

Acute Coronary Syndrome

SP Masud, R Mackenzie

Introduction

Coronary Heart Disease (CHD) is the most common cause of death in the UK. One in four men and one in six women die from the disease (1). CHD is also the most common cause of premature death. The British Heart Foundation (BHF) estimate that CHD accounts for 23% of deaths before the age of 75 in men and 14% in women (2). In addition, the BHF estimate that there are just under 2 million people living in the community who have, or have had, angina and over 1.2 million people who have already had at least one myocardial infarct.

With improved understanding of the pathophysiology of CHD, the 'acute coronary syndrome' concept and terminology has become widely accepted. The term refers to the clinical presentation of acute myocardial ischaemia. It encompasses the spectrum of conditions resulting from rupture or erosion of an atheromatous plaque and it reflects the uncertainty surrounding actual diagnosis at initial presentation (3,4,5). Acute coronary syndrome is typically caused by a sudden reduction in coronary blood flow and myocardial oxygen supply following varying degrees of intra-coronary thrombosis, vasoconstriction and micro-embolisation. The clinical symptoms and signs in any one patient will reflect the location of the obstruction and the severity and duration of ischaemia. The syndrome most commonly presents with acute chest pain (Figure 1).

Chest pain is one of the most frequent rea-

sons for consulting a doctor, attending an Accident and Emergency (A&E) Department or contacting the ambulance service. The underlying cause appears to vary according to whether a patient seeks help from a General Practitioner, the ambulance service or an A&E department (Table 1). Strikingly, patients who dial 999 have been found to be more likely to have an acute coronary syndrome. Paramedics are, therefore, more likely to be the first point of contact for these patients. In addition, patients with chest pain who do call for an ambulance are at higher risk of cardiac arrest and death (6). A third of patients with evolving myocardial infarction and two thirds of patients with established myocardial infarction die before they reach hospital (1,7). Pre-hospital practitioners must, therefore, be capable of making a rapid assessment of chest pain, identifying patients with possible myocardial ischaemia and instituting effective interventions to improve perfusion as soon as possible. Myocardial ischaemia may clearly progress to myocardial infarction and necrosis. Although ischaemia and established infarction are manifestations of the same underlying disease process, it would be wrong to assume that their management is the same. The mainstay of treatment for ST-segment elevation acute myocardial infarction is well established and involves early fibrinolysis whereas that for acute coronary syndrome without ST-segment elevation involves the use of anti-thrombus and anti-platelet agents and assess-

Maj SP Masud MBBS
DipIMC RCSEd
DMCC RAMC

MDHU Northallerton,
Friarage Hospital,
Northallerton,
North Yorkshire,
DL6 1JG.

E-mail:
masud999@doctors.org.uk

Maj R Mackenzie PhD
MRCP DipIMC
RCSEd RAMC

254 (City of
Cambridge) Field
Ambulance (V),
450 Cherry Hinton
Road, Cambridge,
CB1 8HQ.

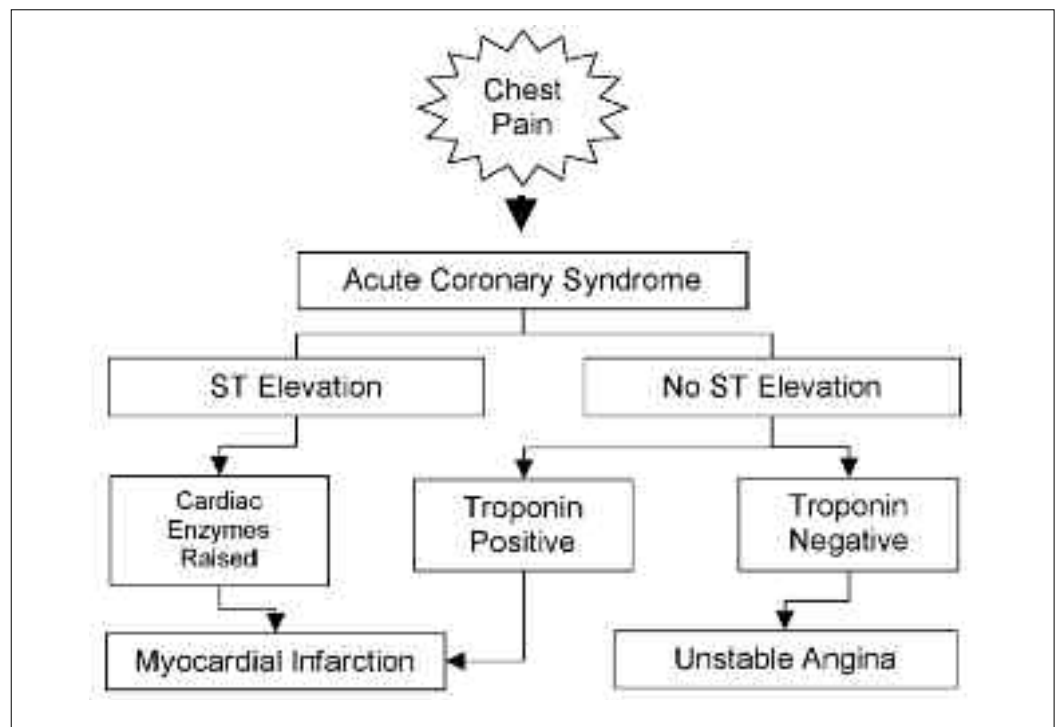


Fig 1. Acute Coronary Syndrome terminology (adapted from reference 5).

