associated with increased mortality in the setting of incessant tachycardia. This was also the experience of more recent investigators using surgery. Other investigational approaches never fulfilled their initial promise.

Therefore, the work of Cao and Gonska is very important. It confirms earlier reports on sustained ventricular tachycardia, and proves not only that it is possible to localize the area of slow conduction and interrupt re-entry in patients with ischaemic heart disease (and possibly with large aneurysms), but also that this can be achieved very successfully in critically ill patients; the complication rate is surprisingly low. The fact that there are recurrences of tachycardia during the follow-up is no surprise, but as implantable cardioverters/defibrillators are available these situations can be managed. These results are so encouraging that a more aggressive approach may be possible — also as regards paroxysmal sustained tachycardia — before proceeding to the implantation of devices. Briefly, the paper by Cao and Gonska is the herald of more work in the cath lab in the foreseeable future.

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more expensive treatment (simvastatin compared with cholestyramine) was the more cost-effective one.

The principal findings of Michel et al. are a cost of DFI 69 126 per additional survivor at 4 years and a cost of DFI 15 799 per life-year gained over 20 years. Note that additional survivors are of course very much more expensive than life-years gained, so that these figures are not comparable. However, they also give an incremental cost-effectiveness ratio of DFI 22 887 per life-year gained over 4 years. This makes for an interesting comparison whereby it is more cost-effective to treat for 20 years than it is to treat for 4 years. We have concerns about an extrapolated 20-year survival in patients with left ventricular systolic dysfunction post-infarction. Notwithstanding, it is good to have at least a suggestion that angiotensin-converting enzyme inhibitors do not merely delay the costs of treating heart failure.

Michel et al. conducted a detailed sensitivity analysis, both univariate and multivariate. Univariate sensitivity analysis reveals that the economic analysis is most sensitive to the incidence of chronic heart failure, the efficacy of treatment, the cost of treatment and the dose of treatment. It hardly needs to be said that treatment will become more cost-effective when the drug patent expires. It is also somewhat obvious that cost could be reduced by using lower doses, but in the absence of evidence about the efficacy of such lower doses, it is impossible to comment further on their cost-effectiveness.

Michel et al. also examine the application of their results nationwide. They find that treatment would reach a steady state in 2035, 37 000 patients would be treated, 2825 life-years would be gained during each year of treatment, and this would cost DFI 42 million or 10-3% of the national budget for treatment of heart failure.

To many clinicians an isolated cost per life-year gained has little meaning. With many studies it is difficult to place such a result in a meaningful context. This is because a major weakness of health economics at present is a lack of comparability of studies. Different groups report on different treatments, analysed by different methods, in different health-care systems. To make choices, clinicians need to be able to make direct comparisons of treatments. A particular strength of the current analysis is that it is one of a stable of such analyses from the Institute of Medical Technology Assessment, including one of the use of angiotensin-converting enzyme inhibitors in chronic heart failure.

The most obvious comparison of the current paper is thus with the same institute's economic analysis based on an application of the results from the treatment arm of SOLVD to the Netherlands[5]. In broad agreement with other studies, the Rotterdam group found that angiotensin-converting enzyme inhibitors in symptomatic heart failure could 'save' money overall, so that the comparison is not a favourable one. One reason for this is that the clinical benefits of a preventative treatment of asymptomatic patients are less than those of treatment of symptomatic patients (CHF hospitalizations prevented per 1000 patient years of treatment=10 in SAVE, cf. 65 in SOLVD-treatment). It is not surprising that economic comparison is also unfavourable. This is not, however, the whole story. The same institute has shown the potential for cost-savings with warfarin post-myocardial infarction, based on the results of the ASPECT trial[6]. This showed that secondary prevention could represent a 'win–win' situation, that is, one where costs decrease and benefits increase. On the other hand, comparison between the ASPECT analysis and the SAVE analysis has repercussions for the rigour of the latter. While the difference between the two economic analyses stems principally from the greater cost of captopril compared with warfarin, it also reflects the fact that the SAVE analysis takes no account of an effect on revascularization, whereas the ASPECT trial did.

