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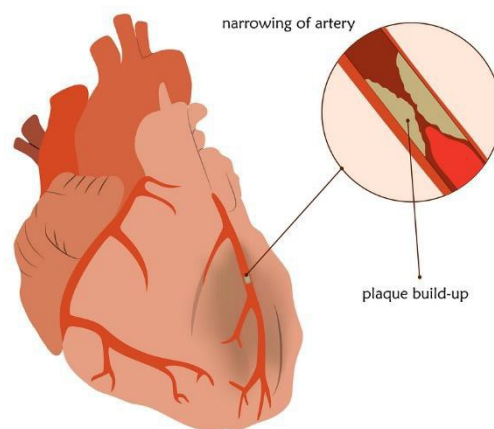
Please note that links within this page may take you directly to the British Heart Foundation website, where you can find person-centred information on heart conditions and related terms.

Definition

Coronary heart disease (CHD – also referred to as coronary artery disease or ischaemic heart disease) is a type of cardiovascular disease (CVD) where oxygenated blood is unable to perfuse the myocardium (muscle tissue of the heart) due to narrowing of the coronary arteries.

Most commonly, this narrowing of the coronary arteries is a result of atherosclerosis.¹

Coronary artery disease



Atherosclerosis is a condition where plaques form within the lining of the arteries. These plaques are formed of white blood cells, lipids (cholesterol and fatty acids), calcium and fibrous connective tissue. This is collectively known as atheroma. Atheromatous plaques cause the lumen of the arteries to narrow and blood flow to become restricted.

Sometimes atheromatous plaques in the coronary arteries can rupture, causing a blood clot to form (coronary thrombus). If this happens, the blood flow in that artery may be partially or completely blocked.²

Healthy artery



Plaque formation



Plaque rupture



There are three main presentations of CHD: angina, myocardial infarction and **heart failure**. We will not be covering heart failure on this page, as it will be covered separately.

Angina

Angina is chest pain caused by an insufficient blood supply to the myocardium. Pain may also be experienced in the neck, shoulders, jaw or arms.^{2,3}

Although commonly a symptom of CHD, angina is less commonly a result of a **heart valve disease** (for example, aortic stenosis – narrowing of the aortic valve opening), **hypertrophic cardiomyopathy**, atrial fibrillation, anaemia or hypertensive heart disease (raised pressure inside the heart).² The British Heart Foundation (BHF) also describes **microvascular angina** and **coronary artery spasm**.

Angina is classified as either the more common stable angina (angina pectoris) or more serious unstable angina.⁴ The National Institute of Health and Care Excellence (NICE) clinical knowledge summary (CKS) **Angina** states the following:

‘Stable angina usually occurs predictably with physical exertion or emotional stress, lasts for no more than ten minutes (usually less) and is relieved within minutes of rest, as well as sublingual nitrates.

Unstable angina is new onset angina or abrupt deterioration in previously stable angina, often occurring at rest. Unstable angina usually requires immediate admission or referral to hospital.’²

Myocardial infarction

Myocardial infarction (MI) is often referred to as a heart attack. It is defined as the death (necrosis) of an area of myocardium due to an inadequate supply of oxygenated blood.

MI is usually caused by atheromatous plaque rupturing and forming a thrombus, as discussed under atherosclerosis (although it should be noted that a coronary thrombosis does not always cause an MI).

The size of the thrombus and extent to which it affects blood flow affects whether it leads to no necrosis, a relatively small amount of necrosis, or a more extensive area of necrosis. Collectively, this range of effects is called acute coronary syndrome (ACS).⁵

Acute coronary syndrome (ACS)

NICE describes the sub-divisions of ACS as follows:

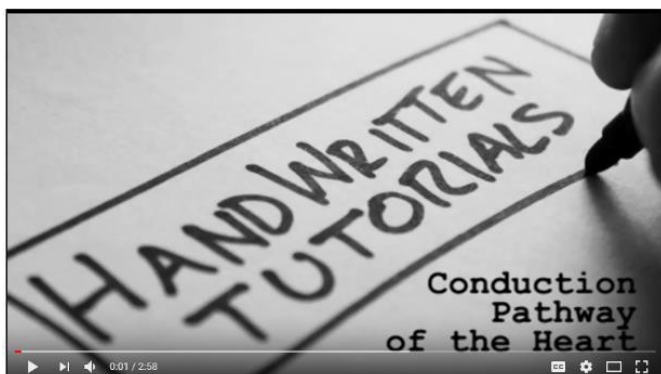
- ST-elevation ACS (STE-ACS). This is typically caused by a complete blockage of an artery. ST relates to the changes observed on an electrocardiogram (ECG); in this case, there is persistent elevation of the ST wave. Most people with this type of ASC develop an ST-elevation MI (STEMI).
- Non-ST-elevation ACS (NSTEMI-ACS). This typically reflects a partial or intermittent blockage of an artery. Here the ECG does not show persistent ST-segment elevation, although there may be other ECG changes or no changes.

