Maximal Heart Rate and Treadmill Performance of Healthy Women in Relation to Age

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SUMMARY Maximal treadmill exercise heart rate, work capacity and electrocardiographic response were studied in 95 asymptomatic, predominantly sedentary women between the ages of 19 and 69 years. Average maximal heart rate (MHR) was found inversely related to age, such that MHR = 216 – 0.88 (years of age) ± 10 beats/min (X±1 SD).

Treadmill exercise endurance was 7.64 min ± 1.99. The reduction of treadmill endurance with advancing age was not statistically significant.

Asymptomatic ST-segment depression occurred in 6% of subjects. In 5% the ST segment sloped upward, and in 1% it was flat. Mean age of women with ST depression was 52 years, compared with 39 years mean age of all subjects. Premature beats during exercise were found in 20 of 95 subjects, and were not related to age.

Graded exercise testing of women employing target heart rates should use heart rate tables developed especially for women. These tables do not require correction for athletically trained or sedentary life-style.

IN ORDER PROPERLY TO EVALUATE the results of maximal or near maximal treadmill tests for coronary artery disease or for the quantification of cardiovascular reserve in valvular heart disease or cardiomyopathies, normal response values are required for comparison. These reference values should include the maximal treadmill work capacities of normals, the heart rates attained in performing maximal exercise and the electrocardiographic responses of normals to such testing.

Maximal exercise heart rate and work capacity throughout childhood and adulthood in women and men are sparsely documented. Published studies on women have tended to concentrate on fairly narrow age ranges as in studies of college students, studies limited to individuals actively engaged in physical education programs, or studies limited to symptomatic patients who should not be classified as normal. One of the largest groups of women was studied by Cumming and colleagues, 357 women between the ages of 20 and 83 years. However these authors did not state what evidence they employed to confirm that their subjects were normal, nor was it reported whether the volunteers were taking any medications at the time of testing. Only if the range and variability of maximum exercise heart rate in women is known is it possible to apply the graded exercise test principle appropriately to women, i.e., to know with reasonable confidence what level of tachycardia represents 90% of average maximum exercise heart rate for a given age, in order to recognize when in the course of an exercise test a near maximal intensity of exercise is taking place. Additionally, it would be desirable to establish normal ranges of treadmill exercise endurance with respect to the age of normal women volunteers.

Subjects and Methods

The volunteers studied were 95 asymptomatic women between the ages of 19 and 69 years. Their mean age was 38.9 years, and there was quite even distribution of their ages as shown in figure 1. Their mean height was 164.8 cm ± 5.8 (in this and subsequent values, the number preceding the ± symbol represents the mean; the number following it, one standard deviation of the mean). Mean
weight was 59.8 ± 7.8 kg. Both height and weight values were also smoothly distributed. Volunteers were recruited from medical center personnel, local churches and high school faculties by making general announcements of the study and the need for subjects. No subject was currently under treatment for an acute illness although three were taking mild antihypertensive medications. All denied episodic chest discomfort, palpitation, fainting spells or undue dyspnea on exertion, and none had been told that she had cardiovascular disease (other than elevated blood pressure). Habitual physical activity level was assessed by asking whether the subject engaged in any type of vigorous physical exertion in sessions lasting at least thirty minutes each, and taking place at least three times a week. A negative answer was interpreted as indicating athletically untrained or sedentary state. Physical examination procedure included indirect blood pressure measurement, peripheral arterial and venous pulse examination, cardiopulmonary auscultation and examination of the feet and ankles. All blood pressures were between 100 and 160 mm Hg systolic and between 60 and 90 mm Hg diastolic. No abnormalities of heart, lungs or musculoskeletal systems were noted, and no pitting edema was present. Resting 12 lead ECGs or Frank lead ECGs on all subjects were within normal limits.

The exercise test procedure was explained to each volunteer. We stressed the importance of keeping us apprised of symptoms when they appeared. Each understood she was agreeing to continue exercise as long as she could tolerate doing so; that the experiment would be only as valid as her conscientious effort to perform maximum exertion. Each subject was made aware of her right to withdraw her participation at any moment, and each gave informed consent to be tested. Each subject had the treadmill operation demonstrated to her, and each familiarized herself with the treadmill and the other equipment in the laboratory. None of these women had undergone maximal or near maximal treadmill exercise testing before.