Michel et al. attempt to put their figures into some sort of context themselves with a comparison against the cost-effectiveness of examples of primary prevention such as cervical cancer screening, cholesterol-lowering treatment and breast-cancer screening. In fact, the cost-effectiveness quoted compares favourably not just with other measures of preventative practice, but also with other more therapeutic manoeuvres[5].

The economic analysis of Michel et al. marks a new chapter in the economic analysis of the use of angiotensin-converting enzyme inhibitors and potentially a new chapter in the use of angiotensin-converting enzyme inhibitors in the period following myocardial infarction. These authors make a case for the cost-effectiveness of selective preventative therapy with angiotensin-converting enzyme inhibitors after myocardial infarction, which at least stands comparison with other examples of secondary prevention, as well as symptomatic treatment or primary prevention.

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Chlamydia and coronary heart disease

See page 682 for the article to which this Editorial refers

Miettinen et al. report a prospective study among two populations in East and West Finland among diabetic and non-diabetic subjects from the general population. A strong association was found between elevated levels of antibodies to *Chlamydia pneumoniae* in non-diabetic subjects from East Finland and the development of myocardial infarction after 7 years of follow-up. This association could not be explained by possible confounding factors such as smoking habit, age, sex, hypertension and lipid levels. In contrast, this association could not be detected among diabetic subjects in either East or West Finland in which the incidence of myocardial infarction was high, or in West Finland among non-diabetic subjects where the incidence of myocardial infarction was particularly low. It is possible that the heightened risk associated with diabetes smothers the excess putative risk of *Chlamydia* infection.

This is the latest of a series of reports linking *Chlamydia pneumoniae* with coronary heart disease and atherosclerosis. Some of these reports, based on case-control studies, must be interpreted cautiously because of possible selective survival/fatality effects. *Chlamydia pneumoniae* (TWAR strain) has become established as a common cause of atypical pneumonia in adults; extrapulmonary manifestations include myocarditis and endocarditis. The incubation period is long, asymptomatic infection may be far more common than symptomatic infection, and the prevalence of antibodies is high in the general adult population. Chronic infection and recurrence may be a feature in common with other diseases caused by related organisms, which include trachoma and psittacosis. An epidemiological association between an epidemic outbreak of *Chlamydia* infection (psittacosis-LGV agent) and primary myocardial and respiratory disease was reported from the United States (Illinois) in 1971.

Other micro-organisms, particularly those associated with myocarditis, endocarditis and also with chronic infections, have also been linked to atherosclerosis. These include cytomegalovirus (CMV) and *Helicobacter pylori*.

A recent cross-sectional population study in south London looked at the prevalence of *Helicobacter pylori* and *Chlamydia pneumoniae* antibodies in relation to the presence of cardiovascular disease. The associations with acute-phase reactants and other risk markers for coronary heart disease were also studied. The authors noted significant associations between both *Helicobacter pylori* and *Chlamydia pneumoniae* antibodies and fibrinogen concentrations, and also between *Helicobacter pylori* and total leukocyte count. The authors propose that the link with cardiovascular disease may be in response to chronic low-grade infection. More specifically, possible mechanisms would include: invasion and activation of macrophages, and possibly other white blood cells, by micro-organisms, stimulation of plasma elements such as fibrinogen or tissue elements such as arterial smooth muscle cells. Factors such as smoking habit and social class may be important confounding factors. Cytomegalovirus has been shown to invade the vascular endothelium in immunocompromized subjects but cytomegalovirus vasculitis is most frequently associated with the gastrointestinal tract.

This is a promising area of multidisciplinary research which may involve pathologists, microbiologists, physicians and epidemiologists. A full understanding of the epidemiology of each infectious agent under investigation is required. Modern