Chest pain in the emergency department

Angus Cooper, David W Hodgkinson, Richard M Oliver

The assessment of patients with chest pain is challenging for any emergency physician because of the spectrum of illness covered by this symptom. Patients may have a serious life-threatening condition or a trivial self-limiting illness. This article presents an approach to the assessment and early management of patients presenting with acute non-traumatic chest pain in the emergency department.

Dr Angus Cooper is Specialist Registrar in Accident and Emergency Medicine, **Dr David W Hodgkinson** is Consultant in Accident and Emergency Medicine and **Dr Richard M Oliver** is Consultant Cardiologist and General Physician in the Ipswich Hospital NHS Trust, Ipswich IP4 5PD

Correspondence to: Dr DW Hodgkinson

Acute chest pain is a common presenting complaint in the accident and emergency (A&E) department, accounting for 2–5% of all patient attendances. The differential diagnosis is extensive (*Table 1*). The consequences of missing a potentially life-threatening condition may be serious and the early recognition of patients presenting with acute coronary syndromes is essential.

The advent of thrombolysis as an effective treatment for acute myocardial infarction (AMI) has placed great importance on the need for early diagnosis and rapid treatment. A national patient education programme has raised public awareness and encouraged early access to emergency medical services. Patients who contact their general practitioners are increasingly advised to dial 999. These measures mean that more patients are presenting directly to A&E departments (Hood et al, 1999).

A&E departments have responded to this challenge by placing a new emphasis on the triage and assessment of patients with chest pain (Mackway-Jones and the Manchester Triage Group, 1997). Staff are being encouraged to move away from the classical sequential approach to patient assessment (history/examination/differential diagnosis/investigation/confirmed diagnosis/management plan) to a parallel assessment process that allows rapid identification of AMI and other potentially life-threatening conditions.

TABLE 1.
Differential diagnosis of acute chest pain

Potentially life-threatening	Acute coronary syndromes	Acute myocardial infarction
		Unstable angina
	Aortic dissection	
	Pulmonary embolus	
	Ruptured oesophagus	
	Tension pneumothorax	
Others	Pleurisy	
	Myopericarditis	
	Oesophageal reflux/spasm	
	Dyspepsia	
	Costochondritis	
	Intercostal muscle strain	
	Herpes zoster	
	Nerve root/spinal pain	

TRIAGE (ASSESSMENT AND PRIORITIZATION)

Patients with chest pain should be triaged rapidly using a standard methodology and allocated an appropriate clinical priority. The Manchester triage system identifies those patients with severe pain, a history suggestive of cardiac chest pain and/or abnormal physiology (Mackway-Jones and the Manchester Triage Group, 1997). These patients are triaged for immediate or very urgent medical assessment (within 10 minutes). They should be taken to a resuscitation room, where immediate observations and an electrocardiogram (ECG) can be performed.

On arrival some patients may have atypical pain (epigastric/abdominal) or even be asymptomatic, particularly if they have had appropriate pre-hospital treatment. It is important that this group of patients is identified, and triaged appropriately, by recognition that their chest pain may represent an acute coronary syndrome.

Primary assessment

An accurate history is essential in identifying the cause of the patient's symptoms. The clinical state of the patient may, however, dictate rapid assessment and emergency treatment before taking a detailed history.

The purpose of this rapid initial assessment is to identify and treat any immediately life-threatening condition. A structured approach is shown in *Table 2*.

A 12-lead ECG should be recorded as soon as possible as part of the primary assessment (*Figures 1–3*). If this ECG confirms AMI, further history taking and examination should be directed specifically towards identifying possible contraindications to thrombolysis. If there are no contraindications the patient should receive thrombolysis in the A&E department without further delay (*Table 3*). Unnecessary delays are created both by transfer of patients and repetition of clinical assessment before thrombolysis. Ideally the doctor at the first point of contact should have the clinical expertise and experience to make these decisions.

