Ageing of the cardiovascular system during 33 years of aerobic exercise


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Abstract

Background: increasing age affects aerobic capacity, with an average loss of 10% or more per decade.
Aim: to determine the effect of ageing on the circulatory system in middle-aged men during 33 years of physical training.
Methods: 15 men initially aged 45 years took part in an exercise training programme for 25–33 years. Nine serial measurements were made at rest and during maximal effort. Aerobic training consisted of swimming, jogging, walking and cycling 3–4 times per week. Sessions were for 61–70 min at 77–84% of heart rate reserve.
Results: there was no change in resting heart rate, blood pressure, percentage fat or body composition. Minimal cardiovascular losses at maximal work included 5.8–6.8% in maximal oxygen uptake per decade, 25 beats in maximum heart rate and 26 beats in heart rate reserve.
Conclusion: exercise training has a favourable effect on ageing of the cardiovascular system in older men, resulting in minimal loss of oxygen uptake, no rise in resting blood pressure and no change in body composition.

Keywords: ageing, blood pressure, cardiovascular, exercise, longitudinal study

Introduction

With increasing age there is loss of aerobic capacity. This functional loss can result in poorer quality of life, reduced chance of survival in emergency and greater potential for developing hypokinetic diseases; it may result in dependency and increased health care requirements [1–4].

Many researchers [5–12] have found declines in circulatory capacity, ranging from 5 to 22% per decade and 0.28 to 1.32 ml·min⁻¹·kg⁻¹·year⁻¹. However, these are mostly cross-sectional reports on selected populations. Can intervention forestall this? We need longitudinal studies on ageing and functional losses to answer this question [13].

Ageing may be defined as the reduction in the capacity of a bodily system. Measurements of maximum oxygen uptake (or VO₂ max) are a common method of evaluating the capacity of the cardiovascular system [14].

The purpose of our longitudinal investigation was to determine the effect of ageing on the cardiovascular system in initially middle-aged men during the course of 33 years of intervention by physical training.

Subjects

Fifteen randomly chosen men (mean age 45 years, range 33–56), were initially tested in 1964. Twelve subjects were free of disease, two had hypertension controlled by medication and one was later diagnosed as having diabetes mellitus. Fourteen were physically active for most of their lives. Some had been university athletes, but only one (a swimmer) competed in endurance events. Before entry, over half were participating in recreational sports (such as badminton, volleyball, tennis, handball, swimming and basketball). None smoked in 1964; however, four had smoked several years before this study. By the year 25, two had developed atrial fibrillation, one mitral valve prolapse and one a myocardial infarction. Another had coronary artery bypass surgery at age 83.

Methods

Training

Initially, 14 of the 15 men jogged and one swam. Later, six men walked, two swam, three cycled and four
jogged. This change was a result of musculoskeletal problems in some men and choice by others. We measured energy expenditure in kilocalories per week. This method permitted us to compare different modes of exercise, as well as to relate energy cost to other investigations. Daily training records were kept, including type of exercise, frequency per week, duration per session and intensity of exercise by heart rate reserve.

**Resting measurements**

Before all testing, each subject signed a consent form as required by the university human subjects committee. All testing was done in the early morning after the subject had fasted overnight. The laboratory temperature was 20–24°C and the relative humidity 40–70%. We recorded resting heart rate, blood pressure, height, weight and percentage body fat. Body mass index was calculated from body weight in kilograms divided by height in metres squared. Heart rate was determined by ECG and blood pressure by auscultation. Body fat was estimated by skinfold thickness taken by the same single anthropometric expert using the formula of Yuhasz [15].

**Dynamic measurements**

We assessed VO2 max using the Douglas bag method [16], combined with the analysis of expired CO2 and O2 in duplicate by the method of Scholander [17]. Pulmonary ventilation was measured with a wet test gas meter (American Meter Company) and calibrated with a Tissot. Volumes were reduced to STPD by standard tables. We calculated VO2 in litres per min, ml·min⁻¹·kg⁻¹ and ml·min⁻¹·kg⁻¹·year⁻¹.

