Psychoneuroendocrinology (2013) 38, 2686—2693

Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/psyneuen

Cortisol rapidly affects amplitudes of heartbeat-evoked brain potentials—Implications for the contribution of stress to an altered perception of physical sensations?

André Schulz [a,b,*], Florian Strelzyk [b,c], Diana S. Ferreira de Sá [b], Ewald Naumann [c], Claus Vögele [a], Hartmut Schächinger [b]

[a] Research Unit INSIDE, Research Group Self-Regulation and Health, University of Luxembourg, Luxembourg
[b] Institute of Psychobiology, Division of Clinical Psychophysiology, University of Trier, Germany
[c] Department of Psychology, Psychophysiological Laboratory, University of Trier, Germany

Received 19 February 2013; received in revised form 3 June 2013; accepted 20 June 2013

KEYWORDS
Cortisol;
Interoception;
Heartbeat detection;
Heartbeat-evoked potentials;
HPA axis;
Non-genomic mechanism;
Stress;
Symptom perception;
Visceral perception

Summary Little is known about the impact of stress and stress hormones on the processing of visceral-afferent signals. Clinical data suggest that cortisol may lower the threshold for interoceptive stimuli, while a pharmacological administration of cortisol decreases the sensitivity for physical symptoms. To clarify the role of cortisol for the processing of interoceptive signals, we investigated 16 healthy men on two occasions, once during the infusion of 4 mg of cortisol and once during the infusion of a placebo substance. Heartbeat-evoked potentials (HEP; derived from resting EEG and ECG, during open and closed eyes), which are psychophysiological indicators for the cortical processing of cardioceptive signals, were measured over 6-min periods once before, and four times after the infusion (1—7, 11—17, 21—27 and 31—37 min). We found that HEP amplitudes were higher during open than during closed eyes between 1 and 17 min after cortisol infusion. There was no effect of cortisol on heart rate. We conclude that cortisol may rapidly modulate the cortical processing of cardioceptive neural signals. These results may have relevance for the effects of stress on the development and maintenance of psychosomatic symptoms.

1. Introduction

Interoception, the perception of bodily processes, plays an important role in the subjective experience of emotions (Wiens, 2005), in consciousness (Damasio, 2003), and in somatic symptom generation (Eley et al., 2004). Stress affects interoception in that activation of the autonomic

* Corresponding author at: Research Unit INSIDE, Research Group Self-Regulation and Health, University of Luxembourg, Route de Diekirch, B.P. 2, L-7220 Walferdange, Luxembourg.
Tel.: +352 46 66 44 9549; fax: +352 46 66 44 9535.
E-mail address: andre.schulz@uni.lu (A. Schulz).

0306-4530/$ — see front matter © 2013 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.psyneuen.2013.06.027

nervous system, as one of two major physiological stress systems (Chrousos and Gold, 1992), mobilizes the cardiovascular system and may therefore enhance cardioceptive neural traffic (Eichler and Katkin, 1994; Schandry et al., 1993; Schulz et al., 2011, 2013). Little is known, however, about the impact of HPA stress axis activation (the second physiological stress system) on the processing of visceral-afferent signals.

While clinical data suggest that cortisol may lower the sensory threshold for the detection of interoceptive stimuli (Rief et al., 1998), exogenous administration of cortisol has been shown to decrease pain sensitivity (Michaux et al., 2012), and metyrapone-induced acute hypocortisolism to increase pain sensitivity (Kuehl et al., 2010b). One possible explanation for these contradictory findings could be the difference between acute and chronic exposure to stress and stress hormones, resulting in hyposensitivity to physical symptoms in acute stress and hypersensitivity under chronic stress conditions (Pruessner et al., 1999; Rief and Barsky, 2005). Nevertheless, the role of cortisol for the processing of non-pain related physical symptoms remains unclear.

Glucocorticoids, such as cortisol, may affect cells in the entire body by binding on mineralocorticoid and glucocorticoid receptors, and by slowly inducing changes in gene transcription (de Kloet et al., 1998). This may be observed earliest after 20—30 min and can last for several hours (Groner et al., 1983). Furthermore, glucocorticoids may also elicit rapid (i.e. within minutes), non-genomic effects on the cell, and it has been proposed that the mineralocorticoid membrane receptor is responsible for these effects (de Kloet et al., 2008; Groeneweg et al., 2011). Recent findings on cortisol rapidly affecting thalamic activity suggest an impact of glucocorticoids on the processing of sensory information due to non-genomic mechanisms (Strelzyk et al., 2012), which may also play a role for the perception of physical symptoms (Cameron, 2001).