It is extremely difficult to obtain an accurate history from a patient in severe pain, therefore pain assessment should be addressed early and rapid control achieved using appropriate medication. In acute coronary syndromes this should include the use of aspirin, nitrates and intravenous opiates. Pain control will reduce the risk of life-threatening complications in acute coronary syndromes.

The absolute and relative contraindications to thrombolysis are listed in *Table 4*.

TABLE 2. Primary assessment

Airway	Open and maintain airway if necessary
	Provide maximal supplementary oxygen
Breathing	Identify and treat inadequate ventilation
	Monitor arterial oxygen saturation
Circulation	Identify inadequate tissue perfusion (shock)
	If present establish cause and start treatment
	Obtain intravenous access and monitor cardiac rhythm
Disability	Assess conscious level as Alert, responds to Voice, responds to Pain or Unresponsive (AVPU)
Exposure	Measure temperature

Secondary assessment

This should only be undertaken when life-threatening conditions have been identified and treated or after thrombolysis has been administered to patients presenting with AMI.

A detailed history is mandatory. Even if the patient is apparently able to give a clear history of the presenting condition it is useful to confirm details with relatives, friends, witnesses, and members of the emergency services who have attended.

It is usually necessary to clarify the severity, character and type of chest pain. The patient may

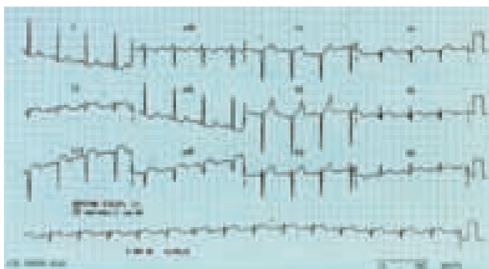

Figure 1. Acute inferolateral myocardial infarction.

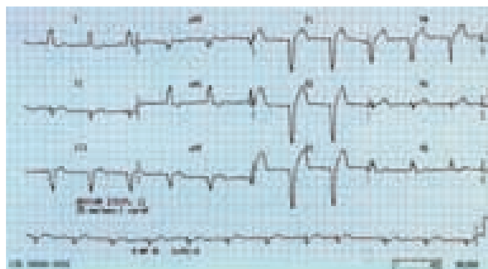

Figure 3. Left bundle–branch block.

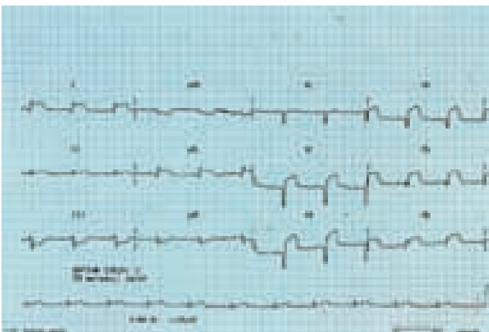

Figure 2. Extensive acute anterolateral myocardial infarction.

TABLE 3. Indications for thrombolysis

Thrombolysis is indicated in all patients who present within 12 hours of an episode of cardiac chest pain who have one of the following ECG findings	ST elevation > 2 mm in 2 or more adjacent chest leads
	ST elevation > 1 mm in 2 or more adjacent limb leads
	New left bundle–branch block or high clinical suspicion of acute myocardial infarction
Where the history of pain is longer than 12 hours thrombolysis is still indicated if the pain has been maximal in the last 12 hours and ECG changes are present	
ECG = electrocardiogram	

use words which have different meanings (e.g. 'sharp' may mean severe). The site of pain, radiation, precipitating and relieving factors, and relationship to posture and movement may all help the process of making a diagnosis. It is particularly important to record the time of onset and duration of chest pain in patients presenting with an acute coronary syndrome.