Continuous heart rate and rhythm monitoring was provided by the use of an exercise ECG patient cable attached to self-adhering fluid-coupled electrodes on the chest. The first 51 subjects were monitored by means of Frank X,Y,Z leads and Wilson V_s. On the following 44 subjects a single bipolar lead, CM-5, which resembles V_s, was used. The ECG was continuously displayed on a multichannel oscilloscope and recorded on magnetic tape. Heart rate was monitored with a digital cardiograph but the rates reported were all measured from ECG strips recorded at 50 mm/sec at one minute intervals. ECG recorder chart speed accuracy (± 1%) was reconfirmed at every test.

Treadmill exercise took place in successive uninterrupted three minute stages at speeds and elevations as follows: stage 1, 2.7 kph, 10% grade; stage 2, 4.0 kph, 12% grade; stage 3, 5.5 kph, 14% grade; stage 4, 6.8 kph, 16% grade; stage 5, 8.0 kph, 18% grade.

Treadmill speed (± 2%) and elevation accuracy (± 0.5%) were confirmed before and after the study. The exercise laboratory temperature was maintained between 21°C and 23°C.

Exercise was terminated upon request of the subject; further inquiry determined whether this was because of inability to continue or for a discretionary reason. Exercise was also terminated if the subject became in need of holding onto the handrails in order to maintain her position on the treadmill. Exercise would have been stopped upon recognition of any disorder of cerebral or myocardial perfusion, blood pressure regulation or neuromuscular coordination, cardiac rhythm or conduction had any of these occurred.

Observation and recording were continued for six minutes after exercise with the subject in a sitting position. Blood pressure was measured indirectly during each stage of exercise and every two minutes after exercise.

Results

All exercise tests herein reported represented convincingly good effort on the part of each subject. No instances of hypotension occurred. No tests were interrupted by anyone other than subjects themselves. No disagreeable side effects or misadventures were encountered.

These women did not appear to stress themselves as severely as have men volunteers in similar test situations participating in maximal exercise studies. These subjects persevered until they had to grasp handrails to stay in place, or refraining from this actually lost ground. Despite this, they usually did not develop impressively fast, labored breathing, cool, clammy skin or staggering gait just before stopping as had most of our men volunteers.

Treadmill exercise duration ranged from 3.5 min to 12.5 min, mean 7.64 min ± 1.00 SD. Exercise duration was weakly related to maximum heart rate (r = 0.22) but interestingly the expected inverse relationship to subject age was insignificant (P = 0.3). None of the other variables we studied was significantly related to exercise duration. Height and weight of subject were not related to exercise endurance or other variables but were related to each other (r = 0.48). Specifically, there was no established tendency toward increased weight with advancing age.

All heart rate observations were related to subject age and to each other. Resting heart rate was inversely but weakly related to age (r = -0.33). At the end of each 3 min exercise stage the relationship to age strengthened, and at peak exercise the maximum heart rate showed its closest correlation with age (r = -0.76). Maximum heart rate in women may be predicted by the equation, MHR = 216 - 0.88 (years of age) (fig. 2). This prediction has a coefficient of variation of 5.5% (standard deviation, ± 10 beats/min; r² = 0.56). No significant relationship was found between
resting heart rate and maximum exercise heart rate. On the other hand, by adding both resting heart rate and the heart rate attained at the end of the first 3 minute exercise stage, approximately 10% improvement in prediction is possible: 

$$\text{MHR} = 162 + 0.266 \times (\text{resting heart rate}) + 0.164 \times (3 \text{ min exercise heart rate}) - 0.70 \times (\text{age})$$

With this equation the coefficient of variation is reduced to 5.0% and standard deviation is reduced to 9 beats/min. (Even this is hardly an exact prediction, since the 95% confidence limit is ± 18 beats, or a total of 36 beats/min in range.)

The mean heart rate and one standard deviation from the mean at the end of the first 3 minute stage of exercise is 139 ± 19 beats/min; at the end of the second stage, 165 ± 18 beats/min; and at the end of the third stage, 176 ± 14 beats/min (fig. 3). Only two women completed the fourth stage and none completed the fifth or higher stages.

