

# CHEST PAIN

# 1 Introduction

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Chest pain is a very common complaint and often creates fear, for sufferers and relatives, of serious heart or lung disease. Accurate diagnosis depends mainly on a careful history of the site and radiation of the pain, its character and duration, together with aggravating and relieving factors, aided by physical examination and special investigations.

Chest pain can be classified as central (including precordial) and lateral, each category being subdivided into acute, usually presenting as an emergency, and chronic – of days' or weeks' duration at presentation. In terms of disability analysis, chronic chest pain is far more common and important, so the causes of this will be described before the acute causes.

For completeness, a brief account will be given first of the causes of superficial chest pain. Ischaemic heart disease (IHD), including myocardial infarction (MI), angina, unstable angina and other variants, is described (with differential diagnoses) in a separate protocol. The differential diagnoses for each condition, apart from IHD, are the others described in the same section.

In most cases of chest pain, especially if chronic, taking a careful history will indicate the likely correct diagnosis. The features of angina and MI that differentiate them from non-cardiac chest pain are described in the IHD protocol.

## 2 Superficial Chest Pain

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Most varieties of superficial chest pain are due to infection and inflammation, and are obvious on examination.

### 2.1 Viral

*Herpes zoster*, causing shingles, involves thoracic nerve roots in at least 50% of cases. Pain and paraesthesiae may occur for a few days before the eruption appears. Vesicles, on an erythematous base, are unilateral and occupy the area of one or more dermatomes. Some patients have fever, malaise and axillary node enlargement in the acute phase. The lesions form scabs and heal in 1-2 weeks, without scarring unless they have become secondarily infected. Particularly in the elderly, post-herpetic neuralgia may occasionally cause severe pain for a long time after the eruption has disappeared, which may cause some functional disability.

### 2.2 Bacterial

Superficial inflammation may have spread from a deeper lesion such as an empyema.

### 2.3 Phlebitis

In *Mondor's disease*, phlebitis of the subcutaneous anterior thoracic veins causes either pleuritic-type pain or pain on raising the arms. The inflamed vein may be palpable as a tender cord. Resolution occurs within 1-2 months.

## 3 Chronic Central Chest Pain

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The most important cause of central chest pain is ischaemic heart disease (IHD): this causes stable angina, unstable angina and myocardial infarction, which are described in a separate protocol. Causes of chronic central chest pain other than stable and unstable angina are discussed below.

### 3.1 Pain Of Oesophageal Origin

#### 3.1.1 Description

Pain from the oesophagus is felt in the midline of the chest with radiation to the jaw, back, shoulders and possibly the inner arms. There may be an inconsistent relationship to exertion – mimicking angina. The pain characteristically wakes the patient in the early hours of the morning.

##### 3.1.1.1 Aetiology

The pain may be due to isolated oesophageal spasm, developing achalasia of the cardia, or hiatus hernia with reflux and oesophagitis (in which heartburn is characteristic – sternal pain radiating upwards from the xiphoid). Acid reflux into the oesophagus may reduce coronary blood flow reserve to the point of inducing true angina (linked angina).

##### 3.1.1.2 Prevalence

Gastro-oesophageal reflux occurs to some degree in everybody, but is only considered a disease when it causes significant symptoms or complications. Idiopathic achalasia has an annual incidence of about 1-2/200,000.

### 3.1.2 Diagnosis

#### 3.1.2.1 Clinical features

The pain is associated with taking food (often only certain foods), temporarily relieved by belching or swallowing saliva, and often worse in postures favouring regurgitation – lying, bending forward or driving a small car, especially with a tight seat belt. The dyspepsia is relieved by antacids.

#### 3.1.2.2 Investigations

- Endoscopy.
- Reproduction of the pain by instillation of dilute hydrochloric acid into the lower oesophagus.
- 24-hour pH monitoring used occasionally.
- Barium swallow – BUT most hiatus herniae are asymptomatic so this finding does not exclude myocardial ischaemia. Trial of omeprazole may be

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diagnostically useful and has practical and economic advantages over sophisticated investigations.

### 3.1.3 Treatment

Antacids, H<sub>2</sub> antagonists, proton pump inhibitors and motility promoters. Confusingly, the pain of oesophageal dysmotility can, like angina, be relieved by nitrates or calcium antagonists.

