RELATIONSHIP OF HEART RATE WITH HYPERTENSION IN ADULT POPULATION OF MINGORA CITY, DISTRICT SWAT

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ABSTRACT
The aim of this study was to evaluate the relationship of heart rate with hypertension in adult population of Mingora city, district Swat. The study comprised of 100 subjects, 34 control, 33 obese and 33 hypertensive. Weight, height, body mass index, heart rate, and blood pressure of hypertensive subjects were compared with obese subjects and control subjects. Hypertensive subjects had faster heart rate than obese normotensive subjects, which in turn had higher value than control subjects. Faster resting heart rate is a potent predictor of cardiovascular morbidity and mortality.

INTRODUCTION
Resting heart rate (RHR) is one of the simplest cardiovascular parameters, which usually averages 60 to 80 beats per minute (b.p.m.), but can occasionally exceed 100 b.p.m. in unconditioned, sedentary individuals and can be as low as 30 b.p.m. in highly trained endurance athletes. Extrapolation of data obtained in different studies allows to provisionally identify some figures, i.e. heart rate normality values between 60 and 80 beats/minute and tachycardia as values greater than 100 beats/minute.

Epidemiological evidences demonstrate that RHR, or its corollaries, namely post-exercise heart rate recovery, which is mediated primarily by vagal tone, and heart rate variability (HRV, beat-to-beat variability also mediated by autonomic nervous system, especially parasympathetic) correlates with cardiovascular morbidity and suggests that RHR determines life expectancy. Multiple studies have identified RHR as an independent risk factor for cardiovascular disease (comparable with smoking, dyslipidemia or hypertension). However, it is often overlooked. Tachycardia tends to intensify pulsatile nature of arterial blood flow and to produce oscillations in shear-stress direction, a phenomenon which in the long term is condusive to atherosclerosis.

Several hypothesis have been advanced for explaining the relationship between an elevated heart rate and cardiovascular risk. Two of them have been confirmed by experimental and clinical studies. The first one refers to the evidence that:

- The heart rate represents an integrated index of autonomic cardiovascular function and elevated heart rate values are markers of an adrenergic overdrive, which exerts, particularly in an ischaemic, failing or hypertrophic heart, pro-arrhythmogenic effects, thereby predisposing to fatal arrhythmias and sudden death.
- The second pathogenetic hypothesis, not mutually exclusive of the first one, says that an elevated heart rate 1) increases shear stress, 2) impairs arterial compliance and 3) favours the development of atherosclerotic vascular lesions.

The association between heart rate and cardiovascular events: is present at all ages and even in subjects older than 70 years. It takes place in patients with and without cardiovascular complications. It appears to be independent of other risk factors for the atherosclerotic disease and is consistent as the association between other “classic” risk factors and cardiovascular disease.

Elevated heart rate in resting condition is a risk factor for atherosclerosis and cardiovascular events. Glostrup Count study found a highly significant association between resting heart rate and the incidence of myocardial infarction. Similar findings were observed in a study of Israeli government employees. Another study reported a much rate of higher cardiovascular death in subjects who had heart rate consistently more than 100 beat per minutes than in subjects with a heart rate below that value. In the Framingham study, the predictive power of heart rate for all causes of mortality was equal to that of smoking or of systolic blood pressure, while in another prospective study, the predictive power of heart was second only to smoking.

Various studies indicate that heart rate is a major risk factor for atherosclerosis and for car-
diovascular and total mortality. However, the importance of its clinical variable is still neglected by the scientific community, probably because the mechanism of the relationship between heart rate and mortality is poorly understood. In particular, heart rate is considered by physicians to be a mere marker of risk rather than a true risk factor.\(^{11}\)

The relationship between tachycardia and hypertension and the metabolic abnormalities reported in many studies points to a role of sympathetic overactivity in determining the high heart rate and metabolic disturbances.\(^{12}\) In addition to the high blood pressure, the elevated heart rate is also related to high haematocrit, hyperinsulinaemia, increased blood glucose, increased body mass index and lipid abnormalities.\(^{13}\)

**SUBJECTS AND METHODS**

The study was carried out on the subjects, randomly taken from different union councils of Mingora city, district Swat. It comprised of 100 subjects, 34 control (Group A), 33 obese (Group B) and 33 hypertensive (Group C). Weight, height, body mass index, heart rate and blood pressure of the hypertensive subjects were compared with both obese and control subjects. Both males and females between age of 40-70 years were included in the study. Subjects who had a medical history of disease other than overweight / hypertension or on any medication known to affect metabolism, were excluded from the study.

