

prognosis. Stress testing is useful both in establishing the diagnosis of ischemic heart disease and in assessing its severity.

### ***Nuclear stress testing and other imaging***

Radionuclide techniques can be used to increase the sensitivity of exercise testing. Left ventricular dysfunction can result from necrotic tissue, myocardial hibernation after injury, or myocardial stunning. Approximately 20%–40% of patients with left ventricular dysfunction on echocardiography or stress testing still have viable myocardial tissue, which may improve with reperfusion. Several agents are available for injection during testing; these include thallium-201, technetium-99m (Tc99m) sestamibi, and technetium-99m tetrofosmin.

Thallium accumulates in healthy myocardium and reveals a perfusion defect in areas of myocardial ischemia. Reversible thallium or Tc99m sestamibi defects are those that are present during exercise but resolve during rest. This correlates with myocardial ischemia. In contrast, a fixed (persistent) thallium or Tc99m defect is present during both exercise and rest and represents a region of prior infarction or nonviable tissue. For patients unable to exercise vigorously enough to reach the required heart rates during the exercise stress test, a thallium scan or ECG in conjunction with a pharmacologic stress test may provide information similar to that of an exercise examination. Tomographic imaging of myocardial perfusion is possible with thallium-201 or Tc99m by means of a technique called *single-photon emission computed tomography (SPECT)*, which provides better imaging of infarcts, enhanced detection of multivessel disease, and fewer artifacts.

Other imaging technologies that may add clinically useful information include the following:

- *Positron emission tomography (PET)*, which differentiates metabolically active myocardium from scar tissue.
- *Coronary CT angiography*, which is useful in evaluating occlusive vascular disease and ruling out atherosclerotic disease. This test compares well to invasive coronary angiography and may be preferred in stable patients with equivocal results on other noninvasive testing.
- *Coronary artery calcium scoring*, which utilizes multidetector CT scans to measure coronary artery calcification, is a metric that correlates with atherosclerosis and is highly sensitive but not specific. It may be an alternative to invasive angiography in some patients.
- *Cardiac magnetic resonance imaging (MRI)*, which provides excellent imaging, and perfusion testing with gadolinium. MRI may be contraindicated in some patients with ICDs or pacemakers, but it can be safely used in the presence of coronary stents. Cardiac CT and MRI are also useful in assessing congenital or acquired coronary abnormalities.


### **Invasive Cardiac Diagnostic Procedures**

As noninvasive imaging techniques have improved, the indications for invasive coronary angiography have decreased. Nevertheless, *coronary angiography* and *ventriculography* provide valuable information about the presence and severity of CHD and about

ventricular function. These techniques can indicate the specific areas of coronary artery stenosis or occlusion, the number of involved vessels, the ventricular systolic and diastolic volumes, the ejection fraction, and regional wall-motion abnormalities. *Radionuclide ventriculography* scans can also be performed for these purposes. This information helps the cardiologist and cardiac surgeon plan appropriate treatment for the patient. *Intravascular ultrasound imaging* at the time of cardiac catheterization is useful for studying the intraluminal coronary anatomy and the effects of stents or angioplasty.

Common indications for coronary arteriography are ACS, post-MI angina, stable angina unresponsive to medical therapy or revascularization, a markedly positive exercise stress test result, and evidence of extensive myocardium at risk from ischemia that might benefit from revascularization.

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 **Ophthalmic considerations** Many of the adult patients seen and treated by ophthalmologists are in the age group at risk for IHD and its many complications. They often undergo stressful eye surgery under local or general anesthesia, and ophthalmologists need to be cognizant of these patients' risks of myocardial ischemia, MI, CHF, and arrhythmias. In addition, ophthalmologists need to be aware that patients with nonproliferative or proliferative diabetic retinopathy have an increased risk of MI, stroke, and death from cardiovascular disease. This information should be shared with the primary care provider of patients with significant diabetic retinopathy but without a diagnosis of IHD so that appropriate screening tests for cardiovascular disease can be considered.

Targher G, Bertolini L, Zenari L, et al. Diabetic retinopathy is associated with an increased incidence of cardiovascular events in Type 2 diabetic patients. *Diabet Med.* 2008;25(1):45–50.

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## Management of Ischemic Heart Disease

The goals of disease management for the patient with CHD are to reduce the frequency of or eliminate angina, prevent myocardial damage, and prolong life. The first line of attack should include eliminating or reducing risk factors for atherosclerosis. Smoking cessation, dietary modification, weight loss, exercise, and improved control of diabetes and hypertension are critical steps. Regression of atherosclerotic lesions following intensive lipid-lowering therapy has been reported; and, unless contraindicated, statins are recommended by the American College of Cardiology (ACC) and American Heart Association (AHA) for all CHD patients. Antiplatelet therapy with low-dose daily aspirin has also been advocated for all patients with CHD because it significantly reduces the risk of MI.

Aspirin appears to offer equal benefit to women and men in reducing primary MI risk, and it is also useful in secondary prevention. Aspirin may also help protect against stroke; however, in low-risk patients, the risk of bleeding complications may outweigh the benefits. Aspirin use should be guided by an assessment of the patient's risk of stroke or MI versus their risk of bleeding complications. Hormone therapy, antioxidant vitamin supplementation, and folic acid therapy do not appear to provide any benefit in preventing cardiovascular disease.

