Determinants of peak aerobic capacity after heart transplantation

See page 1823 for the article to which this Editorial refers

The search for predictive factors of peak aerobic capacity after heart transplantation is an interesting challenge. Although heart transplantation vastly improves the physical capacity of the heart recipients, they still have a reduced peak aerobic capacity (Vo$_2$) compared to healthy sedentary people. Heart recipients' aerobic capacity is, of course, related to biometric characteristics (predicted values based on age, height and weight) of the recipient but also on the age of the donor. The reduced aerobic capacity commonly observed after heart transplantation is explained by several factors. First, it can be related to pre-transplant deconditioning due to end-stage cardiac insufficiency: breathlessness and muscular fatigue at exercise, due to pulmonary congestion and severe left ventricular failure result in progressive reduction in the daily activity of pre-transplant patients, leading to muscular detraining.

Alterations in skeletal muscle histology and biochemistry, including fibre atrophy and decreased oxidative enzyme capacity, are the consequences of this drastically reduced physical activity. Detraining may also be influenced by events before and during the harvesting procedure, the surgical procedure (ischaemia duration) or the number and severity of rejection episodes.

Surgical cardiac denervation is responsible for the reduced cardiac output response to exercise, through a reduced peak heart rate and a reduced peak systolic volume. Differences in heart rate response between transplant recipients studied early and late after heart transplantation have been described in previous studies. There is a higher resting and better chronotropic response in long-term survivors compared with recent recipients. Gradual development of catecholamine super-sensitivity after autonomic denervation at surgery and later direct sympathetic re-innervation are possible explanations for these modifications. With time, myocardial function frequently becomes impaired, and this alteration has been mainly ascribed to cardiac allograft vasculopathy, which represents the most critical long-term problem. Diastolic rather than systolic dysfunction is one of the significant limiting factors of exercise tolerance. Coronary function is often altered and is correlated with the degree of intimal thickening. Indeed, myocardial blood flow reserve during exercise and in response to the cold pressor test may be reduced by endothelium-dependent and -independent abnormalities. Sympathetic re-innervation remains controversial, but could be important, since cardiac adrenergic signals seem to take place in regulating myocardial blood flow and myocardial function.

The study published in the present issue investigates the impact of some of these parameters on the peak Vo$_2$ of their patients. The main observation is the correlation between peak Vo$_2$ and heart rate reserve or peak heart rate. As stated by the authors, there is no evidence that the reduced peak heart rate is the cause or the consequence of the reduced exercise capacity. Indeed, a similar reduction of peak Vo$_2$ was observed by several authors in recipients of different organs. Levy et al. reported reduced aerobic capacity in heart and lung transplant recipients (Vo$_2$ = 15.6 ± 3.2 ml min$^{-1}$ kg$^{-1}$), in double lung recipients (Vo$_2$ = 23.5 ± 9.1 ml min$^{-1}$ kg$^{-1}$) and in single lung recipients (Vo$_2$ = 12.2 ± 2.8 ml min$^{-1}$ kg$^{-1}$). Kidney recipients may also have a reduced exercise capacity (Vo$_2$ = 29.0 ± 7.8 ml min$^{-1}$ kg$^{-1}$) as shown by Kempeneers and co-workers. In the same way, peak heart rate at the end of a typical maximal exercise test never reaches higher values than 90% of maximal predicted values (220 - age) both in patients with a denervated heart (cardiac and cardiopulmonary recipients) and in patients with an innervated heart (lung and kidney recipients).

All transplant recipients have in common a reduction of their muscular strength related to the pathology and the duration of their pre-transplant insufficiency and the corticotherapy commonly used in the immunosuppressive therapy. It is therefore possible that transplant recipients do not have the muscle strength needed to sustain an exercise of sufficiently high level to reach the maximal predicted value of heart rate during a short (less than 12 min) triangular exercise test. This strongly suggests that all transplant recipient should be incorporated in a structured training programme soon after transplantation and for the years following the transplantation procedure. The transplant recipient should
consider regular exercise of moderate intensity as important as taking daily immunosuppressive therapy.

Additional studies on large cohorts of non-selected transplant recipients are warranted to evaluate reliable predictive factors. Longitudinal studies rather than cross-sectional studies should be performed to predict factors of maximal aerobic capacity after heart transplantation. Finally, the impact of regular training has also to be investigated.

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References


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Low energy intracardiac cardioversion of atrial fibrillation

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Over the last ten years, atrial fibrillation has generated ever increasing interest. This may be for different reasons. On the one hand, epidemiologists have clearly demonstrated the very high prevalence of the disease and pointed to the severe consequences it can have both in terms of alterations in haemodynamics and risk of cerebrovascular embolism. On the other hand, the electrophysiology of atrial fibrillation has been investigated in great depth and its mechanisms are nowadays much better understood.

External electrical defibrillation has been used for more than 30 years for restoration of sinus rhythm in cases of atrial fibrillation. The technique has changed little over time and has well recognized weaknesses. First of all, it is far from being efficacious in 100% of cases. In addition, the relatively high energy shocks can be detrimental to the myocardium. They are painful and require that the patient be anaesthetized. More recently, several investigators have demonstrated that internal defibrillation with the use of transvenous catheters is feasible as well as effective[1,2]. The paper by Alt et al.[3] which appears in this issue compares the treatment of atrial fibrillation with low energy intracardiac cardioversion and conventional external cardioversion. The message of the study, which involves a large number of patients is twofold. The authors first demonstrate that internal defibrillation is not only feasible in humans, but is also of greater efficacy than the classical extracardiac transthoracic cardioversion. The second message is that those patients who failed to return to sinus rhythm after an external DC shock, but were successfully treated by internal defibrillation have no tendency for earlier relapse of their arrhythmia than others. One could have logically expected that patients resistant to external defibrillation constitute a group of more difficult cases who might show a higher recurrence rate; this does not appear to be the case. One possible explanation for this observation is that the difference in response to internal vs external DC shocks is related to extra-cardiac (biometrical)