An Assessment of the Exercise Capacity of Cardiac Patients


An attempt is made to relate the level of exercise at which disability occurs in cardiac patients with known oxygen consumption figures for various tasks. The limit to the maximum level of exercise in some patients with rheumatic heart disease is set by circulatory parameters, as in normal subjects, in others it is set by the excessive ventilatory cost of exercise.

The rehabilitation of the patient with heart disease must include placing him in appropriate employment. Gainful work serves not only economic ends but has important psychologic benefits. One of the important decisions the clinician must therefore make is an assessment of the level of effort that the patient can safely undertake in employment and sport. Traditionally the evaluation is made on subjective symptoms and physical signs. With reliance chiefly on the patient's statement of the grade of effort that produces breathlessness and discomfort the physician places him in one of the categories of functional capacity outlined by the New York Heart Association.

The clinical assessment has obvious limitations. The main weakness is apparent when one attempts to relate the level of effort at which the patient claims that disability occurs to a physiologic measure of work, namely, liters of oxygen consumed per minute. Unless this is done it is virtually impossible to match precisely an evaluation of the patient's functional capacity with the grade of effort required to carry out various types of work or sport. Some attempts have been made in this direction by Nylin and Bruce and Rodgers but the level of oxygen consumption at which disability occurs is not readily obtainable by their methods. The present studies are an endeavor to define precisely the maximum exercise capacities of cardiac patients in terms of liters of oxygen consumption per minute.

The maximum capacity for effort of normal men can be defined as the maximum level of "aerobic" oxygen consumption. Åstrand and Taylor and associates have demonstrated clearly that this is the rate of oxygen consumption at which a further increase in the grade of work does not call forth any addition to the level of oxygen consumption. It merely results in an increase in oxygen debt and a shortening of the time of effort. In the laboratory this figure is determined by exercising the subject at increasingly severe grades of work until a level of oxygen consumption, as defined above, is reached. The method is tedious and in sick patients severe grades of work must, of necessity, be avoided.

Recent observations by Berggren and Christensen and Åstrand suggested that the maximum aerobic oxygen capacity can be determined without imposing high work loads. It was shown in normal subjects that heart rate is a linear function of oxygen consumption up to the maximum level of aerobic oxygen consumption, and maximum heart rate of 194 ± 1.6 beats per minute is not exceeded in adults during severe exercise and that this rate is closely correlated with the maximum level of aerobic oxygen consumption. Hence if heart rate is plotted as a function of oxygen consumption at 3 or more grades of work and a linear curve is fitted to these plots and extrapolated to 190 beats per minute, then the oxygen consumption at this heart rate should closely approximate the maximum level of aerobic oxygen consumption of the individual measured by the more laborious method. Moreover, the 3 grades of work can be submaximal efforts.

Our first aim, therefore, has been to test the observations from Christensen's laboratory. Studies were made on 4 normal, trained men to satisfy ourselves as to the linearity of this
functional relationship up to maximal effort, to establish whether the maximum heart rate of the individual correlated closely with the maximum aerobic oxygen capacity, and to determine the variability of oxygen consumption determined in this way.

Having satisfied ourselves on these 3 points, we then measured heart rate, oxygen consumption, and ventilation at 3 grades of work on a group of untrained normal subjects and on 10 ambulant patients with rheumatic heart disease. Particular attention in this study has been paid to the ventilatory volume per minute at different levels of exercise. This is because we and others\textsuperscript{3, 5, 6} have shown that the maximum voluntary breathing capacity in normal individuals always exceeds by a good percentage the minute ventilatory volume observed at the maximum possible level of exercise. On this ground it is concluded that circulatory rather than ventilatory factors set the limit to the maximum level of oxygen consumption of normal individuals. In patients with rheumatic heart disease, however, Stock and Kennedy,\textsuperscript{7} Richards and Dickinson,\textsuperscript{8} and Cotes\textsuperscript{9} have shown a variable decrease in maximum breathing capacity and an increase in the ventilatory cost of one particular grade of exercise. It is unfortunate that these investigators studied only one level of exercise because no light was shed on the probability of the ventilatory cost increasing in a rapidly nonlinear manner as the level of exercise is raised. Should this happen in cardiac patients the limit to the maximum level of oxygen consumption might well be imposed by ventilatory and not cardiac factors as in the normal individual. To test this possibility we have compared the ventilatory function of the cardiac patients during exercise with the normal range of ventilatory volume per minute at different levels of oxygen consumption, up to 1.5 L. of oxygen per minute, of 24 young men living at Johannesburg (altitude 6,000 feet above sea level).

