On checking the pulse

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From the earliest of times physicians have been taught to evaluate the character of the pulse as an essential feature of the physical examination. This recommendation was based entirely on clinical impression; only recently has rigorous scientific evidence been provided in support of this ‘vital sign’. Now, in addition to the resting heart rate, heart rate variability and the heart rate response to exercise have been scientifically demonstrated to have prognostic significance.

The report of Kristal-Boneh et al. provides further evidence on the established value of the resting heart rate as an independent predictor of cardiovascular and overall mortality, now also taking into account haematological factors. The relevance of the latter is unclear since their relationship to the heart rate is obscure. They also examine the heart rate relationship to cancer, as some others have done, where it is difficult to postulate a plausible causal relationship. In any event, with only 45 cancer deaths it is difficult to test the relationship.

The demonstrated relationship of a high heart rate to increased cardiovascular morbidity and mortality may reflect a direct contribution or it may only be a marker for a more direct cause. There are a number of postulated mechanisms for the connection between the heart rate and cardiovascular morbidity and mortality. The heart rate can signify abnormal autonomic control of the circulation characterized by increased sympathetic tone and decreased vagal activity. Epidemiological investigation indicates that the heart rate is related to atherogenic traits such as obesity, hypertension, hyperinsulinaemia, dyslipidaemia, and hyperglycaemia, features of the insulin resistance syndrome. This rapid heart rate clustering with atherogenic risk factors promoted by insulin resistance, and perhaps induced by sympathetic overactivity and stimulation of beta-adrenergic receptors, may explain its association with accelerated atherogenesis. The increased sympathetic tone associated with rapid heart rate also promotes left ventricular hypertrophy, platelet activation and an increased haematocrit that may further promote cardiovascular disease.

Rapid heart rates may also be a consequence of subclinical disease reflecting poor physical fitness, and loss of vigour. This mechanism may explain the relationship of the heart rate to cancer mortality found in some studies. In some persons rapid heart rates at rest may reflect loss of cardiac reserve that may antedate overt heart failure by many years. Compensatory tachycardia may be necessary to maintain adequate cardiac output.

There may also be an independent action of the heart rate to induce cardiovascular disease by increasing the mean arterial pressure, arterial rigidity and ventricular dysrhythmia.

More than 25 years ago Medalie and co-workers showed that the resting heart rate was related to coronary mortality in male government employees. Since then, numerous studies have shown that a rapid resting heart rate is a powerful and independent predictor of cardiovascular and overall mortality, taking other relevant risk factors into account. This heart rate-mortality relationship has been found to be particularly strong for sudden death. The predictive power of the heart rate for cardiovascular events appears to be weaker for stroke and for women.

As long as 54 years ago, a rapid heart rate was shown to predict the onset of hypertension and since then a number of epidemiological investigations have found that an elevated heart rate is followed over time by a greater rise in blood pressure and development of hypertension. This hypertension–heart rate association rivals that of body weight as a precursor of hypertension. In the Framingham Study, the association of increased heart rate with cardiovascular mortality was shown to also apply in the hypertensive segment of the population.

What are the clinical implications of the consistent finding that a rapid heart rate is an independent cardiovascular risk factor for cardiovascular mortality? It would appear that such persons should have more aggressive attention to other risk factors that tend to accompany the tachycardia. A substantial proportion of hypertensive persons, in particular, have not only rapid heart rates, but also increased sympathetic activity and insulin resistance, making it prudent to determine their cardiovascular risk factor profile so that more optimal comprehensive therapy can be prescribed. In hypertensive persons with rapid heart rates drugs that reduce the heart rate along with the blood pressure, without adversely influencing the blood lipids would appear to be indicated.

Can reducing the heart rate pharmacologically help? About 30 years ago, it was shown that reducing
the heart rate of mice prolonged their life span and, more recently, that reducing the heart rate of monkeys with beta-blockers or sino-atrial node ablation retards coronary atherosclerosis[3]. In humans, mortality reduction has been shown to occur, in persons with existing coronary disease, if the heart rate is reduced with beta-blockers by more than 14 beats.min⁻¹. Proof of the efficacy of reducing the heart rate to avoid atherosclerotic cardiovascular disease awaits long overdue clinical trials in hypertensive and non-hypertensive candidates for cardiovascular disease.

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References

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Prognosis in heart failure: the value of parameter-changes over time

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Reliable prediction of prognosis and adequate risk stratification are of primary importance in chronic heart failure. In recent years the prognostic value of a great number of clinical, haemodynamic, ECG, laboratory and exercise parameters have been investigated[1]. However, these efforts resulted only in a few parameters with consistently high power in predicting the progression of chronic heart failure. These conflicting results are not surprising, considering that the prognostic value of a given parameter may depend on and can be modified by several factors, such as the stage and aetiology of heart failure, the biological characteristics of patients and the treatment applied. Generally applicable prognostic factors in chronic heart failure are not yet available.

That is why the changes in parameters over time can be at least as important in prognostication as the values of parameters detected in a single point of time. However, the studies evaluating the significance of these changes are few. The study by Florea et al.[2], investigating the prognostic value of changes over time in exercise variables and standard echocardiographic parameters deserves special attention, as one of the few.

The paper by Florea et al.[2] dealt with a patient population with mild to moderate heart failure, treated with ACE inhibitors, diuretics, digitalis and direct vasodilators. The exercise duration, the mean peak oxygen consumption (VO₂), the ventilatory response to exercise (VE/VO₂) slope, the diameter of the left atrium and the left ventricular ejection fraction measured in a single point of time were all found to be predictive for the disease outcome in this study. However, among the parameter changes over time only the modification in peak VO₂ proved to be of prognostic benefit. The question arises why other parameters failed to prove predictive. The difficulties in evaluating the changes, as well as some limitations in applying them for prognostication, are surely contributing factors impeding the usability of those parameters. An inherent, unavoidable limitation of such an investigation is that the follow-up, i.e. the survival study must be performed on patients alive after the period of observation. By applying this set-up, patients considered to be most severely ill are...