Heart failure post-myocardial infarction: a review of the issues

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In most patients with heart failure due to left ventricular systolic dysfunction, the underlying cause is coronary heart disease. To reduce progression to heart failure in a patient with acute myocardial infarction, it is important to achieve the earliest possible reperfusion, whether by thrombolysis or primary percutaneous coronary intervention. Every patient with acute myocardial infarction should have an assessment of their left ventricular function, the potential for reversibility should be considered, and reversible ischaemia should be identified. Left ventricular dysfunction does not only occur with ST segment elevation myocardial infarction but is also commonly associated with non-ST segment elevation myocardial infarction. Secondary prevention is crucial and this requires long term commitment by the patient and the health care system. Heart failure and left ventricular dysfunction are treatable but require a multidisciplinary, integrated network approach.

Far from implying that progression to heart failure is an inevitable consequence of acute myocardial infarction, the title of this conference highlights the opportunity that the overt occurrence of a myocardial infarction presents for the secondary prevention of major cardiac events. This contrasts with the unfortunate fact that, for many patients, the first clinical expression of underlying coronary heart disease is chronic heart failure (CHF) caused by previous covert myocardial infarction.

In the great majority of patients with heart failure due to left ventricular systolic dysfunction, the underlying cause is coronary heart disease. Effective primary and secondary prevention of coronary heart disease is therefore a major priority. Data from the INTERHEART study show that most, if not all, cases of myocardial infarction are predictable from what is already known about the preventable risk factors. It is also important to seek to improve out-of-hospital and in-hospital management of patients with acute myocardial infarction, and to appreciate that management is changing in relation to conventional versus invasive strategies. There is a need for effective implementation of evidence based treatments to try to prevent progression from acute myocardial infarction to left ventricular dysfunction and heart failure.

Of immediate importance in acute myocardial infarction are pain relief, sustaining life, and myocardial damage limitation—to contain the size of the infarct and also to prevent reinfarction. In addition, all patients with acute myocardial infarction, asymptomatic left ventricular dysfunction, or heart failure should undergo risk assessment in order to determine the options for further treatment. It is important to accept that when a patient is admitted with acute myocardial infarction, they are basically entering a system of acute and chronic disease management which has both “medical” and “interventional” components.

There has been substantial improvement in survival after acute myocardial infarction. In Scotland, during the decade 1986–1996, 30 day mortality after hospital admission with acute myocardial infarction improved from 25% to about 15%. This figure, which is likely to be even better now, is what would be expected for patients who survive to reach hospital and are admitted to a modern coronary care unit. The increased likelihood of surviving the initial episode is one reason why heart failure is becoming more common.

There is intense debate over the best method of limiting the damage after acute myocardial infarction. Thrombolysis is the standard treatment but interventional cardiologists have shown the benefit that can be achieved from primary percutaneous coronary intervention (PCI). A review of 23 randomised trials comparing the two treatments for acute ST segment elevation myocardial infarction showed primary PCI to be more effective than thrombolysis in restoring myocardial perfusion. Mortality may be reduced by the invasive strategy and, of course, improves left ventricular systolic function.
This randomised study involved 311 patients with early treatment was shown in the GREAT trial, a small trial carried out in a semi-rural area of north east Scotland in the early 1990s to compare pre-hospital and in-hospital thrombolysis. It has been shown that ‘‘the earlier the better’’ for thrombolytic treatment, with an estimated 10–50 lives lost/1000 patients for every hour delayed. The benefit of early treatment was confined to patients with non-ST segment elevation myocardial infarction. For example, in the CAPRICORN study evaluating use of a β blocker in myocardial infarction, in which one of the entry criteria was an ejection fraction of 40% or less, over 20% of the 1959 trial patients had non-ST segment elevation myocardial infarction. In other words, these patients with apparently mild myocardial infarction had left ventricular dysfunction.

Left ventricular function must therefore be assessed by echocardiography in all patients after an acute myocardial infarction and this should become a routine procedure in the coronary care unit.

NEW TECHNOLOGIES
One of the new technologies in acute myocardial infarction is the use of troponin measurement to identify those patients with non-ST segment elevation myocardial infarction who could benefit from early invasive treatment. Six month follow up of the TACTICS-TIMI 18 study showed advantages for troponin positive patients from an invasive strategy (early coronary angiography and revascularisation) rather than conservative management. In troponin positive patients there was a significant (p < 0.001) reduction in the composite end point of death, myocardial infarction, or rehospitalisation for acute coronary syndrome with the early invasive strategy. For troponin negative patients there was no significant difference between the two treatment strategies. Troponin measurement can therefore be used to identify high risk patients.

Risk assessment can be further refined by combining troponin measurement with measurement of natriuretic peptides. Figure 2 shows a consecutive series of patients with myocardial infarction in the Western Infirmary Glasgow. It shows that patients can be separated into low risk and high risk groups on the basis of their B-type natriuretic peptide (BNP) concentration.

In a recent review, Jernberg et al reported that in patients with non-ST elevation myocardial infarction who were at low risk according to the BNP concentration there was no mortality advantage from an invasive treatment strategy rather than a conservative treatment approach. However, in patients with raised BNP concentrations there was a substantial advantage from the intensive strategy. It is therefore important to use all the available tools to identify an individual’s risk and then to decide the most appropriate treatment.

