Chronotropic Competence in Endurance Trained Heart Transplant Recipients: Heart Rate is not a Limiting Factor for Exercise Capacity

RUDDY RICHARD, MD, PhD,* JEAN-CLAUDE VERDIER, MD,* ALAIN DUVALLÉT, MD,† SULLY-PIERRE ROSIER, MD,* PHILIPPE LEGER, MD,‡ ALEXIS NIGNAN, MD,‡ MICHEL RIEU, MD, PhD* Reserve.

Paris, France

Objectives. The purpose of this study was to show that the chronotropic potential of the well trained heart transplant recipient (HTR) does not limit exercise capacity.

Background. Chronotropic incompetence is considered to be the main limiting factor of the functional capacity of heart transplant recipients. However, no systematic study had been published on patients who had spontaneously undergone heavy endurance training for several years.

Methods. Heart rate (HR) and respiratory gas exchanges (VO₂, VCO₂, VE) were measured in 14 trained HTRs (T-HTRs) during exercise tests on a bicycle, on a treadmill and by Holter electrocardiography during a race.

Results. Peak values observed in T-HTRs during the treadmill test were higher than those reached during the bicycle test (VO₂peak: 39.8 ± 6.9 vs. 32.5 ± 7.8 ml·kg⁻¹·min⁻¹, p < 0.001; HRpeak: 169 ± 14 vs. 159 ± 16 bpm, p < 0.01). During treadmill exercise VO₂peak and HRpeak values observed were very close to the mean predicted VO₂max and HRmax. The maximum heart rate during the race (HRrace) was greater than HRpeak values during the treadmill test (179 ± 14 vs 169 ± 14 bpm, p < 0.01) and slightly above the mean predicted values (HRrace/HRmax × 100 = 101 ± 10%). The treadmill exercise test yields more reliable data than does the bicycle test.

Conclusions. Extensive endurance training enables heart transplant recipients to reach physical fitness levels similar to those of normal sedentary subjects; heart rate does not limit their exercise capacity.

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From the *Département de Physiologie de la Faculté de Médecine Cochin-Port-Royal (Université René Descartes) and †Service d’Explorations Fonctionnelles et de Physio-Pathologie de l’Exercice, Centre Hospitalier Cochin-Tarnier, 89 rue d’Assas, 75006, Paris; and ‡Service de Chirurgie Cardio-Vasculaire, Groupe Hospitalier La Pitié-Salpêtrière, 75 Boulevard de l’Hôpital, 75013, Paris, ‡Laboratoire de Biochimie A, Groupe Hospitalier Cochin, 27 Rue du Faubourg Saint-Jacques, 75079, Paris Cedex 14, France.

Address for correspondence: Dr. Ruddy Richard, Laboratoire de Physiologie des Adaptations, Faculté de Médecine Cochin-Port-Royal, 24 rue du Faubourg Saint-Jacques, 75014, Paris, France.
Patients (Table 1)

Physical capacities were evaluated by exercise-tolerance tests before the race. The 28 patients (all male) included in the study were: 1) 14 sedentary orthotopic heart transplant recipients (S-HTRs) aged 43 ± 9 years, all of whom were patients in our medical department, and 2) 14 endurance-trained orthotopic heart transplant recipients (T-HTRs) aged 43 ± 12 years, who were recruited from several French departments of cardiovascular surgery. These subjects had trained regularly over a long period of time (36 ± 24 months). They spent 4 ± 2 hours per week in endurance-type physical activities (primarily running). Each session consisted of 30 minutes of training with an energy output of 4 to 8 metabolic equivalent of the task (METs).

Seven of the endurance-trained HTRs (T1-HTRs) had taken part in the Paris-La Plagne race in 1993, 1994, or 1995. Several days before coming to our laboratory for a treadmill test, they had completed a bicycle test, during which VO_2 peak and HR peak were measured, in their home cardiology department.

The other seven trained heart transplant recipients (T2-HTRs) participated in the 1996 race and were investigated exclusively in our laboratory where they performed a bicycle exercise test 24 hours before the treadmill test.

All twenty-eight HTRs were on immunosuppressive therapy (cyclosporine, azathioprine, predimisone). None required beta-adrenergic blocking agents. Seven were treated by antihypertensive medication (nicardipine: a calcium channel blocker) and three others were on low-dose diuretic therapy (furosemide).

