Chapter 7
Chronic Stable Angina

The annual incidence of angina pectoris in the United Kingdom is 0.5%; the male to female ratio is 2:1. More than 1.4 million people suffer from angina, and 300,000 have a heart attack every year.

DIAGNOSIS

Q: How does atypical angina differ from typical angina?
Angina pectoris is a clinical syndrome characterized by discomfort in the chest, jaw, shoulder, back, or arms. It is typically aggravated by exercise or emotional stress and relieved by nitroglycerin. Anginal pain occurs when at least one coronary artery (obstructive coronary artery disease [CAD]) is involved, but can also present in patients with valvular heart disease, hypertrophic cardiomyopathy, and uncontrolled hypertension. Although the term angina usually refers to chronic stable type, there are three types: chronic stable angina, atypical angina, and unstable angina. Unstable angina is now classified as an acute coronary syndrome (ACS).

Anginal symptoms are initially experienced on exertion, which is termed angina of effort. Most symptomatic patients have the lumen of one or more segments of their artery reduced by at least half. Over half the patients show recanalization through the thrombus. There are many variations in the size and shape of the thrombus. Angina can be classified according to the severity of the symptoms (Table 7.1).

Chest pain is the most common mode of presentation. The pain lasts for no more than 10 to 15 minutes. The character of pain may be of crushing, compressing, stabbing, choking, or burning type. The site of pain is usually retrosternal, spreading to both sides of the anterior chest but most commonly to the left. The pain classically radiates along the inner aspect of the left upper limb but may do so to the neck, shoulder, and jaw. Tingling along the left upper limb or fingers may be felt. Breathlessness may accompany pain or may be the only symptom. Fatigue and perspiration may coexist.
The predisposing factors include eating a heavy meal, cold weather, stress, and sexual intercourse. Stress may be in the form of anger, fright, anxiety, or arguments. Coronary spasm may occur both in normal and diseased vessels, which may precipitate anginal symptoms. The symptoms depend on the severity of the spasm. The cause of coronary spasm is not clear. It is possible that certain sites in the coronary artery are more prone to spasm. Since an atheromatous lesion has acquired hypersensitivity to vasoconstrictors, one possibility is that the endothelium at this site is deficient in the production of endothelial-derived relaxant factor. Endothelial damage itself increases sensitivity to vasoconstrictor stimuli. Anginal pain should be distinguished from other causes of chest pain, such as anxiety and hyperacidity, and from pain of musculoskeletal origin. The physical examination is usually unremarkable. However, during an anginal attack increased blood pressure and increase heart rate may be noticed due to an augmented sympathetic response. Mitral regurgitation due to muscle dysfunction may be detected.

Atypical angina (vasospastic, Prinzmetal’s angina) is a rare form of angina and is more commonly prevalent in women. Prinzmetal first described it. Unlike stable angina, coronary spasm has no relation with exercise but is the cause of myocardial ischemia. On the contrary, the pain is sometimes relieved with exercise. The pain usually comes while resting or sleeping. The electrocardiogram may show ST elevation. The pain is relieved by glycerine trinitrate. Specialist investigations using provocative tests

<table>
<thead>
<tr>
<th>Class</th>
<th>Canadian Cardiovascular Society*</th>
<th>New York Heart Association</th>
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<tbody>
<tr>
<td>I</td>
<td>Angina occurs with strenuous exercise</td>
<td>Patient has cardiac disease, no symptoms</td>
</tr>
<tr>
<td>II</td>
<td>Angina occurs on climbing stairs rapidly</td>
<td>Patient has cardiac disease, no symptoms at rest but occur on ordinary activities</td>
</tr>
<tr>
<td>III</td>
<td>Angina occurs on climbing one flight of stairs</td>
<td>Patient has cardiac disease, no symptoms at rest but occur on less ordinary activities</td>
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<tr>
<td>IV</td>
<td>Symptoms present at rest, inability to do ordinary activity comfortably</td>
<td>Patient has cardiac disease, symptom at rest, inability to perform significant activity</td>
</tr>
</tbody>
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* From Campeau.17

