increases oxygen demand, arterial tone is increased, or both. Aggressive reduction of total cholesterol and low-density lipoprotein (LDL) cholesterol (via diet plus statins) slows the progression of CAD, may cause some lesions to regress, and improves endothelial function and thus arterial response to stress. An exercise program emphasizing walking often improves the sense of well-being, reduces risk of acute ischemic events, and improves exercise tolerance.

Medications for angina

The main goals of angina treatment are to

- Relieve acute symptoms
- Prevent or reduce ischemia
- Prevent future ischemic events

(See also table Medications for Coronary Artery Disease.)

To relieve symptoms during an acute attack, sublingual nitroglycerin is the most effective drug.

Nitroglycerin is a potent smooth-muscle relaxant and vasodilator. Its main sites of action are in the peripheral vascular tree, especially in the venous or capacitance system, and in coronary blood vessels. Even severely atherosclerotic vessels may dilate in areas without atheroma. Nitroglycerin lowers systolic BP and dilates systemic veins, thus reducing myocardial wall tension, a major determinant of myocardial oxygen need. Sublingual nitroglycerin is given for an acute attack or for prevention before exertion. Dramatic relief usually occurs within 1.5 to 3 minutes, is complete by about 5 minutes, and lasts up to 30 minutes. The dose may be repeated every 4 to 5 minutes up to 3 times if relief is incomplete. Patients should always carry nitroglycerin tablets or aerosol spray to use promptly at the onset of an angina attack. Patients should store tablets in a tightly sealed, light-resistant glass container, so that potency is not lost. Because the drug deteriorates quickly, small amounts should be obtained frequently.

To prevent ischemia, several classes of medications are used:

- Antiplatelet agents: All patients diagnosed with coronary artery disease or at high risk of developing CAD
- Beta-blockers: Most patients, unless contraindicated or not tolerated
- Long-acting nitrates: If needed
- Calcium channel blockers: If needed

Antiplatelet agents inhibit platelet aggregation. Aspirin binds irreversibly to platelets and inhibits cyclooxygenase and platelet aggregation. Other antiplatelets (eg, clopidogrel, prasugrel, ticagrelor) block

adenosine diphosphate–induced platelet aggregation. These agents can reduce the risk of ischemic events (MI, sudden death), but they are most effective when given with aspirin. Patients unable to tolerate one agent should receive the other alone.

Beta-blockers limit symptoms and prevent infarction and sudden death better than other medications. Beta-blockers other than labetalol and carvedilol should not be used in patients with vasospastic angina because they may cause coronary vasospasm from unopposed alpha-receptor activity. Beta-blockers block sympathetic stimulation of the heart and reduce systolic BP, heart rate, contractility, and cardiac output, thus decreasing myocardial oxygen demand and increasing exercise tolerance. Beta-blockers also increase the threshold for ventricular fibrillation. Most patients tolerate these agents well. Many beta-blockers are available and effective. Dose is titrated upward as needed until limited by bradycardia or adverse effects. Patients who cannot tolerate beta-blockers are given a calcium channel blocker with negative chronotropic effects (eg, diltiazem, verapamil). Patients who are at risk of beta-blocker intolerance (eg, those with asthma) may be tried on a cardioselective beta-blocker (eg, bisoprolol) perhaps with pulmonary function testing before and after medication administration to detect drug-induced bronchospasm.

Long-acting nitrates (oral or transdermal) are used if symptoms persist after the beta-blocker dose is maximized. If angina occurs at predictable times, a nitrate is given to cover those times. Oral nitrates include isosorbide dinitrate and isosorbide mononitrate (the active metabolite of the dinitrate). They are effective within 1 to 2 hours; their effect lasts 4 to 6 hours. Sustained-release formulations of isosorbide mononitrate appear to be effective throughout the day. For transdermal use, cutaneous nitroglycerin patches have largely replaced nitroglycerin ointments primarily because ointments are inconvenient and messy. Patches slowly release the drug for a prolonged effect; exercise capacity improves 4 hours after patch application and wanes in 18 to 24 hours. Nitrate tolerance may occur, especially when plasma concentrations are kept constant. Because risk of myocardial infarction is highest in early morning, an afternoon or early evening respite period from nitrates is reasonable unless a patient commonly has angina at that time. For nitroglycerin, an 8- to 10-hour respite period seems sufficient. Isosorbide dinitrate requires a 12-hour respite period to minimize the risk of tolerance. If given once a day, sustained-release isosorbide mononitrate does not appear to elicit tolerance.