Coronary angiography and radionuclear angiograms had previously been performed during an exercise test in each participant’s cardiovascular surgery department; no abnormalities were found.

There were no significant differences in age, size and weight between the S-HTR and T-HTR groups. Average post-transplant delays were similar for the two groups, but great variations existed among individuals (one to seven years). The pathologies that led to heart transplants were comparable in the two groups, as was the immunosuppressive therapy.

The mean physical activity time of the T-HTRs was 4 ± 1 h per week, and the average work power was 4 ± 1.5 METs.

Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>S-HTRs</th>
<th>T-HTRs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Delay (months)</td>
<td>38.4 ± 6 (6-109)</td>
<td>45.9 ± 23 (21-75)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>43.1 ± 9 (26-54)</td>
<td>43.3 ± 11.6 (19-59)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69 ± 8 (56-84)</td>
<td>69 ± 11 (50-88)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171 ± 4 (163-178)</td>
<td>174 ± 5 (163-181)</td>
</tr>
<tr>
<td>BMI</td>
<td>24 ± 3 (19-31)</td>
<td>23 ± 3 (18-28)</td>
</tr>
<tr>
<td>HR sup (bpm)</td>
<td>87 ± 17 (60-110)</td>
<td>89 ± 12 (66-105)</td>
</tr>
<tr>
<td>HR ortho (bpm)</td>
<td>90 ± 16 (66-113)</td>
<td>93 ± 11 (77-108)</td>
</tr>
<tr>
<td>Pmax (bpm)</td>
<td>177 ± 9 (166-194)</td>
<td>177 ± 12 (161-201)</td>
</tr>
<tr>
<td>Ergocycle VO_2 pmax (ml/kg/min)</td>
<td>37 ± 5 (31-49)</td>
<td>38 ± 6 (29-49)</td>
</tr>
<tr>
<td>Treadmill VO_2 pmax (ml/kg/min)</td>
<td>*</td>
<td>42 ± 6 (32-54)</td>
</tr>
</tbody>
</table>

Delay (months) = months after transplantation; BMI = body mass index; HR sup (bpm) = heart rate in supine position; HR ortho (bpm) = heart rate in standing position; HR pmax (bpm) = maximum predicted heart rate; VO_2 pmax (ml/kg/min) = maximum predicted oxygen uptake.

Protocol

Heart rate was recorded at the end of a 10 min supine resting period and for the first 30 s after an active change to the standing position.

Exercise testing. Bicycle test: All the HTRs performed a “symptom-limited” incremental test on a cycloergometer (Monark-818 E). In our laboratory, where S-HTR and T2-HTR groups were tested, the warm-up load was 30 Watts and the power increase was 30W/3min. ECG was recorded on line (with a Marquette Max 1, twelve derivation system) and heart rate (bpm) was measured using a cardiac monitor (BHL 6000). Respiratory gases were analyzed with a breath-by-breath system (CPX/D Medgraphics). The following gas exchanges were assessed every 15 seconds: oxygen uptake (VO_2: ml/min^-1), carbon dioxide output (VCO_2: ml/min^-1), minute respiratory volume (VE: ml/min^-1), respiratory exchange ratio (R: VCO_2/VO_2).

Treadmill test: The 14 T-HTR runners performed a treadmill test (Gymrol ST 2800) 24 h before the start of the race. The initial warm-up speed was 6 km/h^-1 and the increase in velocity was 1 km/h^-1 every 3 minutes; the slope of the treadmill was 2%. The parameters analyzed were the same as those collected during the bicycle test.

Competition monitoring. During the race, electrocardiograms of the T-HTRs were recorded with a 1993 ELA Medical System, Elatec V303P29 (Holter electrocardiography). Furthermore CMS-type electrocardiographic derivation was simultaneously visualized in real time using a telemetric system (lifescope 6 Nikon Kohden). The records were interpreted after the race. Only records of a minimum duration of two minutes were used in order to average R-R periods in 10 s sequences.

Measurements. In all patients (S-HTRs and T-HTRs) peak values of VO_2, VCO_2, QR, HR, and oxygen pulse (\(O_2\)-pulse = VO_2/HR: mlO_2/bpm) corresponded to the values...
observed at the end of exercise. Predicted maximal oxygen uptake (VO2peak, ml/kg·min−1) was calculated according to Wasserman’s equations (17). HRpeak was expressed in absolute value or as a percentage of the predicted maximum heart rate calculated according to the Astrand formula (HRpeak = 220 – age [years] ± 10) (16). Peak HR during the race was defined as the maximum HR recorded for any given subject during at least two consecutive minutes of any relay.

