Origin of heart failure: cardiac or generalized myopathy?

See page 1191 (issue 16) for the article to which this Editorial refers

The clinical syndrome of heart failure continues to amaze cardiologists as in the last 10 years we have observed a disruption of almost all of the ‘classical’ and ‘fundamental’ concepts which accompanied our clinical understanding of this syndrome:

- the neuroendocrine response, which was once believed to be a compensatory reaction of the organism to counteract the ‘failure’ conditions, is now considered a detrimental response of the body. As such, it is recommended that this response be treated\(^{[1]}\);
- positive inotropic drugs, which were considered the logical treatment solution for the failing heart, do — paradoxically — increase mortality when used chronically. As such, they are now dismissed as treatment of choice\(^{[2]}\);
- Beta-blockers, previously considered as dangerous drugs due to their negative inotropic effects, are now thought to exert positive inotropic effects, when administered as long-term treatment; these drugs also improve prognosis. As such, their use is recommended\(^{[3]}\);
- the old and logical recommendation ‘of-resting-in-bed’ is now considered obsolete and patients are submitted to routine physical training programmes.

This latter consideration comes from the idea that peripheral muscle metabolism and function are abnormal in heart failure. Thus, it has also been suggested that the disease itself can be a disease of the muscles and not only of the heart muscle. With this in mind, the issues highlighted in the paper by Opasich et al.\(^{[4]}\) (issue 16, pp. 1191–1200) address a relatively important concept and attempt to characterize the role of each peripheral muscle in determining the symptoms of the heart failure syndrome. The profile depicted in the paper by Opasich et al. is that ‘heart failure is not only a disease of the cardiac muscle but also a multi-organ disease involving skeletal and respiratory muscle impairments’.

This is probably the main message of the paper which, however, raises a series of problematic issues, such as: ‘Since myopathy is generalized in heart failure, why do we continue treating only one muscle, i.e. the heart?’ Should specific treatments for skeletal and respiratory muscles not also be used? Attempts to this end have recently been followed using the administration of metabolic agents\(^{[5]}\). However, efficacy was observed only in very selected subgroups of patients.

In addition, the excellent clinical results obtained with transplantation and some of the results obtained with re-vascularization in advanced ischaemic cardiomyopathy suggest that the abnormalities of the peripheral muscle may indeed be secondary to heart abnormalities, thus being reversible with the normalization of heart function.

Accordingly, the results of the paper by Opasich et al.\(^{[4]}\) show that the degree of myopathy differs among the cardiac, peripheral and respiratory muscles and, for instance, that the only true determinant for respiratory muscle weakness is age. This is another extremely important message of the paper as it provokes the following question: ‘How can we continue to diagnose heart failure according to the presence of dyspnoea, since dyspnoea could be a physiological pattern of ageing?’ To complete (or perhaps complicate) the puzzle, the authors also report that effort dyspnoea is not relieved by respiratory training. This suggests that it is not respiratory insufficiency which limits physical exercise. Therefore, what is it that actually limits physical performance? This remains a mystery.

The last issue raised by the results of the paper\(^{[4]}\) is that both skeletal and respiratory muscle forces are reduced and the degree of impairment is unrelated to clinical severity, neuroendocrine activation or the cardiac dysfunction occurring in heart failure. This finding suggests that the myopathy has an autonomous profile and is not a ‘passive’ consequence of the clinical syndrome. The results of the study show that peripheral weakness can also be present in mild heart failure and give no support to the hypothesis that central haemodynamic impairment is the cause of muscle weakness. On the contrary, the authors suggest that the level of exercise capacity of healthy subjects can be the determining factor for the muscle weakness which affects their quality of life when they become heart failure patients. In other words: ‘If things have to go wrong, it is worse if the starting point is already compromised’. The paper by Opasich et al. allows us the right to conclude: ‘Moderate exercise is good for you, for your patients and, considering the high prevalence of heart failure, also for us!’

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References


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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 Ståhle 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 Ståhle 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 sympatho-vagal interaction.

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