Calcium channel blockers may be used if symptoms persist despite use of nitrates or if nitrates are not tolerated. Calcium channel blockers are particularly useful if hypertension or coronary spasm is also present. Different types of calcium channel blockers have different effects. Dihydropyridines (eg, nifedipine, amlodipine, felodipine) have no chronotropic effects and vary substantially in their negative inotropic effects. Shorter-acting dihydropyridines may cause acute hypotension and reflex tachycardia and are associated with increased mortality in patients with CAD; they should not be used alone to treat stable angina pectoris. Longer-acting formulations of dihydropyridines have fewer tachycardic effects; they are most commonly used with a beta-blocker. Among longer-acting dihydropyridines, amlodipine has the weakest negative inotropic effects; it may be used in patients with left ventricular systolic dysfunction. Diltiazem and verapamil, other types of calcium channel blockers, have negative chronotropic and inotropic effects. They can be used alone in patients with beta-blocker intolerance or asthma and normal left ventricular systolic function but may increase cardiovascular mortality in patients with left ventricular systolic dysfunction.

Ranolazine is a **sodium channel blocker** that can be used to treat chronic angina. Because ranolazine may also prolong QTc, it is usually reserved for patients in whom symptoms persist despite optimal treatment with other antianginal agents. Dizziness, headache, constipation, and nausea are the most common adverse effects.

Ivabradine is a **sinus node inhibitor** that inhibits inward sodium/potassium current in a certain gated channel (funny or "f" channel) in sinus node cells, thus slowing heart rate without decreasing contractility. It can be used for symptomatic treatment of chronic stable angina pectoris in patients with normal sinus rhythm who cannot take beta-blockers or in combination with beta-blockers in patients whose angina is inadequately controlled by beta-blocker alone and whose heart rate is > 60 beats/minute.

Revascularization

Revascularization, either with PCI (eg, angioplasty and stent placement) or CABG, should be considered if angina persists despite pharmacologic therapy and worsens quality of life or if anatomic lesions (noted during angiography) put a patient at high risk of mortality. The choice between PCI and CABG depends on the extent and location of anatomic lesions, the experience of the operator and medical center, and, to some extent, patient preference (1).

PCI is usually preferred for 1- or 2-vessel disease with suitable anatomic lesions and is increasingly being used for 3-vessel disease. As stent technology improves, PCI is being used for more complicated lesions (eg, lesions that are long or near bifurcation points). PCI can also be used for left main coronary artery disease (LMCA) in the appropriate clinical setting (2, 3, 4).

CABG is very effective in selected patients with angina. CABG is superior to PCI in patients with diabetes and in patients with multivessel disease amenable to grafting (5, 6, 7). The ideal candidate has severe angina pectoris and localized disease, or diabetes mellitus. About 85% of patients have complete or dramatic symptom relief. Exercise stress testing shows positive correlation between graft patency and improved exercise tolerance, but exercise tolerance sometimes remains improved despite graft closure.

CABG improves survival for patients with left main disease, those with 3-vessel disease and poor left ventricular function, and some patients with 2-vessel disease (8). However, for patients with mild or moderate angina (CCS class 1 or 2) or 3-vessel disease and good ventricular function, CABG appears to only marginally improve survival. Several studies show better long-term outcomes following CABG than with PCI for patients with diabetes and proximal left anterior descending disease (8). For patients with 1-vessel disease, outcomes with pharmacologic therapy, PCI, and CABG are similar; exceptions are left main disease and proximal left anterior descending disease, for which revascularization appears advantageous.