TABLE 7.1. Grading of angina by the Canadian Cardiovascular Society and the New York Heart Association.1
(e.g., hyperventilation, cold-pressor testing, or ergometrine challenge) may be required to make the diagnosis. Ventricular arrhythmia and heart block may occur. Calcium channel blockers (CCBs) may prevent coronary spasm, but beta-blockers may worsen the symptoms. However, patients may remain symptom-free for months or years. Like unstable angina, there is a subsequent risk of heart attack.

**Q: Who should be referred for investigations for angina?**

Referral is required for the following situations:

- All newly diagnosed cases of angina, for an objective assessment of myocardial ischemia.
- For diagnostic or prognostic reasons or where a positive diagnosis would have major implications for the patient’s livelihood.
- For treatment for patients of any age with severe, unstable, or rapidly progressive symptoms, for patients with secondary angina from remediable cause, or for patients with unacceptable symptoms despite adequate medical treatment.

Urgent referral, depending on the severity of symptoms, is required for the following situations:

- Patients with recent-onset angina or stable angina but with severe symptoms, or with a previous history of heart attack or heart failure.
- Patients who suffer angina pain at rest, a sudden increase in severity or frequency of angina, or recent onset of angina not responding to medical treatment.

**Q: What investigations are advocated for a patient suspected of having angina?**

Clinical history and examination are very important. The speed with which the investigations are undertaken depends on the urgency, taking into account the age of the patient, the presence of other risk factors, the severity of chest pain, the likelihood of the diagnosis, and the findings on clinical examination. Chest x-ray helps to exclude congestive heart failure, valvular lesions, pericardial disease, or aortic dissection/aneurysm. However, the use of routine chest x-ray is not well established.

Blood tests should include tests for anemia, thyroid function, urea, electrolytes, lipid profile, glucose and cardiac enzymes, troponin, erythrocyte sedimentation rate (ESR) and high-sensitivity C-reactive protein (hsCRP). Resting electrocardiogram (ECG) is
indicated in all cases as an initial test, although a normal ECG does not exclude CAD. Evidence of a prior Q-wave on ECG and evidence of left ventricular hypertrophy also increases the probability of angina. If the resting ECG is normal, depending on the likelihood of the diagnosis, treadmill stress testing is arranged. The treadmill test is most valuable when the pretest probability is intermediate, for example, when a 50-year-old man has atypical angina and the probability of CAD is about 50%.\textsuperscript{2} When the probability of CAD is high, a positive test result only confirms the high probability of disease, and a negative test result may not decrease the probability of CAD enough to make a clinical difference. When the probability of CAD is very low, a negative test result only confirms the low probability of disease; a positive test may not increase the probability of disease enough to make a clinical difference.\textsuperscript{2} If there is high suspicion of CAD, then exercise testing could be combined with thallium testing (myocardial perfusion imaging [MPI]) or angiography.

Echocardiography is usually not indicated in most patients, unless valvular disease or hypertrophic cardiomyopathy is suspected. Coronary angiography is very important in the diagnosis where myocardial ischemia is suspected and noninvasive testing is contraindicated. The recommendations are for cardiac stress imaging as the initial test in the following situations\textsuperscript{2}:

1. Exercise ECG for the diagnosis of obstructive CAD
2. Exercise myocardial perfusion imaging or exercise echocardiography when the patient has intermediate pretest probability of CAD or has either Wolff-Parkinson-White syndrome or more than 1 mm of rest ST depression
3. Exercise MPI or exercise echocardiography if there is a past history of revascularization
4. If a patient is not able to exercise, then adenosine or dipyridamol MPI or dobutamine echocardiography is indicated if patient has either an intermediate pretest probability of CAD or a past history of revascularization.