TABLE 4.
Contraindications to thrombolysis

Absolute	Aortic dissection
	Current haemorrhage
	Previous haemorrhagic stroke
	Known intracranial neoplasm
Relative	Blood pressure > 200/110 mmHg
	Proliferative diabetic retinopathy
	Bleeding diathesis/anticoagulants
	Major surgery/trauma in last 2 weeks
	Cerebrovascular accident/head injury within 2 months
	Proven current peptic ulcer
	Gastrointestinal haemorrhage within 2 weeks
	Traumatic cardiopulmonary resuscitation (rib fractures)
	Severe liver disease/varices

A brief systems enquiry should be targeted at specific symptoms. This will include upper gastrointestinal symptoms (abdominal pain, nausea and vomiting) as well as respiratory symptoms (shortness of breath, cough, haemoptysis) and assessment of exercise tolerance. Neurological symptoms and signs may be a presenting feature in dissection of the thoracic aorta.

An assessment of risk factors should include:

- Cardiac — hypertension, smoking, diabetes, hypercholesterolaemia
- Thromboembolic — major pelvic/hip surgery, recent immobility (British Thoracic Society Standards of Care Committee, 1997)
- Aortic dissection — hypertension, Marfan's syndrome
- Family history/past medical history.

A more detailed examination is now indicated. A number of clinical findings should be actively sought (*Table 5*). In many patients with life-threatening illness clinical examination may be normal.

Further appropriate investigations should also be initiated at this stage. This should include a repeat ECG at 15–30 minutes in all patients with possible acute coronary syndromes (especially if the initial ECG is normal). Repeat ECG examination should continue every

TABLE 5.
Clinical signs associated with specific conditions causing chest pain

Signs	Comment	
Cardiovascular	Difference in blood pressure in upper limbs or absent/diminished peripheral pulses	Found in dissection of thoracic aorta
	Raised jugular venous pressure	Seen in acute myocardial infarction with right ventricular dysfunction, major pulmonary embolus, aortic dissection, cardiac tamponade or superior vena caval obstruction
	Right ventricular heave	Pulmonary embolus
	Pericardial rub	Pericarditis
	Loud 2nd heart sound (pulmonary)	Pulmonary embolus
	3rd/4th heart sound	Ventricular dysfunction. Non-specific sign of acute or chronic heart disease
	Early diastolic murmur	Associated with aortic root involvement in aortic dissection or ruptured sinus of valsalva
Respiratory	Dull percussion, decreased air entry, bronchial breathing	Pneumonia
	Hollow percussion, decreased air entry	Pneumothorax. Look for signs of tension (respiratory distress, shock, trachea deviation)
	Pleural rub	Pleurisy and sometimes in pericarditis
Gastrointestinal	Surgical emphysema in neck	Oesophageal rupture/mediastinal or pericardial air
	Right hypochondrial or epigastric pain	Usually reflects abdominal pathology but may also be the result of hepatic congestion from heart failure
Neurological	Focal neurological signs	If new may indicate aortic dissection
Musculoskeletal	Costochondral tenderness	Beware co-existing pathology of cardiac/other origin
Skin	Herpetic rash	Pain may be out of proportion to skin changes
	Hyperaesthesia	Occurs early in herpes zoster. Also in other causes of nerve root irritation

30–60 minutes thereafter, particularly if the patient continues to experience chest pain. A change in the ECG may dictate further specific treatment. This assessment process should not delay the appropriate transfer of the patient to the coronary care unit (CCU) if an acute coronary syndrome is suspected.

At the end of this secondary assessment it is usually possible to make a diagnosis with a degree of certainty. This will allow an appropriate management plan to be formulated. If this is not possible a list of differential diagnoses should be drawn up with a plan for further investigation (either as an inpatient or outpatient) and specialist referral if indicated. As a general rule patients presenting to the A&E department should not be discharged home without a clear diagnosis being made. All the potentially life-threatening causes of chest pain must be excluded. Remember, a normal ECG does not exclude an acute coronary syndrome.