We used a cycle ergometer (Monark) and treadmill (Quinton, Young) to attain VO2 measurements. Subjects training on a bicycle were tested on a cycle ergometer. We increased the rate to 80–90 revolutions per min during the last 2–3 min of the test, while reducing the resistance slightly. We used the Fox-Costill treadmill protocol [18] to test the joggers and a modified Balke method [19] for the walkers and swimmers. Maximal test duration ranged from 6 to 12 min.

Maximal heart rate was obtained from the ECG tracing, while O2 pulse was calculated by dividing the VO2 max in ml/min by the maximum heart rate.

Mean arterial pressure was determined by dividing the pulse pressure by 3 and adding the diastolic pressure.

We performed serial evaluations at 10, 15, 18, 20, 23, 25, 28 and 33 years. At 28 years, the number of subjects was reduced from 15 to 12 due to three deaths (two from lymphoma at 83 and one with complications of Alzheimer’s disease at 78). Before the 33-year data collection, another man died from pneumonia aged 74.

**Statistics**

The Student t-test was used to determine the significance between the various measurements.

The percent loss per decade was determined by finding the percent loss per year and multiplying by 10.

**Results**

Since four men died, we will present the results at 0, 25 and 33 years. No changes in data were observed between the 11th and 12th subjects at 28 years and these are, therefore, not included. We designated the original 15 subjects as group A and the 11 subjects still alive after 33 years as group B.

**Training**

Over the 33 years, there was some increase in the number of training sessions per week and their duration, and in the total energy expended per week.

<table>
<thead>
<tr>
<th>Group</th>
<th>Year</th>
<th>Frequency (per week)</th>
<th>Intensity (% of HRR)</th>
<th>Duration (min)</th>
<th>Energy expenditure (kcal/week)</th>
<th>Energy expenditure (x10⁶ J/week)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (all)</td>
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<td>3.0</td>
<td>84</td>
<td>61</td>
<td>2290</td>
<td>9.6</td>
</tr>
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<td></td>
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<td>(0.6)</td>
<td>(3.7)</td>
<td>(10.8)</td>
<td>(576)</td>
<td>(2.4)</td>
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<tr>
<td></td>
<td>25</td>
<td>4.6</td>
<td>77</td>
<td>66</td>
<td>2370</td>
<td>9.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.1)</td>
<td>(3.4)</td>
<td>(11.8)</td>
<td>(742)</td>
<td>(2.5)</td>
</tr>
<tr>
<td>B (survivors)</td>
<td>0</td>
<td>3.2</td>
<td>85</td>
<td>62</td>
<td>2440</td>
<td>10.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.6)</td>
<td>(4.7)</td>
<td>(12.5)</td>
<td>(629)</td>
<td>(2.6)</td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>4.6</td>
<td>77</td>
<td>70</td>
<td>2550</td>
<td>10.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.3)</td>
<td>(6.2)</td>
<td>(14.1)</td>
<td>(943)</td>
<td>(3.9)</td>
</tr>
</tbody>
</table>

HRR, heart rate reserve.
While the intensity of exercise declined by 7%, the increase in frequency and duration compensated in kcal expended. There was little difference in the training regimens at 25, 28 and 33 years. No one withdrew from the study (Table 1).

**Resting results**

Few changes occurred in resting measurements. We found little variation in heart rate, blood pressure, body mass index, mean pressure, % fat, and lean body mass. There was a loss of 2 cm in height and about 3 kg in body weight. None of these differences was significant (Table 2).

**Dynamic results**

Groups A and B each sustained small losses in oxygen uptake at year 15. Thereafter, oxygen uptake levelled off in group A until the year 25 (the seventh and final evaluation for this group) with an overall deficit of 7.6 ml·min⁻¹·kg⁻¹ (P ≤ 0.05). In group B, oxygen uptake remained constant from year 15 to year 33, when it fell to 8.9 ml·min⁻¹·kg⁻¹ (P ≤ 0.05; Tables 3 and 4). Maximum heart rate showed a continual, but gradual, reduction throughout the study. In group A it fell by 25 beats (1.0 beats/year) in 25 years (P ≤ 0.001), while in group B it fell 31 beats (0.9 beats/year) in 33 years (P ≤ 0.001).