### 3.1.4 Prognosis

Prognosis depends on the exact nature of the oesophageal disorder.

### 3.1.5 Main Disabling Effects

The disabling effects depend on the efficacy of medical and surgical treatment and should be minimal. However, as with other conditions described in this protocol, disability analysts may see people whose disease has not been optimally managed, or whose condition has not responded to best current therapy – such factors may increase the degree and duration of disability, leading to time off work in some cases.

## 3.2 Other Gastrointestinal Conditions

### 3.2.1 Gastric Distension

Gastric distension (often due to aerophagy) can cause substernal discomfort.

### 3.2.2 Peptic Ulcer

The relationship of peptic ulcer pain to meals, or lack of food, is important.

### 3.2.3 Gallbladder Disease

The pain of gallbladder disease is often related to eating fatty foods. Gallbladder disease and ischaemic heart disease are both common, so they often co-exist – whether more often than by chance remains a matter of debate. ST/T wave changes and a response to nitrates may occur with cholecystitis. Gallbladder pain can radiate into the front of the chest and simulate angina, but even in the presence of gallbladder disease, central chest pain with a constant relationship to exertion must be regarded as angina, until proved otherwise.

### 3.2.4 Chronic Relapsing Pancreatitis

The recurrent epigastric and left upper abdominal quadrant pain often radiates to the back and sometimes to the central anterior chest. The attack frequency may cause the pain to become almost continuous; the pain can be very severe, so medical

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attention may be sought. Diabetes mellitus and/or pancreatic insufficiency may develop; the latter results in malabsorption, loss of weight and steatorrhoea – impaired digestion of fat and protein leading to the passage of bulky fatty stools with heavy nitrogen loss.

### 3.2.5 Investigations

Endoscopy, Ba swallow and meal, cholecystogram, serum amylase and stomach acid secretion studies help to clarify the diagnosis. X-ray often shows calcification in chronic pancreatitis.

### 3.2.6 Treatment

Once the correct diagnosis has been established, routine management applies.

### 3.2.7 Prognosis and Main Disabling Effects

The prognosis and main disabling effects are determined by the nature of the underlying gastrointestinal disorder. Chronic pancreatitis is the most likely condition to cause long-term debility.

## 3.3 Musculoskeletal Causes

### 3.3.1 Recurrent Mild Trauma

This may be related to occupation (or a leisure pursuit), is often a dull ache to the side of the midline, and may correlate with particular movements – especially if anterior thoracic muscles are the sites of origin.

### 3.3.2 Spondylosis or Spondylitis

Pain from *thoracic or even cervical spondylosis* can be referred to the anterior chest, simulating angina. There is usually some limitation of spinal movement, often muscle weakness and sometimes reduced or absent arm reflexes. The distribution of the pain often corresponds to that of one or more dermatomes. Radiological proof of spondylosis does not exclude angina, but relief of the pain by wearing a cervical collar provides good evidence of a skeletal origin.

*Chronic mild trauma* to interspinous ligaments (e.g. in scoliosis) may be a cause of otherwise unexplained precordial pain.

*Ankylosing spondylitis* can cause diffuse anterior chest wall pain, often associated with local tenderness over the sternum and costal cartilages.

### 3.3.3 Less Common Musculoskeletal Conditions

#### 3.3.3.1 Tietze's syndrome

Tietze's syndrome causes pain of sudden or gradual onset in one or more upper

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costal cartilages. The pain is worse on deep breathing or coughing and the affected cartilage is swollen and tender.

### 3.3.3.2 Xiphoidalgia

Xiphoidalgia is a similar condition involving the xiphisternum, which may be related to recurrent mild trauma.

### 3.3.3.3 Sternal Pain

Pain arising in the sternum itself may be due to myelomatosis, metastases, ankylosing spondylitis, osteomyelitis or fracture (usually pathological).

### 3.3.3.4 Precordial catch

Precordial catch causes sudden sharp pain at or near the cardiac apex, which occurs while seated and lasts for a few minutes only, being relieved by a single, painful deep inspiration.

### 3.3.4 Treatment and Prognosis

Treatment and prognosis are those of the underlying disorder.