NSTEMI-ACS is further divided into:

- non-ST-elevation MI (NSTEMI): there is a rise in the **blood troponin** level
- unstable angina: blood troponin level does not rise.⁵

ECGs show the electrical activity of the heart. To understand what this means, it is important to understand the conduction pathways of the heart. If you would like to learn more about this, then watch the following video by Handwritten Tutorials:

Handwritten Tutorials – Conduction pathway of the heart



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Prevalence and incidence

The BHF publishes data on mortality, morbidity, treatment and costs associated with cardiovascular disease. They state that 'CHD is one of the UK's leading causes of death' and 'it is also the leading cause of death worldwide'.⁶

The BHF states that in England in 2015, there were 56,493 deaths due to CHD. Of these, 34,103 were men and 22,390 were women. The total number of deaths is similar to those attributable to dementia and Alzheimer's (58,299), but less than those attributable to cancer (138,509) and respiratory disease (70,111).⁷

For further information, visit the BHF's **Heart statistics publications** page.

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Signs and symptoms

Stable angina

The NHS outlines the following as symptoms of angina:

- *'feels tight, dull or heavy – it may spread to your left arm, neck, jaw or back*
- *is triggered by physical exertion or stress*
- *stops within a few minutes of resting.*⁴

ACS

The NHS outlines the following as symptoms of a heart attack, which differ considerably from the symptoms listed for angina:

- *'chest pain – a sensation of pressure, tightness or squeezing in the centre of your chest*
- *pain in other parts of the body – it can feel as if the pain is travelling from your chest to your arms (usually the left arm is affected, but it can affect both arms), jaw, neck, back and abdomen*
- *feeling lightheaded or dizzy*
- *sweating*
- *shortness of breath*

- *feeling sick (nausea) or being sick (vomiting)*
- *an overwhelming sense of anxiety (similar to having a panic attack)*
- *coughing or wheezing.*⁸

The NICE CKS **Chest pain** states to suspect ACS if:

- *'Pain in the chest or other areas (for example the arms, back, or jaw) lasts longer than 15 minutes.*
- *Chest pain is:*
 - *associated with nausea and vomiting, sweating or breathlessness, or a combination of these.*
 - *associated with haemodynamic instability (for example the person has a systolic blood pressure less than 90 mmHg).*
 - *of a new-onset, or is the result of an abrupt deterioration of stable angina; with pain occurring frequently with little or no exertion, and often lasting longer than 15 minutes.*
- *Do not use the person's response to glyceryl trinitrate to confirm or exclude a diagnosis of acute coronary syndrome.'*

Note that generally the term **haemodynamic instability** is used to describe abnormal or unstable blood pressure.

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Causes/risk factors

Risk factors for CHD due to atheromatous plaques correlate with many of the risk factors for the overall development of CVD. They can be classified as modifiable or non-modifiable.

Modifiable risk factors

- Low blood level of high-density lipoprotein cholesterol (HDL-C)
- High blood level of non-HDL-C
- **Hypertension**
- **Diabetes**
- Smoking
- Being overweight/obese
- Inactivity
- Unhealthy diet
- Excessive alcohol consumption
- Excessive stress^{9,10,11}

For more information about modifiable risk factors, access BHF, **Healthy hearts – Risk factors for coronary heart disease** and NICE CKS, **CVD risk assessment and management**.

NHS Health Check is a scheme that *'aims to promote and improve the early identification and management of individual behavioural and physiological risk factors for vascular disease and the other associated conditions'*.¹² For more information about NHS Health Checks, access CPPE's **Health checks** gateway page.

For more information about the prevention of CVD, access NICE public health guideline, **Cardiovascular disease prevention [PH25]**.

