



# UNSTABLE ANGINA

**Introduction:** Unstable angina results from the abrupt, total or sub-total, transient and typically recurrent obstruction of a coronary artery. It is caused by rupture or erosion of the cap of an atheromatous plaque with superimposition of a platelet-rich thrombus. Spontaneous lysis or fragmentation of the thrombus, allows the ischaemia to resolve but distal embolism may cause myocardial necrosis. The latter is reflected in the release of cytosolic proteins (such as the troponins T and I) and arrhythmias. Persistence of an occlusive thrombus may lead to a fully developed infarction with Q waves on the electrocardiogram (ECG).

**Diagnosis:** Traditionally the diagnosis has required the exclusion of myocardial infarction. However, the use of sensitive markers of myocardial necrosis has shown that a continuum exists from myocardial ischaemia to non-Q wave infarction which it is practical to consider as a single condition. The initial diagnosis is made from a history of new onset (less than 2 weeks) of severe angina, sudden worsening of previous effort angina, or angina at rest, especially if prolonged and relatively unresponsive to nitrates. The essential next step in diagnosis is an electrocardiogram to exclude myocardial infarction with ST elevation for which immediate thrombolysis is indicated. In unstable angina (and non-Q wave infarction) the ECG typically shows ST segment depression or T wave inversion but it may be normal, particularly if some time has elapsed since the last episode of pain.

**Prognosis:** New or worsening angina precedes about 60% of incidents of acute myocardial infarction and sudden cardiac death. For patients with unstable angina or non-Q wave infarction the risk of death or (further) infarction is between 10 and 15% during the first 30 days and a further 35-50% will experience recurrent ischaemia. Those at greatest risk of early complications are older, have elevated troponin T or I levels in their blood, evidence of left ventricular impairment or recurrent episodes of symptomatic or asymptomatic ischaemia (as demonstrated by continuous ST segment monitoring).

**Treatment:** Patients in whom the diagnosis is suspected should be referred urgently to hospital. Anti-anginal drugs, which act mainly by reducing oxygen demand,

tend to be relatively ineffective but glyceryl trinitrate may overcome superimposed coronary artery spasm and should be administered immediately if the patient is in pain. Beta-blockers can reduce the frequency and severity of attacks of ischaemia and are usually combined with an oral long-acting or intravenous nitrate. In refractory cases a calcium antagonist is often added but short-acting dihydropyridines should not be given without a beta-blocker. Diltiazem or verapamil are the agents of choice if a beta-blocker is contra-indicated.

Aspirin reduces the risk of complications by more than 50% and should be given promptly. Heparin is also effective, and the combination of heparin and aspirin may be better than aspirin alone. Low-molecular weight heparins combined with aspirin are at least as effective as unfractionated heparin and are easier to administer. New anti-platelet agents, that inhibit the glycoprotein IIb/IIIa receptor responsible for platelet aggregation, reduce still further the frequency of complications when added to treatment with aspirin and heparin. They are particularly valuable as an adjunct to angioplasty in high-risk patients with refractory ischaemia.

Coronary angiography followed by angioplasty or coronary bypass grafting is undertaken for patients with episodes of ischaemia continuing despite medical therapy. All patients judged clinically to be at high risk should undergo angiography as the prognosis for those with multiple vessel disease accompanied by impaired left ventricular function is improved by surgery.

**Follow-up:** After stabilisation, whether treated medically, by angioplasty or by surgery, secondary risk reduction measures should be implemented. These include help to stop smoking, continued aspirin therapy, treatment of hypertension and, in most cases, cholesterol lowering with a statin, dietary advice and increased physical activity. Patients stabilised with medical treatment remain at risk from their coronary artery disease and, in most cases, should undergo assessment by treadmill exercise testing.

### **Further reading**

Purcell H & Fox KM            Improving outcome in acute coronary syndromes –  
as good as it gets?            European Heart Journal 1999; 20/21 (1533-1537)

Waller D                            Unstable angina and non-Q-wave myocardial infarction  
Prescribers' Journal 1999; 39/4 (193-201)

Kennon S & Timmis A            Management of unstable angina: what role  
intervention, ask the RITA-3 trialists            Heart 1999; 81/6 (565-566)