is the clustering and severity or risk factors that influence endothelial function rather than the presence of the single risk factor. The severity of each risk factor has its importance in the single patient since for example in patients with diabetes the incidence of erectile dysfunction varies according to the severity, duration of the disease and the use of insulin. In any case, since none of the study patients had erectile dysfunction at baseline it is clear that the distribution of risk factors did not have had any relevance in causing a different incidence of ED between groups.

Jaarsma et al. also suggest that the difficulty to discuss ED due to embarrassment or to ignorance may make easier to ‘blame it to a pill’ in the ‘atenelol-know side effects’ group patients but forget to consider that all patients had already received a questionnaire at baseline when they reported no ED.

As stated in the manuscript ED was diagnosed and defined according to the validated IIEF questionnaire taking into account the score obtained from the answers on the erectile function domains. Patients were diagnosed as having ED if they had a score compatible with the international diagnosis of ED moderate or severe. None of the patients reported a score compatible with mild ED (“minimal” is not an internationally accepted definition) in the erectile function domains of the IIEF questionnaire.

Jaarsma et al. mention that a 16% incidence of ED is high compared with the general population without knowing the exact incidence of the problem in a population of normal subjects of the same age as that of our patient population is around 65%.

The comment of Jaarsma et al. on the fact that the conclusions of our article suggest that “patients might benefit if health care providers withhold information from patients about the potential sexual side effects of medications” is an over- and misinterpretation of our conclusions. Since by saying that the expectations of side effects may influence the occurrence of erectile dysfunction we never said that doctors should withhold a complete information to patients. Conversely we strongly believe that the results of our study will help doctors to give a clear explanation on the side effects of the drug thereby increasing patient compliance.

References


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(A. Silvestri)


Beta-blocking agents and erectile dysfunction after acute myocardial infarction: guilty or innocent?

We read with interest the paper by Silvestri et al.1 on the report of erectile dysfunction (ED) after therapy with beta-blockers. An acknowledged preliminary look at an important topic. The author found that the incidence of ED was 3% in the group not knowing which drug they were taking, 16% in the group knowing that they were receiving a beta-blocker and 31% in the group also knowing the side effects of the drug (p = 0.01). I agree when they suggest that report of ED in patients receiving beta-blockers may be mostly psychological in origin. We also presented at ESC Congress 2002 the study: Beta-blocking agents and erectile dysfunction after acute myocardial infarction: guilty or innocent?2 We surveyed 37 male patients that prior to the AMI had an active sexual life, without ED. They filled out two questionnaires: The International Index of Erectile Function (IIEF – Erectile Function – a summation of Questions 1–5) and the Self-Report Questionnaire (SRQ) that was developed by the WHO for the screening of diseases, such as anxiety-related disorders and depression.3 A ranking higher than or equal to seven signaled “mental distress”. Of them, 91% reported resumption of sexual activity after AMI. After six months, 15 (40%) presented with ED, with a mean score of 14.7 (IIEF). Of nine patients with distress, eight presented with erectile dysfunction and of 28 without distress, 7 presented with erectile dysfunction (89% × 25%, p = 0.001). Distress was a associated variable of erectile dysfunction postinfarction and we did not detect a difference with regard to the incidence of ED among the groups with or without beta-blockers (36% × 55%, p = 0.4). Distress may reduce erotic focus and therefore reduce psychogenic incitement, thereby interfering with the normal sexual cycle, jeopardizing male erection. Furthermore, by means of brain signals, psychological problems may also inhibit the parasympathetic activation or aggravate the sympathetic response, influencing the sexual normal cycle of men.4 Also, erectile dysfunction in hypertensive individuals may be related more to hypertension level than to drug treatment and beta-blocking agents is not more likely to be associated with sexual dysfunction than placebo.5

References


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(L.J. Vacanti)


Beta-blockers, psychological influences and erectile dysfunction: Reply

The letter of Vacanti et al. highlights the growing interest and importance on the
relationship between cardiovascular disease (CVD) and erectile dysfunction (ED). The problem of ED was underestimated before the introduction of PDE5 inhibitors and was rarely investigated in patients with CVD. We agree with Vacanti et al. that distress in patients after an acute myocardial infarction (MI) may be associated with ED. However, we believe that the problem of ED in patients with CVD is more complex and encompasses several different pathogenetic causes. Psychological influence has its importance but the atherosclerotic process and the pharmacological effect of CV drugs have a pivotal role.