Heart rate reserve decreased by 22 beats per min (P ≤ 0.001) in group A and by 26 beats per min (P ≤ 0.001) in group B (Table 3). Mean arterial pressure in group A increased from 111 to 115 mmHg (P = 0.52), while in group B it rose slightly from 109 to 112 mmHg (P = 1.03).

Pulmonary ventilation increased in group A from 90.7 to 95.2 l, STPD (< 0.05) and in group B from 93.4 to 86.7 l, STPD (< 1.72).

About a 6% decline occurred in O₂ pulse: in group A it fell from 19.0 to 17.8 units (P < 0.01), in group B it fell from 19.3 to 18.2 (P < 0.10).

The mean group respiratory exchange ratio at the nine test intervals ranged from 1.03 to 1.12.

**Table 2. Resting measurements for groups A (all 15 subjects) and B (the 11 still alive at 33 years)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Year</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Heart rate (beats/min)</th>
<th>Blood pressure (mmHg)</th>
<th>% fat</th>
<th>Lean body mass (kg)</th>
<th>Body mass index</th>
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</thead>
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<tr>
<td>A (all)</td>
<td>0</td>
<td>45.0</td>
<td>177.0</td>
<td>76.1</td>
<td>63</td>
<td>123 80 94</td>
<td>13.3</td>
<td>66.3</td>
<td>24.3</td>
</tr>
<tr>
<td></td>
<td>(6.5)</td>
<td>(7.1)</td>
<td>(8.8)</td>
<td>(9.7)</td>
<td>(13.7)</td>
<td>(12.0) (12.0)</td>
<td>(4.9)</td>
<td>(5.0)</td>
<td>(2.4)</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>70.2</td>
<td>175.0</td>
<td>72.9</td>
<td>60</td>
<td>120 76 91</td>
<td>12.9</td>
<td>63.2</td>
<td>23.8</td>
</tr>
<tr>
<td></td>
<td>(7.1)</td>
<td>(8.0)</td>
<td>(9.7)</td>
<td>(8.5)</td>
<td>(9.7)</td>
<td>(6.2) (3.4)</td>
<td>(3.3)</td>
<td>(4.4)</td>
<td>(2.4)</td>
</tr>
<tr>
<td>B (survivors)</td>
<td>0</td>
<td>43.0</td>
<td>177.0</td>
<td>74.9</td>
<td>62</td>
<td>119 75 93</td>
<td>12.9</td>
<td>62.5</td>
<td>23.9</td>
</tr>
<tr>
<td></td>
<td>(6.0)</td>
<td>(7.8)</td>
<td>(5.5)</td>
<td>(6.9)</td>
<td>(12.5)</td>
<td>(7.5) (7.8)</td>
<td>(2.2)</td>
<td>(5.5)</td>
<td>(2.0)</td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>76.1</td>
<td>174.0</td>
<td>72.1</td>
<td>57</td>
<td>118 79 94</td>
<td>12.1</td>
<td>63.4</td>
<td>23.8</td>
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<td>(5.7)</td>
<td>(7.9)</td>
<td>(6.1)</td>
<td>(4.4)</td>
<td>(9.1)</td>
<td>(5.7) (6.6)</td>
<td>(1.8)</td>
<td>(5.3)</td>
<td>(1.9)</td>
</tr>
</tbody>
</table>