Treatment references

1. Lawton JS, Tamis-Holland JE, Bangalore S, et al: 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: a report of the ACC/AHA Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol* 79(2):e21–e129, 2022. doi: 10.1016/j.jacc.2021.09.006

2. Nerlekar N, Ha FJ, Verma KP, et al: Percutaneous Coronary Intervention Using Drug-Eluting Stents Versus Coronary Artery Bypass Grafting for Unprotected Left Main Coronary Artery Stenosis: A Meta-Analysis of Randomized Trials. *Circ Cardiovasc Interv* 9(12):e004729, 2016. doi:10.1161/CIRCINTERVENTIONS.116.004729
3. Stone GW, Sabik JF, Serruys PW, et al. Everolimus-Eluting Stents or Bypass Surgery for Left Main Coronary Artery Disease [published correction appears in *N Engl J Med* 2019 Oct 31;381(18):1789]. *N Engl J Med* 2016;375(23):2223-2235. doi:10.1056/NEJMoa1610227
4. Stone GW, Kappetein AP, Sabik JF, et al. Five-Year Outcomes after PCI or CABG for Left Main Coronary Disease [published correction appears in N Engl J Med. 2020 Mar 12;382(11):1078]. N Engl J Med. 2019;381(19):1820-1830. doi:10.1056/NEJMoa1909406
5. Farkouh ME, Domanski M, Sleeper LA, et al: Strategies for multivessel revascularization in patients with diabetes. *N Engl J Med* 367(25):2375–2384, 2012. doi:10.1056/NEJMoa1211585
6. Influence of diabetes on 5-year mortality and morbidity in a randomized trial comparing CABG and PTCA in patients with multivessel disease: the Bypass Angioplasty Revascularization Investigation (BARI). *Circulation* 1997;96(6):1761-1769. doi:10.1161/01.cir.96.6.1761
7. BARI 2D Study Group, Frye RL, August P, et al. A randomized trial of therapies for type 2 diabetes and coronary artery disease. *N Engl J Med* 2009;360(24):2503-2515. doi:10.1056/NEJMoa0805796
8. Serruys PW, Morice MC, Kappetein AP, et al. Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease [published correction appears in *N Engl J Med* 2013 Feb 7;368(6):584]. *N Engl J Med* 2009;360(10):961-972. doi:10.1056/NEJMoa0804626

Prognosis for Angina Pectoris

The main adverse outcomes of angina pectoris are unstable angina, myocardial infarction, and sudden death due to arrhythmias. Annual mortality rate is approximately 1.4% in patients with angina, no history of MI, a normal resting ECG, and normal BP (1). However, females with CAD tend to have a worse prognosis. Mortality rate is higher when systolic hypertension is present, than when the ECG is abnormal, and is even higher when both are present. Type 2 diabetes also increases the mortality rate for each scenario.

Prognosis worsens with increasing age, increasingly severe anginal symptoms, presence of anatomic lesions, and poor ventricular function (2). Lesions in the left main coronary artery or proximal left anterior descending artery indicate particularly high risk. Although prognosis correlates with number and severity of coronary arteries affected, prognosis is surprisingly good for patients with stable angina, even those with 3-vessel disease, if ventricular function is normal.

Prognosis references

1. Fox K, Garcia MA, Ardissino D, et al. Guidelines on the management of stable angina pectoris: executive summary: The Task Force on the Management of Stable Angina Pectoris of the European Society of Cardiology. *Eur Heart J* 2006;27(11):1341-1381. doi:10.1093/eurheartj/ehl001
2. Virani SS, Newby LK, Arnold SV, et al. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease: A Report of the American Heart

Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines [published correction appears in *Circulation* 2023 Sep 26;148(13):e148] [published correction appears in *Circulation* 2023 Dec 5;148(23):e186]. *Circulation* 2023;148(9):e9-e119. doi:10.1161/CIR.0000000000001168

Key Points

- Angina pectoris occurs when cardiac workload exceeds the ability of coronary arteries to supply an adequate amount of oxygenated blood.
- Symptoms of stable angina pectoris range from a vague, barely troublesome ache to a severe, intense precordial crushing sensation; they are typically precipitated by exertion, last no more than a few minutes, and subside with rest.
- Do stress testing with ECG for patients with normal resting ECG or with myocardial imaging (eg, echocardiography, radionuclide imaging, PET, MRI) for patients with abnormal resting ECG.
- Do coronary angiography when revascularization (percutaneous intervention or coronary artery bypass grafting) is being considered.
- Give nitroglycerin for immediate relief of angina.
- Maintain patients on an antiplatelet agent, a beta-blocker, and a statin, and add a calcium channel blocker for further symptom prevention if needed.
- Consider revascularization if significant angina persists despite pharmacologic therapy or if lesions noted during angiography indicate high risk of mortality.