When the probability of severe angina is low, noninvasive tests are most appropriate. However, when the pretest probability is high, direct referral for coronary angiography is a suitable choice. Coronary angiography is most useful in the following situations (also, see Chapter 5):

1. If a patient suspected of CAD survived sudden cardiac death
2. Uncertain diagnosis after noninvasive tests
3. If the patient is unable to undergo noninvasive tests.
4. Patients in whom coronary artery spasm is suspected and provocative tests may be necessary.
5. Patients with a pretest probability of left main stem or three-vessel disease.
6. Occupational requirement for a firm diagnosis.
7. To exclude anatomical anomalies in young patients as the cause of angina.

**PROGNOSIS AND TREATMENT**

**Q: What are the tests for risk assessment and prognosis in a patient with chronic stable angina?**

The following factors are associated with worse prognosis:

- One of the strongest and most consistent prognostic markers is the maximum exercise capacity. Poor exercise tolerance, either due to myocardial ischemia or left ventricular dysfunction, is a bad marker.
- Another important prognostic marker is related to exercise-induced ischemia. ST-segment depression and elevation (in leads without pathological Q waves and not in aVR) best summarize the prognostic information related to ischemia.\(^2\)
- Left ventricular dysfunction or ejection fraction is less than 40%.
- Exercise stress test shows ST segment depression >2mm, poor exercise tolerance, or a fall of blood pressure.
- Myocardial perfusion test shows thallium uptake in the lungs.
- Angiography shows left main-stem involvement or multivessel disease, especially in the presence of left ventricular systolic dysfunction.
- Chest x-ray shows cardiomegaly, left ventricular aneurysm, or pulmonary venous congestion.
- A family history of myocardial infarction (MI) and diabetes is an independent predictor of death for coronary heart disease (CHD).
- Increasing age.
- Male gender, but after menopause females have a similar risk.

Stress imaging tests such as the radionuclide MPI test or two-dimensional echocardiography at rest and during stress are valuable for the purpose of risk stratification and planning the best route of management. A normal thallium scan is highly indicative of a benign prognosis even in patients with known CHD. Coronary angiography is usually not indicated unless other measures show
high risk. Stress echocardiography is also able to provide additional prognostic information.

**Q: What are the drugs of choice for the treatment of angina?**
The aim of treatment is to decrease the frequency of anginal attack, control symptoms, prevent MI, and prolong survival. Treatment of angina can be considered under the following headings:

- Managing risk factors (e.g., smoking cessation; treating hypertension, diabetes and elevated LDL-C; weight reduction in all obese patients, and consuming a Mediterranean-type/cardioprotective/TLC diet).
- Relief of symptoms with medical treatment.
- Coronary revascularization.
- Rehabilitation.

In stable angina, aspirin (75 mg daily) reduces the risk of acute MI (AMI) and sudden death by 34%. Similarly, 325 mg aspirin reduced mortality and morbidity by 20% in the International Study of Infarct Survival (ISIS-2). If a patient is allergic to aspirin, clopidogrel is an alternative.

Four groups of drugs are used in the prevention and treatment of angina: nitrates, beta-blockers, CCBs, and potassium channel activators. Nitrates, CCBs, and potassium channel activators are vasodilators. There is no direct evidence that any of these agents has a significant effect on the incidence of sudden death or heart attack. There is little evidence for the intrinsic superiority of one group of drugs over another in terms of symptom relief. It is, however, possible that the combination of two antianginal drugs is more efficient than using one. There is also the added advantage of reduced side effects. Precaution should be taken when discontinuing these drugs. They should be slowly tapered off unless serious side effects are experienced. Rapid-acting nitrates relieve acute angina and may also be used prophylactically in situations likely to induce an attack. Beta-blockers and long-acting nitrates can be used for long-term management of chronic stable angina.