**Methods**

Repeated determinations were made on the 4 trained African men at different levels of work both on stepping stools and on a bicycle ergometer. On the latter, maximal effort was studied. The second group of subjects consisted of members of the laboratory staff; some played games regularly, others were quite sedentary. The third group was that of the cardiac patients. They were all ambulatory and, except for cases 11, 12, 15, and 18, did not complain of breathlessness on exertion. The fourth group consisted of 24 untrained young African recruits to the gold mines.

All submaximal effort studies were made by the subjects stepping on and off a stool 1 foot in height. Work was continuous and rigidly standardized by means of a metronome. There were 3 work periods, each of 20 to 30 minutes' duration. The rates of stepping were 1/20 seconds, 1/10 seconds, 1/5 seconds, and 1/2 1/5 seconds. Most cardiac patients were not put to the highest grade of stepping and cardiac cases 15 and 18 were unable to perform adequately at the third rate of work. Oxygen consumptions were measured by collecting expired air over 3 minutes, midway during the work period, in a Douglas bag or metering it directly into a Kofranyi-Michaelis apparatus. Aliquot samples were analyzed in a Haldane apparatus. Samples were required to check to at least 0.05 volumes per cent. Ventilation volume during work was taken from the 3-minute collection of expired air and expressed at body temperature, atmospheric pressure, and saturated air (B.T.P.S.). Maximum breathing capacities (M.B.C.) were determined over 15 seconds, the expired air being breathed into a Douglas bag. M.B.C.'s were repeated until 2 determinations checked closely. Heart rates were counted at the apex of the heart by auscultation, except in the cases of the men with fibrillation and the 4 normal men at the high rates of work. In those instances the rates are a mean of at least 5 estimations of ventricular rate from an electrocardiogram.

**Results**

**Heart Rate as a Function of Oxygen Consumption**

Our observations on 4 trained men at various grades of work up to maximal effort on a bicycle ergometer have demonstrated conclusively that heart rate is a truly linear function of oxygen consumption, and that the oxygen consumption at the maximum heart rates correlates closely with the maximum aerobic levels determined by successive increments in the rate of work.\textsuperscript{10} At a given rate of work the standard deviation of individual oxygen consumption is 0.11 L. per minute, so that differences between observed oxygen consumptions that indicate a real difference (with 95 per cent confidence) are

\[
0.11 \times \sqrt{2} \times 1.96 = 0.31 \text{ L. per minute}
\]
Differences of this order in maximum aerobic oxygen consumptions are therefore also significant.

Linear curves were fitted to the plots of heart rate as a function of oxygen consumption at the 3 grades of submaximal work and were extrapolated to 190 heart beats per minute. The fitted curves for the cardiac men are given in figure 1 and for the cardiac women in figure 2. Comparisons between individuals of the maximum aerobic oxygen capacities obtained in this way are more meaningful when corrected to a standard weight. For this purpose we have arbitrarily selected 150 lbs. The corrected mean value of maximum aerobic oxygen consumption for the 4 trained men is 3.6 (range 2.8 to 5.0) L. of oxygen per minute; for the 5 laboratory staff men the mean is 3.4 (range 2.7 to 3.7) L. of oxygen per minute; for Åstrand's athletic young men 4.0 L. of oxygen per minute; and for his athletic young women, 3.2 L. of oxygen per minute.

The maximum aerobic capacities of the cardiac patients derived from figures 1 and 2 are considered here as percentages of Åstrand's normal athletic young men and women. This is done to bring out more clearly the departure from normality of the cardiac patients. Two of the cardiac women have approximately 50 per cent of the normal maximum aerobic oxygen capacity and the remainder were within 75 per cent of normal. Two cardiac men, cases 16 and 18, have only 25 per cent of normal. The remainder were more than 50 per cent of normal. All of the cardiac patients fall outside the range of ±2 S.D. from Åstrand's means.