### 3.3.5 Main Disabling Effects

With appropriate treatment these conditions should not cause significant long-term disability (unless, rarely, a regional pain syndrome develops).

## 3.4 Non-coronary Cardiac Pain

### 3.4.1 Prolapsed Mitral Valve Cusp

Prolapsed mitral valve cusp is a relatively common lesion, which is often associated with a mid-systolic click and late systolic murmur. The pain is very variable in site, duration and severity and has no clear-cut diagnostic features. Diagnosis is confirmed by echo- or angio-cardiography.

### 3.4.2 Pulmonary Hypertension and Pulmonary Stenosis

Chronic pulmonary hypertension (PHT), either primary or secondary (as in mitral stenosis or the Eisenmenger syndrome), can produce a pain indistinguishable from angina. The cause is probably myocardial right ventricular ischaemia resulting from severely limited cardiac output and/or excessive right-sided systolic load with severe PHT. When advanced, other features of PHT may include: - dyspnoea; syncope on exertion; oedema; poor peripheral circulation (cold extremities); central and/or peripheral cyanosis; elevated JVP; sinus tachycardia or atrial fibrillation; 3<sup>rd</sup> (right ventricular) or 4<sup>th</sup> heart sounds; a loud pulmonary 2<sup>nd</sup> sound; tricuspid regurgitation; sometimes a lowered systemic BP; and, possibly, hepatomegaly and ascites.

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Severe pulmonary stenosis can cause a similar pain.

### 3.5 Chronic Respiratory Disease

Dyspnoea, especially when associated with airways obstruction, may be described as “tightness in the chest”, so that leading questions may result in the label of a “tight pain” across the chest and hence an erroneous diagnosis of angina.

### 3.6 Psychological Conditions

#### 3.6.1 Da Costa’s Syndrome

Chronic anxiety is the commonest underlying disorder in Da Costa’s syndrome (synonyms: neurocirculatory asthenia; effort syndrome; soldier’s heart; cardiac neurosis; disordered action of the heart).

##### 3.6.1.1 Diagnosis

The other features are chest pain, dyspnoea, palpitations, fatigue, dizziness, sighing and hyperventilation. The pain is most common in the left submammary region, but may be nearer the midline or anywhere on the left side of the chest, even radiating to the left arm. In character it is composed of sharp stabbing twinges superimposed on a background dull ache that persists for many hours. The important point distinguishing it from angina is that the pain often occurs after, rarely during, exertion. It is not normally relieved by anti-anginal medication.

##### 3.6.1.2 Aetiology

The mechanism of the pain is unknown, but it is sometimes triggered by a minor musculoskeletal abnormality. The pain convinces the patient that his heart is diseased and is perpetuated by the anxiety thus engendered.

##### 3.6.1.3 Treatment and prognosis

Hence the condition responds best to a confident diagnosis with firm reassurance; it can be aggravated by unnecessary investigation, which tends to increase concern rather than ameliorate it.

##### 3.6.1.4 Main disabling effects

Properly managed, Da Costa’s syndrome should not be a cause of long-term disability.

#### 3.6.2 Psychological Problems in Patients with Non-cardiac Chest Pain

Atypical non-cardiac chest pain is common and disabling, often persisting despite negative investigations; fundamental factors include inappropriate health beliefs and the continued misinterpretation of minor physical symptoms as evidence of heart



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disease.<sup>[1]</sup> Patients with non-cardiac chest pain often display illness behaviour. Summaries of papers describing follow-up and psychological treatment for various groups of patients with non-cardiac chest pain are given at Appendix A.

## 4 Chronic Lateral Chest Pain

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Almost all the tissues of the lateral chest wall (pleura, muscles, ribs and intercostal nerves) can be the sites of painful lesions.

### 4.1 Pathology involving the Intercostal Nerves

#### 4.1.1 Spinal Disease

Spinal disease can cause referred chest pain anterolaterally.

#### 4.1.2 Nerve Root Compression

Nerve root compression can occur in:

- Pathological fracture of the thoracic spine.
- Tuberculosis.
- Spondylosis with disc protrusion (uncommon in the thoracic spine).
- Neurofibromatosis (does not often cause pain).

