Another striking issue to which the authors refer is the extremely high prevalence of seropositivity to *H. pylori* in the general population of North Glasgow: 65% in men aged 45–54 years, more than 80% in men aged 55–74 years. This small fraction of seronegatives (less than 20%) in the age and sex group most likely to have developed athero-thrombotic disease, limits the possibility of detecting an existing association between *H. pylori* seropositivity and coronary heart disease.

Others have argued whether seropositivity to *H. pylori* is to be interpreted as an indicator of chronic infectious disease and/or as an indicator of childhood poverty.

The increase with age of seropositivity may be a cohort effect although not entirely. However, if we accept that *H. pylori* infection is usually acquired in youth and is a marker of deprivation at that time, it becomes very difficult to separate the effect of childhood deprivation and of socio-economic conditions during adulthood on the risk of developing atherothrombotic diseases.

Social class, educational level or any other indicator of socio-economic class are strongly related to a large variety of health problems including coronary heart disease, although the association with coronary heart disease was inverse in the 1950s and has changed completely in Northern Europe in the last 40 years. The absence of disease specificity and the time trend suggest that social class is related to health through multiple factors associated with a large variety of living conditions and behavioural factors starting in the prenatal phase and continuing until death.

Adjustment for social class status means adjustment for a battery of factors that are not necessarily independent of the study objective i.e. the association between *H. pylori* seropositivity and the atherothrombotic process.

The observation that the association between *H. pylori* seropositivity and coronary heart disease prevalence is no longer statistically significant after adjustment for social class cannot exclude a possible link between *H. pylori* infection and the atherothrombotic process, through mechanisms that are also related to variables that make up part of the Pandora’s box we label as social class.

This study by McDonagh et al. has certainly contributed to the discussion; what is strongly needed are prospective cohort studies on the issue of chronic infectious diseases and coronary heart disease incidence in specific social class subgroups of the population. The complex entity of social class should be left undisturbed. What should be sought for specifically is the association between pre-existent chronic infection and subsequent incidence of atherothrombotic diseases adjusted only for the major disease-specific coronary risk factors that have been well identified.

G. DE BACKER
Cardiac Rehabilitation Center,
University Hospital,
Gent, Belgium

References


Myocardial Infarction trial (TIMI) grade 3 flow[1], with salvage of myocardium and preservation of left ventricular function. It is also well accepted that achievement of patency of the infarct-related artery between 6 and 12 h results in a reduction in mortality, even though this is beyond the 6 h time window for myocardial salvage. Can achievement of patency beyond this time be beneficial?

In this issue, Meneveau et al.[2] report a non-randomized study in which they aimed to determine whether angioplasty of stenoses or occluded infarct-related arteries 7–14 days after infarction in patients treated with thrombolytic therapy affected the process of left ventricular remodelling by 4–6 months. Angioplasty of patent arteries with stenoses had no effect on left ventricular function, whereas angioplasty of occluded infarct-related arteries which subsequently did not reocclude in 19 patients resulted in a non-significant 7% decrease in the end-systolic volume index and a non-significant 15% increase in ejection fraction. However, if reocclusion did occur, as it did in 53% of patients, end-systolic volume increased non-significantly by 4% and ejection fraction by 5%. The success rate of angioplasty in patients with occluded vessels was 53%, and 37% of these vessels had reoccluded by 4–6 months. The frequency of stenting is not reported, but is likely to have been low.

Viable hibernating ischaemic tissue may recover contractile function with late reperfusion. The time window for salvage of viable myocardium and prevention of left ventricular remodelling may be 2–3 weeks[3] or even longer if collaterals are present.

The major effect of late reperfusion is reduction of left ventricular remodelling and end-systolic volumes. End-systolic volumes have been shown to be a more important long-term prognostic factor than ejection fraction[4]. Even in the absence of myocardial salvage, late reperfusion results in less infarct expansion, decreases left ventricular dilatation, increases left ventricular mass and preserves ventricular geometry. Blood flow in an infarct-related artery may have a number of beneficial effects besides salvage of myocytes. By changing the nature of necrosis from the coagulation type to contraction band necrosis, there is persistence of sarcolemmal tubes which prevent collapse of the necrotic zone. Reperfusion promotes more rapid healing, with an increase in mature type I collagen, which imparts rigidity with greater resistance to distension. This effect may offset the lower collagen content that is found in reperfused infarcts. In addition, more rapid healing may promote phenotypic conversion of fibroblasts to myofibroblasts which are capable of contraction and can potentially resist distension. Also, late reperfusion provides a blood-filled coronary vascular bed, and the arteries and veins may act as a scaffold to reduce remodelling and dilatation. Furthermore, an open artery late after myocardial infarction is associated with greater electrical stability, with a lower incidence of signal-averaged electrocardiographic late potentials and ventricular arrhythmias. Another potential mechanism of benefit is the capacity of a patent infarct-related artery to provide collaterals to another coronary artery territory should further infarction occur.

Observational reports prior to the thrombolytic era suggested that late infarct artery patency connotes a favourable outcome after acute myocardial infarction. The univariate long-term prognostic importance of a patent infarct-related artery after thrombolytic therapy has also been shown by a number of groups.

The first study to show the late survival benefit of a patent infarct-related artery independent of left ventricular function followed 312 patients for 3–5 years[5]. Coronary angiography was performed 4 weeks after a first infarction. There was a relationship between the distribution of the infarct-related artery and prognosis, depending on whether or not the ejection fraction was normal. For patients with ejection fractions of <50% even small occluded arteries supplying <25% of the left ventricle adversely affected prognosis, whereas if the ejection fraction was >50%, only occluded infarct-related arteries supplying >25% of the left ventricle adversely affected prognosis.

Welty et al. recently reported a follow-up of 505 patients for 34 months after angioplasty for post-infarction myocardial ischaemia 2–90 days after myocardial infarction[6]. The hazard ratio for death among patients with closed vs those with open infarct-related arteries was 6·1 (95% confidence interval 1·8–20). The status of the infarct-related artery was not associated with a difference in mortality in patients with ejection fractions of ≥50%. Inducible or spontaneous ischaemia after myocardial infarction is associated with increased mortality, and only patients with post-infarction ischaemia were included in this study.

The risk of angioplasty in patients with occluded arteries is low, and success rates outside acute infarction and in the absence of stenting are around 70–80% depending on angiographic variables and the duration of the occlusion. However, without stenting, restenosis is common, with reported rates of up to 77%. In a recent small, randomized trial of stenting of coronary occlusions more than 2 weeks old in 119 patients, stenting reduced restenosis from 74% with angioplasty to 32% (P<0·001), and reocclusion within
6 months was reduced from 26% to 12% \((P=0.06)\)\(^{[7]}\). This study used low stent deployment pressures (<12 atmospheres) and conventional warfarin anticoagulation for 3 months. Improved results would be expected with high deployment pressures and ticlopidine rather than warfarin therapy.

The non-randomized study by Meneveau et al. shows that left ventricular function may be improved when occluded infarct-related arteries are opened and remain patent, which should result in an improved survival. However, this needs to be shown in a large trial with sufficient statistical power for this endpoint. The current study also lacks the power to quantitate the risks of such an approach. Information on cost-effectiveness would also be helpful. A large, randomized trial is urgently needed to compare angioplasty with stenting, and perhaps adjunctive IIb/IIIa receptor antagonists vs conservative management in patients with occluded arteries and without inducible ischaemia. It will be our folly if we delay too long before doing such a trial to obtain answers to the many tantalizing questions raised by this study.

**H. D. WHITE**

*Green Lane Hospital, Auckland, New Zealand*

### References


