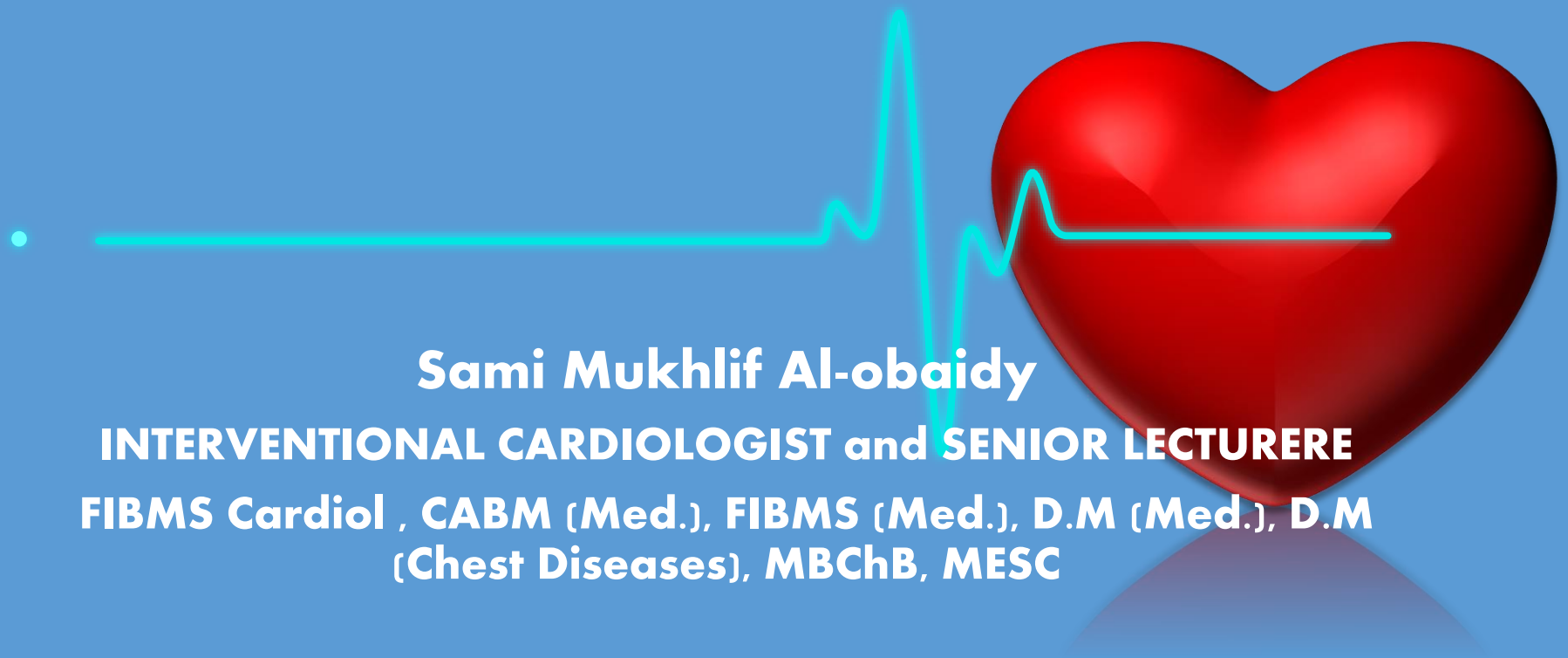
CORONARY ARTERY DISEASE
ANGINA PECTORIS



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Angina pectoris is a symptom complex caused by transient myocardial ischaemia, which occurs whenever there is an imbalance between myocardial oxygen supply and demand.

16.38 Factors influencing myocardial oxygen supply and demand

Oxygen demand: cardiac work

- Heart rate
- Blood pressure
- Myocardial contractility
- Left ventricular hypertrophy
- Valve disease

Oxygen supply: coronary blood flow*

- Duration of diastole
- Coronary perfusion pressure (aortic diastolic minus coronary sinus or right atrial diastolic pressure)
- Coronary vasomotor tone
- Oxygenation:
  - Haemoglobin
  - Oxygen saturation

*Coronary blood flow occurs mainly in diastole.

