More activity, fewer drugs

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The subject of the paper by Bacharova et al. [1] is of great interest, because it constitutes an important contribution to the very topical discussion about the low levels of physical activity in the general population, not only among the elderly and middle-aged but also among children and teenagers, which makes the prognosis for the future worse.

The problem of sedentary life-styles, closely linked to obesity, arterial hypertension, dyslipidemia and hyperinsulinemia, is becoming ever more serious. These traits are, in turn, well-known modifiable risk factors of metabolic syndrome X, diabetes mellitus type II and cardiovascular disease, which in consequence further affect overall mortality. For instance, in the year 2000 cardiovascular disease was a cause of 16.7 million deaths worldwide [2].

Interestingly, it was shown that a reduction in the influence of risk factors results in a more effective decrease in cardiovascular mortality than advances in the diagnostics and therapy of this disease [3]. The INTERHEART Study in 52 countries revealed that six risk factors, namely hyperlipidemia, smoking, hypertension, diabetes, visceral fatness and psychosocial factors, in combination with three cardioprotective factors, these being fruit and vegetable consumption, low doses of alcohol and regular physical exercise, determined over 90% of myocardial infarction morbidity [4].

It has now been demonstrated beyond doubt by, for example, the pioneer epidemiological studies on the Framingham cohort that atherosclerotic cardiovascular disease is to a great extent a product of a “faulty life-style” [5]. A harmful modern life-style was for decades typical mainly of Western societies, the members of which were said to “eat too much of a too rich diet, exercise too little, grow fat and smoke too much”, as stated in 1970 by the Inter society Commission for Heart Disease Resources [6]. Unfortunately, in recent years these bad habits of civilisation have quickly spread to the developing countries too. According to the epidemiological prognosis, by 2020, owing simply to the share of the developing countries, ischemic heart disease will become the main cause of death in the world [7].

What makes the situation worse is that the youngest section of the population is also in real danger of incurring many of the severe conditions resulting from an unhealthy life-style. Because of the hold exerted by TV and PCs energy expenditure per day is currently 600 kcal less than it was in children 50 years ago [8]. The number of obese children is constantly growing worldwide, and this is caused by overeating, junk food and a sedentary life-style. This increases the risk of metabolic syndrome X, related pathogenetically with diabetes mellitus II and cardiovascular disease [9]. Up to 60% of obese children aged 5–10 years manifest one additional risk factor for cardiovascular disease, such as hyperlipidemia, hypertension or hyperinsulinemia, and in 25% of them two or more risk factors are identified [10].

It has, on the other hand, been ascertained that there is a clear correlation between children’s levels of physical activity and the significance of many risk factors, such as fat body mass, body mass index, total cholesterol, triglycerides, apolipoprotein B, insulin and blood pressure [11, 12].

It is 20 years since Kannel et al. [5] made the suggestion that intensity of physical activity is an important ingredient in a multifactorial cardiovascular risk profile, which not only modifies positively the other atherosclerosis risk factors, but maintains the integrity of the cardiovascular system. Ten years later, lack of physical activity, with special stress on recreational activity during leisure time,
was commonly accepted as one of the major independent risk factors of cardiovascular disease [13].

It has been reliably documented that systematic and long-term physical training, especially endurance one, leads to many positive cardiovasoprotective changes [14]. These changes include the physiological adaptation of the cardiovascular system, an increase in general physical capacity and alterations to modifiable risk factors. With respect to this, it has been found that physical training can lower systolic and diastolic blood pressure [15]. Furthermore, even moderate exercise causes a significant reduction in serum levels of total cholesterol, LDL-cholesterol and triglycerides and an elevation of HDL-cholesterol [16]. It has also been demonstrated that regular physical activity decreases blood levels of insulin and glucose [17] and reduces body mass index [18]. Endurance training shows additional anti-atherosclerotic effects by improving coronary endothelium function [19], lowering levels of CRP [20] and homocysteine [21] as well as prothrombotic status [22].

The findings of many long-standing epidemiological projects (e.g. the Framingham Study, MRFIT, Harvard Alumni Study and Nurses’ Health Study) have revealed that regular physical activity has an unassailable position in the prevention of cardiovascular disease [23–25] and of other conditions such as arterial hypertension, diabetes mellitus, osteoporosis, neoplasm and depression [26]. It has also been documented that regular physical training with an energy expenditure of 700–2000 kcal per week has resulted in a 25–30% reduction in all-cause mortality and in a 30–50% reduction in the risk of ischemic heart disease [13, 27].