**NITRATES**
Glycerine trinitrate (GTN) is a short-acting drug, whereas glycerine mononitrate and glycerine dinitrate are longer-acting. Nitrates cause coronary dilatation, which improves myocardial circulation, and venodilatation, which reduces venous return to the heart, thus reducing the cardiac workload. The latter is the predominant effect. Nitrates thus reduce myocardial ischemia, relieve pain, and
improve exercise tolerance. In patients with exertional stable angina, long-acting nitrates improve exercise tolerance, increase the time to onset of angina, and decrease ST-segment depression during exercise tolerance. However, there is no evidence that they reduce mortality in a patient with chronic stable angina. Headache is the most troublesome symptom. Hypotension, especially postural hypotension, may occur. This tends to become less frequent after a few days. Nitrates are available as sublingual tablets or spray, oral modified-release preparations, transdermal patches, or 2% ointment of glycerine trinitrate for topical use. Skin reactions can occur with local applications, caused by the drug or its vehicle.

Glycerine trinitrate taken as sublingual tablets in the dose of 300 or 500µg, or sublingual spray in the dose of 0.3 to 1.0mg relieves chest pain in 10 seconds, and the effect lasts for 20–30 minutes. Oral tablets lose effective in 12 weeks; therefore, if they these are used infrequently, sublingual spray is preferred. Isosorbide dinitrate can also be taken sublingually as a 5mg tablet or 1.25mg spray, the effect lasting up to 1 hour. Sustained-release preparations of isosorbide mononitrate and dinitrate can be taken once a day. Isosorbide mononitrate is used in the initial dose of 20 mg two or three times a day, or 40mg twice a day (10mg twice a day for those who have not received nitrates previously), typically 120mg/d for extended-release preparations. Isosorbide dinitrate is used in the dose of 5 to 20mg sublingually, and 30 to 120mg orally, in divided doses; maintenance dose 40 or 80mg, 8–12 hours. If nitrates fail to control symptoms, an alternative antianginal drug should be used.

**BETA-BLOCKERS**

A randomized controlled trial in chronic stable angina has shown that treatment with beta-blockers is efficacious in reducing symptoms of angina and episodes of ischemia, and improving exercise capacity. Beta-blockers are the drug of choice in angina and they may be used alone or in combination with nitrates. The long-term trials show that there is 23% reduction in the odds of death among MI survivors randomized to beta-blockers. All beta-blockers are equally effective in angina. The heart rate should be reduced to 55 to 60 beats per minute. In more severe cases, however, heart rate should be reduced to 50 per minute, provided there are no symptoms arising from bradycardia, and heart block does not occur. Beta-blockers restrict the heart rate during exercise. Patients with angina should not increase heart rate above 100 per minute during exercise. Metoprolol, atenolol, and propranolol are quite effective in symptomatic relief. Carvedilol, bisoprolol, and metoprolol slow-
release are as effective and may also be better tolerated in patients with peripheral vascular disease (PVD) and those with left ventricular dysfunction. These drugs are described in Chapter 4.

**CALCIUM-CHANNEL BLOCKERS (CCBs)**

Dihydropyridines (e.g., nifedipine and amlodipine), which are potent vasodilators, relieve myocardial ischemia by venodilatation (reduces myocardial demand), arterial vasodilatation (reduces resistance against the left ventricular contraction), and coronary dilatation (increases myocardial oxygen supply). Nondihydropyridines (e.g., diltiazem, verapamil) are also vasodilators, though not as potent, but they also exert their effect by depressing the myocardium, thus reducing myocardial demand. Nondihydropyridines are preferred over dihydropyridines in the absence of heart failure, as a second-line agent when there are contraindications to the use of beta-blockers, or if the patient is intolerant. They also have modest prognostic benefits. Short-acting CCBs are associated with increased incidence of MI and mortality; therefore, long-acting preparations (including slow-release) should be used. Verapamil should be avoided in Wolff-Parkinson-White syndrome and left ventricular dysfunction. Verapamil reduces cardiac events and angina post-MI. Diltiazem is used as 60–120 mg tid; slow-release 90–180 mg bid. Verapamil is used as 80 mg tid, increasing to 120 mg tid.