Pathogenesis

Atherosclerosis is by far the most common cause of angina pectoris. Angina may also occur in aortic valve disease and hypertrophic cardiomyopathy, and when the coronary arteries are involved with vasculitis or aortitis. Approximately 10% of patients who report stable angina on effort have normal coronary arteries on angiography.

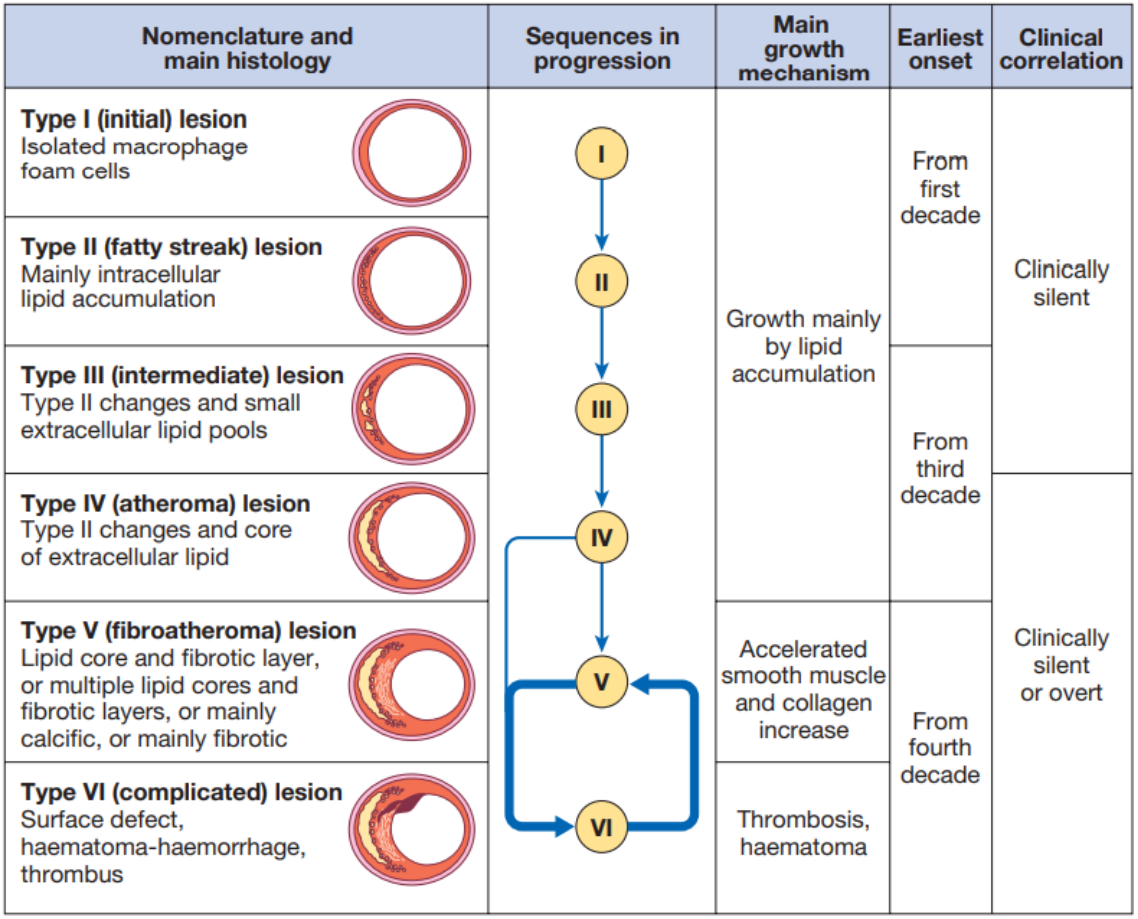

Fig. 16.54 The six stages of atherosclerosis. American Heart Association classification. *From Stary HC, Chandler B, Dinsmore RE et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. Circulation 1995; 92:1355–1374. © 1995 American Heart Association.*

RISK FACTORS FOR ATHEROSCLEROSIS???