Table 1. Aetiology of chest pain in different clinical settings (adapted from reference 6).

Aetiology	Point of first contact (% of patients)			
	General Practitioner	Dispatch (999 call) centre	Ambulance crew	Emergency Department
Cardiac	20	60	69	45
Musculoskeletal	43	6	5	14
Pulmonary	4	4	4	5
Gastrointestinal	5	6	3	6
Psychiatric	11	5	5	8
Other	16	19	18	26

ment of the risk of infarction. In this second article on the pre-hospital management of acute medical emergencies, the pre-hospital assessment and management of acute coronary syndrome and acute myocardial infarction is reviewed.

Clinical presentation

The presentation of an acute coronary syndrome reflects the underlying spectrum of CHD. Patients do not present with a diagnosis of myocardial infarction, but with acute chest pain suggestive of coronary pathology, characterised by presence or absence of ECG changes, and by presence or absence of biochemical markers of myocardial injury (6,8). The immediate assessment of patients with chest pain has been reviewed elsewhere (6,8,9). Figure 2 illustrates the wide range of conditions that should be considered in these patients. The key decision is whether the pain is likely to be ischaemic in origin and is based on the history (including risk factors), presentation and clinical examination. A history of typical ischaemic pain lasting 20 minutes or more supports a working diagnosis of acute coronary syndrome. As with all visceral sensations, myocardial pain is poorly localised and can be indistinguishable from other sources of visceral pain (such as the oesophagus). Chest pain is however, more likely to be ischaemic if the patient has a history of ischaemic heart disease, if the pain is described as radiating to the left or right shoulders or arms and if it is associated with autonomic features such as nausea and sweating (8). In contrast, chest pain which is described as pleuritic, sharp,

stabbing or positional, or which can be reproduced by palpation is less likely to be ischaemic. Elderly patients and those with diabetes and hypertension may experience 'painless' or 'silent' ischaemia. In such cases the patient may present with shortness of breath, syncope or collapse from hypotension or dysrhythmias. In most cases, the clinical examination has little to add in terms of confirming or refuting the presence of an acute coronary syndrome.

When faced with a patient with acute chest pain, the pre-hospital practitioner should undertake a primary survey to identify any immediate threats to life (9). Ventricular fibrillation and cardiac arrest are common in patients with acute myocardial ischaemia and a defibrillator must be immediately available. Cardiac arrest, hypotension and arrhythmias should be managed according to current UK Resuscitation Council guidelines (10). Acute pulmonary embolism and aortic dissection are the two most important alternative diagnoses to consider. Most patients with acute pulmonary embolism have identifiable risk factors and pain which is pleuritic in character (11-13). Aortic dissection is missed in more than 10% of patients. It typically manifests as severe pain of abrupt onset which is described as 'tearing' or 'ripping'. Focal neurology, pulse deficits and a 20 mmHg difference in systolic blood pressure between both arms are the most significant clinical findings (14,15). If aortic dissection is suspected, pre-hospital treatment involves opioid analgesia and rapid transfer to hospital.

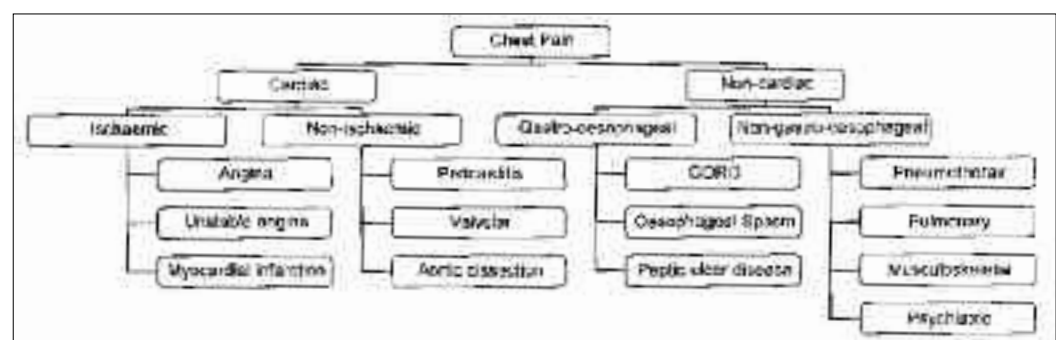


Fig 2. Schematic illustration of the wide aetiology of chest pain (adapted from reference 8).