Non-modifiable risk factors

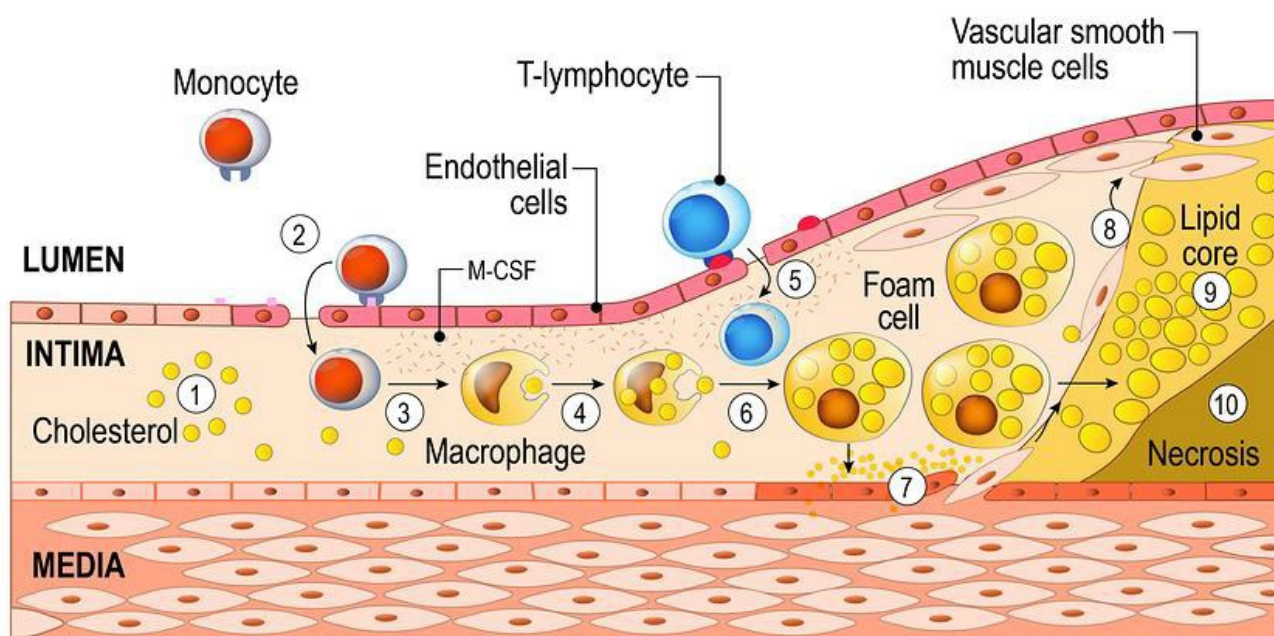
- Increasing age
- South Asian ethnic background and those with an African Caribbean background are at greater risk of hypertension
- Family history of heart disease (family history of hypertension or hypercholesterolaemia increases the risk of developing these risk factors)^{9,10,11}

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Pathophysiology (mechanism of disease)

As discussed in the above definition section, CHD is very often due to atherosclerosis and narrowing of the coronary arteries.

Atherosclerosis is thought to be driven by a chronic inflammatory process within the arterial wall.¹³ The diagram below shows a simplified outline of the way in which atherosclerosis is believed to occur. Each step is numbered, and the numbers correspond to the descriptions below.



Step 1 – it is thought that atherosclerosis starts when low-density lipoproteins (LDL), a type of **cholesterol**, accumulate in the **intima** layer of the coronary artery wall, under the layer of **endothelial cells** (endothelium) on its inner surface.¹⁴

Step 2 – it is thought that an oxidised form of LDL (ox-LDL) plays an important role in the formation of atheromatous plaques by increasing inflammation.¹⁵ The ox-LDL ‘activates’ the endothelial cells causing **monocytes** (a type of white blood cell) to adhere (stick) to them.^{14,15}

Step 3 – the monocytes (which can differentiate, or change into, other types of cells when responding to stimuli) differentiate into **macrophages** (type of white blood cell).

Step 4 – the macrophages start to take up the cholesterol.¹⁶

Step 5 – **T-lymphocytes** (a type of white blood cell) enter the intima. Their role in atherosclerosis is not fully understood, but it is thought that these cells can modulate the type and intensity of the inflammatory response, and may increase the activity of the macrophages.¹⁷

Step 6 – due to the uptake of cholesterol, the macrophages differentiate into **foam cells**. These foam cells are too large to pass back out of the intima through the endothelial cells. They stay in the intima and stabilise the chronic inflammation.¹⁴ The accumulation of foam cells is known as a fatty streak.¹⁸

Steps 7 and 8 – the complex inflammatory process is thought to cause **vascular smooth muscle cells** to migrate from the **media** layer towards the endothelial cells.¹⁹ When these smooth muscle cells reach the endothelial cells, they produce fibrous tissue which forms a protective fibrous cap to try and contain the plaque.²⁰

Step 9 – a **lipid core** made from cholesterol develops. It is thought that this results from a combination of lipids being deposited when foam cells undergo apoptosis (natural cell death), and the aggregation of free lipids within the media.²¹