It is proven that patients with cardiovascular risk factors for CVD have a significantly higher incidence and severity of ED than patients without risk factors, and that in patients with CVD the incidence and severity of ED is related to the degree of coronary atherosclerosis. Since CV risk factors impair endothelial function even when they are subclinical, it is reasonable to believe that the impairment of endothelial function may unmask ED before any clinical manifestation of atherosclerosis. In a vascular bed highly dependent on NO-induced vasodilatation as the penile circulation, the impairment of endothelial function causes ED. As shown by Schachinger et al. in the coronary and peripheral circulation, where other mechanisms of regulation of blood flow are present, angiographically evident atherosclerosis follows by several years the initial impairment of endothelial function. In deed, recent studies have suggested that in patients with a recent MI, ED preceded the clinical manifestation of CVD by several years and that amongst patients with vasculogenic ED, otherwise asymptomatic for CVD, there is a high prevalence of coronary atherosclerosis. On these grounds, it is plausible that psychological and pharmacological influences may act on a dysfunctional endothelium precipitating ED that is in any case a sign of an impaired endothelial function.

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Comments on the European guidelines on cardiovascular disease prevention

Dear Editor

We have read the European guidelines for the prevention of cardiovascular disease (CVD) published in the September 2003 issue of the European Heart Journal with enthusiasm. Evidence-based clinical practice guidelines have become an invaluable tool for clinicians, especially in the field of Cardiovascular Medicine with such a high rate of continuously growing knowledge. Interestingly, the Third Task Force urges the need for the establishment of national strategies for CVD prevention. Moreover, it is encouraging that the guidelines also address the difficulties involved in the behavioral counselling and propose some strategies on how to deal with these difficulties.

The Task Force suggests the use of the SCORE model published earlier by Conroy et al. This model has several advantages, as it provides a quick and simple means to calculate the global CVD risk, while it allows the projection of the risk of a certain individual to a higher age as well as the estimation of the effect of risk factor modification on this risk. Furthermore, the SCORE charts offer a graphical and quite comprehensible way to explain the risk factor modification concept to the patients and motivate them to participate actively in the counseling procedure. However, the SCORE model underwent a considerable criticism by Assmann et al. in a recent issue of the Journal. In contrast to what Assmann et al. state in the conclusion of their letter, we believe that a simplified system does not necessarily compromise the provision of individually-tailored guidance. Moreover, despite the fact that some risk factors are more epidemiologically “specific” for the one or the other component of the cardiovascular system, we do agree with the SCORE approach in treating cardiovascular system as a whole entity, as the differentiation among coronary, cerebral and peripheral arterial disease is neither feasible nor rational in the context of a preventive strategy. It seems to us that the main weakness of the SCORE model is the omission of several CVD risk factors, including both traditional and recently established ones, as also stressed by Assmann et al.

Apart from the cases with an obviously high total CVD risk, including patients with already established CVD or diabetes and individuals with considerably high cholesterol or blood pressure levels, the guidance proposed by the Task Force in all other individuals is based on the total risk as calculated by the SCORE charts, using a threshold of 5% to define cases in high risk. However, individuals with a risk $\geq 5\%$ according to SCORE charts represent only a subgroup of the high risk cases. As a result, the guidance based only on the SCORE model is apparently inappropriate in individuals bearing risk factors that have not been incorporated in the SCORE charts. Therefore, although the SCORE system is quite an appealing tool for an initial risk assessment, treatment decisions and the total CVD prevention strategy should be based on the entire collection of risk factors, which are quite comprehensively presented by the Task Force in their recent report.

References


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