Immediate care

The initial pre-hospital management of an acute coronary syndrome includes supplemental oxygen, aspirin, nitrates and opioid analgesia before and during transfer to hospital (6,16-18). Supplemental oxygen should be administered to patients with severe pain, shortness of breath, signs of left ventricular failure or oxygen saturation by pulse oximetry of less than 92%.

Aspirin, by blocking platelet aggregation, has consistently been shown to reduce the risk of myocardial infarction and death in acute coronary syndromes. A dose of 75 to 150 mg of aspirin (chewed or dispersed in water) is as effective in acute coronary syndromes as the higher dose (150 to 300 mg) recommended for myocardial infarction. Given that patients presenting with acute chest pain are unlikely to have a clear diagnosis prior to aspirin administration, the higher dose is recommended (provided there are no contraindications).

Nitrates are used to relieve ischaemic pain. They act as peripheral venodilators and therefore, decrease myocardial preload and oxygen consumption. They also dilate coronary arteries, increase coronary collateral flow and inhibit platelet aggregation. Current recommendations advise that a short acting nitrate may be given for acute pain if there is no bradycardia or hypotension. In the pre-hospital setting, the best way to administer nitrates is either via the sublingual or buccal routes. If sublingual glyceryl trinitrate is not effective, buccal glyceryl trinitrate (available as 2mg, 3mg and 5mg buccal tablets) should be given.

Pain and anxiety are associated with adverse sympathetic nervous system activation and should be controlled with intravenous opioid analgesia. Morphine or, preferably, diamorphine should be administered and titrated to effect. Antiemetics should be administered concurrently (e.g. intravenous cyclizine or, if left ventricular function is compromised, metoclopramide).

The 12 lead electrocardiogram

Although the sensitivity of the electrocardiogram (ECG) for ischaemia has been reported to be as low as 50% (19), it remains central to the assessment of patients with acute chest pain. If there is any clinical suspicion of ischaemia then a 12 lead ECG should be recorded. The availability of ECG recording will vary according to the operational environment but most UK land and air ambulances now carry appropriate ECG machines and rural or remote immediate care practitioners who attend emergency calls for chest pain must also ensure that they have this capability. Recording an ECG in the pre-hospital setting has been associated with a reduced in-hospital delay and a lower mortality amongst patients with chest pain (6,22). The

ECG should, therefore, be regarded as an adjunct to the clinical examination and obtaining and interpreting the ECG must be considered a priority (i.e. within 5 minutes of initial patient contact). The addition of the ECG to the history and examination, together with a screening checklist and strict ECG criteria, has been shown to dramatically improve diagnostic accuracy in patients with chest pain.

The ECG should be analysed for evidence of ischaemia (20) and infarction (19,21). Acute evolving myocardial infarction is defined as new ST-segment elevation ≥ 0.2 mV at the J point in leads V1 through V3 and ≥ 0.1 mV in other contiguous leads. The presence of ST-segment elevation has been shown to be the most sensitive and specific marker for acute myocardial infarction. If the pain is consistent with ischaemia, has lasted more than 20 minutes and is associated with ST-segment elevation in two or more adjacent leads, the working diagnosis is acute myocardial infarction. However, only 30 to 40% of patients with chest pain who subsequently develop acute myocardial infarction have ST-segment elevation on the initial ECG. In addition, other causes of ST-segment elevation can be misleading and should be considered in atypical cases (Table 2).

While ST-segment elevation indicates infarction, ST-segment depression > 1 mm (0.1 mV) in two or more contiguous leads is highly suggestive of ischaemia (as are inverted T waves (> 1 mm) in leads with prominent R waves). A number of other ECG features of ischaemia may also be evident (19-21).

Clinically established infarction is defined by the presence of any Q wave in leads V1 through V3 or Q waves ≥ 0.03 s in leads I, II, aVL, aVF, V4, V5 or V6, but these are often not evident on the initial ECG. Although a

Table 2. Causes of ST-segment elevation on 12 lead electrocardiography (from reference 21).

- | |
|---|
| <ul style="list-style-type: none"> u Acute myocardial infarction u "High take-off" u Benign early repolarisation u Left bundle branch block u Left ventricular hypertrophy u Ventricular aneurysm u Coronary vasospasm/Prinzmetal's angina u Pericarditis u Brugada syndrome |
|---|

completely normal ECG makes the final diagnosis of myocardial infarct very unlikely, a strong clinical suspicion should never be ignored. A normal initial ECG does not exclude the possibility of an evolving acute coronary syndrome with high risk of infarction. Repeated ECGs are often necessary throughout the initial assessment of the patient with ongoing ischaemic pain.

The presence of new Left Bundle Branch

Block (LBBB) in a patient with typical ischaemic symptoms suggests occlusion of the left anterior descending coronary artery and anterior myocardial infarction. These patients have a high risk of cardiogenic shock and death. Establishing the diagnosis of infarction as soon as possible is, therefore, critical. However, typical ECG changes may be masked by LBBB. Although a number of other features of the ECG may help in diagnosing infarction, these are difficult to interpret (23). All patients with typical history of ischaemia and LBBB should, therefore, be considered for fibrinolysis.