#### 4.1.3 Mononeuritis, Mononeuritis Multiplex and Polyneuritis

*Diabetes mellitus* can cause a mononeuritis, mononeuritis multiplex (involvement of 2 or more peripheral nerves, though less than a polyneuritis), or a true polyneuritis; each of these may lead to asymmetric chest pain.

*Alcoholic poisoning, polyarteritis nodosa and leprosy* can cause mononeuritis multiplex or polyneuritis.

#### 4.1.4 Tabes Dorsalis

Tabes dorsalis is one manifestation of late neurosyphilis, presenting 10 – 25 years after primary infection with *Treponema pallidum*. The characteristic “lightning pains” can occasionally involve the chest, being “needle-like” or “knife-like”, the sudden stabbing nature invariably being illustrated by gesture. Usually only one area is affected at any time and the zone involved often remains tender for a while. The bouts occur irregularly and tend to be provoked by damp weather. Other symptoms include visual and sphincter disturbances, impotence, ataxia (wide stamping gait, worsened by eye closure), gastric or other tabetic crises and trophic lesions (e.g. arthropathies and perforating foot ulcers). Examination may reveal: Argyll-Robertson pupils (small, irregular, fixed to light but contracting on accommodation-convergence), ptosis and optic atrophy; loss of knee/ankle jerks and vibration/position/deep pain sensation in the legs; also Charcot’s joints – painless and unusually mobile despite the gross pathology. Penicillin treatment in the early stages reverses the disease, but delay makes progressive changes only partially reversible. Symptomatic treatment may be needed for confusion, ataxia, Charcot

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joints, urinary retention and lightning pains. Tabes dorsalis is now rare in the UK.

### 4.2 Aortic Aneurysm

Aortic aneurysms can be either 'true', with intact aortic wall, or 'false' – a contained rupture (with the wall being made up of adventitia and peri-aortic fibrous tissue) - following previous trauma (aortic transection) or chronic dissection.

#### 4.2.1 Aneurysm of the Ascending Aorta

Aneurysm of the ascending aorta may erode the sternum, thus causing chest pain (which may radiate to the neck and jaw), but much more often causes no symptoms at all. It is thought to be due to an abnormality of medial connective tissue, which can be either: - idiopathic; part of Marfan's or Reiter's syndrome; or a feature of rheumatoid disease, giant cell arteritis or syphilitic aortitis.

#### 4.2.2 Aneurysm of the Arch and Descending Aorta

Aneurysm of the arch and descending aorta can erode vertebrae and lead to nerve root pressure, causing very severe pain, which may radiate to the interscapular area. Aortic root dilatation may result in aortic regurgitation.

#### 4.2.3 Pressure on Mediastinal Structures

Pressure on mediastinal structures causes symptoms and signs as follows:

- Left recurrent laryngeal nerve – left vocal cord paralysis and hoarseness.
- Trachea – cough and stridor.
- Oesophagus – dysphagia.
- Left main bronchus – collapse of the left lung and infection; there may be a tracheal tug.

#### 4.2.4 Imaging Techniques

- Plain CXR may suggest the diagnosis.
- MRI and CT scans are the investigations of choice and can monitor post-operative follow-up.
- Contrast aortography now superseded by the above.

#### 4.2.5 Treatment

- Surgical repair using synthetic graft, because of risk of rupture.
- 5 - 50% mortality – greatest risk for aneurysms of the aortic arch.
- Morbidity: -
  - (a) *Neurological* - paraplegia/paraparesis if descending thoracic.
  - cerebral if aortic arch involved.

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### **(b) Renal failure - after repair of descending thoracic or thoracoabdominal aorta.**

#### **4.2.6 Prognosis**

10-year survival around 40%. Re-operation may be needed for aneurysmal dilatation at the site of anastomosis between synthetic graft and native aorta.

### **4.3 Intrathoracic Malignant Disease**

Intrathoracic malignant disease (primary or secondary) may cause pain in various ways:

#### **4.3.1 Bronchial Carcinoma**

Bronchial carcinoma leads to pleural pain caused by:

- Direct invasion of the pleura, often with effusion.
- Lung infection distal to a blocked bronchus.