LIFE-THREATENING CAUSES OF CHEST PAIN

Acute coronary syndromes

Cardiac chest pain is generally described as gripping, tight or heavy (Jesse and Kontos, 1997). However, not every patient will present with the classical features and/or description of cardiac chest pain. Problems with recall or communication may affect the description of the chest pain. Some patients attempt to minimize the severity or significance of their symptoms, particularly if relatives are present. The intensity may vary from mild discomfort to very severe pain and is usually unrelated to the level of ischaemia, its duration or the amount of myocardium involved. Myocardial ischaemia and even infarction may occur without pain (particularly in patients with diabetes mellitus or the elderly). Certain atypical presentations should alert the clinician to the possibility of acute coronary syndromes.

Cardiac pain may be sensed only in a referred area (e.g. shoulder, arm, neck, or jaw) with no associated chest pain. Inferior AMI may present with abdominal or epigastric pain.

Patients with cardiac chest pain often say that they have ‘indigestion’ (exertional chest pain after meals). Their chest pain may even be eased by belching.

Cardiac chest pain may be indistinguishable from oesophageal pain reflecting common nerve root innervation.

Stable angina: Patients with stable angina very rarely present to the A&E department. By definition they attend because there is something different about this episode of angina, i.e. it was longer or more severe than usual or there was no response to nitrates.

Unstable angina: Patients with unstable angina must be identified in the A&E department and admitted to a CCU. They should be treated with the same degree of urgency as patients presenting with AMI (*Figure 4*).

The features to identify are:

- An abrupt onset of new angina (whatever its duration)
- Episodes of chest pain at rest
- Sudden worsening of established angina (severity, duration, poor response to nitrates, pain with lower workload).

Thoracic aortic dissection

Thoracic aortic dissection classically causes sudden severe central chest and interscapular pain. It is often described as ripping, tearing or searing. Abdominal pain may also be experienced. A dissecting abdominal aortic aneurysm may cause chest pain. If the dissection occludes the origins of major arterial branches of the aorta the patient may present with AMI, neurological deficit or limb ischaemia. A history of hypertension is present in the majority of cases.

Pulmonary embolus

A massive pulmonary embolus causes central chest pain, dyspnoea, cyanosis, collapse and hypotension. Less extensive pulmonary emboli may cause only transient symptoms and signs (breathlessness, brief collapse with full recovery). The patient may have recovered and have few if any abnormal physical signs at the time of presentation. Unilateral pleuritic chest pain is a feature of pulmonary embolism. The pain may be described as sharp, stabbing or exacerbated by inspiration. It is usually associated with dyspnoea although the patient may not report this at rest. It is necessary to enquire about shortness of breath on activity or exertion (British Thoracic Society Standards of Care Committee, 1997).

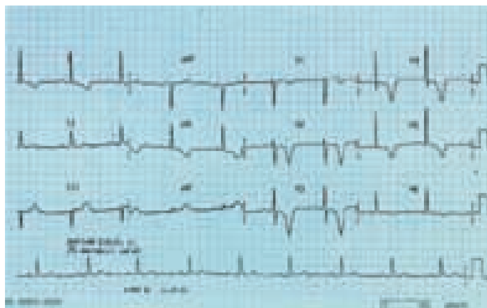

Figure 4. Anterolateral subendocardial ischaemia.

Pneumothorax

A simple pneumothorax usually causes sudden unilateral pleuritic chest pain. This is often only felt anteriorly but may radiate to the back. A tension pneumothorax causes increasing respiratory distress, hypoxia and tachycardia. Cyanosis, hypotension, distended neck veins and tracheal deviation are late signs of a tension pneumothorax. A pneumothorax should always be suspected in a patient with chronic lung disease who develops sudden respiratory distress (with or without chest pain).

Ruptured oesophagus

A patient with spontaneous rupture of the oesophagus (Boerhaave's syndrome) presents with severe central chest pain, often radiating to the back. This diagnosis should always be considered when a patient has severe pain requiring large doses of opiates. The onset of pain is almost always preceded by violent/forceful vomiting or oesophageal instrumentation. There may be associated haematemesis. Early diagnosis is essential to reduce what is otherwise a condition with a high mortality (*Figure 5*).