Cardiac Troponins

No single determination of any biochemical marker of myocardial necrosis reliably identifies or excludes myocardial infarction within 6 hours of symptom onset. However, cardiac Troponin T (cTnT) or Troponin I (cTnI) are more sensitive and specific markers of myocardial necrosis than traditional cardiac enzymes and they do reliably identify and exclude myocardial infarction at 12 hours (17,18,24).

The Troponin complex regulates calcium dependent interactions between actin and myosin within the cardiac myocyte. In patients with myocardial necrosis, an initial rise in peripheral blood is seen after 3 to 4 hours. Assays for cTnT and cTnI are essentially the same. Positive results reflect the 99th percentile for healthy controls for each of the available assay systems. Qualitative point of care Troponin testing kits are available for pre-hospital use. Where rapid evacuation or transport to hospital is available, these pre-hospital assays have little value (unless used to help 'rule in' acute infarction). Where evacuation is delayed or difficult, near patient testing may be of much greater value. Patients with acute coronary syndrome and elevated Troponin but no ST-segment elevation can be considered to have experienced significant infarction and necrosis. Those with acute coronary syndrome but no evidence of necrosis or ST-segment elevation have unstable angina (Figure 1).

Pre-hospital management of unstable angina

The management of acute coronary syndrome without ST-segment elevation has been extensively reviewed (17). In contrast to myocardial infarction, fibrinolysis has been shown to be detrimental and the mainstays of treatment are anti-ischaemic, anti-platelet and anti-thrombin drugs. Anti-ischaemic agents induce coronary artery vasodilatation or decrease myocardial oxygen utilisation by decreasing heart rate, contractility or blood pressure. They include nitrates (see above) and beta-blockers.

Beta-blocking agents inhibit the effects of circulating catecholamines and thus reduce myocardial oxygen consumption. A meta-

analysis has suggested that beta-blocker treatment in acute coronary syndrome is associated with a 13% relative reduction in risk of progression to myocardial infarction. There is no evidence to suggest that any specific beta-blocker is more effective than any other. Current recommendations are that patients without contra-indications should receive intravenous or oral beta-blockers if there is significant delay to hospital. If there are concerns regarding tolerance (e.g. patients with pre-existing pulmonary disease or left ventricular dysfunction) a short acting intravenous agent (e.g. atenolol or metoprolol) should be used.

Given the importance of intra-coronary thrombosis in the evolution of an acute coronary syndrome, anti-thrombin therapy is a major component of treatment. Thrombus consists of fibrin and platelets. Thrombus formation can be reduced by anti-platelet agents (e.g. aspirin as discussed above) and drugs which inhibit thrombin (e.g. heparin). Low molecular weight heparin (LMWH) is a sub fraction of standard heparin. In patients with acute coronary syndrome treated with aspirin, there is evidence that LMWH is better than placebo in reducing risk of infarction. Patients with an elevated Troponin but no indication for fibrinolysis also have a better prognosis if treated with LMWH. In comparison to unfractionated heparin, LMWH has a more predictable anti-thrombin effect with lower complication rates. LMWH can also be administered subcutaneously based on weight adjusted dose and does not require laboratory monitoring. Enoxaparin is currently recommended as the LMWH of choice in acute coronary syndrome (25).

Pre-hospital management of acute myocardial infarction

The pre-hospital management of acute myocardial infarction (acute coronary syndrome with ST-segment elevation) has also been extensively reviewed (16,18). In addition to oxygen, aspirin, nitrates and opioid analgesia, beta-blockers and fibrinolysis should also be considered in the pre-hospital and early hospital phases. Early intravenous administration of beta-blockers has been shown to be of benefit in acute myocardial infarction and atenolol 5-10 mg over 5 minutes or metoprolol 5-15 mg should be administered unless there is pulmonary oedema, hypotension (< 60 mmHg) or bradycardia. Beta-blockers may be particularly helpful when there is tachycardia (in the absence of heart failure), hypertension, or chest pain unresponsive to opioids.

Fibrinolytic agents (a more accurate term than thrombolysis) have been repeatedly shown to decrease the amount of intra-coronary thrombus and significantly improve survival in patients with acute coronary syndromes and ST-segment elevation. For

patients within 12 hours of the onset of their symptoms, the overall evidence of benefit from fibrinolysis is overwhelming. Pre-hospital fibrinolysis significantly decreases the time to fibrinolysis, morbidity and all-cause hospital mortality (26–29). In contrast, a deleterious effect has been consistently observed in patients without ST-segment elevation. Fibrinolytic therapy is not recommended for patients with acute coronary syndromes without persistent ST-segment elevation.