**POTASSIUM-CHANNEL ACTIVATORS**

Nicorandil is the first potassium-channel activator indicated for angina. Potassium (K\(^+\)) channels conduct ions in and out of the cell and act like switches regulating cellular excitability. Nicorandil lowers arterial resistance and reduces cardiac afterload by opening K\(^+\) channels. It also dilates venous capacitance vessels and reduces cardiac preload. In patients with coronary artery disease, it dilates both stenotic and nonstenotic coronary arteries and improves coronary blood flow. It may also prevent coronary spasm. Unlike nitrates, tolerance is not a problem.

Nicorandil is licensed in the U.K. for the prevention and long-term treatment of angina. It is used in the dose of 10 to 20 mg twice a day with maximum of 30 mg twice a day. It is effective as monotherapy but can be used with other agents. It is not proven whether it affects the risk of death or heart attack in patients with coronary artery disease. Nicorandil should not be used in patients with left ventricular failure with low filling pressure. Sildenafil and oral hypoglycemic agents (e.g., glibenclamide) interacts with nicorandil and should not be co-prescribed.
Class I (i.e., American College of Cardiology (ACC)/American Heart Association [AHA] class I measures show evidence, or there is general agreement, that a given procedure or treatment is useful and effective):2,9:

- Aspirin and lipid-lowering drugs reduce MI and death in anginal patient.
- Beta-blockers as initial therapy, whether or not the patient previously suffered MI, though for the former the evidence is stronger. Beta-blockers reduce mortality post-MI. However, their use in other cases is based on their capacity to reduce mortality in hypertension.
- CCBs and/or long-acting nitrates as initial therapy when beta-blockers are contraindicated, not successful, or the patient suffered side effects.
- CCBs and/or long-acting nitrates in combination with beta-blockers when initial therapy with beta-blocker is not successful.
- Sublingual nitroglycerin for immediate relief for symptoms.
- Lipid-lowering therapy in patients with proven or suspected CAD and LDL-C >130mg/dL (3.4mmol/L) with a target LDL-C of <100mg/dL (2.6mmol/L).
- Angiotensin-converting enzyme (ACE) inhibitors in patients with CAD, who has diabetes and/or LVSD.

Class IIa (ACC/AHA class IIa measures show evidence or opinion in favor of usefulness/efficacy):2,9:

- Clopidogrel when aspirin is contraindicated.
- Long-acting nondihydropyridine CCBs instead of beta-blockers as initial therapy.
- Lipid-lowering therapy in patients with proved or suspected CAD (see Chapter 2).
- ACE inhibitors in all patients with significant CAD or other vascular disease.
- Surgical laser transmyocardial revascularization (TMR) as an alternative therapy for chronic stable angina in patients refractory to medical therapy who are not candidates for percutaneous interventions or revascularization.

**Q: How should one proceed with the choice of antianginal drugs?**

- First step: glycerine trinitrate.
- Second step: add beta-blocker especially if previous history of MI; if contraindicated, add CCBs.
- Third step: add CCBs; if contraindicated, add long-acting nitrate.
If a patient suffers pain at rest and nocturnal pain, suggesting vasospasm, initiate therapy with long-acting nitrates and CCBs. All patients should have sublingual spray for prophylaxis before exercise and for symptomatic relief of symptoms. A cardioselective beta-blocker is the first choice, except in patients with ventricular arrhythmia, for whom sotalol is preferred. For thyrotoxic patients propranolol may be the drug of choice. In patients for whom a beta-blocker is contraindicated and in those who suffer from coronary artery spasm, hypertension, diabetes, PVD, asthma, or Raynaud’s phenomenon, CCBs should be tried. Diltiazem and verapamil are equally effective. Compliance with amlodipine is better. Nifedipine, amlodipine, and felodipine are all effective. If a patient is unable to take beta-blockers and CCBs, then long-acting nitrates should be considered. If one drug is ineffective, another drug should be substituted; if necessary, combination therapy should be used. Triple therapy is of doubtful benefit. Nevertheless, at least two and preferably all three classes of drugs should be tried before giving up on medical treatment. Combined with beta-blockers or CCBs, nitrates produce greater antianginal and antiischemic effects in patients with stable angina. Use of ACE inhibitors and lipid-lowering drugs should be considered.