Magnetic resonance imaging (MRI), using injection of gadolinium contrast agent, remains experimental but studies indicate that in the future this technique might be used to identify tissue that has been stunned rather than killed by an infarction. Such tissue is potentially viable and may therefore respond to revascularisation.

While it may not be difficult to identify myocardial infarction if the patient presents with pain, many patients have truly ‘‘silent’’ myocardial infarction. With MRI, we can for the first time visualise the extent of infarction (fig 3). Thirty four per cent of patients attending our heart failure clinic with a diagnosis of idiopathic cardiomyopathy were found to have MRI evidence of a previous myocardial infarction.
infarction, without any history of chest pain. The new
technique is therefore providing clinicians with much more
information about causes of heart failure.

**REMODELLING AND PHARMACOLOGICAL TREATMENT**

The processes involved in progression from acute myocardial
infarction to left ventricular dysfunction and heart failure include
the development of myocardial stunning and hibernation, remodelling, and chronic neuroendocrine activation.

In a fully remodelled heart following infarction, the heart
is dilated and hypertrophied, and there are areas of fibrosis
within the myocardium. The neuroendocrine hypothesis
proposes that these deleterious long term effects are caused
by excess release of angiotensin II, aldosterone, and
noradrenaline (norepinephrine) and can be prevented by
blockade of these hormones with appropriate drug treatment.

Post-myocardial infarction pharmacological management
involves four “standards”: antiplatelet treatment, statin,
angiotensin converting enzyme inhibitor, and β-blocker.
The role of selective aldosterone receptor blockade is also now
of interest because of the proven negative effects of this
hormone on the heart.

**USE OF DEVICES IN HEART FAILURE**

In addition to the many pharmacologic agents available for
heart failure management, there are many non-pharmacologic
approaches. One developing area in the treatment of
chronic heart failure, perhaps less so in acute myocardial
infarction, is the use of devices.

By resynchronising the contraction of the ventricle following
its injury by myocardial infarction, or other disease
process, it is possible to improve cardiac function and to keep
patients out of hospital. The MUSTIC 
and MIRACLE trials
both showed clinical benefit from biventricular pacing. There
is still debate about whether cardiac resynchronisation
therapy (CRT) will improve mortality and that question is
being investigated in the CARE-HF study. Nevertheless,
many patients are already receiving this form of treatment
when all other options have failed.

A more difficult issue is the use of implantable cardioverter-defibrillators (ICDs) to prevent sudden cardiac death.
There is now evidence of benefit from two trials: MADIT II
and SCD-HeFT (reported at the 2004 American College of
Cardiology scientific sessions). The use of ICDs together with
CRT has been investigated in the COMPANION study and
the trial results suggest that mortality might be improved,
but this is not proven.

It is essential that we use all the techniques at our disposal
to identify patients who are most suitable for treatment with
these electrical devices. However, often it is difficult to obtain
funding for these technologies because of the “up front”
costs, although they may be cost effective in the longer term.

**Learning points**

- Coronary heart disease is the major cause of heart failure due to left ventricular systolic dysfunction. Since coronary heart disease is often asymptomatic, recognition of acute myocardial infarction offers a major opportunity in secondary prevention.

- Progression to chronic heart failure after a myocardial
infarction is multifactorial, involving the extent of
myocardial damage at the time of the index event,
recurrent ischaemia and the development of myocardial
stunning and hibernation, remodelling and chronic
neuroendocrine stimulation.

- Adequate investigation to identify left ventricular
dysfunction at the time of myocardial infarction is
important as is the detection of potentially reversible
myocardial dysfunction that might benefit from revasculisation.

- Patients with significant left ventricular dysfunction after
a myocardial infarction require particularly careful
evaluation as they are at high risk of major cardiac
events, including sudden death and heart failure.

- Effective anti-remodelling treatment should be initiated
as soon as possible and monitored in a chronic disease
management strategy.
CONCLUSION
A patient who has had an acute myocardial infarction may or may not progress to develop left ventricular dysfunction and heart failure. The processes involved in this include myocardial stunning and hibernation, remodelling, and neuro-endocrine activation. Adequate investigation and prompt treatment are essential. The treatment of heart failure and left ventricular dysfunction post-myocardial infarction requires the same multidisciplinary, integrated network approach that we advocate for patients with chronic heart failure.

There are important issues on the journey from the coronary care unit to heart failure. It is apparent that the earliest possible reperfusion is important. Much has been achieved, using different strategies, and the time to thrombolysis has improved dramatically, but further improvement is required. One of the main problems is that patients often delay calling for medical help, which is now the major cause of delay in receiving reperfusion treatment. Public education programmes have not proven successful and we must find new ways of trying to alert patients to the need for prompt action so that they can get into the system earlier and so stand to benefit from treatment.

REFERENCES
5 Rawles J. GREAT. 10 year survival of patients with suspected acute myocardial infarction in a randomised comparison of prehospital and hospital thrombolysis. Heart 2003;89:564–6.