Two cardiac patients, 15 and 18, are instructive in that they demonstrate a limitation of effort due to ventilatory disability at a level of oxygen consumption well below that assessed from a heart rate of 190 beats per minute as the maximum. In other words, they are limited by ventilatory and not circulatory factors as is the case in normal men and in the other 8 cardiac patients. Case 18, table 1, on June 7, was fibrillating at 100 to 120 beats per minute at rest, and at a stepping rate of 1 step/10 seconds and an oxygen consumption of 0.79 L. of oxygen per minute he had a heart rate of 190 beats per minute. He was very breathless and was unable to perform at 1 step/5 seconds for sufficiently long to measure oxygen consumption. He was acutely distressed but his heart rate did not rise above 190 beats per minute. By July 6 he had been established in regular rhythm on quinidine. He was then able to do 1 step/5 seconds for 10 minutes even though very breathless at a heart rate of 158 beats per minute. The oxygen consumption of 1.1 L. per minute at 1 step/5 seconds is the maximum level of effort, rather than 1.5 L. of oxygen per minute obtained from extrapolation of heart rate to 190 beats per minute.

Similarly, case 15 was very breathless at 1 step/5 seconds at an oxygen consumption of 0.89 L. per minute with a heart rate of 124 beats per minute. This figure is his maximum level of oxygen consumption rather than 2.2 L.

![Fig. 1 Top. The curves fitted to heart rates at 3 submaximal rates of oxygen consumption (L. per minute, abscissa) for 5 cardiac women and extrapolated to 190 beats per minute (ordinate).](image)

![Fig. 2 Bottom. The curves fitted to heart rates (ordinate) at 3 rates of oxygen consumption (abscissa) for 5 cardiac men.](image)
of oxygen per minute obtained by extrapolating heart rate to 190 beats per minute. Case 15 was fibrillating, but the rate was well controlled on digitalis. He had been in cardiac failure previously and now had ascites and edema, which was treated by weekly injections of mercurial diuretics.

Ventilation-Volume (B.T.P.) as a Function of Oxygen Consumption

The ventilation volume per minute as a function of oxygen consumption of our 24 men at 6,000 feet altitude can be compared with Åstrand’s observations on 40 young men at sea level. At 1 L. of oxygen consumption per minute the mean ventilation of our men is 29.3 ± 0.5 L. per minute, which is significantly different from Åstrand’s 20.6 ± 0.5 at sea level. The difference of 45 per cent is undoubtedly due to the effect of altitude.

In figure 3 are given the ventilatory volumes of the 24 men at rest and at 3 rates of work. Tolerance limits have been calculated that include almost all observations in the experimental population,* hence any curves of ventilation that fall outside these tolerance limits can be regarded as abnormal. All of the laboratory staff and the 4 trained men fall within the limits. With 2 exceptions the cardiac patients also fall within the limits. Two features characterize the responses of the exceptions, no. 15 and 18. First, ventilation was greatly in excess of normal, even at low levels of oxygen consumption and second, the minute ventilation increased in a markedly nonlinear fashion with rise in oxygen consumption.

Both no. 18 and 15 were slightly breathless at the lowest level of work and more breathless at the intermediate level; and no. 18, in his first session, could not do the highest level, but was able to carry on for 10 minutes, although very breathless, on the second occasion when regular rhythm was established. Both of these men had markedly diminished M.B.C.’s and covariance matrix of α and β. The tolerance region is defined by an ellipse

\[ \Gamma_{11}(\alpha - A)^2 + 2\Gamma_{12}(\alpha - A)(\beta - B) + \Gamma_{22}(\beta - B)^2 = C(\theta_1\theta_2 k) \]

where \( C \) is a constant (depending on the sample size \( k \)) such that we may state with 100 \( \theta_1 \) per cent confidence that this ellipse contains at least 100 \( \theta_1 \) per cent of the population of (\( \alpha, \beta \)) pairs. As \( k \to \infty, C \to \chi^2_{2}(\theta_1). \) This ellipse having been defined, 2 bands may be calculated that are the tolerance limits to the individual straight lines \( 0 = \alpha + \beta V. \) This is done by finding the minimum and maximum of \( \alpha + \beta V \) subject to the condition that (\( \alpha, \beta \)) lies on the ellipse given above.

* The problem is to set limits within which we expect subjects for the same population to fall.