Q: What are the indications of angioplasty?

Percutaneous transluminal coronary angioplasty (PTCA) was originally introduced as a balloon angioplasty, a procedure that involved using a catheter-borne balloon that was inflated at the site of coronary stenosis. The scope of this procedure has widened to include the use of stents, atherectomy and laser therapy. The following are the important indications for angioplasty:

- Stable angina in patients with suitable coronary anatomy who are uncontrolled on or intolerant of medical treatment.
- Two- or three-vessel disease with significant proximal left anterior descending artery disease, in patients who have normal anatomy suitable for catheter-based therapy, normal left ventricular function, and who do not have treated diabetes.
- Unstable angina not responding to medical treatment.
- Unstable angina or AMI followed by a positive exercise test.
- AMI complicated by cardiogenic shock.
- AMI where thrombolytic drugs are contraindicated.
- One- or two-vessel disease without a significant disease of proximal left anterior descending artery but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
Disadvantages
Angioplasty has similar mortality to coronary artery bypass grafting (CABG), with 1% mortality for the treatment of single-vessel disease, and 2% when more than one vessel is dilated. About 3% of people need an emergency heart bypass due to damage to the coronary artery during angioplasty. Almost one third of patients experience restenosis within 3 months and again require angioplasty. Of those who have angioplasty on more than one vessel, almost half have restenosis of one or more vessels. Almost one fifth of patients who have angioplasty require heart bypass within 3 years.

STENTS
A coronary stent (scaffold) is an artificial support device in the coronary artery to keep the vessel open. It was developed to overcome the two primary limitations of balloon angioplasty: sudden closure of the coronary artery and late restenosis. Stents prevent narrowing of the coronary artery by providing a scaffolding lattice to tack back the inner surface of the coronary artery. It prevents late restenosis by mechanically enforced remodeling and resetting of the vessel size of the stented segment. In 20% to 40%, restenosis gradually occurs.

There are several different coronary stents available, and the scaffolding lattice of each stent differs markedly in configuration. Coronary artery stenting is currently applicable only to relatively large arteries (>3 mm diameter). The stents can be categorized into the mesh stents, characterized by strong and extensive scaffolding of the vessel wall (Wallstent, Palmaz-Schatz, and AVE Micro) and the coil stents, characterized by a low metallic surface area and predominantly transverse strut orientation (Gianturco-Roubin, Wiktor, Multilink, and Cordis). Drug-eluting stents (DESs) are now used and have been shown to dramatically reduce the risk of restenosis compared with bare metal stents. In the Sirolimus-Eluting Coronary Stent (SIRUS) Trial, 1058 patients undergoing elective coronary stent implantation were randomized to a bare stent or the sirolimus DES. The patients were followed-up for a year. The sirolimus stent reduced the restenosis rate by 75%, from 36.3% to 8.9%, and reduced the rate of repeat revascularization from 28.4% to 13%. No significant advantage was found with regard to mortality and MI.

Coronary stenting usually follows balloon angioplasty, which requires inserting a guide catheter at the ostium of the coronary artery through the femoral artery. The guide wire is then manipulated beyond the lesion, after which the balloon catheter is inserted over it. When this catheter is positioned at the site of blockage, it
is slowly inflated to widen the coronary artery and is then removed. The stent-mounted catheter is then threaded into the artery. When this is correctly positioned in the coronary artery, the balloon is inflated, expanding the stent against the wall of coronary artery. The balloon catheter, guide wire, and guide catheter are then removed, leaving the stent. A cardiac angiography follows to ensure that the stent is keeping the artery open. Aspirin is taken for few days before the procedure in the dose of 300mg a day. There is a small risk that the stented artery may close. Thrombosis, bleeding, and artery damage are rare complications.

