Commentary

Need for a Revision of the Normal Limits of Resting Heart Rate

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The current definition of sinus tachycardia is a heart rate >100 beats per minute (bpm). This limit was set arbitrarily when heart rate was not yet regarded as a risk factor for cardiovascular disease, probably with the main purpose of distinguishing between a disease state (fever, thyrotoxicosis, anemia, congestive heart failure, etc) and a normal condition.

Tachycardia as a Cardiovascular Risk Factor
In recent years, interest has been aroused by the awareness that fast heart rate is a potent precursor of hypertension, atherosclerosis, and their sequelae. In addition, many leading epidemiological studies have shown that tachycardia is associated with an increased risk of death from cardiovascular and noncardiovascular causes. This relationship has been found in general populations, in elderly individuals, and in hypertensive cohorts. In all of these studies, the heart rate value above that in which a significant increase in risk was observed was below the 100 bpm threshold (Table 1). Only in the study by Levy et al was tachycardia defined as a heart rate >99 bpm, but in that study the cutoff between normal and high heart rate was chosen arbitrarily, and the highest heart rate value measured during the examination was taken to define the subject’s heart rate. In all the other studies, the threshold level between normal and fast heart rate was between 79 and 90 bpm.

The normalcy limits of a clinical variable can be established according to different criteria. For many parameters, such as most biochemical indexes, the 95% confidence interval is calculated to identify the upper normal limit of the variable. This statistical approach does not appear suitable for those clinical variables in which the relationship with the level of risk is a continuous one. A typical example is observed with blood pressure, in which the upper normal limits were set arbitrarily. Blood pressure was considered abnormal by the World Health Organization when it was greater than the level at which the increase in risk became considerable. This level roughly corresponded to the highest quintile of the blood pressure distribution in the populations of the industrialized countries. In most of the studies reported in the Table, subjects were considered to have tachycardia if they were in the highest quintile of the heart rate distribution, and an increase in the risk of coronary events and/or cardiovascular and total mortality was found in the subjects of the top quintile. In the Framingham study, for example, a 6-fold increase in the relative risk of sudden death was seen in the subjects of the top heart rate quintile in comparison with those of the bottom quintile. In the Framingham study, a linear relationship was found between heart rate and mortality, although in other studies, a J-shaped relation or a sigmoidal relation was observed. However, in all studies, the excess in risk was present chiefly in the subjects of the highest heart rate quintile.

Is There a Level of Heart Rate That Separates Two Populations With Normal and High Heart Rate?
Some years ago, Spodick et al attempted to redefine the normal limits of heart rate on the basis of the results obtained in a population of subjects aged 50 to 80 years. By the addition of 2 SD to the mean heart rate value, Spodick et al found upper normal limits of 93 bpm for resting heart rate in the men and of 95 bpm in the women, which are above those found to be associated with an increased risk of mortality by most investigators. Moreover, the Spodick approach implies the existence of a normal distribution for heart rate in the general population. Recent results obtained in our laboratory indicate that this is not the case. In fact, in the Belgian, the HARVEST, and the Tecumseh populations we found that the heart rate distribution was significantly skewed among men and women from Tecumseh. Similar results were obtained in a recent analysis of the Mirano population, in which both men and women showed a skewed distribution of resting heart rate (unpublished observations). Mixture analysis showed that in all these populations, the distribution of heart rate was explained by the mixture of 2 homogeneous subpopulations, a larger one with “normal” heart rate and a smaller one with “high” heart rate. Mixture analysis is a statistical test used in the biological sciences to investigate whether a mixture of normal distributions better explains the variation of a trait than a single distribution when overlap exists between the subpopulations. This is an entirely objective way to establish a cutoff level between normal and abnormal values, which avoids the necessity for establishing...
Heart Rate Values Above Which a Significant Increase in Risk Was Found: Data From 9 Epidemiological Studies