A tolerance region will be set for \( \alpha \) and \( \beta \) in combination by using the fact that

\[ \Gamma_{11}(\alpha - A)^2 + 2\Gamma_{12}(\alpha - A)(\beta - B) + \Gamma_{22}(\beta - B)^2 = C(\theta_1\theta_2 k) \]

is a \( \chi^2 \) variable if the joint distribution of \( \alpha \) and \( \beta \) is normal and \( \Gamma_{ij} \) are the coefficients of the inverse of the

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**Table 1.—Exercise Capacity Tests on Subject No. 18**

<table>
<thead>
<tr>
<th>Date</th>
<th>Rate of work</th>
<th>Heart rate</th>
<th>Ventilation rate (B.T.P.S) (L./min.)</th>
<th>Oxygen cons. (L./min.)</th>
<th>Oxygen per cent absorbed</th>
<th>Clinical state</th>
</tr>
</thead>
<tbody>
<tr>
<td>7/6/55</td>
<td>Mild</td>
<td>132</td>
<td>142</td>
<td>145</td>
<td>131</td>
<td>32.3</td>
</tr>
<tr>
<td>6/7/55</td>
<td>Mild</td>
<td>110</td>
<td>115</td>
<td>116</td>
<td>116</td>
<td>24.7</td>
</tr>
<tr>
<td>7/6/55</td>
<td>Moderate</td>
<td>141</td>
<td>170</td>
<td>180</td>
<td>180</td>
<td>45.1</td>
</tr>
<tr>
<td>6/7/55</td>
<td>Moderate</td>
<td>128</td>
<td>136</td>
<td>138</td>
<td>132</td>
<td>33.6</td>
</tr>
<tr>
<td>7/6/55</td>
<td>Moderately hard</td>
<td>180</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>6/7/55</td>
<td>Moderately hard</td>
<td>153</td>
<td>158</td>
<td>—</td>
<td>—</td>
<td>70.0</td>
</tr>
</tbody>
</table>
Fig. 3. Ventilation, liters per minute B.T.P.S. (ordinate), as a function of oxygen consumption at rest (abscissa) and at 3 rates of work of 24 healthy young men with the tolerance limits. X---X is the data of cardiac no. 18. O——O is the data of cardiac no. 15.

hence a small ventilatory reserve (M.B.C. — ventilation per minute/M.B.C.) at the high level of work.

Of the other cardiac patients among the women only no. 11 and 12 complained of breathlessness during the tests. No. 11 did so at a ventilatory rate of 25.0 L. per minute. Her M.B.C. was 101.4 L. per minute. Her ventilatory reserve was thus 75 per cent when breathlessness was observed. By contrast the 4 trained men were breathless only when more than 60 per cent of the ventilatory reserve was employed.

**Discussion**

In normal men oxygen consumption increases linearly with rate of work up to a maximum value designated the maximum "aerobic" oxygen consumption. Further increase in rate of work does not produce any rise in oxygen consumption. Ventilation volume per minute at the maximum level of oxygen consumption is less than the maximum voluntary breathing capacity, hence one can infer that even though these subjects are breathless at this level of effort, nonetheless there is adequate ventilatory reserve. This being the case, the limit to the rate of oxygen consumption per minute in