#### **4.3.2 Primary Tumours of the Pleura**

Primary tumours of the pleura (e.g. mesothelioma) can either:

- Cause pleuritic pain directly, or
- Involve ribs and intercostal nerves producing severe pain.

#### **4.3.3 Metastases in the Thoracic Spine**

Metastases in the thoracic spine can cause intercostal pain.

#### **4.3.4 Metastases in the Ribs**

Metastases in the ribs can be extremely painful.

#### **4.3.5 Mediastinal Tumours**

Mediastinal tumours can cause poorly localised (sometimes lateral, though often central) chest pain without other pressure symptoms.

#### **4.3.6 Treatment and Prognosis**

As well as analgesia, treatment may involve surgery, radiotherapy and/or chemotherapy. Particularly if secondaries are the cause of pain, the prognosis is likely to be poor – in which case treatment may be palliative.

## 5 Acute Central Chest Pain

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The most important cause of central chest pain is ischaemic heart disease (IHD) causing stable angina, unstable angina and myocardial infarction (MI) - these are described in a separate protocol. Causes of acute central chest pain other than MI are discussed below.

### 5.1 Pericarditis

#### 5.1.1 Description

##### 5.1.1.1 Aetiology

Localised pericarditis is common in *myocardial infarction*. Other less common causes are:

- *Viral* (often Coxsackie B), *bacterial, fungal and parasitic infections*.
- *Connective tissue disorders* – SLE, rheumatic fever, rheumatoid disease or systemic sclerosis.
- *Dressler's* (post-MI) *and other similar syndromes* - (e.g. post-cardiotomy).
- *Chronic renal failure/uraemia*.
- *Untreated hypothyroidism*.
- *Malignancy*.
- *Idiopathic* – usually sporadic, but may occur in outbreaks (some patients develop myocarditis).

##### 5.1.1.2 Prevalence

Except for the acute idiopathic form, the prevalence is related to those of the underlying conditions.

#### 5.1.2 Diagnosis

##### 5.1.2.1 Clinical features

(a) *Pain* - in, and to the left of, the sternal region, which may radiate to the epigastrium, neck, back, shoulders and, occasionally, to the arms. Severity varies from mild discomfort to extreme agony; described as "stabbing" or "knife-like" - so it mimics the pain of myocardial ischaemia.

Aggravated by: coughing; deep breathing; twisting movements (e.g. turning over in bed); hyperventilation associated with exertion; recumbent (supine) position; and swallowing. Relieved by sitting up/forwards.

(b) *Friction pericardial rub* – *high-pitched scratching sound*, audible throughout the cardiac cycle; intensity varies with position – often evanescent. A *pericardial effusion* may develop – detectable on echocardiography.

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(c) *Fever and systemic upset.*

### 5.1.2.2 ECG

- S-T elevation, concave upwards, in all standard leads except aVR.
- Later, over 2 – 3 weeks, T-wave inversion (again mimicking myocardial ischaemia).

### 5.1.2.3 Virology/serology/renal or thyroid function tests

- To detect/confirm underlying cause.

## 5.2 Dissecting Aortic Aneurysm

### 5.2.1 Description

Acute aortic dissection is a catastrophic disorder often leading to sudden death. A tear in the aortic intima allows it to be dissected or stripped from its subintimal layers, destroying the tunica media. Involvement of the ascending aorta influences prognosis and management, so the Stanford classification divides dissections into Type A, involving the ascending aorta, and Type B, in which the ascending aorta is spared.

#### 5.2.1.1 Aetiology

There is weakness of the aortic media with breakdown of elastic and collagen tissues (cystic degeneration). Age-related degeneration, perhaps accelerated by hypertension, is the commonest process, but Marfan's, Ehlers-Danlos and Noonan syndromes are complicated by aortic dissection because of their effect on connective tissue. Occasionally, dissection follows disruption of atheromatous plaque or aortic arteritis.

#### 5.2.1.2 Prevalence/Incidence

Patients are usually in the 6<sup>th</sup> or 7<sup>th</sup> decade of life and two-thirds are males.

### 5.2.2 Diagnosis

#### 5.2.2.1 Clinical features

- Very severe “tearing” anterior chest pain (hardly influenced by opiates) radiating to the neck, interscapular area and (later) the abdomen; it rarely spreads to the arms. With thoracic aortic dissection, the pain may begin in the back.
- Nausea, vomiting, profuse sweating and syncope.
- One or more peripheral pulses may be absent (or disappear while under observation).

