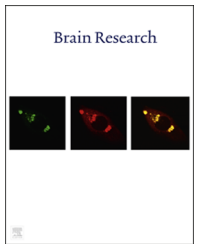
BRAIN RESEARCH 1554 (2014) 49–58

Available online at www.sciencedirect.com

ScienceDirect

www.elsevier.com/locate/brainres

Brain Research



Review

Emotional stress and sympathetic activity: Contribution of dorsomedial hypothalamus to cardiac arrhythmias

Marco Antônio Peliky Fontes[a,*], *Carlos Henrique Xavier*[c], *Fernanda Ribeiro Marins*[a], *Marcelo Limborço-Filho*[a], *Gisele Cristiane Vaz*[a], *Flávia Camargos Müller-Ribeiro*[a], *Eugene Nalivaiko*[b]

[a]*Departamento de Fisiologia e Biofísica, INCT, Instituto de Ciências Biológicas, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil*

[b]*School of Biomedical Sciences and Pharmacy, University of Newcastle, Callaghan, New South Wales, Australia*

[c]*Departamento de Ciências Fisiológicas, Instituto de Ciências Biológicas, Universidade Federal de Goiás, Goiânia, GO, Brazil*

ARTICLE INFO

Article history:
Accepted 24 January 2014
Available online 31 January 2014

Keywords:
Sympathetic activity
Emotional stress
Dorsomedial hypothalamus
Cardiac arrythmias

ABSTRACT

Maintenance of homeostasis in normal or stressful situations depends upon mechanisms controlling autonomic activity. Central requirement for changes in sympathetic output resulting from emotional stress must be adjusted to the input signals from visceral sensory afferent (feedback response) for an optimum cardiovascular performance. There is a large body of evidence indicating that emotional stress can lead to cardiovascular disease. Reviewing the descending pathways from dorsomedial hypothalamus, a key region involved in the cardiovascular response to emotional stress, we discuss the interactions between mechanisms controlling the sympathetic output to the cardiovascular system and the possible implications in cardiovascular disease.

© 2014 Elsevier B.V. All rights reserved.

Contents

*Corresponding author at: Laboratório de Hipertensão, Departamento de Fisiologia e Biofísica-ICB, Universidade Federal de Minas Gerais, Belo Horizonte, MG 31270 901, Brazil. Tel.:+55 31 3409 2953.
E-mail address: peliky@icb.ufmg.br (M.A.P. Fontes).

0006-8993/$ - see front matter © 2014 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.brainres.2014.01.043

1. Introduction

The impact of emotional stress, mainly rage and fear, on sympathetic activity was recognized by Cannon in the early decades of the twentieth century. At that time he already predicted the *dire results* of a long-term exposure to *sympathico-adrenal division* (Cannon, 1957). Currently, psychological stress is considered a component of cardiovascular risk. There is strong and consistent evidence relating emotional stress to the development of cardiovascular diseases, including hypertension, coronary heart disease and cardiac arrythmias (Matta et al., 1976; Rozanski et al., 1999; Lampert et al., 2000; Bunker et al., 2003; Esler et al., 2008). It is well known that the central sympathetic drive increases in consequence of the persistent emotional stressors leading to disease. However, the individual vulnerability or hyperresponsiveness also needs to be considered and this may involve particular early life experiences and/or genetic background. In any case, the exact groups of neurons and mechanisms involved remain to be revealed. Undoubtedly, identifying the central pathways involved in the cardiovascular response to emotional stress is a critical step to understand how emotional stress triggers and alters autonomic responses, thereby leading to cardiovascular disease. This review is focused on the central integration controlling sympathetic output to the cardiovascular system evoked by emotional stress and on the possible contribution of the dorsomedial hypothalamus, in the generation of cardiac arrhythmias.

2. Mechanisms controlling sympathetic activity

Blood pressure is controlled, to a large extent, by sympathetic nerves that are tonically active, driving the so called sympathetic tone or sympathetic cardiac and vascular outflow. The sympathetic tone is set by a network of discrete populations of neurons located in different regions of the central nervous system. Three of these regions have mostly been studied in the context of cardiovascular regulation, the rostral ventrolateral medulla (RVLM), the nucleus of solitary tract (NTS) and the hypothalamus [for concise reviews see Dampney (1994) and Guyenet (2006)]. Sympathetic tone changes in different physiological and pathological conditions. Elevations in sympathetic tone levels can increase cardiac chronotropism, inotropism, vascular resistance and changes in kidney function, increasing the baseline blood pressure.

The sympathetic tone to the cardiovascular system is regulated in the short and long term (Dampney et al., 2002), and mainly involves homeostatic neural mechanisms which are (i) the feedback mechanisms, and (ii) the central control mechanisms. The feedback mechanism adjusts the sympathetic output moment-to-moment, according to the demand of the cardiovascular system which is signaled by receptors inside the system. The feedback mechanism includes an input signal to the central nervous system from different classes of visceral sensory afferents, such as chemoreceptors and baroreceptors. Undoubtedly, the baroreflex is the most important feedback mechanism of short term arterial pressure regulation. Baroreceptors are mechanoreceptors that are activated by distention of the arterial wall caused by increases in blood pressure. Afferent fibers from the baroreceptors terminate within the nucleus tractus solitarius (NTS) that, via a glutamatergic input, excite second order neurons. The second order neurons from NTS projects to and excites GABA containing neurons in the caudal ventrolateral medulla (CVLM) that exert a powerful inhibitory influence on RVLM neurons. RVLM neurons are the most important population of presympathetic neurons, responsible for driving the tonic activity to the sympathetic preganglionic neurons as demonstrated in anesthetized (Dampney et al., 2000) and conscious animals (Menezes and Fontes, 2007). The resting activity level of RVLM neurons is considered critical for long-term blood pressure control (Guyenet, 2006). The sympathetic tonic drive from RVLM to the heart, arterioles and kidney is restrained by baroreceptor stimulation (Fig. 1; pathway 1). Activation of baroreflex also results in excitation of vagal preganglionic neurons localized within the nucleus ambiguous in most species. The excitation of vagal preganglionic neurons increases the parasympathetic tone to the heart (Dampney, 1994).

The central control mechanisms influencing the sympathetic tone are more complex and are associated to behavioral strategies, such as those involved in exercise or in response to emotional stress. The onset of exercise, for example, involves cortical command that drives motor and autonomic (including sympathetic) outflow (Williamson, 2010; Matsukawa et al., 2012). During emotional stress responses, the sympathetic output to the cardiovascular system is initiated from higher cognitive and affective circuits. This involves responses to external environment stimuli such as those triggered by visual, auditory or other sensory input signaling threat (acute stress, e.g., view of a predator). It is important to note that sympathetic activity can also be influenced by emotional memories (e.g., anxiety, chronic emotional stress). Thus, emotional stress can exert influence on the sympathetic activity over the short and long term (Fig. 1; pathway 2). There are other factors that can influence sympathetic activity in the long term, such as hormones and diseases (Brooks and Osborn, 1995; Patel and Zhang, 1996; Dampney et al., 2002) but they will not be discussed in this review.

The stressful threatening stimuli or related emotional memories activate limbic system structures, including the amygdala that plays an important role in the emotional behavior. As suggested by LeDoux and colleagues, a subcortical pathway, including a direct input from the thalamus to the lateral amygdala, mediates rapid and subconscious

Download English Version:

https://daneshyari.com/en/article/4324397

Download Persian Version:

https://daneshyari.com/article/4324397

Daneshyari.com