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Mean heart rate (beats/min.)</th>
<th>Oxygen consumption (L/min.)</th>
<th>Ventilation B.T.P.S. (L/min.)</th>
<th>M.B.C. (L/min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laboratory Staff</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>108</td>
<td>0.73</td>
<td>31.37</td>
<td>163.0</td>
</tr>
<tr>
<td>116</td>
<td>1.01</td>
<td>30.83</td>
<td></td>
<td></td>
</tr>
<tr>
<td>148</td>
<td>1.64</td>
<td>48.22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>96</td>
<td>0.69</td>
<td>23.02</td>
<td>80.3</td>
</tr>
<tr>
<td>112</td>
<td>1.06</td>
<td>31.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>132</td>
<td>1.57</td>
<td>44.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>84</td>
<td>0.60</td>
<td>18.91</td>
<td>142.5</td>
</tr>
<tr>
<td>96</td>
<td>0.86</td>
<td>28.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>124</td>
<td>1.64</td>
<td>44.94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 (unfit)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>92</td>
<td>0.71</td>
<td>22.19</td>
<td>180.1</td>
<td></td>
</tr>
<tr>
<td>108</td>
<td>1.16</td>
<td>32.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>132</td>
<td>1.68</td>
<td>42.20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 (fit)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>0.84</td>
<td>20.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>1.16</td>
<td>27.54</td>
<td></td>
<td></td>
</tr>
<tr>
<td>120</td>
<td>1.84</td>
<td>43.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>148</td>
<td>2.72</td>
<td>63.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>188</td>
<td>3.48</td>
<td>90.87</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Women Cardiacs |
| 9 | 116 | 0.41 | 13.97 | 142.5 |
| 128 | 0.57 | 18.08 | |
| 156 | 0.76 | 23.03 | |
| 10 | 108 | 0.31 | 10.28 | 74.0 |
| 120 | 0.51 | 19.45 | |
| 148 | 0.84 | 26.85 | |
| 11 | 104 | 0.43 | 13.76 | 101.4 |
| 112 | 0.58 | 21.65 | |
| 128 | 0.83 | 25.03 | |
| 12 | 100 | 0.19 | 7.81 | 95.9 |
| 108 | 0.30 | 11.51 | |
| 132 | 0.67 | 25.76 | |
| 13 | 104 | 0.50 | 14.66 | 98.6 |
| 120 | 0.84 | 23.43 | |
| 144 | 1.09 | 29.59 | |

| Men Cardiacs |
| 14 | 108 | 0.65 | 19.73 | 178.1 |
| 120 | 0.80 | 23.43 | |
| 132 | 1.07 | 30.14 | |
| 15 | 96 | 0.47 | 22.61 | 76.7 |
| 112 | 0.77 | 35.60 | |
| 124 | 0.89 | 46.60 | |
| 16 | 116 | 0.50 | 16.99 | 153.4 |
| 128 | 0.67 | 20.55 | |
| 164 | 0.97 | 30.37 | |
| 17 | 121 | 0.57 | 18.77 | 86.3 |
| 132 | 0.67 | 21.78 | |
| 144 | 0.96 | 30.33 | |

* See table 1.
maximal exercise is set by circulatory and not ventilatory factors.

The circulatory parameters concerned in the rate at which the body utilizes oxygen, aerobically, during exercise are heart rate, stroke volume and arteriovenous oxygen difference. The relationship may be expressed as follows:

\[
\text{O}_2 \text{ consumption (L. per minute)} = \frac{\text{Heart rate (beats per minute)}}{\text{Stroke volume (ml. per minute)}} \times \frac{\text{A-V difference (ml. per 100 ml. blood)}}{\text{(ml. per 100 ml. blood)}}
\]

The interrelationship of these variables at different rates of oxygen consumption is contained in the data obtained by the direct Fick determinations of Donald and associates,10, 12 Dexter and associates,13-14 and Freedman and associates,15 on normal and cardiac volunteers. Direct Fick studies were made at a maximum work rate of 2.0 L. oxygen per minute. Recently Asmussen and Nielsen16 obtained data at or near the maximum working capacity using the uptake of an inert gas and a dye-dilution technic. From these various studies it is now possible to make a relatively coherent statement on the way in which the 3 circulatory parameters contribute to an increase in oxygen consumption.

In both Nielsen’s and Freedman’s studies stroke volume increases with rise in oxygen consumption up to a maximum, which occurs at an oxygen consumption of between 1.4 and 1.6 L. per minute. Heart rate and A-V difference, on the other hand, both increase linearly with rise in oxygen consumption right up to the maximum aerobic oxygen consumption. At this level of oxygen consumption, heart rate in normal adults reaches a limiting value of 190 beats per minute.3 The limiting value of A-V difference is not known so precisely. Even at high levels of cardiac output during exercise a percentage of the total outflow goes to areas such as brain and liver where the extraction rate of oxygen is low. The figure usually given for maximum A-V difference of 140–150 ml. per L. of blood therefore seems reasonable. The actual value will undoubtedly depend upon the volume of muscle engaged in exercise.4 From these data it can be concluded that, at low levels of effort in normal men, stroke volume, heart rate and A-V difference all contribute to the rate at which oxygen is made available to tissues. Above a level of 1.5 L. of oxygen per minute (approximately 6 times the resting level) only heart rate and A-V difference are involved. Both increase as linear functions of oxygen consumption up to the maximum aerobic level.