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- Other evidence of arterial occlusion (e.g. hemiparesis, blindness in one eye, myocardial ischaemia or haematuria).
- Aortic regurgitation (if the aortic ring is involved) – indicates a Type A dissection.
- Rupture into the pericardium or pleural space may cause acute tamponade or pleural effusion.
- In the absence of tamponade, BP little changed or raised – whereas usually lowered in MI.

### 5.2.2.2 Investigations

- ECG – normal unless a coronary artery is involved or pre-existing hypertensive changes are present.
- Radiography – on CXR, not easy to distinguish dilatation of dissection from unfolding of the aorta without a previous film.
- MRI is the investigation of choice, if available, otherwise:
- CT scan, aortography or transoesophageal echocardiography.

### 5.2.3 Treatment

- Adequate analgesia.
- Rigorous control of hypertension (avoiding ACE inhibitors because of possible extension of the dissection to involve the renal arteries), both initially and long-term.
- Emergency surgery (within 24 hours) improves survival for Type A dissections, and may be necessary for complications of Type B dissections (e.g. vital organ compromise – kidney or intestine, haemorrhage, or evidence of extension).

## 5.3 Massive Pulmonary Embolism

### 5.3.1 Description

Acute major pulmonary embolism results from significant obstruction to the proximal pulmonary arteries.

#### 5.3.1.1 Aetiology

Embolism from thrombosis in the pelvic or deep leg veins. Predisposing factors:

- Postoperative period and/or major trauma.
- Enforced recumbency:
  - Low cardiac output (e.g. post-MI, cardiac failure).
  - Probably, (economy-class) long-haul air travel – much current debate.
- Female sex hormones
- Pregnancy, oral contraceptive pill or HRT.
- Hypercoagulability and hyperviscosity syndromes.

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- Malignancy, increasing age, obesity and smoking.

### 5.3.1.2 Incidence

An analysis of death certificates in England and Wales in 1967 revealed 4981 reports in which pulmonary embolism was the suspected primary cause of death, but the number increased to 21,000 when more than one cause had been recorded.

Many patients die *with* rather than *from* emboli, particularly the elderly with malignancy or chronic cardiac/lung disease. Pulmonary embolism was considered to have been the *sole* cause of death in only 7% of adults dying in a district general hospital, though it had contributed towards death in another 7%. The true incidence of pulmonary embolism may be rising as hospital patients become older and less mobile over the decades.<sup>[2]</sup>

Between 300,000 and 600,000 patients suffer a pulmonary embolism in the USA each year, with a mortality of 30% if untreated and 3-8% with treatment.

### 5.3.2 Diagnosis

#### 5.3.2.1 Clinical features

Rapid severe illness with:

- Central chest pain, nearly identical to that of MI.
- Severe breathlessness, tachypnoea and rapid pulse.
- Faintness or loss of consciousness.
- Peripheral cyanosis.
- Very low BP and very raised JVP.
- Gallop rhythm over right ventricle.
- With peripheral emboli: - pleuritic pain, pleural rub and haemoptysis.

#### 5.3.2.2 Investigations

- *CXR* – dilatation of one or both branches of the pulmonary artery.
- *Pulmonary angiography* – shows occlusion of the pulmonary artery.
- *ECG* – simulates changes of antero-inferior infarction, right axis deviation and clockwise rotation.
- *V/Q scan or spiral CT*.

### 5.3.3 Treatment

- Emergency hospital admission.
- Tilt head-down and oxygen.
- CVP line to monitor venous pressure and give IV colloid, inotropes and



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thrombolytic agents - streptokinase, urokinase or tissue plasminogen activator (tPA) – and possibly hydrocortisone.

- Thermodilution pulmonary artery catheter – to monitor cardiac output, pulmonary artery pressure and mixed venous oxygen saturation, facilitating assessment of the rate of resolution of the embolus in response to treatment.

### 5.3.4 Prognosis

High early mortality, so vigorous therapy should be pursued.

