Infarction (TIMI) 14 trial that addition of the IIb/IIIa inhibitor to reduced dose thrombolytic therapy significantly improved myocardial perfusion as assessed by ST segment resolution[6]. Thus, this simple tool (that all clinicians can use by simply obtaining a 12-lead ECG 90 min after the start of thrombolysis — and comparing it to the baseline ECG), allows determination of several key aspects of response to therapy and assessment of prognosis.

The second area of importance of the ST segments is in assessing prognosis. This is true for patients with ST elevation myocardial infarction (with either the baseline ECG, or as noted above, using the change from baseline to 90 or 180 min), and for those with non-ST elevation acute coronary syndromes[7]. In the latter case, presence of just $\geq 0.5$ mm ST segment depression has been found to confer as bad prognostic significance as the more traditional $\geq 1$ mm ST depression. It should be noted that for ST elevation myocardial infarction, the significance of 0-5 mm ST elevation has not been evaluated.

For me, the take home message is: Pay close attention to the ST segments! They are helpful in (1) the initial diagnosis of myocardial infarction, (2) evaluating the response to therapy, and (3) predicting future prognosis. If we carefully scrutinize the 12-lead electrocardiogram, it should assist us in targeting appropriate therapies for the broad group of patients with acute myocardial infarction and acute coronary syndromes.

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Dilated cardiomyopathy, are a few drinks allowed?

The incidence of alcohol as the cause of dilated cardiomyopathy has been reported at between 20% and 45%[1,2]. These data, however, originate from observational studies and do not allow for discrimination between alcohol as the primary cause and alcohol as a contributing or aggravating factor for heart failure in dilated cardiomyopathy. The latter distinction may become important in view of the increasing awareness of inherited gene defects as important causes of dilated cardiomyopathy. Recently the frequency of familial dilated cardiomyopathy has been reported as up to 35% (instead of the formerly assumed 10%)[3]. Therefore, some cases of dilated cardiomyopathy identified as alcohol-induced may have been caused by inherited defects. In such cases excessive alcohol consumption may have promoted dilatation of the heart chambers rather than have caused it. Alcohol may also have contributed to the development of heart failure in patients with asymptomatic dilated cardiomyopathy. The larger than expected genetic origin of dilated cardiomyopathy might also explain the reported individual variations in the amounts of alcohol necessary to induce cardiomyopathy.

Data on the exact minimal amount and duration of alcohol consumption to cause cardiomyopathy are

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inaccurate. In addition, we do not have information on the extent to which the development of heart failure in familial/genetic forms of dilated cardiomyopathy are influenced by alcohol consumption. Based on observations in the past, it is generally accepted that the consumption of more than 80 g of alcohol daily (±8 units of alcoholic beverage) for at least 10 years is required to cause dilated cardiomyopathy.

In daily clinical practice, however, a contributing role of lower amounts of alcohol is observed: each year several patients referred to our centre for heart transplantation improve to NYHA class I or II and are removed from the transplant waiting list after cessation of daily consumption of 2 to 6 units of alcohol for far less than 10 years.

Specific mechanisms by which alcohol might produce myocardial dysfunction are impaired sarcoplasmic reticular uptake of calcium, inhibition of myosin ATPase, elevation of intracellular Na⁺ and water, inhibition of the Na⁺-K⁺ ATPase and alterations in the incorporation of membrane fatty acids and phospholipids. This knowledge, mainly gained from animal experiments, has not up to now resulted in specific treatment modalities for alcohol-induced cardiomyopathy. Based on observational studies in humans, the key to treatment of alcohol-induced cardiomyopathy (or cardiomyopathy influenced by alcohol) is immediate and total abstinence of alcohol as early as possible. Evidence for the benefits of such a regimen in terms of clinical parameters and prognosis is found in a several older and one more recent albeit small study of 23 patients[4–6]. In these studies, patients with alcohol-induced cardiomyopathy who abstain from drinking have a much better prognosis than patients who continue alcohol consumption and patients with alcohol-induced cardiomyopathy who abstain have a better prognosis than patients suffering from idiopathic dilated cardiomyopathy.

In contrast with previous studies Fauchier et al.[7] (this issue) report equal bad outcomes of patients with idiopathic dilated cardiomyopathy and patients with alcohol-induced cardiomyopathy who abstain from alcohol. At a first glance, when reading is limited to the abstract, these results seem strikingly new and disappointing to the cardiologist, but may fulfil the wish of many patients to resume drinking. Looking more closely, however, it appears that in the study of Fauchier et al.[7] the 10 year survival rates of both groups are comparable to the survival rates of the combined study population (including patients with idiopathic as well as with alcohol-induced cardiomyopathy) of the recent study by Prazak et al.[6]. The latter study, however, showed a substantial difference in survival between patients with alcohol-induced cardiomyopathy, who participated in alcohol withdrawal programmes and refrained from alcohol intake and patients with idiopathic cardiomyopathy, in favour of the former patients. An explanation of these discrepancies can be found in the deviating ‘definitions’ of the groups of patients studied by Fauchier et al.[7]; in the group considered to suffer from idiopathic cardiomyopathy almost 60% of the patients still consumed between 2 and 8 drinks per day and in the group considered to suffer from alcohol-induced cardiomyopathy and to refrain from alcohol, less than half of the patients were really abstinent. Apparently, investigators have different views about the amount of alcohol considered ‘social’ and ‘good for your health’ and what is harmful. This may be partly explained by their country of residence.

We cannot rule out that the prognosis of the patients in Fauchier’s idiopathic cardiomyopathy group was negatively influenced by their moderate alcohol consumption. In addition we have to reckon with failure of improvement of the patients in the alcohol-induced cardiomyopathy group because they did not become totally abstinent. A negative influence of alcohol consumption in amounts less than the presumed harmful 80 g per day therefore could explain the findings of Fauchier et al.[7]. The combination of the results of Prazak et al.[6] and Fauchier et al.[7] therefore seem to strengthen the evidence that complete withdrawal of alcohol as early as possible is necessary to improve prognosis in alcohol-mediated cardiomyopathy and provide a negative answer to the question ‘are a few drinks allowed in dilated cardiomyopathy?’.

In order to clarify the influence of alcohol on the development of dilated cardiomyopathy, as well as on the development or aggravation of heart failure in idiopathic or familial cardiomyopathy, a large trial consisting of the follow-up of several cohorts of patients (youngsters, middle aged and elderly patients, men as well as women) for at least 10 years will be necessary. Subjects of interest in such a trial could also be: (a) the influence of alcohol on myocardial dysfunction caused by ischaemic heart disease, (b) the effects of today’s optimal medical therapy including ACE inhibitors (and/or ATII antagonists?), beta-blockers, loop diuretics and spironolactone on the clinical course and (c) the question, why are women more sensitive than men to the toxic effects of alcohol on the heart[8]? In view of the impending growth of the number of heart failure patients and of drinking habits in Western industrialized countries such a trial appears worth the necessary financial costs.

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