16.37 Population-based strategies to prevent coronary disease

- Do not smoke
- Take regular exercise (minimum of 20 mins, three times per week)
- Maintain an 'ideal' body weight
- Eat a mixed diet rich in fresh fruit and vegetables
- Aim to get no more than 10% of energy intake from saturated fat

Coronary artery spasm

Angina may result from vasospasm of the coronary arteries. This may coexist with atherosclerosis, especially in unstable angina.

May occur as an isolated phenomenon in less than 1% of cases, in patients with normal coronary arteries on angiography.

This is sometimes known as **variant angina**; when it is accompanied by transient ST elevation on the ECG, it is termed **Prinzmetal's angina**.

Syndrome X

The constellation of typical angina on effort, objective evidence of myocardial ischaemia on stress testing, and normal coronary arteries on angiography is sometimes known as syndrome X.

Many of these patients are women and the mechanism of their symptoms is often unclear. This disorder is poorly understood; it carries a good prognosis but may respond to anti-anginal therapy.

Other causes

Angina can occur in association with aortic stenosis, HOCM and aortitis. It may also rarely be found in association with vasculitis.

Clinical Features:

The history is the most important factor in making the diagnosis .

Stable angina is characterised by central chest pain, discomfort or breathlessness that is predictably precipitated by exertion or other forms of stress and is promptly relieved by rest

Some patients find the discomfort comes when they start walking and that later it does not return despite greater effort ('warm-up angina').

Physical examination is frequently unremarkable but should include a careful search for evidence of valve disease (particularly aortic), important risk factors (hypertension, diabetes mellitus), left ventricular dysfunction (cardiomegaly, gallop rhythm), other manifestations of arterial disease (carotid bruits, peripheral arterial disease), and unrelated conditions that may exacerbate angina (anaemia, thyrotoxicosis).

16.39 Activities precipitating angina

Common

- Physical exertion
- Cold exposure
- Heavy meals
- Intense emotion

Uncommon

- Vivid dreams (nocturnal angina)
- Lying flat (decubitus angina)

16.40 Canadian Cardiovascular Society (CCS) angina score

Class I

- Angina only during strenuous or prolonged physical activity

Class II

- Slight limitation, with angina only during vigorous physical activity

Class III

- Moderate limitation where symptoms occur with everyday activities

Class IV

- Inability to perform any activity without angina or angina at rest, i.e. severe limitation

16.41 Risk stratification in stable angina*

High risk	Low risk
Post-infarct angina	Predictable exertional angina
Poor effort tolerance	Good effort tolerance
Ischaemia at low workload	Ischaemia only at high workload
Left main or three-vessel disease	Single-vessel or two-vessel disease
Poor left ventricular function	Good left ventricular function

*Patients may fall between these two categories.