<table>
<thead>
<tr>
<th>Author (Study)</th>
<th>HR Cutoff, bpm</th>
<th>Measurement: Type (No.)</th>
<th>Method Used for Cutoff Identification</th>
<th>Study Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levy et al(^1) (US Army officers)</td>
<td>99</td>
<td>Pulse (1)</td>
<td>Arbitrary</td>
<td>5-y cardiovascular mortality in subjects with HR (&gt;99) bpm “far in excess” compared with subjects with HR (&lt;99) bpm</td>
</tr>
<tr>
<td>Medalie et al(^4) (Israeli government employees)*</td>
<td>90</td>
<td>Not specified</td>
<td>Highest tertile</td>
<td>Age-adjusted rate/1000 of MI over 5 y: 61 in top tertile and 40 in bottom tertile (P) defined as “significant”</td>
</tr>
<tr>
<td>Dyer et al(^2) (Chicago-People Gas Co)*</td>
<td>79</td>
<td>From ECG</td>
<td>Highest quintile</td>
<td>Age-adjusted 15-y all-cause mortality rate in the HR quintiles: (\chi^2 = 20.1, P = 0.001)</td>
</tr>
<tr>
<td>Dyer et al(^2) (Chicago-Heart Association)*</td>
<td>86</td>
<td>From ECG</td>
<td>Highest quintile</td>
<td>Age-adjusted 5-y all-cause mortality rate in the HR quintiles: (\chi^2 = 20.4, P = 0.001)</td>
</tr>
<tr>
<td>Dyer et al(^2) (Chicago-Western Electric)*</td>
<td>89</td>
<td>Pulse (1)</td>
<td>Highest quintile</td>
<td>Age-adjusted 17-y all-cause mortality rate in the HR quintiles: (\chi^2 = 21.05, P = 0.001)</td>
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<tr>
<td>Kannel et al(^6) (Framingham)</td>
<td>87</td>
<td>From ECG</td>
<td>Highest quintile</td>
<td>Age-adjusted 26-y sudden death mortality rate in top HR quintile vs others: (P = 0.001)</td>
</tr>
<tr>
<td>Gillum et al(^7) (NHANES)</td>
<td>84</td>
<td>Pulse (1)</td>
<td>Arbitrary</td>
<td>Odds ratios for risk-adjusted() 10-y all-cause mortality for HR (&lt;84) bpm: black men, 1.71 (1.14–2.56); white men, 1.81 (1.26–2.60); black women, 1.95 (1.16–3.27); white women, 1.81 (1.26–2.59); Reference are subjects with HR &lt;74 bpm.</td>
</tr>
<tr>
<td>Palatini et al(^8) (CASTEL)†</td>
<td>80</td>
<td>Pulse (3)</td>
<td>Highest quintile</td>
<td>Odds ratios for risk-adjusted() 12-y cardiovascular mortality for HR (&lt;80) bpm: men, 1.38 (0.94–2.03), (P = 0.005); women, NS. Reference are subjects with HR from 64 to 80 bpm.</td>
</tr>
<tr>
<td>Gilman et al(^9) (Framingham)‡</td>
<td>84</td>
<td>From ECG</td>
<td>Arbitrary</td>
<td>Odds ratios for risk-adjusted() 36-y all-cause mortality for each 40-bpm increment in HR. Men, 1.98 (1.52–2.59); women, 1.87 (1.37–2.56)</td>
</tr>
</tbody>
</table>

HR indicates heart rate; y, years; MI, myocardial infarction; NHANES, National Health and Nutrition Examination Survey; and CASTEL, Cardiovascular Study in the Elderly.

*Male population; †elderly subjects; ‡hypertensive cohort; §data adjusted for age, smoking, blood pressure, cholesterol, and other confounders.

arbitrary threshold levels. With the use of this method in the above mentioned populations, we identified cutoffs varying from 80 to 85 bpm for resting heart rate measured by the physician in the clinic.\(^14\) The percentage of subjects with a high heart rate ranged from 12.3% to 29.8% in the various male and female populations. These results show that 2 subpopulations with normal and high heart rate can be separated within a general population and that the threshold level between the 2 subpopulations is around 80 to 85 bpm.

**Effect of Treatment in Subjects With Tachycardia**

If tachycardia is a strong risk factor for cardiovascular disease, antihypertensive drugs that also decrease heart rate should be more beneficial in hypertensive subjects with fast heart rate. However, no clinical trial has been implemented as yet with the specific purpose of studying the effects of cardiac slowing on morbidity and mortality in hypertension. The only available data on the effect of heart rate reduction in humans stem from retrospective analyses of subjects with myocardial infarction or congestive heart failure. These results suggest that \(\beta\)-blockers are effective in reducing mortality only in subjects with a high baseline heart rate.\(^17\) Carvedilol, for example, has been reported to cause a marked reduction in mortality in subjects with congestive heart failure,\(^18\) but the benefit was clear only in patients with a heart rate \(>82\) bpm. An association between the reduction in heart rate and mortality has been shown also with amiodarone, which improved survival in patients with congestive heart failure, but only in subjects with heart rate \(>89\) bpm.\(^19\) According to some investigators, the upper normal limit of a clinical variable should be defined as the level at which the benefits of treatment outweigh the risks or, in other words, as a treatment threshold.\(^20\) The data obtained in subjects with myocardial infarction or congestive heart failure suggest that for heart rate this level should be set in the range of 80 to 89 bpm.