### ***Antithrombotic agents***

Prevention of stroke, MI, and numerous other thromboembolic diseases requires selective inhibition of the hemostasis process. Numerous medications have been developed to inhibit platelet aggregation or block specific steps in the coagulation cascade (Fig 6-3), including several oral agents, which are frequently used in the outpatient setting (Table 6-1). The drugs that require intravenous or subcutaneous injection are predominantly used in an inpatient setting (Table 6-2).

### ***Treatment of stable angina pectoris***

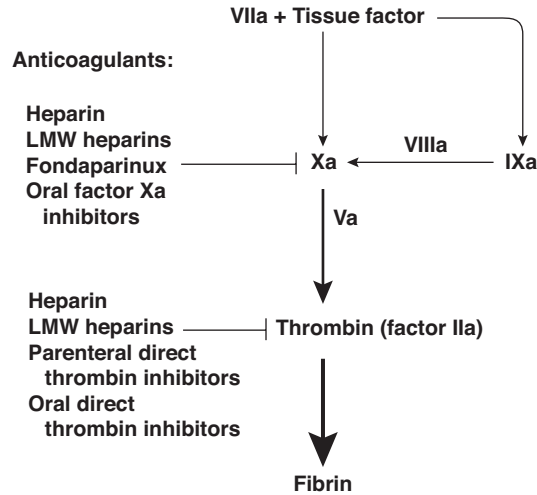
**Medical** Medical management of angina pectoris is designed to deliver as much oxygen as possible to the potentially ischemic myocardium, to decrease the oxygen demand to a level at which symptoms are eliminated or reduced to a comfortable level, or both.

Therapeutic agents include the following:

- *$\beta$ -Adrenergic blockers.* Also called  *$\beta$ -blockers*, these drugs represent the first line of treatment. They reduce heart rate and contractility (decreasing oxygen demand) and have been shown to prolong life in patients with CHD. They should be avoided in patients with Prinzmetal angina.
- *Slow calcium channel blockers.* These agents, including diltiazem, verapamil, and amlodipine, are useful for long-term treatment of angina. They should be used with caution in patients with left ventricular dysfunction.
- *Nitrates and nitroglycerin.* These agents increase oxygen delivery through coronary vasodilation. Systemic effects (eg, venous dilation, reduction in blood pressure) decrease oxygen demand. They should be used with caution in patients taking erectile dysfunction drugs.
- *Aspirin with or without clopidogrel, prasugrel, or ticagrelor.* These drugs can be used for anticoagulation. The regimen of aspirin plus 1 of these other drugs is called *dual antiplatelet therapy (DAPT)*.
- *Statins.* Statins are recommended for use in virtually all ACS patients, regardless of serum lipid levels, if not contraindicated.

Improving the oxygen-carrying capacity of the blood by treating anemia or coexisting pulmonary disease provides some additional benefit. Patients in whom medical therapy is unsuccessful may be candidates for revascularization procedures.

**Revascularization** Procedures for revascularization include *percutaneous coronary intervention (PCI)* with or without stenting or *coronary artery bypass grafting (CABG)*. These approaches may improve coronary blood flow, control angina, and increase exercise tolerance. In high-risk patients, the risk of infarction is reduced, and long-term survival is enhanced. Revascularization is indicated in otherwise healthy patients with advanced left main coronary artery disease, left ventricular dysfunction with 3-vessel disease, or angina that is not adequately controlled with medical treatment. Either PCI or CABG is effective for relieving angina; however, CABG is superior to PCI in terms of survival for some patients who have significant areas of at-risk myocardium or substantial left ventricular dysfunction. Recently, some authors have questioned whether PCI is superior to maximal medical therapy in the treatment of stable angina.



**Figure 6-3** Coagulation cascade. The various anticoagulant drugs interrupt the cascade at different points in the process. LMW=low molecular weight. (Adapted with permission from Leung LLK. *Direct oral anticoagulants and parenteral direct thrombin inhibitors: dosing and adverse effects*. In: UpToDate. Post TW, ed. UpToDate, Inc. Accessed October 10, 2022.)

**Table 6-1 Oral Antithrombotic Agents**

Class	Drug (trade name)	Mechanism of Action
Antiplatelet agents	Aspirin	Blocks cyclooxygenase and inhibits platelet aggregation
Antiplatelet agents (P2Y <sub>12</sub> receptor blockers)	Cangrelor (Kengreal) Clopidogrel (Plavix) Prasugrel (Effient) Ticagrelor (Brilinta) Ticlopidine (not available in the United States)	Prevents activation of glycoprotein IIb/IIIa and inhibits platelet aggregation
Vitamin K antagonists	Warfarin (Coumadin) Other coumarins (not available in the United States) including acenocoumarol, fluindione, phenprocoumon	Reduces the synthesis of numerous clotting factors that require vitamin K
Direct thrombin inhibitors	Dabigatran (Pradaxa)	Prevents thrombin from cleaving fibrinogen to fibrin; can be reversed with idarucizumab (Praxbind)
Factor Xa inhibitors (Note: all drugs in this class end in "-xaban")	Apixaban (Eliquis) Edoxaban (Lixiana, Savaysa) Rivaroxaban (Xarelto)	Prevents factor Xa from cleaving prothrombin to thrombin; rivaroxaban and apixaban can be reversed with andexanet alfa (Andexxa)