We considered the question whether women with greater exercise capacity might have a different age regression of maximum heart rate from that of women with lesser capacity. Age regressions were calculated separately for women with above average treadmill exercise durations and those with below average duration. No significant difference in the slopes of the regressions was found, and indeed the two curves were virtually identical. It should be emphasized that nearly all subjects were classified by us as sedentary rather than physically trained. It is possible that a different result would have occurred if the tested population had included an appreciable number of athletes.

In these asymptomatic women heart rate dropped rapidly following termination of exercise, from a mean MHR of 181 to 145 beats/min after one minute of rest. Deceleration of heart rate receded in the second minute postexercise by a mean of 22 beats to 123/min. This trend persisted to the fourth minute when the mean was 107/min. By this time heart rate slowing became very gradual; at 6 minutes the mean heart rate was 102. All postexercise heart rates, as well as resting and exercise heart rates, clearly had an inverse relation to age, which diminished slightly in the postexercise period; at stop of exercise $r = -0.76$; 2 min postexercise, $r = -0.61$; 4 min postexercise, $r = -0.54$; 6 min postexercise, $r = -0.48$.

We expected women with greatest physical fitness, and therefore with longest treadmill endurance, would show a more rapid deceleration of heart rate after exercise than those with shorter exercise times. No such correlation with treadmill exercise time was noted, however.

**Electrocardiographic Findings**

In this group of asymptomatic women no clinical symptoms or signs suggested the presence of ischemic heart disease during or after the exercise stress. Completely in the absence of chest pain, however, six of 95 subjects (6%) developed ST-segment depression which was greater than 0.1 mV at the J point and at least 0.1 mV 80 msec after the J point* (fig. 4). Five of the six cases were of the slowly ascending ST-segment variety and the remaining one was flat. The mean age of the women with ST depression was 52 years, and only one was as young as 38. Since the mean age of all subjects was 39, those with ST depression were notably older than the average.

Of the six with ST depression, two persons had blood pressures over 130/84. One was a 60-year-old woman with blood pressure 160/88, and the other a 59-year-old with blood pressure 150/90. Neither was receiving antihypertensive therapy.

One-fifth (20 of 95) of the subjects manifested premature beats during the test (fig. 5). The number of premature beats from a single subject ranged from one to fifty. One subject had junctional premature beats, three had atrial premature beats, and 16 had ventricular premature beats. Of those with ventricular premature beats, three had a single instance each of paired multiform premature beats. In all other instances the premature beats were uniform in contour and coupling interval, and not encroaching on preceding T waves. In contrast with ST-segment depression, premature beats were not

*Since the beginning of this study Froelicher has shown that lead CM-5 has about 50% more amplitude sensitivity than Wilson V. Therefore we reexamined all CM-5 recordings to see whether there were any ST depression whose classification might be changed by this difference in sensitivity. There were none.
concentrated in the older subjects but were well distributed throughout the group.

Discussion

Heart Rate Response

The maximum heart rates found in Astrand's study are remarkably similar to those we found, and in fact any difference in age regression of maximum heart rate could be explained by chance variation (table 1). The two groups are dissimilar with respect to exercise history. Astrand's subjects were members of a group regularly taking part in organized exercise since leaving school. Had these women participated in our study we would have classified them as physically trained. Of our 95 volunteers only three fell into the physically trained category, and the remainder were classified as sedentary.

These women volunteers did not appear to become as completely exhausted in the course of performing maximal exercise tests as men volunteers had become in previous studies. This observation raised the question whether maximum effort had indeed taken place. At the time of testing each subject understood the procedure, had been cooperative and continued exercise until unable to maintain position on the treadmill without holding on. Wilmore has found that strong motivation will result in subjects being able to demonstrate increased power or endurance. However indices of maximal aerobic capacity such as maximal oxygen consumption or maximal exercise heart rate are not increased by motivation. He explains that motivation has the power of increasing the degree of anaerobic metabolism tolerated by the body, while not affecting maximal aerobic mechanisms. Our findings are consistent with other studies in which maximal oxygen consumption was documented by actual measurement. Therefore we are convinced that the volunteers we studied actually did perform maximal aerobic exercise, but that they probably did not force themselves into anaerobic metabolism at the peak of exercise to the degree that previous volunteers had done. It should be remembered, however, that physiologic documentation of maximal aerobic performance requires measuring a plateau of oxygen consumption in spite of increasing work output. We did not monitor oxygen consumption.