Heartbeat-evoked brain potentials (HEPs) represent electrocortical potentials, which are related to the perception of cardiac signals, such as heartbeats. HEPs can be measured 250—600 ms after a cardiac R-wave and have their largest amplitude over the right hemisphere (Leopold and Schandry, 2001; Pollatos and Schandry, 2004; Schandry et al., 1986). HEP amplitudes have been demonstrated to reflect an individuals' performance in heartbeat detection tasks (Katkin et al., 1991; Pollatos and Schandry, 2004; Schandry and Montoya, 1996), motivation to perform in those tasks (Weitkunat and Schandry, 1990) and attentional focus on heartbeats (Montoya et al., 1993; Schandry et al., 1986; Schandry and Weitkunat, 1990). As summarized by Shao et al. (2011, p. 1843), HEPs can also been found independently from an individuals' conscious heartbeat perception, since ''afferent signals from the body continuously reach the brain, and consciously or unconsciously, the brain is continuously monitoring interoceptive information''. Taken together, HEP amplitudes are interpreted as psychophysiological indicator for cortical processing of cardioceptive signals, independently from a conscious process of body perception.

The aim of the current study was to investigate rapid, non-genomic effects of cortisol on HEP amplitudes. Since previous observations about cortisol effects on pain perception were based on oral administration of cortisol and over longer time periods (Michaux et al., 2012), it is difficult to attribute the effects to either genomic or non-genomic mechanisms of cortisol. We, therefore, used a different strategy and assessed the effects of intravenous cortisol infusion on HEPs in a within-subject, double blind, placebo-controlled trial. To control for possible effects of cortisol on cardiovascular activity we also monitored heart rate (HR) during all sessions. As open or closed eyes have a large impact on resting EEG (i.e. the increase of alpha band power in closed eyes), we tested all participants in conditions with open and closed eyes. Based on previous studies showing a reduction in pain sensitivity after cortisol administration and an increase in pain sensitivity as a consequence of metyrapone-induced hypocortisolism (Kuehl et al., 2010b; Michaux et al., 2012), we expected cortisol to affect cortical processing of cardioceptive stimuli in that HEPs would be decreased after cortisol infusion.

2. Method

2.1. Participants

Sixteen right-handed, healthy men (mean age = 23.8 years; SD = 2.1 years) took part in the study and received monetary compensation for participation (€ 50,—). Medical status was carefully screened prior to the experiment by using a customized interview performed by a physician (H.S.) and a psychologist (F.S.), as this is required for any bio-behavioral research at the University of Trier. Exclusion criteria were any acute or chronic physical or mental health complaints, smoking, current medication, critical life events in a time period of six months before participation, or major examinations two weeks prior to or after the experiment. All participants provided written informed consent and were made aware of their right to discontinue participation in the study at any time. Study procedures were approved by a community-based Ethics Committee (Landesärztekammer Rheinland-Pfalz).

2.2. Experimental procedure

Participants attended two laboratory sessions, one week apart, and at exactly the same time of day (between 13:00 h and 17:00 h), in a within-subject design. They received cortisol and placebo in a randomized and counterbalanced order, to control for sequence effects. All participants arrived 1 h prior to the experimental session, and a flexible intravenous infusion line was inserted into one of the radial veins, which remained there until the end of the experiment. The 1 h waiting period before the start of the experiment was intended to reduce potential carry-over stress effects related to the initial pain induced by placing the intravenous infusion line. It was repeatedly demonstrated that HEPs can be derived from simultaneous resting EEG/ECG assessment for several minutes without the instruction to focus on heartbeats (Montoya et al., 1993; Schandry et al., 1986; Shao et al., 2011). Since this study was designed to investigate rapid pharmacological effects of cortisol on HEPs, an exact timing of the psychophysiological measurement session was mandatory. Based on our own previous research on rapid cortisol effects on thalamus perfusion (Strelzyk et al., 2012) we divided the experiment into five