**Bradycardia**

Sinus bradycardia is said to exist in the adult when the sinus node discharges at a rate \(<60\) bpm.\(^1\) Sinus bradycardia may occur as a consequence of a disease such as increased intracranial pressure, myxedema, hypothermia, and vasovagal syncope. In epidemiological studies in general populations or hypertensive cohorts, no increased risk of mortality was generally found for the lower extreme of heart rate. Only in the Chicago Heart Association Study were low heart rates \((<60\) bpm) related to an increase in sudden death.\(^3\) However, in that study, subjects with bradyarrhythmias at ECG were
Looking for a New Definition of Tachycardia

Although there are no objective data that allow us to establish new normal limits for resting heart rate, it seems clear that the traditional 100 bpm value is not appropriate to define the threshold below that in which heart rate can be considered safe. The epidemiological studies listed in the Table clearly demonstrate that the association between heart rate and the cardiovascular risk occurs for levels well below the 100 bpm value. Also, the results of the intervention trials in postmyocardial infarction patients or in subjects with congestive heart failure suggest that the limit of normality of heart rate should be set below 100 bpm. On the basis of the data from the literature and the results obtained with mixture analysis in our laboratory, we suggest a new consensus: for men, it appears reasonable to set the upper normal value of heart rate at 85 bpm. Because of the higher heart rate commonly seen in women (3 to 7 bpm greater), a slightly higher threshold should be adopted for them. Conversely, a lower limit should be set in the elderly. Heart rate has been reported to decline slowly with age, with an average decrease of 1 bpm every 8 years. The cutoff between normal and high heart rate found in our laboratory in elderly men (80 bpm) was slightly lower than that found in younger adults by most investigators (Table). Hypertensive individuals, and subjects with congestive heart failure usually have higher values of heart rate than healthy controls. However, this does not mean that the level of risk related to heart rate is shifted toward higher values in these patients. A recent report in subjects with acute myocardial infarction showed that the risk of death sharply increases for heart rate values >80 bpm. Another well recognized factor that affects heart rate is physical training. Tachycardia may be a marker for decreased physical fitness, which in turn may increase risk of cardiovascular death. However, high heart rate turned out to be a predictor of cardiovascular mortality also in the studies that controlled for energy expenditure. Thus, physical activity can be regarded as a useful and physiological method for decreasing heart rate, and its well known cardioprotective action could be at least partially because of its effect on heart rate. The increase in heart rate variability caused by endurance training could also contribute to the beneficial effects conferred by regular exercise. An inverse correlation has been reported between heart rate variability and mortality from myocardial infarction and other cardiovascular causes. Thus, not only the mean heart rate value but also its variability seems to be related to cardiovascular morbidity and mortality.

The above mentioned heart rate limits can be of help for better defining the cardiovascular risk profile of a given individual, but we are still unable to say whether a reduction of heart rate below those levels could confer any benefit in terms of life expectancy, especially in hypertensive patients. As for the opposite extreme of the heart rate range, the data from the literature do not allow the identification of any clinically meaningful limit. In fact, no increased risk of mortality was generally found for the lowest values of sinus heart rate. With the above mentioned approach, Spodick et al identified the level of 50 bpm as the lowest normal limit of heart rate, but there is no indication from the literature that a heart rate below that limit is really hazardous in the absence of sinoatrial dysfunction. It is obvious that a low heart rate, particularly in unfit elderly subjects, may need further evaluation for sinoatrial node dysfunction or other diseases.

Conclusions

Heart rate has been neglected for a long time as a clinical parameter, and it is time that this variable receives the consideration it deserves in clinical practice. Although official upper normal limits for resting heart rate are not yet available, the data of the literature are sound and indicate that these limits should be set well below 100 bpm, the threshold currently used to define tachycardia, to probably around 85 bpm. Heart rate can become a useful tool in clinical practice and research in the future provided the criteria for measurement are strictly standardized by the scientific societies. We suggest that criteria similar to those adopted for blood pressure assessment be used with heart rate. The clinician must consider all of the circumstances that may produce variations in heart rate and attempt to control or avoid them before taking the measurement. At least 2 readings taken over a 30-second period should be averaged. In addition, heart rate should be checked by the use of repeated visits before a final diagnosis is made, because a “white-coat tachycardia” can occur in some patients in the presence of healthcare professionals. We have recently demonstrated that the day-to-day variability of clinic heart rate is 40% greater than that for heart rate recorded over 24 hours. If heart rate is measured by 24-hour recording or with automatic devices, lower values should be expected. The evolution of our understanding of the relationship between heart rate and mortality will dictate that different levels of heart rate are taken, which depends on the method of measurement, as the upper limit of the normal heart rate is reached.

References


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