Editorial

Resting heart rate is the index of cardiovascular health

Heart rate (HR) is the most important parameter assessed by all physicians in clinical practice. Usually, it is estimated by clinical examination of the radial pulse. Radial pulse examination, referred to as "nadi pariksha" in ayurveda, was the mainstay of diagnosis of diseases in ancient days. Physiologically HR reflects the state of neural, endocrinal, metabolic and respiratory balances and the state of emotional and psychological being of the individual. HR is profoundly influenced by the neural component, especially the autonomic nervous system (ANS) as sinu-atrial node (SA node) is innervated by both parasympathetic and sympathetic divisions of ANS. However, at rest, the vagal (parasympathetic) influence on SA node predominates over sympathetic influence. Therefore, resting HR is the index of vagal tone and is considered as an important parasympathetic function test.[1] Even during the states of activation, such as exercise, emotion and excitement, though tachycardia has been explained by sympathetic activation, vagal withdrawal plays a crucial role in increase in HR. Thus, both in resting and activity states, vagus plays a vital role in the modulation of cardiac pacing.

HR is directly influenced by many hormones such as adrenaline, noradrenaline, thyroxine, dopamine, glucagon, insulin, etc. Body metabolism, via metabolic products and change in body temperature influences HR. Hypothalamus, the center of visceral, endocrinal and metabolic functions and also the center of sympathovagal output exerts a deep influence on HR. Hypothalamus is also the link for limbic control of HR that mediates influences during emotion, excitement and stress. Thus, hypothalamo-neuroendocrine-sympathovagal (HNESV) axis, the major central outflow system is the principal regulator of HR. Thus, any factor that affects this system, alters SA nodal discharge. In this system, sympathovagal output to the heart is the key element determining HR. Stress is foremost among all factors to influence HNESV axis and stress is known to reduce vagal tone and increase sympathetic activity. Stress is the commonest cause of sympathovagal imbalance, the pathophysiological basis for resting tachycardia and all-cause morbidity and mortality.[2] Due to high-stress level in the society, presently the resting HR is high in the general population.

Access this article online	
Quick Response Code:	Website: www.ijcep.org
	DOI: 10.4103/2348-8093.149747

Increase in HR decreases the duration of diastole, and therefore the diastolic perfusion time reduces.[3] With tachycardia, there is an increase in myocardial contractility by means of force-frequency relationship. As such increased HR increases the rate of contraction, which enhances the myocardial oxygen demand. Thus, tachycardia has a significant impact on perfusion-contraction matching, which is the dynamic parameter that regulates myocardial blood supply and function. In normal individuals with a healthy heart, metabolism increases as a result of increased HR and contractile function, which in turn results in increased myocardial blood flow, and oxygen extraction. Presence of coronary atherosclerosis and perfusion-contraction mismatching leads to inadequate supply of nutrients and oxygen to the myocardium. With inadequate coronary artery inflow to meet the demands, contractile and diastolic functions in the affected area are correspondingly reduced. An increase in HR results in an increase in myocardial oxygen demand and causes impairment of supply resulting from a reduction of collateral perfusion pressure and collateral flow in the coronaries.[4] This imbalance promotes coronary ischemia, cardiac arrhythmias, ventricular dysfunction, acute coronary syndromes, heart failure or sudden cardiac death. Chronic tachycardia has been found to promote cardiac noradrenaline synthesis and causes an increase in circulating plasma noradrenaline. This has direct cytotoxic effect on cardiac myocytes and has also been reported to promote cardiomyocyte apoptosis and facilitate adverse ventricular remodeling. Elasticity of the larger arteries is reduced in long-standing tachycardia that results in perfusion mismatch.[4] In addition, prolonged tachycardia exacerbates heart failure. Therefore, persistent tachycardia has been associated with all-cause mortality and sudden death.[5]

Among all variables of clinical examination, HR is the most preferred one as the radial pulse is easily accessible. HR at rest is the indicator of basal vagal tone. It has been observed that individuals with less resting HR within the physiological limit have good health. Based on the data of epidemiologic studies and clinical trials, it has been inferred that tachycardia at rest is prognostically detrimental. However, the importance of HR as a prognostic factor, and potential therapeutic effect of the reduction in HR has not been studied. Therefore,

despite having large body of evidence to correlate resting tachycardia with poor prognosis, this concept has not been generally accepted by all. As HR is the reflection of autonomic, hormonal, metabolic and physchosocial factors, resting HR not only reflects the cardiovascular health, but the health in entirety.[6] Therefore, we consider resting HR as the indicator of integral health of the person. The aim of this editorial is to make our readers and physicians aware of the importance of having less basal HR in general. Though the range of resting HR has been accepted as 60–100/min, it has been recently observed that HR < 50 and HR > 90 are associated with adverse effects. Though the range of normal HR is wide, the rise or fall from the individual's regular HR is more important. It has been generally accepted that HR > 75 is deleterious for health.

Work should be done to evaluate the possible pathophysiological basis of the association of tachycardia with different health problems, in addition to its link with adverse cardiovascular health, and the likelihood of therapeutic utility of HR slowing in improving outcomes for a wide range of patients.

G K Pal

Department of Physiology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India

Address for correspondence:
Dr. G K Pal,
Professor and Head Department of Physiology,
Programme Director, Advanced Center for Yoga Therapy, Education and Research, Jawaharlal Institute of Postgraduate, Medical Education and Research, Puducherry - 605 006, India.
E-mail: drgkpal@gmail.com

REFERENCES

1. Pal GK. Autonomic function tests. In: Textbook of Practical Physiology. 3rd ed.. Hyderabad: Universities Press; 2010. p. 282-90.
2. Thayer JF, Yamamoto SS, Brosschot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int J Cardiol 2010;141:122-31.
3. Pal GK. Heart rate and arterial pulse. In: Textbook of Medical Physiology. 2nd ed.. New Delhi: Ahuja Publications; 2011. p. 624-9.
4. Arnold JM, Fitchett DH, Howlett JG, Lonn EM, Tardif JC. Resting heart rate: A modifiable prognostic indicator of cardiovascular risk and outcomes? Can J Cardiol 2008;24 Suppl A: 3A-8.
5. Fox K, Borer JS, Camm AJ, Danchin N, Ferrari R, Lopez Sendon JL, *et al*. Resting heart rate in cardiovascular disease. J Am Coll Cardiol 2007;50:823-30.
6. Pal GK, Pal P, Nanda N, Amudharaj D, Adithan C. Cardiovascular dysfunctions and sympathovagal imbalance in hypertension and prehypertension: Physiological perspectives. Future Cardiol 2013;9:53-69.

How to cite this article: Pal G K. Resting heart rate is the index of cardiovascular health. Int J Clin Exp Physiol 2014;1:243-4.