## 5.4 Respiratory Disease

### 5.4.1 Tracheitis

Tracheitis causes upper sternal pain aggravated by the hyperventilation of exercise (so may mimic angina). Persistent coughing can lead to soreness in the upper airways and trachea.

### 5.4.2 Mycoplasma pneumonia

Mycoplasma pneumonia is never associated with pleurisy, but may cause a substernal pain aggravated by coughing.

## 5.5 Gastrointestinal Disease

Upper abdominal catastrophes, such as perforated peptic ulcer and acute pancreatitis can cause pain felt in the midline of the front of the chest. Abdominal signs, and gas under the diaphragm on X-ray or a raised serum amylase, confirm the respective diagnoses.

## 5.6 Pericardial Fat Necrosis

Pericardial fat necrosis is a rare cause of chest pain simulating that of pericarditis. There is no friction rub and the ECG is normal. CXR may reveal a para-cardiac mass.

## 5.7 Acute Anxiety

In a genuine and terrifying panic attack the patient complains of dizziness, palpitations, dyspnoea and precordial oppression or pain. “Angor animi” (the fear of impending death) is more prominent than in actual myocardial ischaemia, and exacerbates the anxiety. The circumstances of the attack and total absence of objective evidence of organic disease should clarify the diagnosis.

## 6 Acute Lateral Chest Pain

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### 6.1 Pleurisy

#### 6.1.1 Description

Pleurisy is pain related to respiration.

##### 6.1.1.1 Aetiology

- Pulmonary infections (e.g. lobar pneumonia, tuberculosis).
- Vascular lesions (e.g. pulmonary infarction).
- Connective tissue disorders (e.g. SLE).

##### 6.1.1.2 Incidence

The incidence of pleurisy is dependent on those of the underlying conditions.

#### 6.1.2 Diagnosis

##### 6.1.2.1 Clinical features

- (a) *Pain* in the cutaneous areas supplied by the intercostal nerves (which supply plentiful pain fibres to the parietal pleura), including a large part of the anterior abdominal wall. Spasm of the intercostal muscles may be a contributory factor.
- Sharp, superficial, of variable severity.
  - Aggravated by deep breathing and coughing.
  - Exacerbated or relieved by change of posture.
  - Relieved completely by holding the breath in expiration.
  - Inspiration abruptly halted by pain, so that respiration is often very shallow.
  - Pain of diaphragmatic pleurisy referred to the shoulder (a common feature of sub-diaphragmatic lesions – e.g. liver or subphrenic abscess).
- (b) *Pleural friction rub* – a characteristic creaking sound (like that of rubbing leather):
- Present during inspiration and expiration.
  - Poorly correlated to the pain.
- (c) *Pleural effusion* – development of this usually abolishes the pain and the pleural rub.

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### 6.1.2.2 Investigations

CXR and sputum culture.

### 6.1.3 Treatment and Prognosis

Treatment and prognosis are those of the underlying cause.

## 6.2 Bornholm Disease

### 6.2.1 Description

Synonyms: - epidemic pleurodynia/myalgia; Devil's grip.

#### 6.2.1.1 Aetiology

Bornholm disease is due to Group B Coxsackie viruses.

#### 6.2.1.2 Incidence

Bornholm disease occurs sporadically or in outbreaks, affecting mainly children or young adults. In large temperate urban conurbations, outbreaks generally occur in summer or autumn.

### 6.2.2 Diagnosis

#### 6.2.2.1 Clinical features

- Acute onset, the intercostal muscles being most often affected.
- Pain is the presenting symptom and may be extremely severe.
- Respiration becomes shallow, rapid and painful.
- Fever quickly develops; then headache and malaise are common.
- The affected muscles are tender and there may be a pleural rub plus a lymphocytosis.
- The disease lasts 7 - 10 days, so recovery is usually rapid, but relapses are frequent and may continue for several weeks.
- However, no serious complications or deaths have been reported and treatment is symptomatic.
- There should be no long-term disability.

## 6.3 Trichinosis

In trichinosis, involvement of the intercostal muscles can produce pleuritic pain. The diagnosis is supported by finding peri-orbital or generalised oedema and by blood eosinophilia.