A number of fibrinolytic agents have been developed and there is no universal consensus regarding which should be used. Newer fibrinolytic agents which can be given by a single bolus have, however, dramatically simplified the process for pre-hospital practitioners. Tissue-type Plasminogen Activator (t-PA) given with intravenous heparin (the ‘accelerated’ regime) has been reported to result in 10 fewer deaths per 1000 patients treated than streptokinase. The increased risk of stroke with accelerated t-PA (3 per 1000 patients) has been offset by the many advan-

tages of the variants of t-PA. In particular, single bolus, weight-adjusted TNK-tPA (tenecteplase) is equivalent to accelerated t-PA for 30 day mortality and is associated with a significantly lower rate of non-cerebral bleeds and less need for blood transfusion. Tenecteplase is, therefore, the agent of choice for pre-hospital fibrinolysis. It should be given within 6 hours of a myocardial infarction.

In acute myocardial infarction, the addition of unfractionated heparin to t-PA has improved coronary patency in the hours or days following fibrinolysis. Unfractionated heparin is, therefore, recommended along with fibrinolysis in the pre-hospital phase. A ‘front-loaded’ regime involving a bolus of 5000 U of unfractionated heparin prior to fibrinolysis (followed by a 24 to 48 hour heparin infusion) is common. In order that there is no delay in commencing the heparin infusion, hospital staff should be pre-alerted for all patients who have received pre-hospital fibrinolysis. In contrast to acute coronary syndromes without evidence of

Box 1. JRCALC Thrombolytic Therapy Guideline (from reference 30).

Ensure patient fulfils ALL the criteria for drug administration in the Primary and Secondary patient assessment sections of the Thrombolytic Therapy Guideline.

Primary assessment

Can you confirm that:

1. The patient is conscious, coherent, and able to understand that clot dissolving drugs will be used?
2. The patient is aged 75 years or less?
3. The patient has had symptoms characteristic of a heart attack (i.e. pain in a typical distribution and of 30 minutes duration or longer)?
4. The symptoms started less than 3 hours ago?
5. The pain built up over seconds and minutes rather than starting totally abruptly?
6. Breathing does not influence the severity of the pain ?
7. The heart rate is between 50 and 140 beats per minute?
8. The systolic blood pressure is more than 80 mmHg and less than 160 mmHg?
9. The electrocardiogram shows abnormal ST segment elevation of 2 mm or more (0.08 seconds after the J point) in at least 2 standard leads or in at least 2 adjacent precordial leads, not including V1? (ST elevation can sometimes be normal in V1 and V2).
10. The QRS width is 0.14 mm or less, and that bundle branch block is absent from the tracing?
11. There is NO atrio-ventricular block greater than 1st degree? (if necessary after

treatment with i.v. atropine)

Secondary assessment (contra-indications)

Can you confirm that:

12. The patient is not likely to be pregnant, nor has delivered within the last two weeks?
13. The patient has not had a peptic ulcer within the last 6 months?
14. The patient has not had a stroke of any sort within the last 12 months and does not have permanent disability from a previous stroke?
15. The patient has no diagnosed bleeding tendency, has had no recent blood loss (except for normal menstruation) and is not taking warfarin (anticoagulant) therapy?
16. That the patient has not had any surgical operation, tooth extractions, significant trauma, or head injury within the last 4 weeks?
17. That the patient has not been treated within the last 3 months for any other serious head or brain condition ? (This is intended to exclude patients with cerebral tumours).
18. If streptokinase is the thrombolytic of choice can you confirm that streptokinase has not been given previously? (If the patient has had thrombolytic treatment and does not know which agent was used, you should assume that it was streptokinase)
19. That the patient has not had chest compression for resuscitation for a period longer than 5 minutes?
20. That the patient is not being treated for liver failure, renal failure, or any other severe systemic illness ?

infarction, there is insufficient evidence at this stage to support the use of LMWH together with fibrinolysis.

Fibrinolysis is associated with an excess of approximately two non-fatal strokes per 1000 patients treated, one of which will be moderately or severely disabling. Major bleeding elsewhere in the body (usually related to procedures) occurs in 4 to 13%. This represents one of the highest complication rates of any drugs used in pre-hospital care. The Joint Royal Colleges Ambulance Liaison Committee (JRCALC) have, therefore, developed restrictive but clear guidelines for ambulance services (30) (Box 1). These emphasize four important issues: the need to make an accurate ECG diagnosis, the need to exclude contra-indications, the need to obtain consent (Box 2) and the importance of time-to-hospital in deciding whether to initiate fibrinolysis. Safety is strongly dependent on a correct diagnosis and a variety of systems have been developed to either assist in the on scene interpretation of the ECG, or transmit it via a mobile telephone or radio for interpretation in hospital (31). Paramedic administered fibrinolysis is safe and effective (32,33).

It should be borne in mind that primary coronary angioplasty, when performed by experienced cardiologists within 90 minutes of diagnosis of acute infarction is as effective as fibrinolytic therapy in patients meeting standard criteria for fibrinolysis. In the unlikely event that such a service is available locally, transfer to hospital for primary coronary angioplasty should be considered as an alternative to fibrinolytic therapy in patients with cardiogenic shock or contra-indications for fibrinolysis (34).

