Heart Online First, published on January 30, 2018 as 10.1136/heartjnl-2017-312731

Resting heart rate: what is normal?

David Nanchen

Resting heart rate (RHR) is a clinical parameter easily measurable with typical value between 50 and 90 beats per minute (bpm) that varies during the day with a night-time decrease.[1] RHR can go down to 30 bpm in those with good physical condition, but RHR is also partly genetically determined, with slightly higher values in women than in men.[2] The interpretation of RHR by clinicians is traditionally done in the acute setting, typically for evaluation of pulmonary embolism or acute infection. It is now possible to continuously and accurately self-measure RHR using a mobile phone or a watch bracelet, so that monitoring RHR has become very popular in the general population.[3] Therefore, it is important for physicians to know the clinical significance of RHR and its usefulness for chronic disease prevention in healthy adults.

Life expectancy of animal species is inversely correlated with their RHR.[4] Such ecological observation has led scientists to investigate the impact of RHR on disease development in humans. In recent years, many epidemiological studies have demonstrated a significant association between RHR and cardiovascular and non-cardiovascular outcomes.[5–7] Despite this large piece of evidence, there is still no established recommendation for healthy RHR values in adults. One explanation for this lack of consensus may be the paucity of interventional researches designed to target RHR with the aim of reducing cardiovascular risk.[8] Observational studies are limited to confirm a causal relationship between RHR and cardiovascular or non-cardiovascular disease. Confounding factors such as physical fitness and quality of diet including alcohol, caffeine or sodium consumption were rarely measured in previous studies, and many other factors are believed to affect RHR such as the activation of the autonomic nervous system or subclinical inflammatory processes.[9] Measurement errors of RHR when assessed by pulse palpation or automatic blood pressure monitor compared with electrocardiography can lead to misclassification bias because electrocardiography can identify adults with pre-existing atrial fibrillation or other conduction disorders. Furthermore, only few previous population-based studies have assessed multiple measurement of RHR over time.[10] This is important because RHR may change over time due to environmental factors such as physical activity or clinical conditions. Reverse causality is also an issue in observational studies because pre-existing cardiovascular disease or heart failure can increase or decrease RHR due to sympathetic hyperactivity, or drug use such as beta-blockers. Finally, specific cardiovascular or non-cardiovascular outcomes, such as cerebrovascular disease, type of cancer or respiratory disease, have rarely been examined.

Thanks to the study by Seviiri *et al*[7] published in *Heart*, a comprehensive picture of the association between RHR and change in RHR and cause-specific mortality can be drawn. Using a large prospective population-based cohort study in Melbourne of more than 40 000 men and women with a mean age of 55 years, the authors reported the association between baseline RHR and several types of cardiovascular, cancer and non-cardiovascular mortality over a 20-year period of follow-up. Less than 10% reported pre-existing cardiovascular disease and less than 4% reported diabetes at baseline. In about half of the Melbourne cohort, a second measurement of heart rate was available 10 years after the first measurement, allowing the authors to report the association between long-term change in RHR and cause-specific mortality over a 10-year follow-up period. RHR was measured in a sitting position with an automatic blood pressure monitor, averaging the second and third readings measured at 1 min intervals. The authors reported stratified analyses by gender and were able to adjust their models to key lifestyle factors, such as smoking, alcohol consumption, diet or physical activity. They performed sensitivity analyses excluding adults with pre-existing cardiovascular disease at baseline and the first 2 years of follow-up to avoid reverse causality. In the fully adjusted model excluding the first 2 years of follow-up, the authors demonstrated that for each increase of 10 bpm of baseline RHR, the cardiovascular, cancer and other-cause mortality increased by 11%, 8% and 20%, respectively. They also demonstrated that a temporal increase in RHR of more than 15 bpm for men and more than 25 bpm for women was associated with higher mortality risk compared with stable RHR over a 10-year period. In particular, adults who increase their RHR from below 70 bpm to above 70 bpm had a 23% higher mortality risk over the next 10 years compared with adults with stable RHR below 70 bpm.

Can we now translate this high-quality observational data into clinical recommendations for optimal RHR values among healthy adults? Similar to previous epidemiological studies, Seviiri *et al* reported in their fully adjusted models that compared with men with RHR below 60 bpm, cardiovascular, cancer and other-cause mortality were significantly increased in men with RHR equal or above 80 bpm, 60 bpm and 60 bpm, respectively. Compared with women with RHR below 60 bpm, cardiovascular, cancer and other-cause mortality were significantly increased in women with RHR equal or above 80 bpm for all three types of deaths. Therefore, taken together, the most appropriate definition of normal RHR may be below 70 bpm for men and below 80 bpm for women. Even if approximate, these optimal cut-offs are well below the established definition of tachycardia above 100 bpm used in the acute setting.