Our maximum heart rate results are similar also to those of Profant and colleagues in comparable age ranges.

By use of the maximum exercise heart rate age regression found in this study it is possible to construct a graded exercise test target heart rate table for women (table 2), consisting of 90% of the average maximum heart rate found in each half-decade studied. This table is considerably different

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**Table 1. Reported Average Maximal Exercise Heart Rate Responses by Decades**

<table>
<thead>
<tr>
<th>Author</th>
<th>Number of subjects</th>
<th>Age range (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Astrand</td>
<td>44</td>
<td>20-29 187 185 178 170</td>
</tr>
<tr>
<td>Benestad et al.</td>
<td>10</td>
<td>30-39 197 192 179 167 158 145</td>
</tr>
<tr>
<td>Cumming et al.</td>
<td>357</td>
<td>40-49 197 192 179 167 158 145</td>
</tr>
<tr>
<td>Metheny et al.</td>
<td>17</td>
<td>50-59 197</td>
</tr>
<tr>
<td>Profant et al.</td>
<td>144</td>
<td>60-69 184 180 177 167 160</td>
</tr>
<tr>
<td>Sheffield et al.</td>
<td>95</td>
<td>70-79 194 186 178 166 165</td>
</tr>
</tbody>
</table>
from the target heart rate values developed for men. For example, at age 60 the target heart rate for women is 13 beats/min lower than that recommended for sedentary men. As is the case with men, women’s target heart rates are for comparative purposes, to give a general indication as to whether a test subject has put forth near maximal effort. Target heart rate should never be misused to prolong an exercise test after a clinically valid reason for stopping has occurred, nor should it be used as the only basis for terminating a test when a subject does not appear adequately stressed.

Exercise Capacity

Work capacity is known to be greater in men who engage in regular strenuous physical activity than in sedentary men of the same age. This effect has also been observed in women by Astrand and by Bruce’s group. Nearly all of our subjects were classified as sedentary, so we had no opportunity to observe the effect of physical training on the exercise capacity of women. However the treadmill performance of our subjects corresponds closely with the sedentary group reported by Profant et al. and shows lower exercise capacity than their group of physically active women.

Exercise capacity is known to decline with advancing age in both men and women. Our group has found the effect of age to be much less in asymptomatic women than in men; however, while the treadmill endurance time was reduced 10% per decade of age in men, we found a reduction of only 2% per decade in women under 60. Due to sample size (less than 30 persons per decade), this regression of exercise capacity with age was statistically insignificant (P > 0.05), but on the basis of the above and other studies there is good reason to believe the effect of age is real. Another reason for the small effect of age on exercise capacity in our subjects probably lies in the study design itself. Frolicher and colleagues found similar results in studying 192 special project volunteers at the School of Aerospace Medicine. Although they ranged from 25 to 45 years in age, maximal treadmill endurance time for the older subjects was the same as that for the younger ones. This consequence of using an asymptomatic volunteer group was anticipated at the outset and accepted, since our aim was not to study the incidence of disability with age but to characterize the reactions and capacities of normal, healthy individuals. We believe the volunteer-preselection effect was also responsible for our finding no relationship between subject body weight and age. It is known that women’s weight increases with age in the general population (as does men’s), and this was also found in the studies of Astrand, Profant et al. and Andersen. One gathers from these studies that the common trend of aging is to reduce daily physical activity, gain weight and lose exercise capacity. Our study shows that women who are asymptomatic and consider themselves in good health may not follow this pattern at all.