The Grampian Region Early Anistreplase Trial (GREAT)

No review of pre-hospital management of acute coronary syndromes can be complete without reference to this seminal trial (35-37). This randomized, placebo-controlled, double-blind trial compared pre-hospital

with in-hospital fibrinolysis for clinically diagnosed acute myocardial infarction in rural Scotland. The study randomised 311 patients with probable acute MI on clinical grounds (i.e. without ECG evidence of infarct), who saw their general practitioner within four hours of symptom onset, to receive fibrinolysis (anistreplase) either at home or in hospital. The median time to treatment was 105 minutes in the home group and 240 minutes in the hospital group. Mortality was the same at one month, but rapidly diverged thereafter: by 30 months, it was 17% with pre-hospital treatment versus 32% with hospital treatment. On multiple regression analysis, the major independent predictors of mortality at 30 months were age, time of fibrinolytic administration, and time of presentation.

GREAT is one of the few published trials to demonstrate a clear long-term advantage of pre-hospital over in-hospital fibrinolysis for acute myocardial infarction: a one-hour delay in treatment was equivalent to 43 additional deaths (95% confidence interval 7 to 88 deaths) per 1,000 treated. For patients presenting two hours after symptom onset, the authors estimated that each hour's delay would lead to 69 lives lost at 30 months per 1000 patients treated.

The extremely long pre-hospital times in the control group and the 135-minute shortening of time to treatment in the pre-hospital group probably account for the survival benefit and this may not be reproducible across the UK (64% of the pre-hospital treatment group received therapy within two hours, compared to only 1% of controls). The GREAT trial is, however, directly relevant in terms of remote and resource limited pre-hospital care. Pre-hospital treatment halved both the median time to fibrinolysis (101 versus 240 minutes) and mortality at 1 year (10% versus 22%).

Summary

Acute coronary syndromes encompass a heterogeneous group of patients with different clinical presentations, who have differences

Box 2. Consent Information (from the East Anglian Ambulance NHS Trust).

To be read to the patient prior to the administration of thrombolytics:

'It is likely that you have suffered a heart attack, and the best treatment is a clot-dissolving drug called Tenecteplase. The quicker you receive this drug, the lower the risk from the heart attack – which is why doctors recommend the treatment is started as soon as possible. These drugs can cause serious side effects in a small minority of patients which I can explain to you in more detail if you so wish, but the risks attached to this treatment are very much less than the likely benefit. Would you like me to give you the injection or would you prefer to have more details?'

In the event that patients do want more information, they should be told the following:

'Treatment at this stage saves the lives of about 4 patients for every 100 we treat. But it can sometimes cause serious bleeding. The biggest risk is stroke which affects about 1 patient in every 200. Some patients also have allergic and other effects that do not usually cause any major problems.'

in both the extent and severity of underlying coronary atherosclerosis and who have different degrees of risk of progression to myocardial infarction. For each patient, the pre-hospital practitioner should make individual treatment decisions based on the history and examination, the ECG findings, the facilities and diagnostic equipment available and the transfer time to the nearest appropriate hospital. Patients with acute ischaemic chest pain should have oxygen, aspirin, nitrates and opioid analgesia. A 12 lead ECG should be performed within 5 minutes of initial assessment. If the ECG reveals ST-segment elevation or presumed new LBBB, this signifies acute myocardial infarction and in most cases immediate reperfusion therapy should be considered. The evidence of benefit in terms of mortality and morbidity following prompt anti-platelet and fibrinolytic therapy in such cases is unequivocal. Pre-hospital fibrinolysis is now well established and should be undertaken in patients with acute infarction on clinical and ECG grounds if the transfer to hospital is likely to exceed 30 minutes and it is less than 12 hours since the onset of pain.

Patients with no ECG evidence of infarction may still be at considerable risk and should still be conveyed to the nearest appropriate medical facility. Whilst en-route, they should receive aspirin, nitrates, low molecular weight heparin (LMWH) and beta blockers provided there are no contraindications.

The recommended approach to acute coronary syndromes for pre-hospital practitioners is summarised in Box 3.

This article was previously published in *Journal of the Royal Army Medical Corps* Vol 149 No 4 December 2003.

References

1. Department of Health. Heart attacks and other acute coronary syndromes. In: *National Service Framework for Coronary Heart Disease*. London,