The situation with cardiac patients is not so simple. In 8 of the 10 cases studied by us ventilation volume per minute as a function of oxygen consumption fell within the tolerance limits we have calculated for normal men. Moreover, the ventilation rate that would obtain at the maximum aerobic oxygen consumption (obtained by extrapolation of heart rate to 190 beats per minute) was less than the maximum breathing capacity. These cases, therefore, in these 2 respects conformed to normal. Two cardiac patients were, however, quite different. Even at the low rate of work they were breathless and ventilation was greater than normal. At high rates of work they were very breathless and ventilation as a function of oxygen consumption rose nonlinearly, so that no. 18, at an oxygen consumption of 1.0 L. per minute, ventilated at twice the normal volume. The maximum breathing capacities of these men were well below normal. Ventilation at the highest rates of work encroached seriously, therefore, upon ventilatory reserve. Heart rates of the 2 men, no. 15 and 18, were well below 190 beats per minute when they were very breathless. It is probably safe to say that in these 2 men the factor that limits exercise is primarily respiratory and not cardiac capacity per se. An objective method of determining the upper permissible limit of effort would be the level of oxygen consumption at which breathlessness produced discomfort. For no. 15 and 18 this would be 0.89 and 0.79 L. of oxygen per minute respectively. An alternative would be the point in the ventilation curve where it tends to become highly nonlinear. The minute volume ventilated by these 2 cardiac cases is grossly in excess of normal and raises an interesting problem in the control of ventilation. The explanation is unlikely to be a rise in \(pCO_2\) in blood because the percentage of CO\(_2\) in expired air is well below normal, indi-
cating that CO₂ is being blown off as in volun-
tary hyperventilation. There are 2 probable
explanations. One is an accumulation of edema
in pulmonary tissues with a loss of lung elas-
ticity. Hayward and Knott²⁷ have shown this
condition in rheumatic heart patients with
dyspnea. The other is the excessive accumula-
tion of lactic acid in blood and has been
demonstrated by Cotes.⁹ Both mechanisms are
undoubtedly due to an inability to raise the
cardiac output in a normal manner with in-
crease in exercise. Hence it can probably safely
be inferred in these and similar patients that
the level of oxygen consumption at which ven-
tilation departs in a markedly nonlinear manner
from the normal is the level of effort at which
cardiac output also fails to show the normal
response to exercise.

In the other 8 cardiac cases the factor
limiting exercise was not ventilatory function,
as pointed out. Can we assume that the heart
rate of 190 beats per minute was correlated, as
in normal subjects, with the maximum aerobic
oxygen capacity? The question can be put in
another way that clarifies the issue. Is there
any evidence in cardiac patients that at a heart
rate of 190 beats per minute either A-V differ-
ence or stroke volume have not been employed
up to their limits? If they have, then clearly at
a heart rate of 190 beats per minute there is no
other way of transporting more oxygen by the
circulation and 190 beats per minute must be
correlated with the maximum aerobic oxygen
capacity. Dexter's data¹³, ¹⁴ throw light on this
problem if stroke volume, A-V difference, and
heart rates are plotted as functions of oxygen
consumption. The main difference from normal
is that severe cardiac cases are unable to in-
crease stroke volume with increase in effort.
Consequently heart rate and A-V difference as
function of oxygen consumption rise more
rapidly than in normal subjects and reach their
limiting values at relatively low levels of
oxygen consumption. In both severe and mild
cardiac cases extrapolation of A-V difference to
140 to 150 ml. per L. of blood indicates that
this level is reached at lower levels of oxygen
consumption than is the heart rate of 190 beats
per minute. We have shown that heart rate is a
linear function of oxygen consumption and
therefore can safely be extrapolated. The only
question open to challenge is extrapolating
A-V difference linearly. In the cardiac patient
increase in cardiac output is limited because of
the restriction in stroke volume. Consequently,
as oxygen consumption increases, A-V differ-
ence is used up more rapidly, as is clear in
Dexter's data, and not less rapidly than nor-
mal. The error, if any, in extrapolation of A-V
difference will therefore be on the right side,
viz., towards oxygen consumptions at 140 to
150 ml. per L. of blood, which are too low.
Hence we can safely assume that at a heart
rate of 190 beats per minute the other 2
parameters have been fully employed and
therefore this heart rate must be closely cor-
related with the maximum aerobic oxygen
capacity.