**Table 3. Dynamic measurements for groups A (all 15 subjects) and B (the 11 still alive at 33 years)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Year</th>
<th>VO₂ max (l/min)</th>
<th>VO₂ max (ml·min⁻¹·kg⁻¹)</th>
<th>Heart rate (beats/min)</th>
<th>Maximum</th>
<th>Reserve</th>
<th>Mean pressure (mmHg)</th>
<th>Respiratory exchange ratio</th>
</tr>
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<tr>
<td>A (all)</td>
<td>0</td>
<td>3.37</td>
<td>44.2</td>
<td>177</td>
<td>114</td>
<td>111</td>
<td>1.08</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.57)</td>
<td>(6.7)</td>
<td>(11.1)</td>
<td>(10.0)</td>
<td>(10.5)</td>
<td>(0.07)</td>
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</tr>
<tr>
<td></td>
<td>25</td>
<td>2.67</td>
<td>36.6</td>
<td>152</td>
<td>92</td>
<td>115</td>
<td>1.03</td>
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<tr>
<td></td>
<td></td>
<td>(0.66)</td>
<td>(10.6)</td>
<td>(20.0)</td>
<td>(17.2)</td>
<td>(11.1)</td>
<td>(0.07)</td>
<td></td>
</tr>
<tr>
<td>B (survivors)</td>
<td>0</td>
<td>3.46</td>
<td>45.9</td>
<td>179</td>
<td>117</td>
<td>109</td>
<td>1.09</td>
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<tr>
<td></td>
<td></td>
<td>(0.54)</td>
<td>(5.5)</td>
<td>(10.0)</td>
<td>(9.1)</td>
<td>(8.2)</td>
<td>(0.07)</td>
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<tr>
<td></td>
<td>33</td>
<td>2.68</td>
<td>37.0</td>
<td>148</td>
<td>91</td>
<td>112</td>
<td>1.03</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.49)</td>
<td>(5.2)</td>
<td>(13.7)</td>
<td>(15.1)</td>
<td>(14.1)</td>
<td>(0.04)</td>
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</table>

VO₂ max, maximal oxygen uptake.
Discussion

During this study there were few changes in the training intensity and duration. However, there was a large (53%) increase in training frequency, which may have been responsible for maintaining the small reduction in aerobic capacity. Although the mode of exercise changed, it remained aerobic. It is unlikely that this change caused any difference in capacity. The mode, intensity, frequency and duration of exercise appear to have been sufficient to minimize the effect of ageing on VO₂ max.

How can we explain the adherence to this training regimen for 25–33 years? Explanations may include daily recording of the training measures, personal feedback of the test results and the participants’ lifetime interest in sport and exercise.

Buskirk and Hodgson [4] have reported a linear loss in aerobic capacity of 10% per decade and 0.45 ml·min⁻¹·kg⁻¹·year⁻¹ in cross-sectional studies. In contrast, our data show losses of 6.8 and 5.8% per decade, with 0.30 and 0.27 ml·min⁻¹·kg⁻¹·year⁻¹ at 25 and 33 years in groups A and B, respectively. Our results are lower than any reported and represent longitudinal data collected over a long time (33 years). However, these declines are not linear, since reductions occurred at 15, 25 and 33 years. Furthermore, dichotomizing the subjects shows one group, who were 41 years of age at the initial evaluation, who had abrupt losses at ages 55 and 66 years (of 15 and 14% respectively) and another group (initially aged 50) who had similarly large diminutions (17 and 14%) at ages 66 and 76. The reasons for these abrupt changes are unknown.

Several factors may have contributed favourably to minimizing the loss of aerobic capacity of our men while they aged. These factors include pulmonary ventilation, heart rate, heart rate reserve and mean arterial pressure. Ventilation showed little change from initial to final measurement in either group A or B. Maximum heart rate values declined by 12 and 14% in groups A and B, respectively, yet were above age-predicted values [19]. Heart rate reserve was greater than age prediction during the entire study, improving from 7 to 15% [19, 20]. Mean arterial pressure was relatively low throughout the 33-year period, being 20–23% less than values reported for middle-aged trained and untrained men by Hartley et al. [21]. Factors such as the reduction of peripheral vascular resistance by a minimal mean arterial pressure and by adequate ventilation, heart rate and heart rate reserve probably account for much of the minimal loss of VO₂ max with ageing.