Health Scale (Model ZT-120) was used to measure weight and height. Weight was assessed at 2 different points during interview, and the two were averaged for these analyses. It was measured to the nearest 0.5kg. Height was also assessed at two different points during interview, and the two readings were averaged for these analysis. It was measured to the nearest 0.1 cm.\(^{14}\) Body mass index (BMI) was determined by dividing weight (wt) in kilogram by height (ht) in meters square (BMI= wt/ht\(^2\)). BMI 30 or more was taken as cut off value to determine obesity.\(^{16}\)

Heart rate was calculated as mean number of beats per minute. Blood pressure (BP) data was obtained, after at least 5 minutes of rest, with subjects in seated position. A mercury sphygmomanometer (Model SM- 300), with an appropriate sized cuff covering two third of the upper arm was used. The onset of the first tapping sound was taken to indicate the systolic blood pressure, while the point of complete disappearance of the sound (Korotkoff V) was taken to indicate diastolic blood pressure. The mean of three reading was recorded. In adult population, hypertension is usually defined as blood pressure level that exceeds 145-150/ 90-95 mm Hg.\(^{17}\)

**RESULTS**

Anthropometric and cardiovascular parameters of Group “A” (control) were compared with Group “C” (hypertensive). The mean weight of Group “A” was 65 ±7.27 and that of Group “C” was 76 ± 8.13. The p-value was < 0.05 (significant). The mean height of Group “A” was 1.67 ± 0.08 and that of Group “C” was 1.64 ± 0.054. The height difference was not significant. The mean BMI of Group “A” was 23 ± 1.29 and that of Group “C” was 28 ± 1.99. The p-value was less than 0.05 (significant) (Table 1 and Fig. 1).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control “A” (n = 34)</th>
<th>Hypertensive “C” (n = 33)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (Kg)</td>
<td>65 ± 7.27</td>
<td>76 ± 8.13 **</td>
</tr>
<tr>
<td>Height (M)</td>
<td>1.67 ± 0.08</td>
<td>1.64 ± 0.05</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>23 ± 1.29</td>
<td>28 ± 1.99 *</td>
</tr>
<tr>
<td>Heart rate b.p.m.</td>
<td>75 ± 6.5</td>
<td>102 ± 7.4 **</td>
</tr>
<tr>
<td>Systolic B.P mmHg</td>
<td>130 ± 11.8</td>
<td>160 ± 8.27 **</td>
</tr>
<tr>
<td>Diastolic B.P mmHg</td>
<td>85 ± 10.99</td>
<td>105 ± 6.79 **</td>
</tr>
</tbody>
</table>

The values are expressed as mean ± SD (standard deviation). *P < 0.05 (significant); **P < 0.001 (highly significant).

Anthropometric and cardiovascular parameters of Group “A” were compared with Group B. The mean weight of Group “A” was 65 ± 7.27 kg and that of Group “B” was 87 ± 8.43 kg. The p-value was < 0.001 (highly significant). The mean height of Group “A” was 1.67 ± 0.08 and that of Group “B” was 1.65 ± 0.068. The height difference was not significant. The mean BMI of Group “A” was 23 ± 1.29 and that of Group “B” was 31 ± 1.41. The p-value was < 0.001 (highly significant) (Table 2 and Fig. 1).

The mean heart rate of group A was 75 ± 6.5 and that of group ‘C’ was 102 ± 7.4. The p-value was < 0.001 (highly significant). The mean systolic blood pressure of Group “A” was 130 ± 11.8 and that of Group “C” was 160 ± 8.27. The p-value was < 0.001 (highly significant). The mean diastolic blood pressure of Group “A” was 85 ± 10.99 and that of Group “C” was 105 ± 6.79. The p-value was < 0.05 (significant).

The mean heart rate of group A was 75 ± 6.5 and that of group B was 95 ± 6.9. The p-value was < 0.05 (significant). The mean systolic blood pressure of Group “A” was 130 ± 11.8 and that of Group
“B” was 140 ± 6.75. The p-value was < 0.05 (significant). The mean diastolic blood pressure of Group “A” was 85 ± 10.09 and that of Group “B” was 90 ± 7.4. The p-value was < 0.05 (significant).

Anthropometric and cardiovascular parameters of Group B (obese) were compared with Group “C”. The mean weight of Group “B” was 87 ± 8.43 kg and that of Group “C” was 76 ± 8.13 kg. The p-value was < 0.05 (significant). The mean height of Group “B” was 1.65 ± 0.068 and that of Group “C” was 1.64 ± 0.054. The height difference was not significant. The mean BMI of Group “B” was 31 ± 1.41 and that of Group “C” was 28 ± 1.09. The p-value was less than 0.05 (significant) (Table 3 and Fig. 1).