**Q: Do revascularization procedures have a better outcome than medical treatment?**

There is no significant advantage of PTCA over medical treatment. The Randomized Intervention Treatment of Angina (RITA-2) trial compared the effectiveness of angioplasty with medical treatment in patients with one-vessel or two-vessel disease or mild to moderate angina, deemed suitable for either treatment. More patients who received angioplasty died or had an MI than did patients who received medical treatment. Angioplasty also led to a greater rate of nonfatal heart attack. No difference existed between the groups for mortality, 19% of patients who received angioplasty or heart bypass compared with 23% who received medical treatment. At 3 months, angina and exercise tolerance were more improved in the angioplasty group, an effect that attenuated in 1 or 2 years. A study of one-vessel disease and mild angina also has shown that medical treatment is only slightly less efficacious in relieving symptoms and improving exercise tolerance than angioplasty, while the prognosis was comparable in both groups.

In another randomized trial the outcomes of medical treatment, angioplasty, and heart bypass were compared in a patient with stable angina. Mortality and MI rates do not differ for medical therapy and angioplasty in low-risk patients with single-vessel disease. In high-risk patients with multivessel disease, mortality is lower at 5, 7 and 10 years in patients who receive bypass surgery rather than medical therapy. Angioplasty and bypass surgery produce similar reduced rates of mortality and MI, but the need for repeat revascularization is more common after angioplasty. In the presence of heart failure, medical treatment with ACE inhibitors, beta-blockers, and spironolactone is probably superior for most patients.

These studies suggest that medical treatment should initially be given in cases of mild chronic angina. If this fails, revascularization should be considered. (Primary coronary angioplasty is described in Chapter 8.)
Q: What are the indications for coronary artery bypass grafting?

Coronary artery bypass grafting (CABG) is now an established success; some 25,000 operations are performed each year in the U.K., with an operative mortality now approaching 1% in most centers. New minimally invasive operative procedures that do not require cardiopulmonary bypass may extend its use to patients who are at greater risk of surgery. Arterial grafts can be used not only from the internal mammary artery but also from the right gastroepiploic artery, inferior epigastric artery, and radial artery. Arterial grafts have many advantages over saphenous vein grafts. They have a reduced propensity to develop atherosclerosis. Reviews of patients treated with internal mammary artery grafting of left anterior descending artery have shown improved long-term survival, a lower long-term incidence of angina, and higher graft patency rates. Fourteen percent of coronary bypass operations are now reoperations. Repeat grafting is associated with higher operative risk (3%). Symptom-free patients, without complication, can be expected to return to work in 4 to 8 weeks.

The important indications for CABG are as follows:

- Patients with significant left main artery disease.
- One- or two-vessel disease without significant proximal left anterior descending artery disease but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
- Patients with three-vessel disease; the surgical benefit is better with left ventricular dysfunction (ejection <50%).
- Patients with two-vessel disease with significant proximal left anterior descending artery disease and left ventricular dysfunction (ejection <50%).
- Patients with one- or two-vessel artery disease without proximal left anterior descending artery disease who survived sudden cardiac death or sustained ventricular tachycardia.
- Patients who have not been successfully treated by medical therapy.

Q: How do the results of CABG compare with those of angioplasty?