## 6.4 Splenic Pain

Pain arising from the capsule of the spleen is related to respiration and commonly due to splenic infarction. There may be a friction rub resembling that of pleurisy. Splenic pain may occur in Hodgkin's disease and similar conditions.

## 6.5 Spontaneous Pneumothorax

### 6.5.1 Description

#### 6.5.1.1 Aetiology

A pneumothorax results from gas entering the potential space between the visceral and parietal pleura. A spontaneous pneumothorax is caused by rupture of a bulla or cyst on the surface of the lung.

#### 6.5.1.2 Incidence

The annual incidence is about 9 per 100,000. It is more common in asthmatics and also in young men, with a male:female ratio of approximately 4:1. About 20% of patients who have had one pneumothorax are likely to have a recurrence.

### 6.5.2 Diagnosis

#### 6.5.2.1 Clinical features

(a) *Pain*

- Usually abrupt in onset and pleuritic in type.
- Some patients complain of a dull ache or a sense of tightness.
- A few have no pain at all.
- Central chest pain may be caused by dissection of air into the mediastinum with an audible "crunching" sound over the heart.

(b) *Physical signs*

- Reduced chest wall movement and breath sounds on the affected side.
- Hyper-resonant percussion note on the affected side.
- If under tension, deviation of the trachea to the normal side.
- Auscultation may detect the same "crunching" sound.

#### 6.5.2.2 Investigations

CXR with expiratory film.

#### 6.5.2.3 Treatment

Chest drain or aspiration.

### 6.6 Trauma

Fractured ribs.

- Typical history of appropriate trauma.
- Localised tenderness.
- Visible on CXR, either immediately or later after callus formation.
- Significant disability unlikely once sound union has occurred.

## **Appendix A - Psychological Treatment for Chest Pain**

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### **Psychological Treatment for Atypical Non-cardiac Chest Pain**

In a randomised controlled trial cognitive behaviour therapy proved effective. There were significant reductions in chest pain, limitations and disruption of daily life, autonomic symptom distress and psychological morbidity in the treated group. The assessment-only control group were treated subsequently and showed comparable changes. The improvements were maintained in both groups during 4 – 6 months' follow-up.<sup>[3]</sup>

### **Non-cardiac Chest Pain and Benign Palpitations in Cardiac Clinic Patients**

Patients referred from general practice to a hospital cardiac clinic with a presenting disorder of chest pain or palpitations were followed up for 3 years. There were 39 patients with a cardiac diagnosis and 51 with no cardiac or other major physical diagnosis. The non-cardiac group was more likely to be young and female, and to report other physical symptoms and previous psychiatric problems.

Both groups reported progressive improvement in presenting symptoms and disability, but little change in mental state. Three quarters of the non-cardiac subjects described continuing limitation of activities, concern about the cause of their symptoms and dissatisfaction with medical care. Outcome was poor for those who had negative investigations - despite reassurance that they had no cardiac disorder or other serious physical finding.<sup>[4]</sup>

### **Psychosocial Outcome and Group Psychological Treatment in Patients with Chest Pain and Normal Coronary Arteries**

During assiduous 11-year follow-up of 46 patients with chest pain and normal coronary arteries, four died, one from IHD. Chest pain continued in 74%, being frequent, severe or both in half of these, and led to further hospital treatment in 58%; 13% had repeat coronary angiography. Of the survivors, 71% were taking cardiac medication and 29% were unable to work for medical reasons. High levels of chest pain, other physical symptoms, psychological distress and functional disability persisted long after angiography, (being similar to those found in patients with MI or angina) and led to heavy use of medical resources.<sup>[5]</sup>

60 patients with continuing chest pain despite cardiological reassurance after normal angiography received small group psychological treatment in 6 sessions: education; relaxation; breathing training; graded exposure to exercise; and challenging of automatic thoughts about heart disease. Treatment reduced chest pain episodes from median 6.5 to 2.5 per week ( $p < 0.01$ ) and the prevalence of hyperventilation from 54% to 34% ( $p < 0.01$ ). Improvements for anxiety and depression scores ( $p < 0.05$ ), disability rating ( $p < 0.0001$ ) and exercise tolerance ( $p < 0.05$ ) were

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maintained after 6 months. However, patients continuing to attribute their pain to heart disease had poorer outcomes.<sup>[6]</sup>

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