Even if causality is not yet established, clinicians should now screen for abnormal RHR defined as above 70 bpm for men and above 80 bpm for women when they monitor blood pressure in healthy adults. Because it is unknown if targeting RHR using drugs such as beta-blockers or ivabradine will improve prognosis, the most appropriate preventive attitude towards high RHR values may be lifestyle counselling. The expected availability of big data from continuous self-measurement of heart rate by the general population will hopefully inform researchers to design public health strategies to target optimal RHR.[11] Fundamental and clinical researches also need to further disentangle the link between RHR and subclinical inflammation with the aim to improve risk prediction and better identify adults whose risk may be reduced by promising anti-inflammatory drugs.[12] In conclusion, RHR should inform physicians in the acute setting and also have the

Correspondence to Dr David Nanchen, Department of Ambulatory Care and Community Medicine, University of Lausanne, CH-1011 Lausanne, Switzerland; david.nanchen@chuv.ch

potential to frame clinical decision and counselling for prevention of chronic diseases.

Funding DN researches are supported by the Swiss National Science Foundation (SNSF 32473B_163271, Long-term benefit of the multi-center, multi-dimensional secondary prevention program in patients with acute coronary syndromes).

Competing interests None declared.

Provenance and peer review Commissioned; internally peer reviewed.

To cite Nanchen D. *Heart* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/heartjnl-2017-312731

Received 30 December 2017
Revised 14 November 2017
Accepted 5 January 2018

Linked

► http://dx.doi.org/10.1136/heartjnl-2017-312251

Heart 2018;**0**:1–2.
doi:10.1136/heartjnl-2017-312731

REFERENCES

1 Bonnemeier H, Richardt G, Potratz J, *et al*. Circadian profile of cardiac autonomic nervous modulation in healthy subjects: differing effects of aging and gender on heart rate variability. *J Cardiovasc Electrophysiol* 2003;14:791–9.
2 Eppinga RN, Hagemeijer Y, Burgess S, *et al*. Identification of genomic loci associated with resting heart rate and shared genetic predictors with all-cause mortality. *Nat Genet* 2016;48:1557–63.
3 Shcherbina A, Mattsson C, Waggott D, *et al*. Accuracy in wrist-worn, sensor-based measurements of heart rate and energy expenditure in a diverse cohort. *J Pers Med* 2017;7:3.
4 Böhm M, Reil JC, Deedwania P, *et al*. Resting heart rate: risk indicator and emerging risk factor in cardiovascular disease. *Am J Med* 2015;128:219–28.
5 Nanchen D, Leening MJ, Locatelli I, *et al*. Resting heart rate and the risk of heart failure in healthy adults: the Rotterdam Study. *Circ Heart Fail* 2013;6:403–10.
6 Reunanen A, Karjalainen J, Ristola P, *et al*. Heart rate and mortality. *J Intern Med* 2000;247:231–9.
7 Seviiri M, Lynch BM, Hodge AM, *et al*. Resting heart rate, temporal changes in resting heart rate, and overall and cause-specific mortality. *Heart* : Published Online FIrst: 21 December 2017 10.1136/heartjnl-2017-312251.
8 Fox K, Ford I, Steg PG, *et al*. Ivabradine for patients with stable coronary artery disease and left-ventricular systolic dysfunction (BEAUTIFUL): a randomised, double-blind, placebo-controlled trial. *Lancet* 2008;372:807–16.
9 Nanchen D, Stott DJ, Gussekloo J, *et al*. Resting heart rate and incident heart failure and cardiovascular mortality in older adults: role of inflammation and endothelial dysfunction: the PROSPER study. *Eur J Heart Fail* 2013;15:581–8.
10 Nauman J, Janszky I, Vatten LJ, *et al*. Temporal changes in resting heart rate and deaths from ischemic heart disease. *JAMA* 2011;306:2579–87.
11 Lau E, Watson KE, Ping P. Connecting the dots: from big data to healthy heart. *Circulation* 2016;134:362–4.
12 Ridker PM. Canakinumab for residual inflammatory risk. *Eur Heart J* 2017;38:3545–8.

Heart

Resting heart rate: what is normal?

David Nanchen

Heart published online January 30, 2018