consistently increased standardized mortality ratio for cardiovascular disease in Type 1 diabetes[1].

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European Heart Journal (2001) 22, 2047–2049

Predicting the coronary risk in primary and secondary prevention. What is the difference?

See page 2047, doi:10.1053/ehj.2001.2544 for the article to which this Editorial refers

Prevention of coronary heart disease in clinical practice results from assessing an individual’s global risk[1]. For individuals without clinical manifestation of atherosclerotic vascular disease, estimation of risk is currently based on the risk functions derived from the Framingham study[2]. As early as 1994 the European Recommendations on coronary prevention were based on calculated risk scores in the form of a Coronary Risk Chart[3]. This was extended in the new version[1] to include individuals with diabetes. It has been stressed that subjects with established coronary heart disease or other manifestations of atherosclerotic vascular disease are at very high risk over 10 years. Similarly, although diabetics are at high risk of manifesting atherosclerotic vascular disease[4], these risk charts were not intended for patients in the secondary prevention setting.

The role of standard coronary heart disease risk factors in predicting the long-term risk of recurrent coronary events in survivors of myocardial infarction was examined in the Framingham cohort as early as in 1989[5]. The age-adjusted analyses of about 500 survivors of myocardial infarction showed the risk of reinfarction to be positively associated with blood pressure and serum cholesterol, and coronary death with blood sugar, systolic blood pressure, serum cholesterol, heart rate and diabetes. The relative body weight was inversely associated with reinfarction for reasons that are not clear. Women appeared to be at
a higher relative risk of reinfarction and death than men, however, their risk adjusted to conventional risk factors was only a half. Thus, in persons surviving myocardial infarction the conventional risk factors were shown to be relevant, particularly blood pressure, serum cholesterol and diabetes. About half the risk associated with cigarette smoking appeared to be attributed to high fibrinogen concentrations and increased white blood cell count.

Numerous studies have found that several baseline variables are important predictors of risk of death after myocardial infarction or acute coronary syndromes. Most important among them are age, signs of congestive heart failure, diabetes and the presence of ST depression. The specific marker of myocardial damage is troponin T, which is more sensitive than the commonly used creatinine kinase MB isoenzyme level. These are particularly valuable for estimation of short-term risk. On the other hand, the long-term risk of recurrence and death mainly depends on the activity of atherogenetic and thrombogenetic processes. They depend on the inflammatory activity within the atherosclerotic plaque. The sensitive markers are acute phase proteins whose levels increase in response to inflammation. C-reactive protein levels and fibrinogen are suggested to reflect inflammatory activity. Moreover, fibrinogen has a key role in the coagulation cascade, platelet aggregation and plasma viscosity.

There was much evidence on the importance of the role of single risk markers for the prediction of recurrent myocardial infarction and/or coronary death. However, the equation which quantitatively estimates coronary risk is not available for post-myocardial infarction patients, in contrast to high risk individuals, and the primary prevention scene. To elaborate a simple, comprehensible, quantitative model for long-term risk estimation of individuals after myocardial infarction was not an easy matter. In this issue, the GISSI investigators used the GISSI-Prevenzione Trial to assess the absolute and relative risk after myocardial infarction and to construct coronary risk charts suitable for wide use in clinical practice to predict the global risk. The GISSI Prevenzione Study included 11 324 patients without age limit and without signs of congestive heart failure, who were randomized to dietary supplement groups <3 months after myocardial infarction using either capsules containing about 850 mg of n-3 polyunsaturated fatty acids (eicosapentaenoic acid and docosahexaenoic acid in an average ratio 1:2) and 300 mg of vitamin E. The patients were asked to adhere to treatment with aspirin, beta-blockers, ACE inhibitors, and statins, if recommended prior to inclusion in the study. The n-3 polyunsaturated fatty acids treatment resulted in a 10–15% decrease of relative risk of death, non-fatal myocardial infarction and non-fatal stroke. During the 4-year follow-up more than 1000 death were recorded, i.e. the risk was only about 9%, little more than 2% yearly. In spite of the low global risk of the population, about 42% were smokers, 36% hypertensives, 15% diabetics, 15% obese, 12% had previous myocardial infarction and 86% an ejection fraction higher than 40%. More than a half (55%) were in NYHA I. The future risk was calculated in a multivariate model. Except for complications of the myocardial infarction the classical risk factors were also included. The multivariate beta-coefficients for risk were calculated and translated into a numerical score attributable to each of the included factors. Besides age and sex and presence of left ventricular dysfunction, the following risk factors were particularly attributed to global risk: diabetes, smoking, claudication, elevated leukocyte count and fibrinogen, and low HDL-cholesterol. As shown in the coronary risk chart, patients’ risk after myocardial infarction varies considerably. Individuals, males or females, aged less than 50 years, without left ventricular dysfunction, arrhythmias, ischaemia, hypertension, diabetes or claudications, and who were non-smokers with normal laboratory values of HDL-cholesterol, fibrinogen and leukocytes had a 4-year risk of death <2%. In contrast, older patients, whose risk scores were high were at an absolute risk >30%. The absolute risk of death, according to the risk score, varied between 0.9–83.9% in men and 0.7–61.6% in women. The relative risk was 5–7 times higher in younger subjects (<50 years) than in those who were older with the same number of risk score points.

The tables and the risk charts enable the practicing cardiologist, or other physician to consider what changes in absolute risk may be expected when proper therapies reach the treatment targets. Obviously, in younger patients with the highest relative risk it is possible to reduce the risk in units of 10%. However, the absolute risk can only be reduced in units of 1%. By contrast, in older patients, the relative risk will be reduced by units of 1% with proper treatment, and absolute risk by units of 10%.

It is well known, and has been published widely, that patients with established coronary heart disease or other atherosclerotic diseases are priorities for preventive cardiology. Their risk is generally considered to be as high as 20–40% over the next 10 years. This very rough estimate can be rendered more specific by calculating the post-myocardial infarction global risk score, as published in this paper. It deserves great attention by epidemiologists and practising physicians.
The application of global risk scores, to estimate long-term prognosis after myocardial infarction, enables the effective tailoring of medical intervention to prevent coronary recurrences or deaths. The scores and tables were constructed from data of a low risk Mediterranean population. Therefore it would be necessary to evaluate them in the larger European population. The European Heart Survey Studies on acute coronary syndromes or secondary prevention offer an opportunity to verify the validity of global risk scores.

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European Heart Journal (2001) 22, 2049–2051

Angiography and revascularization after thrombolysis: where are we?

See page 2104, doi:10.1053/euhj.2001.2622 for the article to which this Editorial refers

Thrombolysis therapy is the most commonly used reperfusion strategy after acute myocardial infarction. It is still unclear whether coronary angiography after thrombolysis should be available routinely for all patients after thrombolysis or targeted to specific groups.

In this issue, Llevadot and colleagues present an analysis of practice patterns following thrombolysis for acute myocardial infarction using the InTime II dataset[1]. InTime II was a randomized trial of 15 078 patients enrolled from 855 hospitals comparing the effects of two thrombolytic agents, lanoteplase (nPA) and alteplast (tPA) on clinical outcomes. The results of InTime II showed that these two agents had very similar effects on mortality rates, but there was a suggestion that lanoteplase was associated with a higher rate of cerebral haemorrhage[2]. In the present analysis they compare rates of angiography and revascularization across three hospital groups: those with