Primary PTCA for acute myocardial infarction—a logistic comment

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The cause of acute myocardial infarction is currently believed to be due to an occluding fresh thrombus within the vicinity of a rupture of the cap of a stenosing atheromatous plaque. Over the next few hours spontaneous thrombolysis may restore coronary flow in a minority of arteries, whereas thrombolytic regimens will significantly accelerate this process. In the GUSTO angiographic substudy, for instance, TIMI grade 3 flow was restored in 54% of patients randomized to an accelerated schedule of rtPA by 90 min. Such early achievement of patency results in better preservation of left ventricular function and is associated with better early survival. Unfortunately, thrombolytic therapy is not recommended for all patients, in whom early mortality is significantly higher. Whether relying on spontaneous or pharmacologically assisted thrombolysis, however, the stenotic lesion in the underlying infarct-related artery is unaffected and may remain a threat for recurrent thrombosis (and therefore reocclusion) both acutely and in the long term. Hence we have the philosophy of viewing an acute myocardial infarction as an obstructive coronary disease to be dealt with mechanically both in order to restore patency (and therefore flow) and to substantially reduce (by PTCA) or nullify (by CABG) the degree of residual stenosis of the culprit artery.

In order to prove their worth beyond doubt and to obtain a reliable estimate of their inherent risks, thrombolytic drugs have been administered to thousands of patients in randomized controlled clinical trials in many different hospital settings. Alternative means of restoring flow, however, have been similarly assessed in only a few hundred patients in hospitals equipped with dedicated and highly experienced teams of angioplasters and cardiac surgeons providing a continuous on-call service. Their assertions of survival and treatment superiority from a policy of primary PTCA (+ CABG if necessary) with that of thrombolysis (resorting to intervention if clinically necessary) are heard loud and clear. In this issue, the pioneering group from Zwolle further support their interventional stance by reporting on the comparative (or marginally cheaper) costs per patient randomized to a schedule of immediate PTCA over a period of a mean of 31 months. Is their optimism justified?

The early mortality in the control/thrombolysed patients in the combined analysis of the three immediate PTCA trails referred to above is lower than that reported in any controlled trial of any thrombolytic regimen (5.9%) and the success rate of the initial angioplasty in the intervention groups was very high (≥95%), no doubt testimony to the expertise of those involved. But how likely is it in the foreseeable future that such resources and expertise could be more widely available around the clock to assess and deal with the rising tide of patients admitted with chest pain query acute myocardial infarction? Before grappling with that issue a much larger comparative trial of acute intervention vs the 'best' thrombolytic schedule undertaken in many different hospitals with staff of varying expertise is required. Only then shall we get a clearer perspective of the range of risks and benefits from each treatment option.

If such a large trial eventually confirms these preliminary exploits, what of the resource implications of offering such a service on a continual basis country-wide? Using the United Kingdom as an example, there are approximately 300 000 patients with acute myocardial infarctions per year. If we assume for the moment that approximately one third will not reach hospital alive and that roughly one half of those that do will be deemed ineligible for thrombolysis, that still leaves 100 000 patients eligible for primary PTCA instead of thrombolysis. In addition, an unknown proportion of 'thrombolysis ineligible' patients may also be suitable for acute intervention. So we could arrive at a yearly demand for intervention of between 100 000–200 000 purely for acute myocardial infarction at a time when the current total PTCA activity is about 12 000 per annum. Even trying to select 'PTCA preferred patients', such as those with large anterior infarctions, still makes the logistics of providing such a service awesome.

So could other clinical or financial benefits accrue as a direct result of such a policy, for instance fewer days spent in hospital or convalescence before returning to work, fewer recurrent ischaemic events necessitating readmission and fewer drugs for symptomatic treatment? In this issue, the group from Zwolle in The Netherlands assert a firm yes. They randomized 301 patients to either direct PTCA (152) or streptokinase (149). Over a mean follow-up time of 31 months, there were 12 (8%) deaths in the PTCA group (seven cardiac), compared with 20 (13%) in the
SK group (17 cardiac), with $P=0.12$ and $P=0.03$ respectively.

In addition there were four non-fatal reinfarctions in the PTCA group vs 25 in the streptokinase group, presumably all necessitating readmission to hospital. Patients randomized to PTCA had a higher initial and follow-up left ventricular ejection fraction than those randomized to streptokinase (48 ± 12% vs 43 ± 13%, $P=0.006$). It is asserted that this small superiority of the interventional group over streptokinase is a direct result of early more complete and more sustained reperfusion than those patients randomized to streptokinase. Although we are not given a breakdown of hospital stay, drug treatments, work status, physical activity, or symptom status in this paper, the Zwolle group assert a total saving of Dfl.0-288 per patient, or Dfl.1-793 per survivor, or Dfl.9086 per event-free survivor in those assigned PTCA. Multivessel coronary artery disease and a previous myocardial infarction were associated with increased costs.

Neither in this paper nor in the original reports of the three comparative trials cited above are we told what proportion of all suspected or eventually proven infarctions are represented by those patients randomized to PTCA or thrombolysis. This, I believe, is an important omission when trying to budget for the totality of admitted acute myocardial infarction patients rather than just those suitable for PTCA or thrombolysis.

The modest individual savings reported from Zwolle, when multiplied over a whole country, imply that PTCA might not be so financially daunting as first thought. But the initial outlay in terms of plant and sufficient trained personnel will, in my view, restrict such an enterprise to areas of high enthusiasm in countries either with generous health care budgets or those with an acceptance that other medical activity will inevitably suffer financially.

Finally, in order to compete favourably with thrombolysis, interventional treatment must be close at hand — not restricted to distant tertiary referral units with the inevitable delay in arranging and effecting patient transfer. This would mean that each district hospital would need at least two (perhaps three or four) experienced invasive teams in order to offer a round the clock service. Members of such teams will need to maintain dexterity and competence by engaging also in diagnostic coronary work — and perhaps elective coronary angioplasty. Such extension of activity will enter the debate of surgical cover, not for acute myocardial infarction work necessarily, but for the inevitable creeping development.

Providing an acute PTCA service for patients with AMI, should it prove to be the superior strategy, may thus have logistic consequences way beyond the coronary care unit.

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References