The mean heart rate of group “B” was 95 ± 6.9 and that of group “C” was 102 ± 7.4. The p-value was < 0.05 (significant). The mean systolic blood pressure of Group “B” was 140 ± 6.75 and that of Group “C” was 160 ± 8.27. The p-value was < 0.001 (highly significant). The mean diastolic blood pressure of Group “B” was 90 ± 7.4 and that of Group “C” was 105 ± 6.79. The p-value was < 0.001 (highly significant).

Table 2: Shows anthropometric and cardiovascular parameters of Group A and Group B.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control “A” (n = 34)</th>
<th>Obese “B” (n = 33)</th>
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</thead>
<tbody>
<tr>
<td>Weight (Kg)</td>
<td>65 ± 7.27</td>
<td>87 ± 8.43 **</td>
</tr>
<tr>
<td>Height (M)</td>
<td>1.67 ± 0.08</td>
<td>1.65 ± 0.07</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23 ± 1.29</td>
<td>31 ± 1.41 **</td>
</tr>
<tr>
<td>Heart rate b.p.m.</td>
<td>75 ± 6.5</td>
<td>95 ± 6.9 *</td>
</tr>
<tr>
<td>Systolic B.P mmHg</td>
<td>130 ± 11.8</td>
<td>140 ± 6.75 *</td>
</tr>
<tr>
<td>Diastolic B.P mmHg</td>
<td>85 ± 10.99</td>
<td>90 ± 7.4 *</td>
</tr>
</tbody>
</table>

The values are expressed as mean ± SD (standard deviation). *P < 0.05 (significant); **P < 0.001 (highly significant).

Table 3: Shows anthropometric and cardiovascular parameters of Group B Group C.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Obese “B” (n = 33)</th>
<th>Hypertensive “C” (n = 33)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (Kg)</td>
<td>87 ± 8.43</td>
<td>76 ± 8.13 **</td>
</tr>
<tr>
<td>Height (M)</td>
<td>1.65 ± 0.07</td>
<td>1.64 ± 0.05</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>31 ± 1.41</td>
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</tr>
</tbody>
</table>

The values are expressed as mean ± SD (standard deviation). *P < 0.05 (significant); **P < 0.001 (highly significant).

Fig. 1: Graphic presentation of anthropometric and cardiovascular parameters of Group A (Control), compared with Group B (Obese) and with Group C (Hypertensive).
DISCUSSION

Previous studies have demonstrated that resting clinic heart rate is an independent risk factor for adult cardiovascular disease in general and coronary heart disease in particular. However, the pathogenesis of the connection between elevated heart rate and cardiovascular disease remains obscure. A number of mechanisms for this association have been postulated. Data from animal models suggest that the atherogenic action of high blood pressure, which would favour the occurrence of arterial wall lesions. According to some authors, tachycardia may merely indicate poor physiological fitness and/or subclinical loss of cardiac reserve. Moreover, it has been postulated that elevated heart rate may reflect a higher consumption of tobacco or alcohol, which are well-recognized risk factors for cardiovascular disease. A portion of the effect on coronary heart disease has been attributed to high blood pressure, which appeared consistently positively correlated with pulse rate in several studies but the nature of this relationship remains unclear.

The present study recorded significant association between elevated resting heart rate and high blood pressure in both obese and hypertensive subjects. Our data are consistent with the positive relationship between resting heart rate and blood pressure observed in epidemiological studies. These prior studies included both normal-weight and overweight subjects. Chicago people Gas Company study dataset confirmed a significant association between baseline heart rate and the likelihood of developing cardiovascular events, independent of other major risk factors. Also fast heart rate was associated with increase rate of sudden death. In the Framingham Study, it was found that elevated resting heart rate was also strongly associated with mortality in the general population, in a cohort composed of 5070 subjects who were free from cardiovascular disease at the time of entry into the study, cardiovascular and coronary mortality increased progressively with resting heart rate. A Study With Mixture Analysis in Three Populations: European general population (Belgian study), one North American general population (Tecumseh study), and one European hypertensive population, these studies observed almost similar close correlation between blood pressure and heart rate in all populations, and the relationship persisted after adjustment for other factors potentially influencing heart rate.

In conclusion, fast resting heart rate is significantly correlated with higher blood pressure, and increased heart rate is prospectively related to the development of hypertension, atherosclerosis and their sequelae. On the basis of this evidence, the importance of recognizing and treating elevated heart rate in clinical practice becomes clear. The association between heart rate and cardiovascular morbidity suggests that reduction of heart rate should be an additional goal of antihypertensive therapy.

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REFERENCES

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