INVESTIGATIONS

Electrocardiograph

This may not be diagnostic in all the life-threatening causes of chest pain. The clinician should not necessarily be reassured by a normal ECG.

Acute myocardial infarction: Initial ECG may be normal in a high proportion of patients. It may show ST segment elevation, T wave inversion or Q waves, or may show new left bundle–branch block.

Pulmonary embolus: The classically described S1 Q3 T3 pattern is unusual. The most common finding is a sinus tachycardia but there may be signs of right bundle–branch block, right axis deviation, right ventricular strain and/or atrial fibrillation.

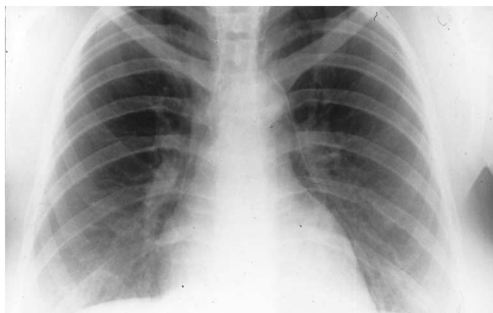

Figure 5. Pericardial air (pneumopericardium) may be the only sign of an oesophageal tear. More often than not the film will be normal early in the illness.

Acute pericarditis: Classically shows widespread convex (saddle-shaped) ST elevation. Changes may not be widespread and can be confused with AMI (*Figure 6*).

Aortic dissection: Hypertensive changes (left ventricular hypertrophy or strain). May show changes of ischaemia or infarction (aortic root involvement may compromise coronary blood flow by involvement of coronary ostia) (*Figure 7*).

Chest radiograph

The radiographic abnormalities associated with specific conditions are outlined in *Table 6*.

Cardiac enzymes

At present no reliable markers of acute myocardial damage are available for rapid use in the A&E department. Cardiac enzymes alone should not be used to make an admission/discharge decision on a patient with acute chest pain.

Arterial blood gases

These are normal in most patients with chest pain. Patients with pulmonary emboli may have a low PaO_2 and low $PaCO_2$. These findings are not specific or diagnostic as they are also found with pneumonia, left ventricular failure and acute asthma. Blood gases, measured while breathing room air, may be normal in a significant proportion of patients with pulmonary embolism.

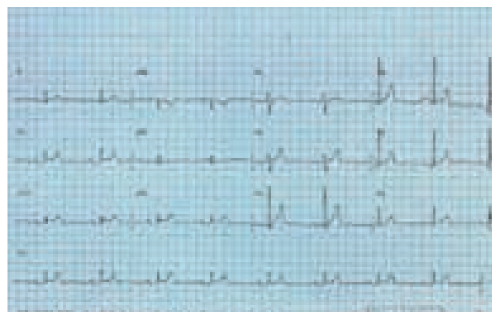

Figure 6. Acute pericarditis.

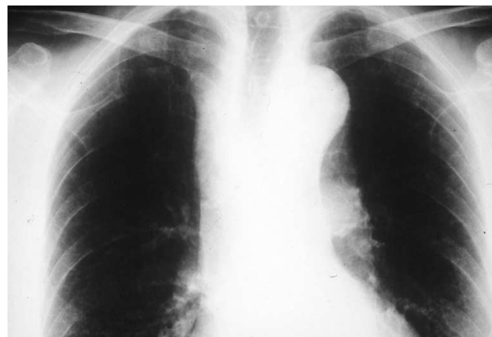

Figure 7. This patient had a history suggestive of a thoracic aortic dissection. The film is normal and this diagnosis can not be made on this film. It was made on computed tomographic imaging.

Echocardiography

Echocardiography is an under-used investigation in the A&E department. In the hypotensive patient with chest pain it may show findings that can rapidly confirm the diagnoses of pulmonary embolism, AMI, aortic dissection or pericardial tamponade (Rubin, 1997).