- Department of Health, March 2000.
2. British Heart Foundation Coronary Heart Disease statistics at www.heartstats.org.
3. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes (1). *N Engl J Med* 1992;326:310-318.
4. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes (2). *N Engl J Med* 1992;326:242-250.
5. Hamm CW, Bertrand M, Braunwald E. Acute coronary syndrome without ST elevation: Implementation of new guidelines. *Lancet* 2001;358:1533-38.
6. Erhardt L, Herlitz J, Bossaert L, et al. Task force on the management of chest pain. *Eur Heart J* 2002; 23: 1153-76.
7. Huikuri HV, Castellanos A, Myerburg RJ. Sudden death due to cardiac arrhythmias. *N Engl J Med* 2001; 345: 1473-82.
8. Panju AA, Hemmelgarn BR, Guyatt GH, Simel DL. Is this patient having a myocardial infarction? *JAMA* 1998; 280: 1256-63.
9. Mackenzie R, Sutcliffe RC. Immediate assessment and management of acute medical emergencies. *J R Army Med Corps* 2002; 148:276-87.
10. See www.resus.org.uk.
11. British Thoracic Society. Suspected acute pulmonary embolism: a practical approach. *Thorax* 1997; 52: S1-S24.
12. Fedullo PF, Tapson VF. The evaluation of suspected pulmonary embolism. *N Engl J Med* 2003; 349: 1247-1256.
13. Task Force on Pulmonary Embolism. Guidelines on diagnosis and management of acute pulmonary embolism. *Eur Heart J* 2000; 21:1301-1336.
14. Klompas M. Does this patient have an acute thoracic aortic dissection? *JAMA* 2002; 287: 2262-72.
15. Erbel R, Alfonso F, Boileau C, et al. Task force report: Diagnosis and management of aortic dissection. *Eur Heart J* 2001; 22, 1642-1681.
16. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Acute myocardial infarction: pre-hospital and in-hospital management. *Eur Heart J* 1996; 17: 43-63.
17. Bertrand ME, Simoons ML, Fox KAA, et al. Management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. The task force on the management of acute coronary syndromes of the European Society of Cardiology. *Eur Heart J* 2002; 23:

Box 3. Priorities for the pre-hospital practitioner.

- u Look for and treat immediate threats to life.
- u Make a rapid provisional diagnosis - do not waste time attempting to establish a diagnosis unless there are therapeutic options such as fibrinolysis immediately available.
- u Relieve ischaemic pain (oxygen, nitrates, opioid analgesia).
- u Analyse a 12 lead ECG within 5 minutes of initial assessment.
- u If non ST-segment elevation acute coronary syndrome (unstable angina) then administer aspirin and consider LMWH.
- u If ST-segment elevation acute coronary syndrome then administer aspirin and consider beta-blockers.
- u Initiate fibrinolysis and unfractionated heparin for ST-segment elevation acute coronary syndrome (myocardial infarction) unless the patient is within 30 minutes of an appropriate hospital.
- u Anticipate and manage cardiac arrest whilst en-route to hospital.

- 1809-40.
18. Ven de Werf F, Ardissino D, Betriu A, *et al.* Management of acute myocardial infarction in patients presenting with ST-segment elevation. The task force on the management of acute myocardial infarction of the European Society of Cardiology. *Eur Heart J* 2003; **24**:28-66.
 19. Morris F, Brady WJ. ABC of clinical electrocardiography: Acute myocardial infarction - Part I. *BMJ* 2002; **324**:831-834.
 20. Channer K, Morris F. ABC of clinical electrocardiography: Myocardial ischaemia. *BMJ* 2002; **324**:1023-1026.
 21. Edhouse J, Brady WJ, Morris F. ABC of clinical electrocardiography: Acute myocardial infarction - Part II. *BMJ* 2002; **324**:963-966.
 22. Canto JG, Rogers WJ, Bowlby LJ, French WJ, Pearce DJ, Weaver WD. The prehospital electrocardiogram in acute myocardial infarction: is its full potential being realized? *J Am Coll Cardiol* 1997; **29**:498-505.
 23. Brady WJ, Morris F. Electrocardiographic diagnosis of acute myocardial infarction in the presence of left bundle branch block. *J Accid Emerg Med* 1999; **16**: 275-9.
 24. American College of Emergency Physicians. Clinical policy: critical issues in the evaluation and management of adult patients presenting with suspected acute myocardial infarction or unstable angina. *Ann Emerg Med* 2000; **35**:521-544.
 25. Goodman SG, Cohen M, Bigonzi F, *et al.* Randomized trial of low molecular weight heparin (enoxaparin) versus unfractionated heparin for unstable coronary artery disease: one-year results of the ESSENCE Study. Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q Wave Coronary Events. *J Am Coll Cardiol* 2000; **36**:693-8.
 26. Morrison LJ, Verbeek PR, McDonald AC, Sawadsky BV, Cook DJ. Mortality and prehospital thrombolysis for acute myocardial infarction: A meta-analysis. *JAMA* 2000; **283**: 2686-92.
 27. Brouwer MA, Martin JS, Maynard C, *et al*, for the MITI Project Investigators. Influence of early prehospital thrombolysis on mortality and event-free survival: the Myocardial Infarction Triage and Intervention Randomized Trial. *Am J Cardiol* 1996; **78**:497-502.
 28. Fath-Ordoubadi F, Al-Mohammad A, Huehns TY, Beatt KJ. Metaanalysis of randomised trials of prehospital versus hospital thrombolysis. *Circulation* 1994; **90**: 325.
 29. Grijseels EW, Bouten MJ, Lenderink T, *et al.* Pre-hospital thrombolytic therapy with either alteplase or streptokinase: practical applications, complications and long-term results in 529 patients. *Eur Heart J* 1995; **16**: 1833-38.
 30. Joint Royal Colleges Ambulance Liaison Committee. Pre-hospital Guidelines 2002 (version 2.2). Available at www.warwickuniversity.nhs.uk
 31. Väisänen O, Mäkitjärvi M, Silfvast T. Prehospital ECG transmission: comparison of advanced mobile phone and facsimile devices in an urban Emergency Medical Service System. *Resuscitation* 2003; **57**:179-185
 32. Keeling P, Hughes D, Price L, Shaw S, Barton A. Safety and feasibility of prehospital thrombolysis carried out by paramedics. *BMJ* 2003; **327**:27-8.
 33. Pedley DK, Bissett K, Connolly EM, *et al.* Prospective observational cohort study of time saved by prehospital thrombolysis for ST elevation myocardial infarction delivered by paramedics. *BMJ* 2003; **327**:22-6.
 34. Boersma E, Akkerhuis M, Simoons ML. Primary angioplasty versus thrombolysis for acute myocardial infarction. *N Engl J Med* 2000; **342**: 890-91.
 35. Rawles J. Halving of mortality at 1 year by domiciliary thrombolysis in the Grampian Region Early Anistreplase Trial (GREAT). *J Am Coll Cardiol* 1994; **23**:1-5.
 36. Rawles J. Magnitude of benefit from earlier thrombolytic treatment in acute myocardial infarction: new evidence from Grampian region early anistreplase trial (GREAT). *BMJ* 1996; **312**:212-215.
 37. Rawles JM. Quantification of the benefit of earlier thrombolytic therapy: Five-year results of the Grampian Region Anistreplase Trial (GREAT). *J Am Coll Cardiol* 1997; **30**:1181-1186.