The current recommendations concerning physical activity in the primary prevention of cardiovascular disease are as follows: endurance training lasting 20–60 minutes at a moderate intensity (40–60% VO₂max or 60–75% HRmax) performed a minimum of three times a week (supplemented by resistance training a minimum of twice a week), consuming at least 1000 kcal per week [28].

Promotion of regular physical training is incorporated into the three-level model of cardiovascular disease prevention which was proposed by Benjamin and Smith in 2002. In this model primary and secondary interventions are supplemented by essential prevention which embraces the entire population, from children to the very elderly [29].

In the context of “mass prevention” referred to above, the subject of the paper “Decrease in QRS amplitude in juvenile female competitive athletes during the initial twenty-one months of intensive training”, which concerns the adaptation of the cardiovascular system to systematic physical exercise performed by teenagers, is very topical. A group consisting of junior (13–17 year-old) female athletes were subjected over 21 months to almost daily gymnastic training comprising both endurance (aerobic) and resistance (anaerobic) exercises.

It is known that as a result of long-term, intensive, systematic and combined physical training the heart usually develops organic changes known as “athlete’s heart,” including enlarged ventricular chambers (eccentric hypertrophy) in conjunction with increased left ventricular wall thickness (concentric hypertrophy). The “athlete’s heart” is often associated with an increased QRS amplitude on 12-lead ECG recordings [30].

In routine medical practice a preliminary diagnosis of left ventricular hypertrophy (LVH) is still based on ECG voltage criteria, which are then verified by echocardiography. ECG is regarded as reflecting left ventricular thickness, according to the assumption that the greater the left ventricular mass, the stronger the cardioelectric field and the higher the QRS amplitude, whereas echocardiography is to estimate the size of the left ventricle. However, because it has been demonstrated that QRS amplitude and left ventricular mass correlate poorly, it is thought that ECG enables only a small proportion of real LVH cases to be detected. Additionally, over 50% of athletes with the augmented left ventricular mass do not manifest a QRS voltage exceeding the normal ECG limits [31].

The main conclusion of the paper under consideration is that, surprisingly, 21-month intensive physical training performed by young girls led to a decrease in QRS complex voltage, which is in contrast to the traditional assumption for diagnosing LVH using ECG criteria. In order to assess the QRS voltage parameters, the authors applied the standard Sokolow-Lyon index (SLI) as well the approximated maximum spatial QRS vector magnitude (QRSmax), which involves changes in the all three spatial components and eliminates the problem of the heart’s location in the thorax. Differences ascertained at the end of the follow-up period turned out to be statistically significant: the mean value of LSI decreased by 24%, and the QRSmax values decreased by over 30%. It should be emphasised that the possible influence of somatometric parameters and vagotonia, which could have accounted for the diminished QRS voltage, were excluded.

Seeking a reason for the decreased QRS voltage observed as the result of the regular and intensive physical training, the authors proposed an
alternative to the classic hypothesis, suggesting that the false negative ECG pattern in the evident left ventricular hypertrophy could be attributed to the relative voltage deficit caused by modifications of the active and passive electrical properties of the myocardium that occur during the initial phase of hypertrophy.

Because of the importance of the issue discussed, the paper considered here should, in my opinion, be treated as a preliminary report. It seems worth increasing the number of the group followed up in a further study (the present group of 12 girls is too small). It could also be beneficial to collect some information about the state of puberty of the subjects analysed, which might yield a possible insight into hormonal influence on left ventricular mass, and about energy expenditure (in calories/metabolic equivalents), determining the intensity level of physical training which could affect the changes in left ventricular mass.

The results reported by Bacharova et al. [1] are of a novel nature (there has only been one published paper to report a decrease in QRS due to the early stages of physical training), and the theoretical background presented by the authors is completed with an interesting concept. There is nothing for it but to express an optimistic hope that the pathomechanisms of exercise-induced cardiovascular system modifications of protective strength will be a challenge for an increasing number of researchers. Every attempt to encourage people to apply a greater dose of physical activity to everyday life should be vigorously supported. The saying that “Physical activity cannot be replaced by any drug” is still extremely relevant.

References