### TABLE 2. Graded Exercise Test Target Heart Rates for Women

<table>
<thead>
<tr>
<th>Stress level</th>
<th>Age (yr) 20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
</tr>
</thead>
<tbody>
<tr>
<td>100% (Max)</td>
<td>198</td>
<td>194</td>
<td>190</td>
<td>185</td>
<td>181</td>
<td>177</td>
<td>172</td>
<td>168</td>
<td>163</td>
<td>159</td>
</tr>
<tr>
<td>90% (GXT)</td>
<td>179</td>
<td>175</td>
<td>171</td>
<td>167</td>
<td>163</td>
<td>159</td>
<td>155</td>
<td>151</td>
<td>147</td>
<td>143</td>
</tr>
</tbody>
</table>

Electrocardiographic Observations

Profant et al. reported the incidence of ST-segment depression in asymptomatic women to vary from zero in the third decade to 67% in the seventh. ST deviations were, however, predominantly of the upsloping variety. Of 144 women tested they observed only two with flat or downsloping ST-segment reaction to exercise. This is comparable to our finding one such instance in 95 women. In addition, of our subjects manifested ST-segment depression of the upsloping variety.

We found premature beats on exercise in one-fifth of our subjects, while Astrand found them in nearly half (19 of 44) of hers. When one compares the occurrence of ventricular premature beats, however, the results are similar: 14% in her subjects and 16% in ours. Ventricular premature beats are known to have an adverse prognostic effect in persons with known coronary heart disease, but the significance of their occurrence in persons with a normal cardiovascular history is unknown.

Conclusions

Women who continue to consider themselves healthy have little reduction in exercise capacity with advancing age, and these women do not tend to become obese. They have a maximum heart rate regression with age that is different from men’s. This should be taken into account when evaluating results of strenuous exercise tests. If graded exercise tests are carried out on women using target heart rates, a heart rate table based specifically on women’s exercise response should be used.

Acknowledgment

The authors are grateful to the ninety-five women who generously agreed to be the subjects of this study, to the technicians of the Allison Laboratory, to David Hurst, Ph.D. and Seng-jaw Soong, Ph.D., who performed numerical analysis of our data, and to Mrs. Florence Driskill and Mrs. Juanita Brasher who assisted in the preparation of this manuscript.

### References


**Alpha and Beta Adrenergic Effects on Human Atrial Specialized Conducting Fibers**

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*With the technical assistance of Nan E. Perry*

SUMMARY We determined the effects of epinephrine on automaticity and action potential characteristics of right atrial specialized fibers (RAF) from human atria obtained during cardiac surgery. RAF were studied with standard microelectrode techniques during superfusion with Tyrode's solution at 37°C. A biphasic response to epinephrine was seen, rate slowing at low agonist concentrations and increasing at high concentrations. The epinephrine-induced slowing of spontaneous rate was due to a decrease in the slope of phase 4 depolarization. At the high epinephrine concentrations RAF hyperpolarized. The α-adrenergic blocker, phentolamine, shifted the dose-response curve upward and to the left and enhanced the hyperpolarization of RAF. The β blocker, propranolol, shifted the curve to the right and decreased the degree of hyperpolarization. Our study suggests the presence of α and β receptors in RAF. The α response consists of a slowing of rate, the β response of an acceleration of rate and hyperpolarization of RAF.

THE EFFECTS OF ADRENERGIC AMINES on the electrophysiologic properties of cardiac fibers have been studied extensively. Both α and β adrenergic effects on mammalian Purkinje fibers1,4 and atria7,8 have been described. Phenylephrine, primarily an α agonist, prolongs Purkinje fiber action potential (AP) duration as does norepinephrine in the presence of propranolol. This action has been referred to as α adrenergic. The β agonist, isoproterenol, and norepinephrine in the presence of phentolamine both decrease AP duration, an action referred to as β adrenergic.1,8 In studies of Purkinje fiber automaticity low concentrations of epinephrine have been shown to decrease spontaneous rate4,4 and K+ uptake,8,9 and high concentrations to increase these variables.4,9 The decreases in spontaneous rate and K+ uptake are blocked by phentolamine and have been interpreted as alpha adrenergic.4,9 The epinephrine-induced increases inautomaticity have been attributed to a selective effect on the kinetics of iK1 (pacemaker current), and both epinephrine effects on automaticity and on iK1 are blocked by propranolol.8

Despite these studies of adrenergic effects on mammalian cardiac fibers no information is yet available concerning the presence of α and β adrenergic receptors in human cardiac specialized conducting fibers. For this reason we studied the effects of epinephrine and of α and β blockade on human atrial fibers. We found changes in automaticity consistent with the presence of α and β receptors.
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Circulation. 1978;57:79-84
doi: 10.1161/01.CIR.57.1.79

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:
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