APPROACH

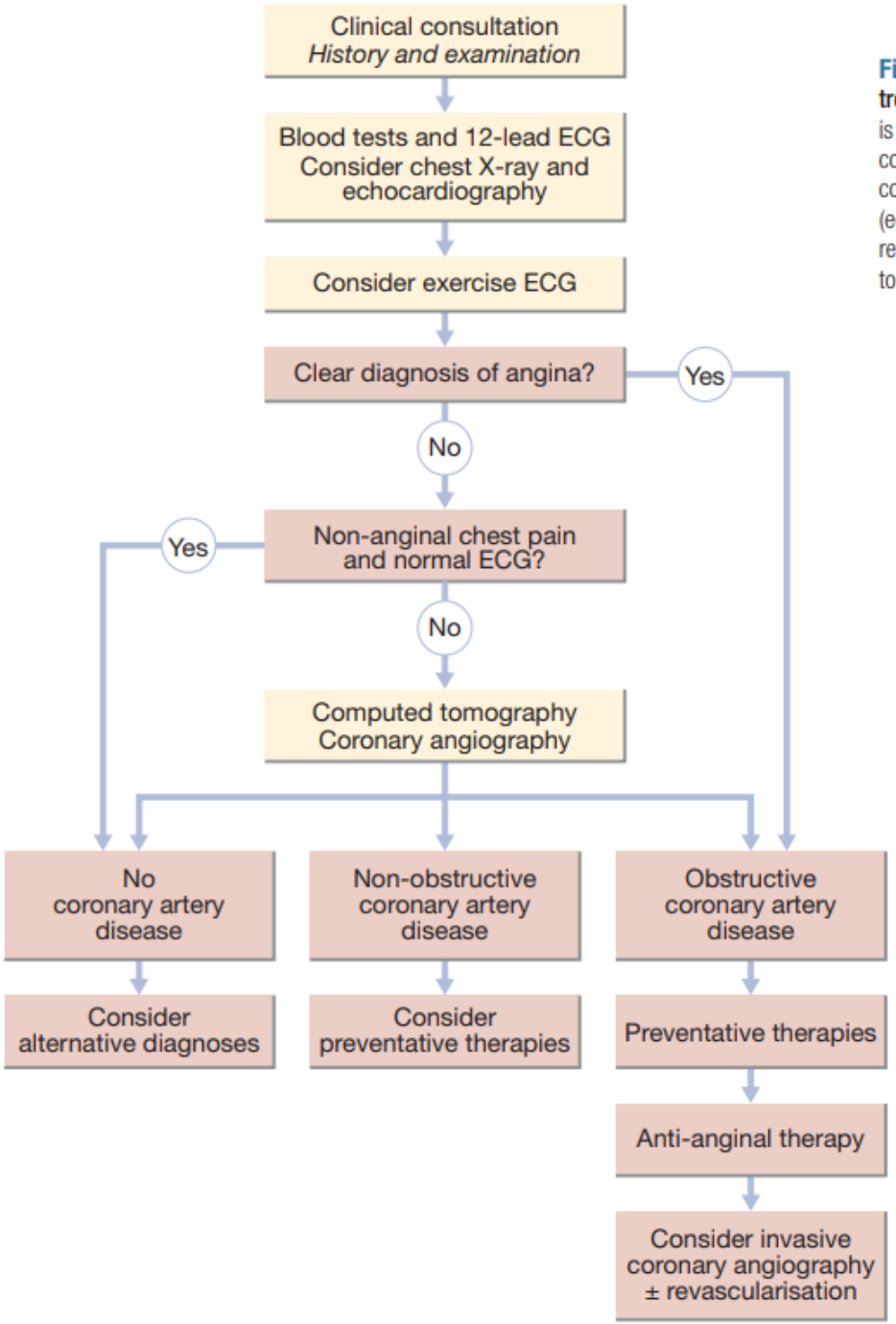

Fig. 16.55 A scheme for the investigation and treatment of stable angina on effort. This scheme is best adopted for patients without prior known coronary artery disease. For patients with known coronary artery disease, further stress imaging (echocardiography, radionuclide perfusion or magnetic resonance perfusion) rather than computed tomography coronary angiography is recommended.

MANAGEMENT

The principles of management involve:
• a careful assessment of the extent and severity of arterial disease
• identification and treatment of risk factors
• advice on smoking cessation
• introduction of drug treatment for symptom control
• identification of high-risk patients for treatment to improve life expectancy

All patients with angina secondary to CAD should receive **antiplatelet therapy**. Low-dose (75 mg) aspirin should be prescribed for all patients and continued indefinitely since it reduces the risk of MI. Clopidogrel (75 mg daily) is an equally effective alternative if aspirin causes dyspepsia or other side effects.

All patients should be prescribed a **statin**, even if cholesterol is normal.

16.42 Advice to patients with stable angina

- Do not smoke
- Aim for an ideal body weight
- Take regular exercise (exercise up to, but not beyond, the point of chest discomfort is beneficial and may promote collateral vessels)
- Avoid severe unaccustomed exertion, and vigorous exercise after a heavy meal or in very cold weather
- Take sublingual nitrate before undertaking exertion that may induce angina

Anti-anginal drug therapy

The goal of anti-anginal therapy is to control symptoms using a regimen that is as simple as possible and does not cause side effects.

Five groups of drug can be used in the prevention and treatment of angina but there is little evidence that one group is more effective than another. It is conventional to start therapy with sublingual glyceryl trinitrate (GTN) and a β-blocker, and then add a calcium channel antagonist or a long-acting nitrate if needed.

If the combination of two drugs fails to achieve an acceptable symptomatic response, revascularisation should be considered

Nitrates

Nitrates act directly on vascular smooth muscle to produce venous and arteriolar dilatation.