Other data were collected from the seven patients of the T2-HTR subgroup. Blood samples were taken from the humeral vein. Determination of plasma lactate levels was performed at rest, at the peak of the exercise tests (bicycle and treadmill) and after 5, 10, 15, 20, and 30 minutes of recovery. An enzymatic procedure was used for the lactate spectrophotometric assay. Plasma catecholamines were measured using a Bio-Rad at rest, at peak work load and after 30 minutes of recovery, by a three-step procedure: catecholamines were adsorbed onto alumina at a pH of 8.6, then diluted with a 0.1 phosphoric acid and finally analyzed by HPLC. An internal standard was included with each extraction to monitor normal plasmatic concentration of epinephrine at rest; Ep rest = plasmatic concentration of epinephrine at rest; La rest = plasmatic concentration of lactate at rest.

The Body Mass Index (21) [BMI = weight (in kg)/height² (in m)] were similar for the two groups of HTRs (Table 1).

T2-HTRs (Table 2) were given Baecke’s questionnaire (19) evaluating time devoted to sport and leisure activities. They spent considerably more time on sports than did 118 “average” French males (18) aged 30–58 years (3.4 ± 0.5 and 2.8 ± 0.8). Their mean index for leisure time was similar to that found for the 118 French men (2.8 ± 0.8 vs. 2.8 ± 0.4) (18).

Exercise testing. Bicycle tests (Tables 3 and 4). S-HTR group: The average maximum work power (Pmax) and the mean VO2peak were respectively 110 ± 27 watts and 22.3 ± 4.3 ml/kg·min−1. Mean VO2peak/VO2pMax and HRpeak/HRpMax ratios were 60 ± 16% and 76 ± 12%; VO2peak values were less than VO2pMax values for all patients. Mean mechanical efficiency, calculated from body weight and from the energetic equivalent of oxygen (20.9 Joules/oxygen liter), was 20.4 ± 1.6%. Mean Oxygen pulse was 11.5 ± 3 mlO2/beat−1 (Table 3).

T-HTR group: Mean Pmax and VO2peak were respectively 169 ± 25 watts and 32.5 ± 7.8 ml/kg·min−1. Mean ratios of VO2peak/VO2pMax and HRpeak/HRpMax were, respectively, 85 ± 11% and 90 ± 8%. In only one patient was the VO2peak value equal to the VO2pMax value. The mechanical efficiency was 21 ± 1.7%. The Oxygen pulse peak was 13.8 ± 1.9 mlO2/beat−1 (Table 3).

Each of the above values was significantly different from the corresponding value for the S-HTR group. The mean values for Pmax, VO2peak and HRpeak were not significantly different in the T2-HTR subgroup from those of the T1-HTR subgroup.

Mean plasma lactate concentration was 5.75 ± 2 mmol·l−1 at the end of the exercise test; recovery half-time was approximately 20 min. Mean peak plasmatic concentrations of epinephrine and of norepinephrine were respectively 1.6 ± 1.1 nmol·l−1 and 23.6 ± 7.4 nmol·l−1 (Table 4).

### Results

**Characteristics at rest.** Resting heart rates were high in both groups (Table 1). The active change from the supine to the upright position did not raise the heart rate significantly; mean increase in HR was 3.4 ± 6 bpm in S-HTRs and 3.6 ± 6 bpm in T-HTRs. However, the heart rate of four subjects increased markedly.

### Table 2. Characteristics of T2-HTRs (session: 1996; n = 7)

<table>
<thead>
<tr>
<th>Fat %</th>
<th>Baecke indices</th>
<th>La (rest)</th>
<th>Ep (rest)</th>
<th>NE (rest)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>Sport</td>
<td>Leisure</td>
<td>mmol/l</td>
</tr>
<tr>
<td>Average</td>
<td>14.7</td>
<td>3.4</td>
<td>2.8</td>
<td>1</td>
</tr>
<tr>
<td>SD</td>
<td>5.6</td>
<td>0.5</td>
<td>0.8</td>
<td>0.3</td>
</tr>
<tr>
<td>Range</td>
<td>8–24</td>
<td>2.8–4</td>
<td>1.7–3.7</td>
<td>0.81–1.60</td>
</tr>
</tbody>
</table>

T-HTR = trained heart transplant recipients investigated in 1996; Fat % = % of body fat; NE rest = plasmatic concentration of norepinephrine at rest; Ep rest = plasmatic concentration of epinephrine at rest; La rest = plasmatic concentration of lactate at rest.