The majority of cardiac cases in this group
did not complain of dyspnea during the tests,
but 2 women did so complain. For example,
no. 11 had a ventilation rate of 25.0 L. at 0.83
L. of oxygen per minute. Her maximum
breathing capacity was 101.0 L. per minute. In
spite of a ventilatory reserve of 75 per cent she
complained of dyspnea. She was excessively
worried about her "heart trouble" and had
avoided any but the mildest form of exercise.
Her musculature was flabby. Dyspnea could
well have been due to a level of ventilation to
which she was unaccustomed. There appears to
be no cogent reason why such a patient should
not lead a more active life. The clinician's
impression was one of cardiac neurosis and our
data are helpful in lending him confidence to
encourage her to pursue a more active life
with a limit set by her maximum oxygen con-
sumption as in the other cardiac patients of
this group.

In assessing the level of exercise and, more
especially, the grade of work that the cardiac
can be allowed to do, these 2 broad categories
are useful. The cases with a ventilatory limita-
tion should not be called upon to work for a
normal day at a rate that produces more than
mild breathlessness. The level of oxygen con-
sumption at which mild and moderate breath-
lessness occurs is clearly apparent in the data
on no. 15 and 18.

The cardiac patient with exercise capacity
limited by circulatory rather than ventilatory parameters is at present in greater danger of being misplaced in regard to the grade of work that is permissible. The criterion generally employed by the clinician for all cardiac patients is the level of work at which breathlessness occurs. In this group of patients, heart rate rises abnormally rapidly with increase in oxygen consumption and the circulatory parameters are exhausted at a level of ventilation that, as in normal subjects, is well below the maximum voluntary breathing capacity. Hence the maximum permissible effort of these cardiac patients must be judged in relation to the maximum aerobic oxygen capacity, as in normal men, rather than by the level of effort at which breathlessness occurs.

The maximum aerobic oxygen capacity defines the level of work that normal and trained men can sustain for only a few minutes before fatigue supervenes. It serves to differentiate between men with different capacities for maximum effort. Thus Åstrand showed that Landy, the 4-minute miler, has a maximum aerobic capacity of over 5.0 L. of oxygen per minute, whereas the average of his young, fit men was 3.9 L. of oxygen per minute. Its usefulness in industrial employment is as a standard against which to assess a permissible level of oxygen consumption for 8 hours of work. Thus Åstrand stated that healthy factory workers should not work at more than half their maximum capacity. While there is no evidence that a similar grade of effort relative to the maximum capacity would have any harmful effect on the cardiac patient, we suggest that, until we have evidence on the point, the maximum permissible level of work be set at one third of the maximum aerobic oxygen capacity for this particular group of cardiac patients. This information should be of use to the hospital social worker in rehabilitating the cardiac patient. The range of permissible work can be judged in regard to meaningful domestic activities and industrial tasks by correcting the maximum aerobic oxygen consumption to 150 lbs. weight, and comparing them with data in tables from laboratories engaged in such studies.

A further use of the maximum aerobic oxygen consumption figure, whether assessed by ventilatory or circulatory criteria, is as an objective measure of the effect of type of heart disease and the regimen of therapy on the exercise status of the patient. Thus no. 18 was fibrillating on the first occasion and had acute discomfort at 0.79 L. of oxygen per minute, but after treatment with quinidine to bring him into regular rhythm he was able to exercise briefly at 1.1 L. of oxygen per minute and his ventilation volume per minute improved considerably. It may be equally useful to judge objectively the effect of valvulotomy. Finally, it is an index with which to judge the progress of the disease with time. Regular examinations, say at yearly intervals, would give a measure of the improvement or deterioration of the cardiac patient.

**Summary**

Heart rate, oxygen consumption, and minute ventilation volume were measured at 3 grades of work on 4 trained men, 5 untrained men, and 10 ambulatory cardiac patients with rheumatic heart disease. Ventilation volume/minute at different levels of oxygen consumption were also measured on 24 young men to set up standards against which to judge abnormality at an altitude of 6,000 feet above sea level.