Another reason for the retention of VO₂ max with increasing age could be a favourable body composition with a low percentage of fat and high percentage lean body mass. Jackson [22] has stated three reasons for the diminution of VO₂ max with increasing age: sedentary living, body composition and lifestyle. Our subjects minimized these three factors, thereby helping to forestall the usual loss in VO₂ max with age.

Paffenbarger et al. [23] reported that an expenditure of 2000 kcal per week reduced the incidence of coronary heart disease. The 2000 plus kcal per week, 10.7 joules (10⁶) in the present study had an average energy expenditure of 30–35 kcal·kg⁻¹·week⁻¹ compared with 25 for Paffenbarger’s subjects. An interesting finding of our study was the stable resting blood pressure over 33 years. Lifestyle factors which affect blood pressure include smoking, alcohol, obesity and sedentary living [24]. These factors were avoided by our men, thus minimizing their chances of developing

### Table 4. Maximal oxygen uptake and age comparisons from published data

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</thead>
<tbody>
<tr>
<td>Present*</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>A</td>
<td>44.2</td>
<td>45.2</td>
<td>40.5</td>
<td>41.3</td>
<td>39.0</td>
<td>38.6</td>
<td>36.6</td>
<td>6.8</td>
<td>0.30</td>
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<tr>
<td>B</td>
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<td>47.3</td>
<td>43.9</td>
<td>43.1</td>
<td>41.3</td>
<td>41.8</td>
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<td>39.7</td>
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<tr>
<td>Astrand [5]</td>
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<td>45.6</td>
<td>43.2</td>
<td>8.0</td>
<td>0.49</td>
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<tr>
<td>Hagerman [6]</td>
<td>65.5</td>
<td>46.8</td>
<td>14.0</td>
<td>0.94</td>
<td></td>
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<tr>
<td>Marti [7]</td>
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<td>63.7</td>
<td>8.0</td>
<td>0.59</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Pollock [8]</td>
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<td>50.0</td>
<td>40.8</td>
<td>12.0</td>
<td>0.67</td>
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<tr>
<td>Robinson [9]</td>
<td>71.4</td>
<td>41.8</td>
<td>12.5</td>
<td>0.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Rogers [10]</td>
<td>54.0</td>
<td>51.8</td>
<td>5.0</td>
<td>0.28</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Trappe [11]</td>
<td>60.3</td>
<td>40.7</td>
<td>15.0</td>
<td>0.90</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*Group A: the original 15 subjects; group B: the 11 who were still alive at 33 years.
hypertension, as shown by the relatively low mean arterial pressures at rest and maximal work. This contrasts with the 60% incidence of hypertension in the US population over 65 [25].

Our seven and nine measurement points contrast to a maximum of two or three in other reports; there is a narrow age range in other studies, with only two including subjects over 65. The one study of equal duration has much younger subjects (25–58 years) and the one study with a comparable rate of VO₂ decline is of a shorter duration (8 years) [10]. Longitudinal data are difficult to obtain. Our data are therefore valuable, despite the small sample size.

Does a reduction of cardiovascular ageing improve longevity? If we include the four deceased members’ ages, the combined ages for 15 men would be 77 years—4–5 years beyond the life expectancy for American men.

What about quality of life? Validated quality of life scales were not available at the onset of this study. From a physical perspective, the present participants are engaging collectively in more than 20 vigorous activities (including hunting, skiing, mountain climbing and tennis). These are self-sufficient individuals, capable of great energy expenditure and leading independent lives.

In conclusion, adherence to a 33-year exercise regimen reduces the effect of ageing on cardiovascular function, as reflected in a relatively high VO₂ max and low measures of resting blood pressure, mean arterial pressure, heart rate reserve and body fat.

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**Key points**

- A 33-year programme of aerobic exercise in middle-aged American men reduced the effect of ageing on cardiovascular function.
- There was no change in resting blood pressure, heart rate or body composition, and little change in pulmonary ventilation, maximum heart rate, heart rate reserve or mean arterial pressure.

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**References**


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