

# Ischaemic heart disease: stable angina

Lavinia Gabara

Percy Jokhi

Nick Curzen

## 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. A range of tests is available to investigate patients with stable angina. Anatomical tests, include CT coronary angiography and invasive coronary angiography, aim to assess the presence and extent of atheroma in the coronary arteries. Functional tests, including stress echocardiography, stress MRI and nuclear perfusion, aim to detect the presence and extent of reversible myocardial ischaemia. The gold standard test to detect coronary disease remains invasive coronary angiography with the addition of fractional flow reserve to assess the significance of stenosis. Appropriate drug therapy significantly improves symptoms and prognosis. Risk stratification requires 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 most patients with stable angina, 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 co-morbidities, surgical risk, bleeding risk and patient preference.

**Keywords** Coronary artery bypass graft (CABG); coronary disease; fractional flow reserve; ischaemia; medical therapy; MRCP; percutaneous coronary intervention; revascularization; stable angina

## Introduction

### Angina is a clinical syndrome

- characterized by discomfort (pain, tightness, heaviness, pressure) in the front of the chest or the neck, shoulders, jaw or arms

**Lavinia Gabara** MRCP is a Fellow in Cardiology at University Hospital Southampton NHS Foundation Trust, UK. Competing interests: none declared.

**Percy Jokhi** MB BChir, PhD, MRCP (UK) is a Consultant Interventional Cardiologist at Lincoln County Hospital, United Lincolnshire Hospitals NHS Trust, UK. Competing interests: none declared.

**Nick Curzen** BM(Hons) PhD FRCP is a Professor of Interventional Cardiology at University Hospital Southampton NHS Foundation Trust & Faculty of Medicine, University of Southampton, UK. Competing interests: none declared.

## Key points

- A detailed history is paramount to establish the likelihood of coronary disease and whether symptoms are compatible with angina and patients with unstable symptoms should be identified at the outset and managed accordingly
- Anatomical tests (CT coronary angiogram, invasive coronary angiogram) and physiological tests for ischaemia/viability (stress echocardiography, stress MRI, nuclear stress tests, fractional flow reserve) are complementary
- The extent and location of coronary disease on angiography (if performed) should be assessed alongside the presence and extent of myocardial ischaemia to determine whether revascularization is appropriate
- Optimal medical therapy, ideally with aspirin, statin,  $\beta$ -adrenoceptor blockade and angiotensin-converting enzyme inhibition, should be considered in all cases, with additional anti-anginal drugs as required for symptom control
- The choice of revascularization method (percutaneous coronary intervention, coronary artery bypass grafting) is determined by the extent and complexity of disease, presence of diabetes, co-morbidities that increase surgical risk, ability to take dual antiplatelet therapy and patient preference

- precipitated by physical exertion
- relieved by rest or glyceryl trinitrate (GTN) within about 5 minutes.

The presence of all three characteristics defines typical angina. If only two features are present, the syndrome is defined as atypical angina. If one or none of the above features are elicited in the history, it is most likely non-anginal chest pain.<sup>1,2</sup>