Step 10 – when cells that have undergone apoptosis are not cleared away properly (due to defective efferocytosis), a **necrosis** occurs and a necrotic core forms.²²

The lipid core and necrosis cause the plaque to swell, and the fibrous cap of the plaque can start to degrade. This can result in the plaque rupturing, at which point platelets stick to the contents of the plaque and form a blood clot.²² Atheromatous plaques that are at risk of rupture tend to have a thin fibrous cap, a high lipid content, few smooth muscle cells, and a high proportion of macrophages and monocytes.²³ This blood clot can block the entire artery, meaning the oxygen-rich blood doesn’t reach the myocardium.

Stable angina

Atheromatous plaques affect the flow of blood through the coronary arteries; they also disrupt the endothelium and affect its ability to fully dilate in response to increased oxygen demand.²⁴ When the demand for oxygenated blood is higher than the supply (without leading to necrosis) – for example, during exercise – this can cause the pain experienced during stable angina.

ACS

If the atheromatous plaque changes acutely, this can trigger ACS. Changes can include endothelial dysfunction, platelet aggregation and spasm leading to plaque erosion, rupture, haemorrhage and thrombosis.²⁵ STEMI is associated with major blockages to coronary arteries and a relatively large amount of damage. NSTEMI is associated with less myocardial damage than STEMI, which may be due to there

being a partial blockage or a smaller vessel being affected. Unstable angina is associated with a partial blocking of an artery.²⁶

When blood flow to the myocardium is reduced or blocked for around 30 to 60 minutes, this can lead to cardiomyocyte (heart muscle cell) damage. This damage leads them to release a protein called **troponin**.

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Prognosis and complications

NICE CKS offers excellent summaries of the prognosis for **angina**, and the prognosis and complications associated with an MI, **MI – secondary prevention – Complications and prognosis**.

A serious complication of CHD is cardiac arrest. For more information about cardiac arrest, visit the BHF's **Cardiac arrest** page.

Post MI, a process called cardiac (or ventricular) remodelling may occur. This can result in left ventricular (LV) dysfunction and heart failure. For more information about remodelling and its prevention, access the following articles:

- The American College of Cardiology Foundation's **Causes and prevention of ventricular remodeling after MI**
- *Heart's Left ventricular dysfunction: causes, natural history, and hopes for reversal.*

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Diagnosis/detection

Stable angina

Stable angina is diagnosed through clinical assessment, recognition of relevant risk factors and potentially an ECG. Those who have experienced chest pain, but do not require a hospital admission, should be referred to a specialist chest pain service to confirm or exclude angina.²⁷ At this point, a diagnostic test such as a computerised tomography (CT) coronary angiogram, non-invasive functional testing or a percutaneous **coronary angiogram** may be performed.²⁸

NICE clinical guidance **Recent-onset of chest pain of suspected cardiac origin: assessment and diagnosis [CG95]** recommends that the typicality of chest pain is assessed as follows:

- *'Presence of three of the features below is defined as typical angina.*
- *Presence of two of the three features below is defined as atypical angina.*
- *Presence of one or none of the features below is defined as non-anginal chest pain.*

Anginal pain is:

- *constricting discomfort in the front of the chest, or in the neck, shoulders, jaw or arms*
- *precipitated by physical exertion*
- *relieved by rest or GTN within about five minutes.'*

The reason for this classification is to help clinicians determine the likelihood of pain being related to CHD; it can also be used to help determine which tests should be undertaken to aid a diagnosis.

ACS

NICE quality standard **Acute coronary syndromes in adults [QS68]** states that '*Acute myocardial infarction can have a poor prognosis so prompt and accurate diagnosis is important to ensure that appropriate treatment and care is offered as soon as possible*'.

ACSs are medical emergencies.²⁹ Both the BHF and NHS advise people who are experiencing the symptoms of a heart attack to immediately dial 999.^{30,31}

Diagnosis of ACS is made by taking a clinical history, conducting an assessment and taking a 12-lead ECG, and can be aided by measuring blood troponin level.²⁸

More information about diagnosis and assessment of those who present with chest pain can be found in NICE guideline **Recent-onset chest pain of suspected cardiac origin: assessment and diagnosis [CG95]**.

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Pharmacological treatment

Stable angina

Read Sections 1.3, *General principles for treating people with stable angina* and 1.4, *Anti-anginal drug treatment* of NICE clinical guidance **Stable angina: management [CG126]**, which outline the antianginal therapies that should be offered, in which order they should be offered, and the additional add-on therapies that are recommended.