Acute Coronary Syndrome

Commentary

Dr C Laird

This is an interesting article based mainly around the pre-hospital management of acute coronary syndrome (ACS). It also looks at the early hospital management of ACS because it has been written for practitioners who may not have the luxury of immediately admitting their patients to hospital. The result is an article which explains to those not routinely involved in anything but the immediate pre-hospital management of myocardial infarction the rationale behind the early management of patients in hospital. I found his part of the article particularly interesting.

I have always believed that intravenous cyclizine can cause vessel spasm and thus have not used it as an anti-emetic when managing acute coronary syndrome. The ECG criteria for giving fibrinolysis are different from the ones I have previously been familiar with and are also different from the JRCALC guidelines. The current JRCALC guidelines state: "if it can be confirmed that the electro-cardiogram shows abnormal ST segment elevation of 2mm or more in at least two standard leads or in at least two adjacent pre-cordial leads not including V1 (ST elevation can sometimes be normal in V1 and V2)." Many medical practitioners would also give fibrinolysis if the patients' ECG shows left bundle branch block which the patient was not previously known to have and there was a convincing history of a myocardial infarction. Thus to help identify new left bundle branch block many coronary care units are now recording the patients' ECG on discharge and either keeping it in the unit or giving the patient a copy of their ECG to take home with them.

Because of the cross-over in this article between pre-hospital care and the early in-hospital management I feel the article tends to suggest the use of beta-blockers in the pre-hospital situation. I know of few pre-hospital practitioners who would actually administer this therapy at this point. I totally agree that tenecteplase is the agent of choice for pre-hospital fibrinolysis and I think the place of tenecteplase is now well established.

With regard to low molecular weight heparin, some of the Out-of-Hours Co-operatives are already using this instead of unfractionated heparin mainly on the basis of the ASSENT-3 trial (1). None of the low molecular weight heparins have a license for

this purpose, and hence it would be unwise to recommend this as routine therapy at the present time. However, because of the short half life of intravenous unfractionated heparin, low molecular weight heparin as it was given in the ASSENT-3 trial (an intravenous bolus of 30mg immediately followed by the first subcutaneous dose of 1 mg per kg) may be particularly useful for patients with journey times of over 30 minutes. It may be of interest to note that a recent JRCALC position statement supports the administration of heparin concurrently with fibrinolysis (2). JRCALC suggest that, for ambulance services, patient group directives may be developed for using unfractionated heparin as an adjunct to fibrinolytic therapy. It is expected that low molecular weight heparin (Enoxaparin) will become the adjuvant heparin of choice when current studies have been completed.

References

1. The Assessment of the Safety and Efficacy of a New Thrombolytic Regimen (ASSENT) – 3 Investigators. Efficacy and Safety of tenecteplase in combination with enoxaparin, abciximab, or unfractionated heparin: The ASSENT-3 randomised trial in acute myocardial infarction. *Lancet* 2001; 358; 605-613.
2. Joint Royal Colleges Ambulance Liaison Committee. Prehospital Thrombolytic Therapy. JRCALC Position Statement. July 2004, (available at www.jrcalc.org.uk).

Dr C Laird

Director of
Education, BASICS
Education Scotland

Sandpiper House
Aberuthven Enterprise
Park, Main Road,
Aberuthven PW3 1EL