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Does it matter where you go with an acute myocardial infarction?

See page 1794 for the article to which this Editorial refers

The management of patients presenting with acute myocardial infarction has evolved considerably over the past decades, and clinical outcome has improved due to a variety of factors. Earlier diagnosis of the acute event, the development of effective reperfusion modalities, and widespread availability of pharmacological therapies such as aspirin, beta-blockers, and ACE-inhibitors have all made important contributions to the care of the patient with evolving myocardial infarction[1–2]. Although the guidelines of the European Society of Cardiology and the American College of Cardiology/American Heart Association are well known, there is uncertainty as to how well these guidelines are implemented in daily practice.

In this issue Gottwik, Zahn and co-workers, report from the pooled data of the Maximal Individual TheraPy in Acute myocardial infarction (MITRA) studies and the Myocardial Infarction Registry (MIR)[3]. They studied 24 814 patients with acute myocardial infarction, treated in 305 hospitals between 1994 and 1998, including the entire spectrum of small as well as large community hospitals, tertiary care centres and university hospitals. Compared to patients treated in hospitals without a cardiology department, patients treated in hospitals with a cardiology department had a better clinical outcome, a lower hospital mortality, a lower rate of heart failure at discharge and a lower proportion of patients with a prolonged hospital stay. This was associated with a higher use of both reperfusion therapy as well as other recommended therapies. Although observational studies should always be interpreted with caution, in particular when randomized trials are not available or not possible, they can make an important contribution. This study is further observational evidence of the fact that clinical outcome after myocardial infarction may differ depending on the setting: type of hospital (with or without cardiology
department), type of doctor (cardiologist or generalist), choice of reperfusion therapy (angioplasty or thrombolysis), and patient load, all have a significant impact on clinical outcome[3–8].

What can we learn from these observations, and how should we respond? Better knowledge of the possibilities and limitations of diagnostic and therapeutic procedures leads to improved clinical outcome. Improvements in training programmes for cardiologists as well as generalists, participation in community-based clinical trials, and development of methods to implement guidelines into clinical practice can contribute in this regard. The major published guidelines for the treatment of myocardial infarction, both in Europe and the United States of America, have appeared in journals that target cardiologists[1,2]; primary care physicians and internists may also benefit from such guidelines. As illustrated by the temporal analysis of the data[3], introduction of quality control and outcome monitoring results in a gradual improvement of clinical outcome in all types of hospitals. There seems to be a relationship between patient load and clinical outcome. Thiemann and co-workers described a cohort of 98,898 patients with acute myocardial infarction, 65 years of age or older and found better outcomes following admission to hospitals that treated large numbers of patients, with a consistent dose-response relationship between hospital volume and survival[9]. Although the hazard associated with low hospital volume may be smaller for patients with myocardial infarction, than for patients undergoing surgical procedures[10,11], the mortality attributable to hospital volume is considerable, because the population at risk is large. The implications of these findings are dependent on many other factors, such as geographic considerations, population density, organizational issues and costs. Furthermore, there are abundant examples of cardiologists and generalists who deliver excellent care in low volume institutions, and high volume institutions with ‘less than optimal’ results. Nevertheless, previous studies of elderly patients have suggested that the initial treatment of acute myocardial infarction (within the first day) was the major determinant of survival at 4 years, and that patients living in the catchment area of a hospital with a catheterization laboratory were more likely to be treated at a high volume hospital and to undergo early cardiac catheterization, with a 1% absolute lower rate of death at 1 year, as compared to other patients[12,13]. In particular, for patients with high risk characteristics regional planning should, whenever possible, ascertain that these patients attend a medical facility with 24 h of emergency cardiac care and have an emergency revascularization programme established. Pre-hospital diagnosis and triage can make an important contribution in this regard[13,14].

Finally, these observational data show, that although therapeutic possibilities for patients with acute myocardial infarction will continue to improve, most benefit for our patients in the coming years will come from dedicated application of all that is available to us today.

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References
The quest for the ideal stent

See page 1808 for the article to which this Editorial refers

The introduction of coronary stent implantation is the second great advancement in the treatment of obstructive percutaneous coronary artery disease, since the introduction of balloon angioplasty. Stent implantation, acting as a scaffolding device, helps to optimize the initial result of balloon angioplasty by tackling the intimal flaps and plaque dissections, so that a ‘smoother’ surface and a larger lumen results, with less resistance against antegrade blood flow. Stent implantation may reduce or prevent (in cases of direct stenting, i.e. without pre-dilatation) distal embolization from the plaque after the disruptive action of balloon angioplasty. In addition, stent implantation almost completely prevents abrupt vessel closure and has significantly reduced late restenosis by eliminating acute elastic vessel recoil and in particular chronic negative vessel remodelling, both of which have been shown to be important components of late balloon angioplasty restenosis.

Jacques Puel was the first to implant a stent in a human in 1986, in Toulouse, France. These early stents were rather crude, causing problems in correct implantation and were associated with a disturbingly high rate of subacute thrombotic occlusion. Since then, stent technology has significantly improved, resulting in stents that are more flexible, very low profile and thus more easily deliverable.

Nowadays, stent implantation is a safe, predictable technique. It has significantly reduced the need for bailout emergency surgery and allows for percutaneous treatment of a wide variety of obstructive coronary lesions.

Yet, despite all these stent ‘blessings’, significant shortcomings still exist. First, subacute in-stent thrombosis, although largely prevented by appropriate stent implantation and adjunctive treatment with thienopyridines (ticlopidine or clopidogrel), still occurs in less than 2% of cases and is often associated with significant morbidity and mortality, calling for a truly non-thrombotic stent.

Second, and the most annoying is the occurrence of ‘stent disease’, i.e. neointimal hyperplasia within the stent, focal or diffuse, which causes clinically significant obstructions in 15% to 20% of the cases. In-stent restenosis is difficult to eradicate because repeat percutaneous treatment itself is associated with a high repeat in-stent restenosis rate of up to 60%. Recently, brachytherapy, the only reasonably effective treatment, has reduced this rate to ~30%.

Therefore the quest for the ideal stent continues. One method of tackling the occurrence of in-stent thrombosis and late in-stent restenosis could be by enhancing the blood and tissue compatibility of the stent by stent coatings.

Stent coatings have been designed to be either passive or active. Passive coatings such as polymer coatings or inorganic coatings (silicon carbide, carbon or gold) provide a biologically inert barrier between the stent surface, vessel wall and circulatory blood, in an attempt to curb antiinflammatory responses and to prevent in-stent thrombosis and neointimal hyperplasia. Active coatings are biologically active because the coatings are loaded with drugs (heparin, paclitaxel or rapamycin) which are released (at a certain rate) to prevent the occurrence of thrombosis or restenosis.

Stents coated with a covalent binding of heparin should not be regarded as active drug eluting stents, because the covalent binding prevents release of heparin into the blood tissue. These stents are biologically active because the coatings are loaded with drugs (heparin, paclitaxel or rapamycin) which are released (at a certain rate) to prevent the occurrence of thrombosis or restenosis.

Stents coated with a coeval binding of heparin should not be regarded as active drug eluting stents, because the coeval binding prevents release of heparin into the blood tissue. These stents are biologically active and have been shown to be effective in reducing thrombosis in animal models and in reducing intimal hyperplasia after injury in animals. Heparin-coated stents were first tested in humans in the Benestent-II Pilot Study. This non-randomized study showed that a heparin-coated Palmaz–Schatz stent implanted in stable patients with