Editorial Comments

Beneficial Effects of Exercise Training in Compensated Heart Failure

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Over the last decade, major advances have occurred in our understanding of heart failure, although knowledge of this complex and increasingly prevalent clinical syndrome remains far from complete. Increasingly, attention has been accorded to changes in the peripheral circulation, both adaptive and maladaptive, that occur in response to decreased cardiac performance. Special attention also has been focused on the complex interactions that take place between the adrenergic nervous system, the renin angiotensin axis, and other vasoactive substances in response to impaired cardiac function. Indeed, success has been reported in the few studies in which this approach has been employed, but little information regarding the mechanisms responsible for the improvement in exercise tolerance is available. In the current issue of Circulation, Sullivan et al describe the effects of 4 hours each week of exercise training, equivalent by heart rate criteria to 75% of peak oxygen consumption, sustained for 16–24 weeks on hemodynamics, peak oxygen consumption, ventricular function and peak increase in blood flow to the leg. The investigators also measured the plasma concentration of lactate in arterial and femoral venous blood.

Successful measurement of radionuclide left ventricular ejection fraction, central hemodynamics, and femoral blood flow along with central and peripheral lactate determinations at rest and during submaximal and maximal exercise in patients before and after an exercise training period is an impressive accomplishment. A more ideal study design, using a matched control group not undergoing exercise training and an identical and unchanging drug regimen in all patients, was not attempted and is unlikely to be realized.

The study of Sullivan and coworkers clearly demonstrates that both central hemodynamic and peripheral metabolic adaptations to heart failure are improved by a period of exercise training comparable with that used in many cardiac rehabilitation programs. The study makes an important contribution to our understanding of how the organism adapts to heart failure and should influence our therapeutic approach to this important clinical problem.

Sullivan et al were unable to demonstrate any change in left ventricular ejection fraction brought about by the exercise training period, confirming that while this index of ventricular function is important in assessing the extent of myocardial damage and for prognosis, it is of no value in assessing the results of treatment in individual patients and is unrelated to the patients' ability to exercise and consume oxygen; consequently, it is unrelated to the symptoms of heart failure. This observation also confirms that usually it is difficult or impossible to prove that the benefits of cardiac rehabilitation programs can be attributed to improved

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In the same period, significant advances have been made in exercise physiology and in methods to assess the results of treatment for heart failure, which have changed radically as a consequence of recent developments in physiology and pathophysiology. Thus, prolonged bed rest for up to 1 year as treatment for heart failure has been abandoned; most physicians prescribe bed rest only during the acute unstable phase and place no subsequent limits on ordinary physical activity.

During this period of rapid advances in the field of heart failure, the concept has evolved that far from being detrimental to patients with heart failure, physical activity may be beneficial and lead to increased exercise tolerance. The absence of correlation between exercise tolerance and ventricular function and the increase in exercise duration observed in control subjects in all studies of pharmacological treatment of heart failure in which serial exercise testing is used, has advanced claims that training by exercise is an important factor in the therapeutic response and has shifted attention away from the heart and toward the exercising muscles.

The foregoing considerations have led to the conclusion that a program of cardiac rehabilitation with a prescribed exercise regimen might benefit

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myocardial function. It has been suggested that measurement of improvement in left ventricular ejection fraction during exercise may be a more sensitive means than measuring this parameter at rest for detecting beneficial effects of a treatment program for heart failure. However, in the study by Sullivan et al, no increase in left ventricular ejection fraction, measured either at rest or during exercise, was brought about by the training program. The signal-to-noise ratio of left ventricular ejection fraction is notoriously low in patients with severely depressed ejection fraction and, typically, drug treatment produces little or no increase and, therefore, is only revealed by large multicenter studies.

The most dramatic effect of exercise training was more than one MET increase in peak oxygen consumption, which was associated with an increase in exercise time of over 100 seconds and an increase of peak workload of almost 100 kpm. Increased peak oxygen consumption was not accompanied by lower pulmonary arterial or pulmonary wedge pressure, and the 1:1 increase in cardiac output observed at peak exercise did not quite reach statistical significance, but peak arteriovenous oxygen difference was significantly increased by exercise training. These data confirm that exercise tolerance and symptoms of heart failure do not correlate directly with increased pulmonary wedge pressure: the relation is more complex, being modified by the type and duration of exercise and by peripheral hemodynamic and metabolic factors.

Peak exercise was associated with a significant increase in oxygen delivery to the exercising leg. However, exercise training did not lower either systemic arterial or femoral venous lactate concentration or decrease the arteriovenous lactate difference or lactate production in the leg during maximal exercise. These results are congruent with the observation that exercise training corrects impaired peripheral vasodilation in patients with heart failure, as well as in normal subjects.

When data were taken at submaximal exercise, quite different results were observed. When interpreting these results, it must be recalled that after training, the patients exercised longer and harder. Compared with maximal exercise, at submaximal exercise, patients’ heart rates were lower, rather than higher after training, and the increases in arteriovenous oxygen difference were not observed. In contrast to peak exercise, no changes in the hemodynamics of the exercising leg were observed, but arterial and femoral venous lactate concentrations, femoral arteriovenous lactate difference, and leg lactate production were substantially reduced.

The different responses reported by Sullivan et al at peak exercise compared with responses at submaximal exercise highlight the need for well-defined means of assessing exercise responses in patients with heart failure. The peak exercise response measures the maximal cardiovascular-pulmonary performance of which the patient is capable; furthermore, patients with heart failure often cannot sustain such maximal effort. Therefore, maximal oxygen consumption during exercise is more a measure of the extent of exercise limitation than it is a mirror of the symptoms experienced by the patients during day-to-day activities, whereas submaximal exercise is a more appropriate means of following the course of heart failure and the effects of treatment.

In spite of the many unresolved questions, Sullivan et al have presented an elegant demonstration of some of the mechanisms underlying a training effect in patients with heart failure. Whether their results can be applied to less-compensated, more-unstable patients is not yet established. Nevertheless, they have made a valuable contribution by confirming that exercise is beneficial, not harmful, in patients with stable compensated heart failure.

References

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