Resting heart rate and excessive heart rate increase during pre-exercise mental stress: which one predicts mortality?

In the study of Juven et al.1 the excessive heart rate (HR) increase during the preparation of an exercise test was indicated as a novel predictor of sudden cardiac death. The authors attributed this increase of HR to the mild mental stress caused during the pre-exercise preparation phase and suggested that a localized release of norepinephrin or a faster vagal withdrawal during this mild mental stress could be potentially related to this phenomenon. However, it is difficult to explain why subjects of the tertile with the lower HR increase were surprisingly found to have significantly higher HR at rest. As Juven et al. have previously described, the crude rate of sudden death increases linearly with the level of resting HR, a fact that does not agree with the present findings. Furthermore, an impaired autonomic balance should also affect the decrease of HR during the recovery period (post-exercise HR recovery). It would be of great interest to examine whether subjects with excessive increase in HR also have a slower decrease of HR during the recovery period, since HR recovery is a well-documented prognostic factor.2 Although HR recovery predicts all-cause mortality and not sudden cardiac death, the common pathophysiological link makes this comparison intriguing. Finally, the magnitude of the mental stress before a diagnostic test might possibly be exaggerated by the ‘first-time’ effect. It would be useful if we knew which of these subjects had undergone an exercise stress test before and how this factor influenced their performance.

References

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We thank Dr Fourlas for his interest on our article. The main result is that the heart rate increase during a mild mental stress in preparation for exercise is a strong predictor of sudden death. Specifically, individuals with the largest heart rate increase during mild mental stress and the smallest increase in heart rate during an exercise test are at higher risk. Dr Fourlas expressed concerns about the several potential mechanisms that we listed in the discussion. We share his opinion, and obviously, there is not one simple physiological explanation for the results that we observed in this long-term epidemiological study. We observed only a very modest correlation between heart rate increase during stress and during exercise. The heart rate increase during exercise is negatively associated with sudden death risk, whereas heart rate increase during mental stress is positively associated with sudden death. This suggests that at least two different physiological mechanisms are involved.

We are pleased to be able to provide data concerning the association between heart rate during recovery and risk for sudden death, which have been obtained in the same cohort and published previously.1 Heart rates at 1, 2, 3, and 4 min after cessation of exercise were all associated with overall mortality and particularly with sudden death, but not with non-sudden coronary death. When subjects with a heart rate recovery at 1 min higher than 40 b.p.m. were taken as reference (higher quintile), subjects with a low heart rate recovery, <25 b.p.m. (lower quintile), had a 2.1-fold risk of sudden death, a 0.9-fold risk of non-sudden coronary death, and a 1.3-fold risk of overall death. As suggested by Dr Fourlas, we still have to compare together the heart rate increase during mental stress and heart rate decrease during recovery. In summary, beside resting heart rate, heart rate changes during specific stimulations such as mental stress, exercise, and recovery provide additional predictive value for sudden death risk and clearly challenge the current physiological explanations.

Reference

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