### Table 3. Peak Values Collected in Sedentary Heart Transplant Recipients (S-HTRs) and in Trained Heart Transplant Recipients (T-HTRs) During Bicycle Tests

<table>
<thead>
<tr>
<th></th>
<th>P watts</th>
<th>HR peak bpm</th>
<th>VO2 peak ml/kg/min</th>
<th>Ox pulse mlO2/beat</th>
</tr>
</thead>
<tbody>
<tr>
<td>S-HTRs</td>
<td>110 ± 27 (70–150)</td>
<td>135 ± 20 (95–162)</td>
<td>22.3 ± 4.3 (15–30)</td>
<td>11.5 ± 3 (7.2–18.3)</td>
</tr>
<tr>
<td>T-HTRs</td>
<td>169 ± 25 (120–210) **</td>
<td>159 ± 16 (126–180) **</td>
<td>32.5 ± 7.8 (23.3–50) **</td>
<td>13.8 ± 1.9 (11.1–17.1) *</td>
</tr>
</tbody>
</table>

P = work power (watts); HR peak = heart rate peak (bpm); VO2 peak = oxygen uptake peak (ml/kg/min); Ox pulse = oxygen pulse peak (mlO2/beat); Comparison between S-HTRs and T-HTRs = *p < 0.01, **p < 0.001.
Table 4. Plasma Lactate, Epinephrine and Norepinephrine Peak Values During Bicycle and Treadmill Testing

<table>
<thead>
<tr>
<th></th>
<th>Bicycle</th>
<th>Treadmill</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>La (peak) mmol/l</td>
<td>Ep (peak) nmol/l</td>
</tr>
<tr>
<td>Average</td>
<td>5.75</td>
<td>1.6</td>
</tr>
<tr>
<td>SD</td>
<td>2</td>
<td>1.1</td>
</tr>
<tr>
<td>Range</td>
<td>2.4–8.6</td>
<td>0.8–3.7</td>
</tr>
<tr>
<td></td>
<td>La (peak) mmol/l</td>
<td>Ep (peak) nmol/l</td>
</tr>
<tr>
<td></td>
<td>6.75</td>
<td>3.2 **</td>
</tr>
<tr>
<td></td>
<td>1.85</td>
<td>1.35</td>
</tr>
<tr>
<td></td>
<td>3.8–9.7</td>
<td>2.3–6</td>
</tr>
</tbody>
</table>

La (peak) = mean plasma lactate peak value; Ep (peak) = mean plasma epinephrine peak value; NE (peak) = mean plasma norepinephrine peak value; Comparison between bicycle and treadmill = *p < 0.05, **p < 0.01, ***p < 0.001.

Treadmill test. T-HTR group (Table 5): Mean VO₂peak and mean maximum running speed were respectively 39.8 ± 6.9 ml/kg *min⁻¹ and 2.8 ± 0.54 ms⁻¹. Mean ratio of VO₂peak/VO₂pMax and HRpeak/HRpMax were respectively 95 ± 10% and 95 ± 9%; VO₂peak values were equivalent or superior to VO₂pMax values in seven patients; mean Oxygen pulse peak was 16.1 ± 2.7 mlO₂·beat⁻¹ this value is significantly different from the average Oxygen pulse peak 13.8 ± 1.9 mlO₂·beat⁻¹ of the same group during the bicycle test.

In the T2-HTR sub-group VO₂peak maximum running speed, HRpeak and Oxygen pulse mean values were not significantly different from those observed in the T1-HTR subgroup. The mean plasma lactate peak value was 6.75 ± 1.8 mmol/l⁻¹ and the recovery half-time was approximately 20 minutes (Table 4). Mean epinephrine and norepinephrine peak values were respectively 3.2 ± 1.35 nmol/l⁻¹ and 34.3 ± 15.6 nmol/l⁻¹. These values were significantly higher than those observed during the bicycle test.