FUTURE DEVELOPMENTS

There is a need for more rapid and accurate identification of life-threatening causes of chest pain, in particular acute coronary syndromes. Acute chest pain units that allow rapid assessment/diagnosis and early safe discharge (within 12 hours) are being evaluated. A combination of cardiac enzymes (troponin and myoglobin) and symptom-limited exercise stress testing may be more widely applied.

The improved speed of access to emergency treatment and rapid identification of patients with AMI or unstable angina may potentially reduce the high mortality/morbidity of these conditions, particularly in the first few hours after onset of symptoms.

The more widespread use and availability of echocardiography in the A&E department may greatly assist with the diagnosis of patients with acutely life-threatening conditions (Rubin, 1997).

CONCLUSIONS

Assessment of patients with acute chest pain in the A&E department should include:

- Rapid triage
- A primary assessment identifying potentially life-threatening conditions and patients with AMI who should receive thrombolysis promptly
- A more detailed secondary assessment.

Patients with potentially life-threatening causes of chest pain may have minimal symptoms at presentation and physical examination and all investigations may be normal. Recognition of these conditions relies on accurate history taking and an appropriate level of awareness.

Patients should not be discharged from an A&E department unless all life-threatening causes of chest pain can be excluded. HM

The authors would like to thank Dr MC Colquhoun, Malvern, Worcestershire for permission to reproduce electrocardiograms from his collection and Dr S Garber and Dr G Picken, Diagnostic Imaging, Ipswich Hospital for their permission to reproduce images from their collection
Conflict of interest: none.

British Thoracic Society: Standards of Care Committee (1997) Suspected acute pulmonary embolus: a practical approach. *Thorax* **52** (suppl 4): S1–S24
Hood S, Birnie D, Swan L, Hillis WS (1999) Repeat questionnaire survey of thrombolytic therapy in accident and emergency departments in the UK. *Br J Cardiol* **6(9):** 508–11
Jesse RL, Kontos MC (1996) Evaluation of chest pain in the emergency department. *Curr Problems Cardiol* **22(4):** 149–236
Mackway-Jones K and the Manchester Triage Group (1997) *Emergency Triage.* BMJ Publishing Group, London
Rubin M (1997) Cardiac ultrasonography. *Emerg Med Clin North Am* **15(4):** 745–62

TABLE 6.
Radiographic abnormalities/features associated with specific conditions

Condition		Feature
Acute myocardial infarction/unstable angina		Usually normal unless complication, e.g. acute pulmonary oedema
Aortic dissection		May be normal
		Widening of the upper mediastinum > 8 cm. Depression of the left main bronchus to an angle < 40° with trachea
		Tracheal shift to the right
		Blurring of the aortic outline. If arch calcification, shadowing present outside
		Obliteration of the medial aspect of the left upper lobe
		Opacification of the angle between the aortic and the left pulmonary artery
		Fluid in left costophrenic angle.
Pulmonary embolus	Early	Raised hemidiaphragm
		Abnormally increased radiolucency resulting from reduced vessels distal to embolus
		Asymmetry of vessels compared with normal side
	Late	Abrupt cut off or partial appearance of pulmonary vessels
		Pleural effusion
		Linear wedge-shaped shadows as a result of infarction. Occasionally infarcts may cavitate
Ruptured oesophagus		Widened mediastinum
		Surgical emphysema
		Pneumomediastinum. Pneumopericardium
		Hydropneumothorax
		Pleural effusion (left usually)

KEY POINTS

- Chest pain is a symptom that covers a wide spectrum of illness from life threatening to minor self-limiting pathology.
- Patients with chest pain must be immediately assessed (triage) on arrival in the emergency department, and acute coronary syndromes and life-threatening pathology rapidly identified.
- Skilled and detailed history taking is still the most valuable tool available for assessment, particularly in patients with a normal examination and series of investigations.
- Patients should not be discharged from an emergency department unless all life-threatening causes of chest pain can be excluded.