

# Ischaemic heart disease: stable angina

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## Abstract

Stable angina is a clinical syndrome reflecting inadequate myocardial perfusion. This is typically, but not always, caused by atherosclerotic coronary artery disease. A detailed history is important to establish the diagnosis, presence of risk factors and unstable symptoms. Stress ECG or stress imaging techniques should ideally be used to determine the presence and extent of myocardial ischaemia. The gold standard test to detect coronary disease remains invasive coronary angiography. Appropriate drug therapy significantly improves symptoms and prognosis, and should be started in all patients. Risk stratification should be performed by clinical evaluation, assessment of the presence and extent of myocardial ischaemia, quantification of left ventricular function, and coronary angiography where appropriate. Revascularization improves symptoms in the majority of patients, and improves prognosis in those with a high ischaemic burden. The choice of revascularization method (percutaneous coronary intervention or coronary artery bypass graft) is influenced by the extent and complexity of disease; presence of diabetes mellitus; comorbidities that increase surgical risk; ability to take dual antiplatelet therapy; and patient preference.

**Keywords** Coronary artery bypass graft (CABG); coronary disease; ischaemia; medical therapy; PCI; revascularization; stable angina

Stable angina is a clinical syndrome characterized by discomfort in the chest, jaw, back or arms, typically associated with exertion or anxiety, and relieved by rest or glyceryl trinitrate (nitroglycerin). It is usually caused by obstructive coronary artery disease (CAD).

## Pathophysiology

Angina reflects transient regional myocardial ischaemia caused by inadequate coronary perfusion. The most common cause is atherosclerotic CAD (Figure 1). Other causes include aortic stenosis and hypertrophic cardiomyopathy (HCM). Conditions such

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## What's new?

- It is likely that the dominance of ischaemia over coronary anatomy as a determinant of prognosis will lead to more emphasis on both patient-level and lesion-level ischaemia to direct revascularization
- Already studies such as FAME, FAME-2 and RIPCORD (ref Curzen et al. *Circulation: Cardiovascular Interventions* 2014 in press) have demonstrated how much influence pressure wire can have in management strategy and clinical outcome
- The ability to detect both anatomy and ischaemia is being investigated using CT angiography with a computer model for FFR; if it proved possible, a non-invasive approach would revolutionize our assessment and management of patients
- Revascularization — with recent improvements in equipment and technique, percutaneous coronary intervention has been shown to be as safe and effective as coronary artery bypass graft for treatment of all but the most complex left main stem and multi-vessel CAD, although there is a greater need for subsequent revascularization due to re-stenosis (SYNTAX trial<sup>1</sup>). Whether PCI is equal to CABG for LMS disease remains unanswered pending ongoing trial results

as poorly controlled hypertension, anaemia or thyrotoxicosis can also precipitate or exacerbate angina.

## Epidemiology

### Prevalence

This increases with age for men (4–7% of those aged 45–64 to 12–14% aged 65–84) and women (5–7% of those aged 45–64 to 10–12% aged 65–84).<sup>2</sup> Estimates suggest there are 1,200,000 men and 900,000 women with angina in the UK.<sup>3</sup>