They help angina by lowering preload and afterload, which reduces myocardial oxygen demand, and by increasing myocardial oxygen supply through coronary vasodilatation.

Sublingual GTN, administered from a metered-dose aerosol (400 μg per spray) or as a tablet (300 or 500 μg), is indicated for acute attacks and will usually relieve an attack in 2–3 minutes.

Patients should also be encouraged to use the drug prophylactically before taking exercise that is liable to provoke symptoms.

Sublingual GTN has a short duration of action and side-effects include headache, symptomatic hypotension and, rarely, syncope.

Headache is common with oral nitrates but tends to diminish if the patient perseveres with the treatment.

Continuous nitrate therapy can cause pharmacological tolerance but this can be avoided by a 6–8-hour nitrate-free period, best achieved at night when the patient is inactive.

If nocturnal angina is a predominant symptom, long-acting nitrates can be given at the end of the day.

Beta-blockers

These lower myocardial oxygen demand by reducing heart rate, BP and myocardial contractility, but they may provoke bronchospasm in patients with asthma

In theory, non-selective β-blockers may aggravate coronary vasospasm by blocking coronary artery β2-adrenoceptors, and so a once-daily cardioselective preparation such as slow-release metoprolol (50–200 mg daily) or bisoprolol (5–15 mg daily) is preferable.

Beta-blockers should not be withdrawn abruptly, as rebound effects may precipitate dangerous arrhythmias, worsening angina or MI. This is known as the β-blocker withdrawal syndrome

Calcium channel antagonists

These drugs lower myocardial oxygen demand by reducing BP and myocardial contractility.

Since dihydropyridine calcium antagonists, such as nifedipine and amlodipine, may cause a reflex tachycardia, it is best to use them in combination with a β-blocker.

In contrast, verapamil and diltiazem can be used as monotherapy because they slow SA node firing, inhibit conduction through the AV node and tend to cause bradycardia.

They are particularly useful when β-blockers are contraindicated.

Calcium channel antagonists reduce myocardial contractility and must be used with care in patients with poor LV function, since they can aggravate or precipitate heart failure.

Other unwanted effects include peripheral oedema, flushing, headache and dizziness

Potassium channel activators

Nicorandil (10–30 mg twice daily orally) is the only drug in this class that is currently available for clinical use. It acts as a vasodilator with effects on the arterial and venous systems, and has the advantage that it does not exhibit the tolerance seen with nitrates.

If channel antagonist

Ivabradine is the first of this class of drug. It induces bradycardia by modulating ion channels in the sinus node. It does not inhibit myocardial contractility and appears to be safe in patients with heart failure.

Ranolazine

This drug inhibits the late inward sodium current in coronary artery smooth muscle cells, with a secondary effect on calcium flux and vascular tone, reducing angina symptoms.