Paris-La Plagne relay race. Individual relay times and speeds of the T-HTR runners varied greatly from 10 to 30 minutes and from 8 to 14 km/h. Lengths of the relays depended on previous physical fitness training and were correlated to the VO₂peak values (r = 0.91; p < 0.01). The mean maximal value of HR registered during the race (HRrace) was significantly higher than the mean HRpeak of the same patients during laboratory exercise tests (the HRrace values were superior to HRpeak values in ten patients); the ratio of mean HRpeak/HRpMax was 101 ± 10%.

No electrocardiographic abnormalities (supraventricular or ventricular rhythm disorders) were observed. There were no disturbances of cardiac conduction that might have suggested myocardial ischemia.

Discussion

This study indicates that the maximum heart rates recorded for endurance-trained HTRs during foot races are not significantly different from predicted maximum heart rates estimated according to the Astrand formula (16). Moreover the study demonstrates that the exercise tests performed on the treadmill yield more relevant information about the functional capacity of patients than do bicycle tests. The results confirm that physical work capacity is much higher in trained than in sedentary HTRs.

Bicycle exercise testing: sedentary and trained HTRs (Table 3). As we pointed out, the T1-HTR subgroup did not perform the bicycle tests in our laboratory and their test protocols were slightly different from ours (incremental work load 10 W/1 min vs. 30 W/3 min in our department).

Due to the inertia of the heart response to exercise in HTRs, such short work periods might result in an underestimation of VO₂peak and HRpeak values (22,23). In fact, the VO₂peak and HRpeak values documented in the T1-HTR subgroup were not significantly different from those collected in the T2-HTR subgroup in our laboratory; they were higher than the values we observed in the S-HTR group and similar to those reported by other researchers. Therefore we considered all the trained HTRs as a single group for the bicycle exercise test. The VO₂peak levels on the bicycle (mean VO₂peak = 32.5 ± 7.8 ml·min⁻¹·kg⁻¹) were higher than those reported by most other authors (2.7–10) but similar to those documented in eight very compliant patients after eight to twelve months endurance-type training (2,24). Nevertheless the VO₂peak values remained lower than the maximum predicted VO₂ level for sedentary men. The results collected in the S-HTR group were

Table 5. Peak Values collected in Trained Heart Transplant Recipients (T-HTRs)

<table>
<thead>
<tr>
<th></th>
<th>HR peak bpm</th>
<th>VO₂ peak ml/kg/min</th>
<th>Ox pulse mlO₂·beat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bicycle</td>
<td>159 ± 16 (126–180)</td>
<td>32.5 ± 7.8 (23.3–50)</td>
<td>13.8 ± 1.9 (11.1–17.1)</td>
</tr>
<tr>
<td>Treadmill</td>
<td>169 ± 14 (135–188) *</td>
<td>39.8 ± 6.9 (31–57) **</td>
<td>16.1 ± 2.7 (12.1–20.7) *</td>
</tr>
<tr>
<td>Race</td>
<td>174 ± 19 (146–201) †</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

HR peak = heart rate peak (bpm); VO₂ peak = oxygen uptake peak (ml/kg/min); Ox pulse = oxygen pulse peak (mlO₂·beat); Comparison between bicycle and treadmill = *p < 0.01, **p < 0.001; Comparison between race and treadmill = †p < 0.05.
similar to those related in other scientific papers (1–6): HRpeak and VO2peak values were low and corresponded to 85% and 69%, respectively, of those observed in the T-HTR group.

**Comparison between bicycle and treadmill exercise tests.** (Table 5) Data published since 1964 (16,25,26) show that the VO2max values of normal subjects running on a treadmill are about 10% higher than those resulting from exercise tests on a bicycle (25–27). The same findings were reported concerning patients with heart failure. In our study, the difference in mean VO2peak values observed between the two kinds of exercise was even greater (ergocycle:32.5 ± 7.8 mlO2·kg⁻¹·min⁻¹; treadmill: 39.8 ± 6.9 mlO2·kg⁻¹·min⁻¹; mean difference; 24.71%). Nonetheless, the difference in VO2max between bicycle and treadmill tests was observed for equal heart rates (25–27). In our study mean HRpeak registered on the treadmill (169 ± 14 bpm) was significantly higher than that documented on the bicycle (159 ± 16 bpm).

Similarly, the mean peak Oxygen pulse value measured during treadmill tests was significantly higher than that observed during bicycle tests (16.1 ± 2.7 mlO2·beat⁻¹ vs. 13.8 ± 1.9 mlO2·beat⁻¹); the mean difference was 17%.