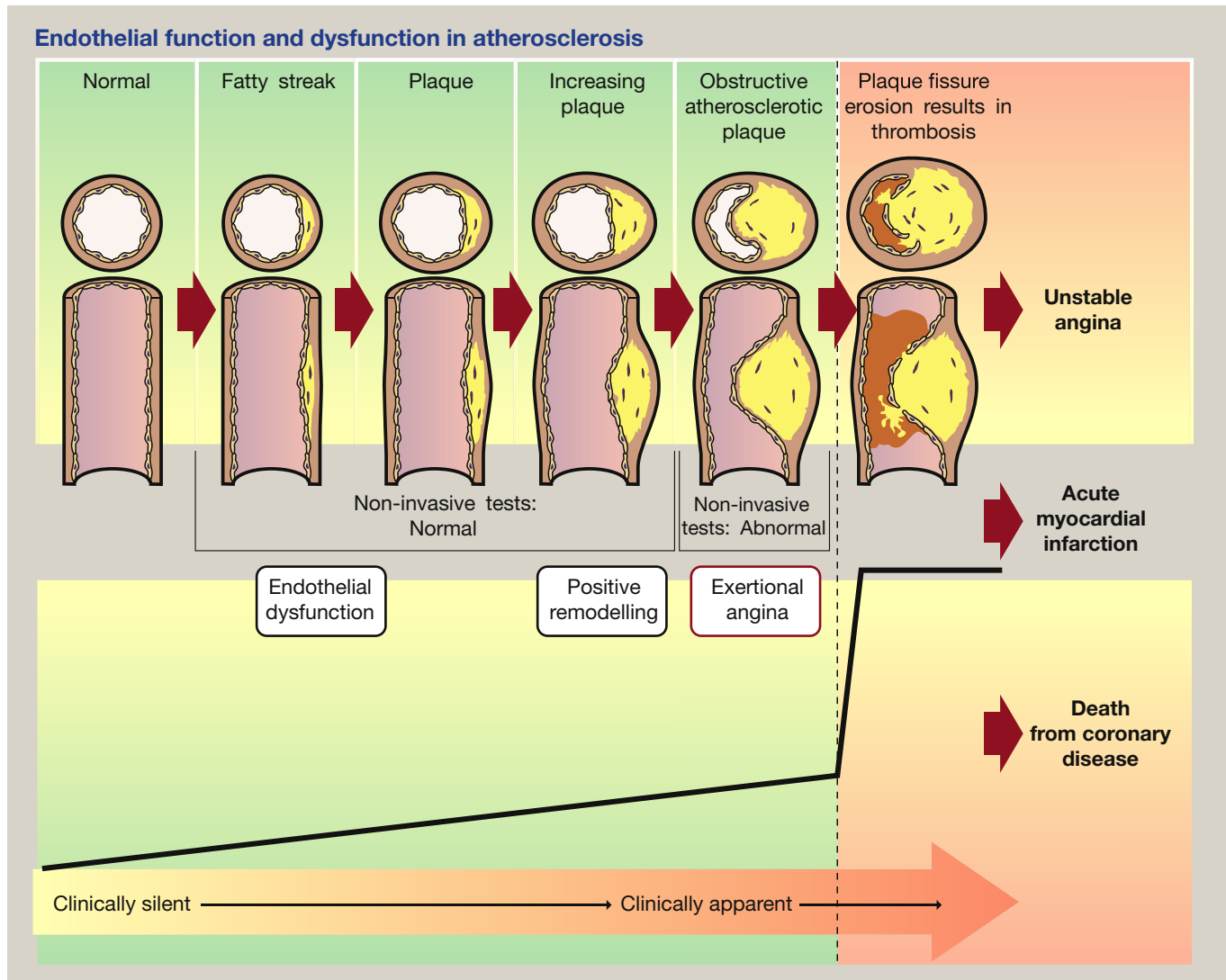
Chronic stable angina implies that symptoms have been unchanged in frequency and severity for at least 2 months. It is usually caused by obstructive coronary artery disease (CAD).

## Pathophysiology

Angina reflects transient regional myocardial ischaemia caused by inadequate coronary perfusion to meet the metabolic demands of the myocardium. The most common cause is atherosclerotic CAD (Figure 1). Others include aortic stenosis and hypertrophic cardiomyopathy. Conditions such as poorly controlled hypertension, anaemia, tachyarrhythmia or thyrotoxicosis can also cause or worsen angina.

## Epidemiology

**Prevalence:** this increases with age for men (from 0.05% at <45 years to 16.96% at >75 years) and women (0.02% <45 years to 11.15% >75 years). Interestingly, angina is more prevalent in middle-aged women than men, probably reflecting microvascular CAD.<sup>2</sup> Estimates suggest that >1.3 million people in the UK are living with angina.



**Figure 1** Typical progression of coronary atherosclerosis. Increasing plaque burden initially occurs external to the lumen, preserving luminal diameter; this is known as positive remodelling, or the Glagov effect. Eventually, however, plaque encroaches into the lumen, resulting in haemodynamic obstruction and angina. Disordered endothelial vasomotor function is also 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. Source: Adapted from: Greenland P, Gidding SS, Tracy RP. Commentary: lifelong prevention of atherosclerosis: the critical importance of major risk factor exposures. *Int J Epidemiol* 2002; 31: 1129–34 and Abrams J. Clinical practice. Chronic stable angina. *N Engl J Med* 2005; 352: 2524–33.

**Incidence:** the annual incidence of angina pectoris in Western populations is approximately 1% in 45–65-year-old men and slightly higher in women in this age group. It rises steeply with age, reaching almost 4% in both sexes at 75–84 years of age.<sup>2</sup>

**Natural history and prognosis:** advances in pharmacology and revascularization, as well as public health prevention strategies including smoking reduction, have led to a progressive decline in death and non-fatal myocardial infarction (MI) in individuals with stable angina. Contemporary data in patients given appropriate treatment suggest annual mortality and rate of non-fatal MI to be 0.6–1.4% and 0.6–2.7%, respectively. However, in patients with a history of MI and significant co-morbidities such as diabetes mellitus and peripheral vascular disease, the annual incidence of death reaches 3.8%.<sup>2</sup> In the UK in 2015, >69,000

deaths were attributed to CAD: >11% of the total annual mortality.<sup>3</sup>

### Diagnosis and assessment

The diagnosis of angina involves clinical assessment, laboratory tests and cardiac investigations. Figure 2 shows the European Society of Cardiology suggested algorithm.

### Clinical assessment

Careful history-taking is mostly essential for diagnosis. Anginal pain is usually described as pressure, tightness, heaviness or burning across the chest that can 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

Download English Version:

<https://daneshyari.com/en/article/8952298>

Download Persian Version:

<https://daneshyari.com/article/8952298>

[Daneshyari.com](https://daneshyari.com)