Eight of the cardiac patients had ventilatory volumes/minute at the 3 levels of oxygen consumption that were essentially the same as normal subjects and therefore ventilatory function, as in normal subjects, did not appear to be the factor that sets a limit to the maximum possible level of oxygen consumption during severe exercise. Hence, as in normal subjects, the maximum level of oxygen consumption was obtained by extrapolation to 190 beats/minute of the heart rates obtained at 3 submaximal grades of work when the heart rates are plotted as a function of oxygen consumption.

Two of the cardiac patients had an abnormal pattern of ventilatory response to exercise. Even at mild effort they had a larger minute volume than normal and this departed further from normal as the level of exercise was raised. The possible implications of this observation are discussed and the primary cause is considered to be an inability to increase the
cardiac output in a normal manner in relation to exercise.

The ventilatory responses to exercise, therefore, serve to distinguish 2 clear categories of rheumatic heart disease patients. The method of assessing the maximum possible level of oxygen consumption of patients in these 2 categories is discussed.

It is suggested that assessment of the individual cardiac patient’s maximum level of oxygen consumption can be employed as an objective index in choosing a safe level of work in employment, in assessing the influence of therapy or surgery, and in following the course of the disease with time.

ACKNOWLEDGMENT

We are pleased to acknowledge the help and useful criticism of Dr. van Lingen and Dr. MacGregor of the Department of Medicine, University of the Witwatersrand. The work was done in the Department of Medicine with Professor Elliott’s permission. Mr. J. S. Maritz did the statistical analyses.

SUMMARIO IN INTERLINGUA

Velocitate del corde, consumption de oxygeno, e ventilation per minuta esseva mesurate a 3 nivello de exercitio in 4 homines accostumate a labor physic, 5 homines non assi accostumate, e 10 patientes cardiac ambulatori qui habeva morbo cardiac rheumatic. Le volumine ventilatori per minuta esseva etiam mesurate in 24 juvene homines normal con le objectivo de establir standards pro le judicamento de anormalitates a un altitude de circa 5500 m supra le nivello del mar.

Oceto del patientes cardiac habeva, a omnele 3 nivello de consumption de oxygeno, volumines ventilatori per minuta que esseva essentialemente identic con le valores in subjectos normal. Per consequente, le function ventilatori in iste casos, exactamente como in subjectos normal, non pareva representar le factor que impone le limite del plus alte nivello possibile de consumption de oxygeno in exercitios sever. Assi, como in subjectos normal, le nivello maximal del consumption de oxygeno poteva esser obtenite super le base del velocitates cardiac obtenite a tres grados submaximal de exercitio per extrapolation a 190 pulsos per minuta in graphicos monstrante le velocitates cardiac como function del consumption de oxygeno.

Duo del patientes cardiac habeva formas anormal del responsa ventilatori a exercitio. Mesmo sub le conditiones de leve grados de effort ille habeva volumines per minuta que exceedeva le norma, e le differentias ab le norma creseva quando le nivello del exercitio esseva elevate. Le signification de iste factos es discutite, e le these es formulate que le causa primari es le incapacitate de augmentar le rendimento cardiac in relation normal al grado del exercitio.

Assi le responsa ventilatori a exercitio pote servir a distinguere clamente duo categorias de patientes de morbo cardiac rheumatic. Es discutite le methodo de evalutar le plus alte nivello possibile del consumption de oxygeno del parte de patientes in iste duo categorias.

Es presentate le idea que le calculation del nivello maximal de consumption de oxygeno in le casos individual de patientes cardiac pote esser usate como indice objective in determinar qual nivello de efforto es admissible in le occupation professional del paciente, in evalutar le influentia de therapia o chirurgia, e in traciar le desenvoluppamento del morbo in le curso del tempore.

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Acetylcholine infused into the main pulmonary artery caused a slight fall in pulmonary arterial pressure when the subject breathed ambient air, but a greater fall in pressure after pulmonary hypertension had been produced by hypoxia. The fall in pressure was associated with either a constant or an increased cardiac output. The evidence suggested that acetylcholine caused vasodilatation in the human lungs. This observation is in conflict with earlier reports of others that acetylcholine can constrict vessels of animal lungs.

Aviado
An Assessment of the Exercise Capacity of Cardiac Patients
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Circulation. 1957;16:384-393
doi: 10.1161/01.CIR.16.3.384
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/16/3/384

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