Consequently, the increases in the HRpeak and in the Oxygen pulse are responsible for, respectively, approximately 1/3 and 2/3 of the greater VO2peak values found on the treadmill compared to those observed on the bicycle. However, several patients displayed markedly different VO2peak values (VO2peak on treadmill > VO2peak on bicycle) although the HRpeak was similar during both tests. Much higher Oxygen pulse peak values were collected on the treadmill. There is a significant positive correlation between peak Oxygen pulse and VO2peak expressed in percentage (r = 0.81; p < 0.01).

Ours results show that the endurance-trained HTRs were able to reach a far higher physical work power level on the treadmill than on the bicycle. The VO2peak values collected during running tests were greater or equal in seven patients to the predicted maximum VO2 for healthy sedentary men. The poor adaptation of T-HTRs to bicycle testing may be explained by the fact that most of them were not accustomed to this type of exercise. Mean norepinephrine and epinephrine peak values observed in the T2-HTR subgroup were significantly higher on the treadmill than on the bicycle. The positive correlation between the norepinephrine peak values and the HRpeak values (r = 0.78; P < 0.05) and VO2peak values (r = 0.91; P < 0.01) suggests that the reactivity of the sympathetic nervous system influences exercise capacity during both treadmill and bicycle tests. The specific mechanical characteristics of the two types of exercise might explain why venous return from contracting muscles to the heart is lower in cycling than in running; it follows that a cyclist’s maximal stroke volume would be lower than that of a runner. This hypothesis, if valid, is even more applicable to HTRs because of the importance of the Frank-Starling mechanism in these patients’ adaptation to exercise.

**Analysis of heart rate recorded during the race** (Table 5). The length and speed of the relays varied greatly between individuals. However, for all but one of the patients, the maximum heart rate values recorded during the race were equivalent or higher than the HRpeak collected during exercise—both treadmill and bicycle. Georges Niset previously made the same observation concerning a marathon runner (15). The mean HRace value collected for the T-HTR group was significantly greater than the mean HRpeak value registered the day before the competition during treadmill testing (169 ± 14 bpm. vs. 179 ± 14 bpm). One subject’s heart rate rose little during the treadmill exercise test (HRpeak = 135 bpm) but increased considerably during the race (HRace = 184 bpm). Throughout the race the motivation and the strain were probably more intense than in the laboratory, and the HR peak was generally higher than that recorded during treadmill exercise.

It should be noted that the mean HR value recorded in the T-HTR group during the competition corresponded to 101% of the maximum predicted heart rate. Therefore HRpeak cannot be considered a limiting factor to the functional capacity of HTRs as has often been suggested (4,13,14). To our knowledge, the present study is the first to demonstrate clearly that endurance-trained HTRs can regain normal chronotropic competence. We found no reinnervation of the transplanted heart as was suggested in previous publications (28–30). Indeed, there was not any significant increase in heart rate after an active change from the reclining to the upright position (Table 1) in the T-HTR group. The same change of position by normal subjects results in an average heart rate increase greater than 10 bpm (31,32).

**Conclusion**

We found strong evidence suggesting that treadmill tests yield more reliable information on cardiorespiratory adaptation to exercise of endurance-trained HTRs than do bicycle tests.

In our study the maximum values of HR recorded during exercise for T-HTRs in the laboratory, as during the race, were very close to the predicted maximum HR for each patient. Chronotropic potential was normal in T-HTRs and did not limit their functional capacity. Furthermore, VO2peak values collected during treadmill tests were close to the predicted maximum VO2 for sedentary men. In contrast, chronotropic incompetence was substantial in S-HTRs and partly explains the very low VO2peak usually observed in these patients.

Physical work can be sustained easily for a long time only when the energy expenditure does not exceed approximately 35 percent of VO2max (33,34); therefore sedentary heart transplant recipients are unable to carry out tasks with an energy cost much greater than 9 mlO2·kg⁻¹·min⁻¹ (2.5 METs), a level frequently reached in everyday life. In contrast the functional capacity of endurance-trained heart transplant recipients is far superior (14 mlO2·kg⁻¹·min⁻¹ corresponding to 4 METs). As a result, their reintegration to the labor and leisure society is improved and their quality of life is vastly enhanced.
We thank Professor C. Cabrol and Professor I. Gandjbakhch for their collaboration. We also thank “Transforme Association” for their assistance. We would especially like to thank Yvonne Laugier-Werth for her help with the English version of this text.

References