### Incidence

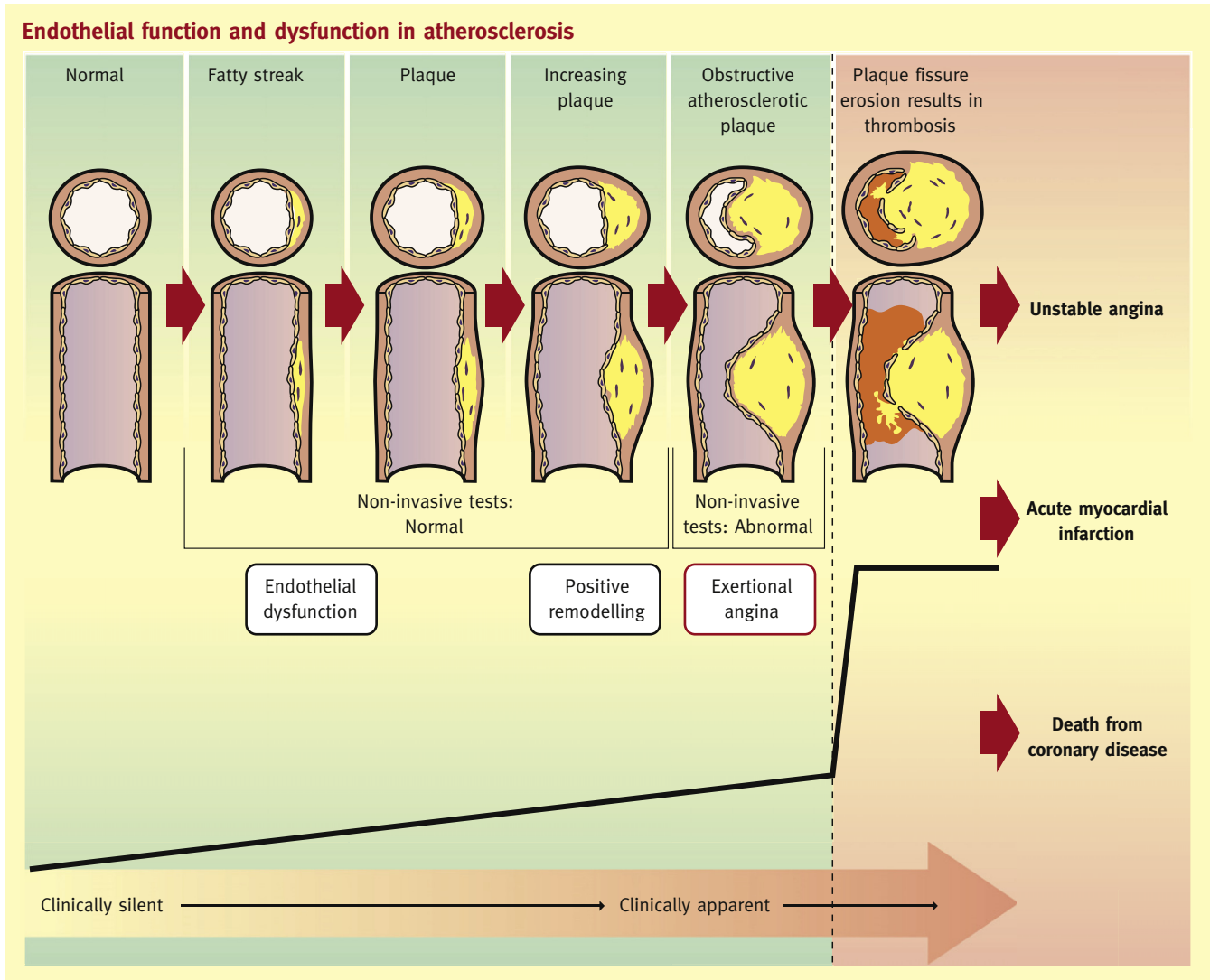
The annual incidence of angina pectoris in men in Western populations is approximately 1%.<sup>2</sup> The incidence, of course, varies with age as shown in Table 1.

### Natural history and prognosis

Advances in pharmacology and revascularization have led to a progressive decline in death and non-fatal myocardial infarction for patients with stable angina. Contemporary data in patients receiving appropriate treatment suggest annual mortality and non-fatal MI rates of 0.6–1.4% and 0.6–2.7%, respectively.<sup>2</sup> However, in patients with a history of MI and significant comorbidities such as diabetes mellitus and peripheral vascular disease, the annual incidence of death reaches 3.8%.<sup>2</sup>

## Diagnosis and assessment

The diagnosis of angina involves clinical assessment, laboratory tests and cardiac investigations. A suggested algorithm is shown in Figure 2.



**Figure 1** Typical progression of coronary atherosclerosis. As plaque burden increases, this initially occurs external to the lumen, preserving luminal diameter; this is known as the Glagov effect, or positive remodelling. Eventually however, plaque encroaches into the lumen, resulting in haemodynamic obstruction and angina. Additionally, disordered endothelial vasomotor function is common and results in diminished vasodilatation, or even vasoconstriction in response to stimuli such as exercise. In acute coronary syndromes, vulnerable plaque is a more important factor than the degree of stenosis; acute coronary syndromes result from ulceration or erosion of the fibrous cap, with subsequent intraluminal thrombosis. (This figure has been adapted from Greenland et al.<sup>4</sup> and Abram.<sup>25</sup>)

### Clinical

Careful history taking is essential, and key to the diagnosis in most cases. Anginal pain is usually described as pressure, tightness, heaviness or burning across the chest and may radiate

to the jaw, arms or back. It is typically brought on by exertion, cold weather or emotional stress, but can also occur after a heavy meal or first thing in the morning. Its duration is brief and the sensation generally resolves with rest or sublingual nitrates. Atypical presentations are more common in women (localized or inframammary pain), the elderly (exertional dyspnoea) and patients with diabetes (dyspnoea or 'silent' ischaemia). The severity of symptoms can be formally graded (Table 2). It is important to distinguish patients with 'unstable' angina or other acute coronary syndromes who may present with:

- recent onset of severe limiting angina
- rapidly worsening symptoms in previously stable patients or
- rest pain.

Risk factors such as smoking, diabetes, hypertension, dyslipidaemia or family history should also be determined.

Physical examination may be normal, or reveal the presence of obesity, hypertension, valvular heart disease, cardiomyopathy, heart failure or peripheral vascular disease.

### Incidence of angina in the UK by gender and age. Rates per 100,000 person years<sup>3</sup>

Age group (years)	Men	Women
0–44	3.9	1.7
45–54	60.5	33.3
55–64	156.3	78.6
65–74	215.4	136.6
75+	136.7	99.9
All	48.7	28.0

**Table 1**

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