Additionally, the Scottish Intercollegiate Guidelines Network (SIGN) national clinical guideline **Management of stable angina [SIGN 151]** provides evidence-based recommendations and best practice guidance on the management of patients with stable angina.

ACS

Initial management

The following NICE guidance outlines recommendations for early treatment options in those presenting with ACS:

- NICE clinical guideline **Acute coronary syndromes [NG185]**.

Ongoing management

Those who experience ACS are usually offered the following therapies.³²⁻³⁴

- angiotensin-converting enzyme inhibitor (ACE inhibitor) to reduce the cardiac remodelling (changes in ventricular size and shape)
- dual antiplatelet therapy (aspirin plus a second antiplatelet agent) to reduce the risk of coronary thrombosis
- beta-blocker to reduce heart rate, limit myocardial oxygen demand and reduce the incidence of arrhythmias and cardiac death³⁵
- high-potency statin to reduce LDL levels.

Read Section 1.4, **Drug therapy for secondary prevention**, which outlines the standard pharmacological therapies and refers to further guidance as necessary.

Age and Ageing's article **Acute coronary syndrome management in older adults: guidelines, temporal changes and challenges** explores the management of older people who have experienced ACS and the available guidance. The *British National Formulary (BNF)* offers a treatment summary for the initial and long-term management of **Acute coronary syndromes**, which also links to the individual monographs for each recommended medicine.

The BHF's **Tests** information booklet offers simple explanations about which tests are commonly offered to help diagnose heart diseases.

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Non-pharmacological treatment

There are several non-pharmacological treatment options which can be offered to those with both stable angina and ACS.

Information and support

As part of long-term management, information and support can be provided. This should include information about a person's condition and the way it may affect their lifestyle. The person should also be encouraged to ask questions about their condition and be offered support to manage it. Information about how to reduce cardiovascular risk should also be offered.^{36,37} Detailed information about the recommended lifestyle advice can be found on the NICE CKS **MI – secondary prevention** page. This information also applies to those who have stable angina.^{36,37}

Cardiac rehabilitation

Cardiac rehabilitation involves exercise and information, and should be offered to all patients who experience ACS. For more information, watch the following BHF video, which can be found on their **Cardiac rehabilitation** page:

British Heart Foundation – Joining a cardiac rehabilitation programme



For a person's perspective of cardiac rehabilitation, access ***Moving forward after a heart attack – Mark's story***.

Coronary angioplasty and stents

Coronary angioplasty, also known as percutaneous coronary intervention (PCI) or percutaneous transluminal coronary angioplasty (PTCA), can be used to improve symptoms of angina or in an emergency situation to open a blocked coronary artery and thus re-perfuse the heart.

Watch the BHF video ***Your guide to angioplasty and stents***, which can be found on their ***Coronary angioplasty and stents*** page, to learn about what happens during an angioplasty and the use of stents.



The location of the stents may be described in terms of which coronary artery they have been placed in. For more information about the anatomy of the coronary arteries, visit the Heart & Vascular Institute's ***Anatomy and function of the coronary arteries*** page. In most circumstances, a drug-eluting stent is used.

The technology for stents is still evolving, and knowing the type of stent used is important for deciding on the duration of dual antiplatelet therapy. Dual antiplatelet therapy should be offered in accordance with the stent's instructions for use (IFU), which are device-specific.³⁸

Coronary bypass surgery

Coronary bypass surgery involves grafting a blood vessel to bypass a blockage in a coronary artery. The graft is referred to as a coronary artery bypass graft (CABG).

Watch the BHF's video *Your guide to coronary bypass surgery, heart disease treatment*, which can be found on their *Coronary bypass surgery* page, to learn about what happens during coronary bypass surgery.



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Patient support

The BHF offers information about specific conditions and support for those with heart conditions. More information can be found on their *Information & support* page.

The NHS has a dedicated *Coronary heart disease* page with information on symptoms, causes, diagnosis, treatment, recovery and prevention.

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Further resources

CPPE's *Coronary heart disease* gateway page provides further learning on stable angina, health checks and links to NICE guidance.

The following *Geriatric Medicine* article looks at the management of angina in the elderly population specifically, *Angina: management in the elderly*.

The European Society of Cardiology's *Antiplatelets therapy: antiplatelet therapy in ischemic heart disease* explores the differences between antiplatelet therapies.

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External websites

CPPE is not responsible for the content of any non-CPPE websites mentioned on this page or for the accuracy of any information to be found there.

All web links were accessed on 11 January 2022.

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Next review due: December 2022