Aerobic training in the elderly after a coronary event

See page 1638 for the article to which this Editorial refers

Spencer, in 1989, calculated that by the year 2000 13% of the population of the United States will be aged 65 or over; while in 1950 this figure was just 8%[1]. It is likely that other comparable societies will show a similar trend, and so it is easy to understand that the dimensions of the problem ‘elderly’ are considerable.

Many studies have demonstrated that ageing involves a decline in cardiovascular function, and if, for instance, some coronary event is superimposed on age, the result will be more evident. Among the age-related changes in cardiovascular function, of great importance are decreases in the responsiveness of the beta-adrenergic receptors, that are also involved after a coronary event[2]. The balance of the autonomic nervous system, as concerning the heart rate response, can be favourably affected by endurance (aerobic) training, and the hypothesis of Stähle and co-workers, that it could improve the heart rate variability, tending also to reduce a risk factor in elderly coronary patients, is encouraging[3]. This behaviour could be explained as a counteraction against the increase of the duration of cardiac contraction that occurs in the elderly[4].

If we consider that, among the functional changes secondary to endurance training in the elderly, we should include: reduced contraction time, the timing of the peak contractile tension and the dynamic behaviour relative to stiffness, which can be lowered to a level comparable with those of young sedentary subjects, it is likely that these changes could have a synergic impact on the improvement of the heart rate variability.

The data of Stähle and co-workers[3], as concerning the resting heart rate, are in agreement with those of Hagberg and Graves[5] who found that only the elderly fail to show a reduction in heart rate at rest following exercise conditioning, while the submaximal heart rates were 10 beats lower in an endurance trained group, for both males and females aged 70–79 years, as compared to a sedentary or power trained (weight lifting) group.

These data could indicate the need for a study investigating the possible effect of power (isometric) training on heart rate variability in the elderly. We have to stress, however, that some authors have observed that the heart rate, even at rest, is reduced in older athletes compared with sedentary subjects, while others failed to discover this reaction in either young subjects, young athletes or middle-aged men.

However, this effect probably results from the fact that these subjects were long-used to exercising. It would appear, therefore, that the response of the heart rate at rest depends on when exercise training is begun.

Another mechanism, indicating the favourable effect of exercise training in coronary patients on heart rate variability, could be that which occurs through increased vagal modulation owing to physical activity.

Malliani and co-workers[6] show that patients, at 2 weeks after a myocardial infarction, in comparison to control subjects, exhibited a significant increase in low frequency components of heart rate variability, as a quantitative index of increased sympathetic modulation, and diminished high frequency components, as a quantitative index of decreased vagal modulation. In the same patients, at 6 and 12 months after the myocardial infarction, the authors observed a progressive decrease in low frequency components and an increase in high frequency components, which suggests a tendency to normalization of the sympato-vagal interaction.
If an exercise training programme, which has the known effects on autonomic balance were added to this spontaneous tendency, it would be easier to accept that physical activity has a beneficial impact on heart rate variability thereby reducing a risk factor.

But if we can accept that physical training could improve autonomic balance, and if a spontaneous trend in this direction is present in coronary patients what happens to myocardial contractility, or more generally the mechanical efficiency of the heart? In other words, improvement of sympatho-vagal interaction due to physical training also plays a counteractive role on ventricular remodelling, and in doing so increases the process of risk reduction after a myocardial infarction? At the moment the question has no clear and definite answer. We probably need larger numbers of patients and a different approach to the analysis of the heart rate variability.

A good suggestion, in our opinion, could be that of our own experience\(^1\) in which to assess heart rate variability as a predictor of myocardial recovery from ischaemia or hypoxia, we used a non-linear dynamic analysis (fractal dimension), which appeared convenient because fractal dimension predicts arrhythmia recurrence in patients treated for life-threatening ventricular arrhythmias.

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**References**


[6] Malliani A, Pagani M, Lombardi F et al. Cardiovascular effects on autonomic balance were added to this spontaneous tendency ischaemia or hypoxia, we used a non-linear dynamic analysis (fractal dimension), which appeared convenient because fractal dimension predicts arrhythmia recurrence in patients treated for life-threatening ventricular arrhythmias.

**Vitamin C and coronary vasoreactivity**

See page 1676 for the article to which this Editorial refers

Vitamin C, ascorbic acid, has many functions including collagen formation and wound healing, but in this editorial I will focus on its antioxidant properties. Although ascorbate is not lipophilic, it can reduce LDL oxidative susceptibility. Therein lies its potential to attenuate the progression of atherosclerotic plaques and coronary heart disease prevention.

Oxidized LDL is thought to be one of the culprits important in the pathophysiology of atherosclerosis. Studies have generally shown reduced coronary heart disease risk in populations consuming foods high in antioxidant vitamins. However, it is not known whether antioxidant vitamins given as supplements to food will reduce cardiac events or prevent the development of atherosclerosis. The US Nurses Health Study reported 30–40% risk reduction in cardiovascular events in subjects taking the antioxidant vitamin E who were in the highest quintiles\(^4\).

Enstrom et al. in NHANES 1 (National Health and Nutrition Examination survey) reported data from >11 000 US adults\(^2\). Individuals reporting high intakes of vitamin C exhibited significantly lower risk of death from all causes, particularly from coronary heart disease over a 10-year period. Prospective trials of the antioxidant beta carotene in a 12 year randomized trial in 22 071 male physicians showed neither benefit nor harm in relationship to the incidence of cardiovascular disease or all-cause mortality. In contrast, the Cambridge Heart Antioxidant Study (CHAOS) of patients who had established coronary heart disease, showed a reduction in cardiovascular events but not in all-cause mortality in physicians using vitamin E (400 IU–800 IU per day)\(^3\).

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