Ravindran Revand and Sanjeev K. Singh*

Algogen-induced vasosensory reflexes modulate short-term heart rate variability parameters in experimental rat models

https://doi.org/10.1515/jbcpp-2020-0361
Received October 30, 2020; accepted January 30, 2021; published online April 20, 2021

Abstract

Objectives: The present work was designed to study the modulatory effects of algogen-induced vasosensory reflex responses on short-term heart rate variability (HRV) parameters in naïve and vagotomized rat models.

Methods: In this study, vasosensory reflex responses were elicited by instilling algogens (bradykinin/histamine), a component of inflammatory mediators into a local segment of medium-sized peripheral blood vessel (femoral artery) while a continuous electrocardiogram (ECG) was recorded. Short-term (5 min) ECG segments obtained from original recordings were examined in detail and relevant data of HRV parameters were pooled. Time domain and frequency domain analyses were performed using dedicated software (LabChart 8, AD Instruments®, Australia) and results were analyzed.

Results: Bradykinin-induced vasosensory reflexes caused significant alterations in both time domain and frequency domain HRV parameters as compared to the time-matched saline control group. Instillation of bradykinin caused a transient increase in NN interval, RMSSD, TSP, HF power (HFP) along with a decrease in the standard deviation of all normal NN intervals (SDNN), SDNN/RMSSD, LF power (LFP), LFP/HFP. Histamine produced a similar pattern of responses, but HRV alterations were less pronounced compared to those with bradykinin. Further analysis revealed that algogen-induced vasosensory reflex responses caused an increase in the parasympathetic influence on the heart accompanied by a decrease in sympathetic influence. In addition, HRV modulation by algogen-induced vasosensory reflexes was significantly attenuated in vagotomized rats, illustrating the principal role of vagus in the reflex HRV modulation.

Conclusions: The present study proposes a novel hypothesis regarding the cardio-protective role of inflammatory mediators during acute stress, by potentiating the vagal impact and attenuating the sympathetic impact on the heart.

Keywords: bradykinin; heart rate variability; histamine; nociception; vagotomy; vasosensory reflexes.

***Corresponding author: Sanjeev K. Singh**, Professor, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India, Phone: +91 7897091535, Fax: +91 542 2367568/2368174, E-mail: drsks07@bhu.ac.in

Ravindran Revand, Department of Physiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Introduction

Biological systems display intricate patterns of variability in the body that can be described by scientific anarchy. The circadian variations in hormone release, body temperature rhythms, and respiratory sinus arrhythmia are notable among them. The complex oscillations in the rate and rhythm of a healthy heart allow the cardiovascular system to adjust promptly to sudden unexpected physical and psychological challenges that could potentially alter homeostasis [1]. Heart rate variability (HRV) is a measure of the differences in the intervals between adjacent QRS complexes produced by sinus node depolarization in a continuous electrocardiographic (ECG) recording [2]. Reduced HRV is a strong, independent, and consistent risk factor of mortality in diseases involving the heart and the autonomic nervous system (ANS) [3–6]. Standard guidelines recommend two types of ECG recordings for HRV analysis: a short-term recording of 5 min or a continuous 24 h recording [2, 7]. HRV analyses using short-term ECG recordings are quicker, easier, and more feasible whereas 24 h ECG recordings provide a more precise picture of the HRV parameters [8–11]. Cardiovascular anatomy and physiology are often affected by inflammatory diseases such as rheumatic fever, systemic lupus erythematosus, and peripheral vascular diseases [12, 13]. Although it is not proven that inflammatory mediators such as histamine, bradykinin, serotonin, and prostaglandins cause cardiovascular diseases, inflammation is a common event that accompanies serious heart diseases such as myocardial infarction, atherosclerosis, heart failure, etc. [4, 12–14]. Several theories have been put forth to explain the involvement of chemical mediators of inflammation in cardiac ailments, the exact mechanism of which is still a topic of ongoing research.