Non-pharmacological treatments

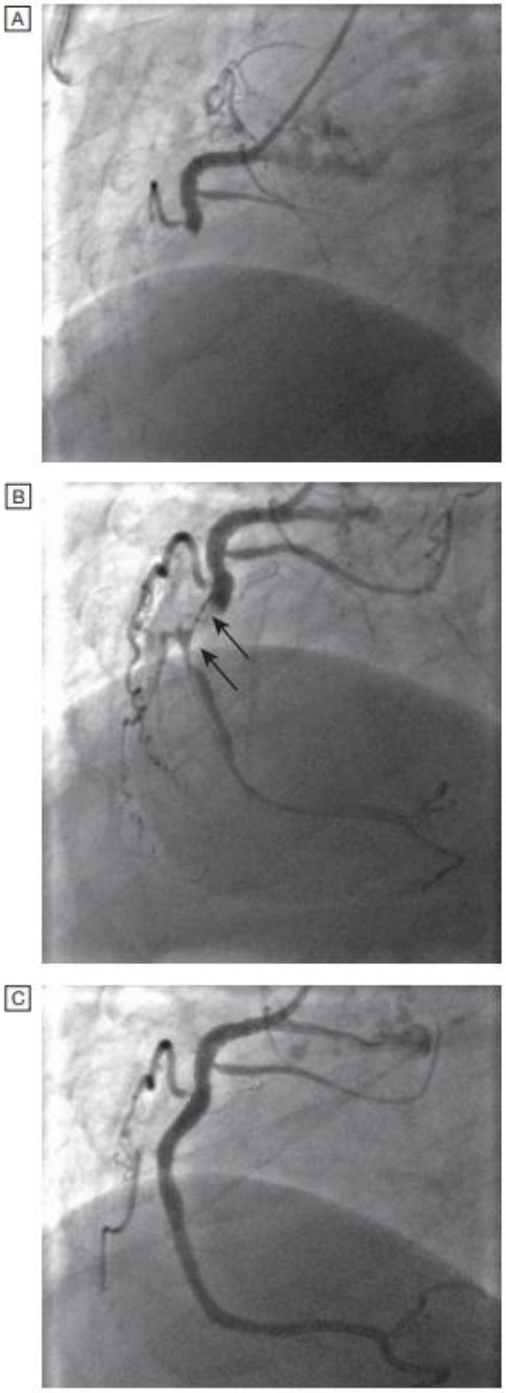

Fig. 16.60 Primary percutaneous coronary intervention. A Acute right coronary artery occlusion. B Initial angioplasty demonstrates a large thrombus filling defect (arrows). C Complete restoration of normal flow following intracoronary stenting.

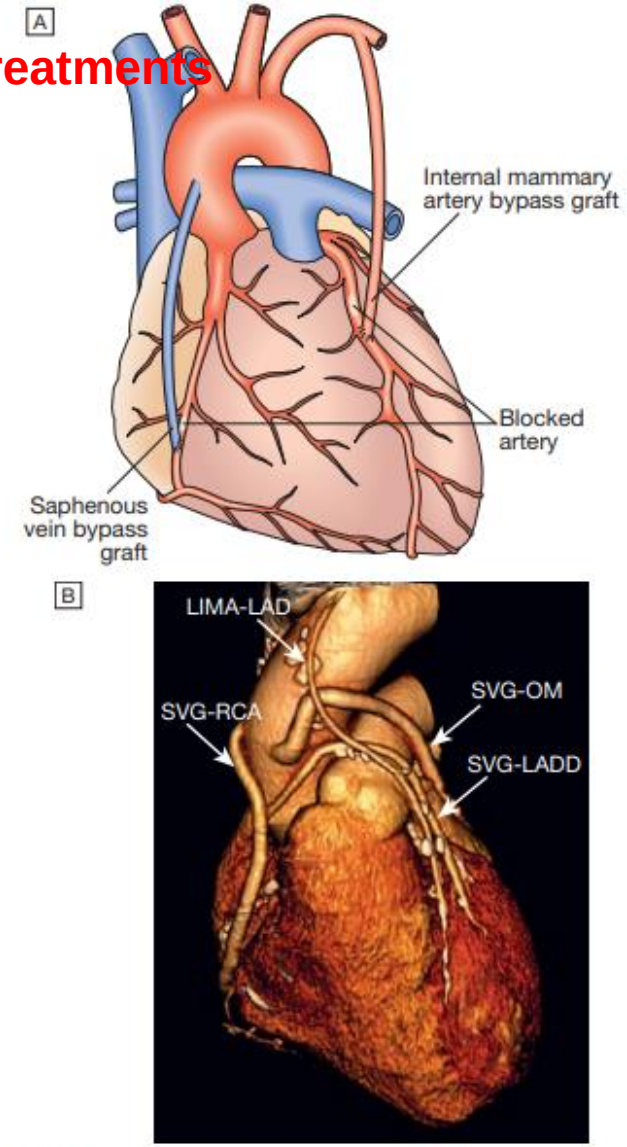

Fig. 16.61 Coronary artery bypass graft surgery. A Narrowed or stenosed arteries are bypassed using saphenous vein grafts connected to the aorta or by utilising the internal mammary artery. B Three-dimensional reconstruction of multidetector CT of the heart. The image shows the patent saphenous vein grafts (SVG) to the right coronary artery (RCA), obtuse marginal branch (OM) and diagonal branch (LADD), and left internal mammary artery graft (LIMA) to the left anterior descending (LAD) coronary artery.