Some patients are suitable for either procedure (CABG or PCTA). Overall there is no evidence of a major difference between the two over 3 to 5 years in the risk of death or heart attack. Occasionally, it is difficult to dilate all stenosed segments of the coronary artery at a single attempt of angioplasty, and restenosis occurs within 6 months in about one third of patients. Therefore, patients initially treated with angioplasty needed more repeat procedures to restore blood circulation (30–50%) than did bypass patients (5–10%).
Both procedures relieve angina in most patients, but overall bypass was slightly more effective at least for the first few years. Thus in the RITA-1 trial, 11% of the CABG group compared with 32% of the angioplasty group had angina 6 months after the procedure; after 2 years the comparable figures were 21% and 32%.\textsuperscript{14} Patients undergoing angioplasty required more medications. However, with regard to physical activity, exercise tolerance, employment status, and quality of life, there was no significant difference. The two United States trials of PTCA versus CABG groups have shown that early and late survival rates have been equivalent for both groups.\textsuperscript{15,16} In the Bypass Angioplasty Revascularization Investigation (BARI) Trial, the subgroup of patients with treated diabetes (with multiple severe lesions) had significantly better survival rates with CABG. In the Emory Angioplasty versus Surgery Trial (EAST), diabetics had equivalent survival rates with both procedures.

Patients who wish to avoid major operation can choose angioplasty provided they understand the higher risk of recurrent angina, with a one in three risk of additional procedures during the next few years. Those who prefer a more certain medium-term result may choose bypass, but with no greater overall risk of mortality or major morbidity. Patients with single-vessel disease also do not have a prognostic benefit with CABG.

Q: What are the newer revascular techniques and their advantages?
The following are some of the newer techniques:

**Coronary Atherectomy Devices**
Since PTCA does not remove the plaque but acts by splitting and shifting the plaque and stretching the coronary artery, this led to the development of new devices that remove the plaque and also cause fewer traumas to the deeper components of the arterial wall. Atherectomy devices include directional, rotational atherectomy, transluminal extraction, and excimer laser angioplasty.

The directional coronary atherectomy is a nonballoon interventional device. It is a cutting device, as it cuts the plaque and leaves smooth lumen. It is a suitable procedure when lesions are ostial, eccentric, or present at bifurcations. Its contraindications include small vessel size, calcified lesions, lesion angulation, and proximal tortuosity of the vessel.

Rotational atherectomy ablates plaque material. Percutaneous rotational atherectomy uses a high-speed metal burr coated with diamond clips to abrade and destroy plaques into fine microparticles. This technique is suitable for harder calcified, fibrotic lesions.
It is contraindicated in patients with left ventricular dysfunction and in those with visible thrombus.

Transluminal extraction atherectomy (TEC) increases luminal size by cutting material and aspirating it. The system comprises a conical cutting head with two stainless steel blades bound to the distal end of a hollow flexible torque tube. A suction bottle, which collects the excised lesions, and a battery-powered motor drive unit are attached to the proximal end of the tube. The TEC is useful in lesions in which thrombus or debris has to be removed from the artery.

**Coronary Laser Angioplasty**

Coronary laser angioplasty involves using excimer laser systems. It is a pulsed laser, that is, the energy is released in short bursts of ultraviolet light separated by relatively long periods of silence, during which laser emission is switched off. This procedure is indicated in saphenous vein graft lesions, long lesions, ostial lesions, and total occlusions. Its contraindications include bifurcation lesions, highly eccentric lesions, severe lesions, angulation, vessel tortuosity, and prior dissection.

**Transmyocardial Laser Revascularization (TMR)**

Transmyocardial laser revascularization with the aid of a laser makes small channels into the myocardium, which lead to improved exercise tolerance. The Atlantic Study showed that TMR improved exercise tolerance in intractable angina on maximum medical treatment. The procedure is performed in the operating theater (with carbon dioxide or holmium: yttrium-aluminum-garnet [YAG] laser) or by a percutaneous approach. Although this technique gives symptomatic improvement in chronic stable angina, no definite benefits have been shown in terms of increasing myocardium perfusion.

**Spinal Cord Stimulation**

This method involves accurate placement of the stimulating electrode in the dorsal epidural space, usually at the C7-T1 level. This method is proposed for patients with chronic stable angina refractory to medical, catheter intervention, and surgical therapy.

**References**