Psychological and physiological responses to stress
Houtveen, J.H.

Citation for published version (APA):

General rights
It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations
If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: http://uba.uva.nl/en/contact, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

UvA-DARE is a service provided by the library of the University of Amsterdam (http://dare.uva.nl)

Download date: 27 Dec 2018
Physiological responses to mental stress are regulated by the sympathetic nervous system (SNS) including the sympathetic adrenal medullary (SAM) axis, the parasympathetic nervous system (PNS), and the hypothalamic pituitary adrenocortical (HPA) axis (e.g., see Johnson & Anderson, 1990; Lovallo & Thomas, 2000). The main function of these autonomous systems is to maintain internal homeostasis (Cannon, 1915). They regulate increased energy consumption during exercise and they also influence other physiological systems, such as immune function (Lovallo & Thomas, 2000). These systems react not only in response to exercise, but they also react in response to mental stress in order to adjust internal homeostatic processes in anticipation of a possible action in the near future (i.e., the fight-flight response).

Several non-invasive techniques exist to estimate SNS, SAM, PNS, and HPA reactivity to stress situations. Sympathetic nervous reactivity may be non-invasively estimated by changes in the galvanic skin response (Boucsein, 1992; Dawson, Schell, & Filion, 2000) or (regarding the beta-adrenergic drive to the heart) by changes in the pre-ejection period (PEP) derived from the impedance-cardiogram (Cacioppo, 1994; Sherwood et al., 1990). Reactivity of the SAM axis may be non-invasively estimated by changes in the level of urinary adrenaline and/or noradrenaline, although an estimation of changes in the level of plasma adrenaline appears to be more valid and reliable (Goldstein, 1995). Parasympathetic reactivity (regarding the vagal control of heart rate) may be non-invasively estimated by changes in respiratory sinus arrhythmia (RSA) derived from the electro-cardiogram (Berntson et al., 1997; Cacioppo, 1994). Finally, reactivity of the HPA axis may be non-invasively estimated by measuring cortisol in saliva using salivettes (Kirschbaum & Hellhammer, 1989).

The original research plan for this thesis was to test a speculative idea based on relatively old literature. According to this idea, individual differences in psychological defense mechanisms (i.e., cognitive avoidance of negative emotions) should be reflected in individual differences in response patterns of the SAM, PNS, and HPA axes to mental stress. However, during an initial review of recent literature undertaken to sharpen the experimental design (chapter 2), severe methodological and theoretical issues surfaced regarding the joint reactivity of the SAM and HPA axes. Only very recently, Sapolsky and co-workers (2000) have provided guidelines on how to deal with these problems. As a consequence, this thesis has additionally focused on responses of the ‘third axis’ (i.e., the PNS
branch of the autonomous nervous system) and on central physiological responses to mental stress. Thus, although the main research question of this thesis remained whether individual differences in cognitive avoidance of negative emotions are reflected in individual differences in physiological responses to mental stress, sympathetic, cardiac vagal, and central EEG responses were measured instead of reactivity of the SAM and HPA axes.

This thesis consists of two main parts. Part I (chapters 2 to 4) deals with theoretical and methodological issues that are of relevance for the assessment of physiological responses to mental stress. Chapter 2 reviews the literature in favor of a dualism in the reactivity of the SAM and HPA axes to mental stress. This dualistic view assumes that specific psychological characteristics of a stress situation lead to differential patterns of reactivity of the SAM and HPA axes. Concisely, it has been suggested that effort induces increased SAM activation, whereas (perceived) distress triggers increased HPA activation (e.g., see Løvallo & Thomas, 2000; Lundberg & Frankenhauser, 1980). This situational specificity is tacitly assumed to reflect a broader (dispositional) difference in the preferred emotional coping style (see Henry & Stephens, 1977). Evidence for both situational and dispositional specificity in the reactivity of the SAM and HPA axes is examined in chapter 2.

Chapters 3 and 4 tackle the two main issues regarding the estimation of PNS regulation of heart rate (i.e., cardiac vagal tone) by means of RSA. Chapter 3 deals with the question of whether frequency domain or time-frequency domain measures should be used to estimate RSA. This chapter examines the error made by violating the assumption of stationarity when using discrete Fourier transformation for spectral decomposition of heart period data. Chapter 4 deals with the complex relationship between cardiac vagal tone and RSA. This chapter examines whether and how RSA is co-determined (apart from the influence of cardiac vagal tone) by the central respiratory drive, tidal volume, and respiratory rate.

Part II of this thesis (chapters 5 to 7) deals with the relationship between individual differences in cognitive avoidance of negative emotions and individual differences in physiological responses to mental stress. Dispositions like a repressive emotional coping style or alexithymia have been suggested to increase autonomous physiological reactivity to mental stress (e.g., see Brown et al., 1996; Scheier & Bridges, 1995; Schwartz, 1990; Sifneos, 1973; Weinberger, Schwartz, & Davidson, 1979). It has, for example, been suggested that individuals with a repressive emotional coping style are characterized by relatively low self-reported negative affect despite having relatively high autonomous physiological responses (Schwartz, 1990; Weinberger, Schwartz, & Davidson, 1979). This combination may ultimately lead to an increase in physical disease (Scheier & Bridges, 1995; Schwartz, 1990; Ursin & Olff, 1993). The study described in chapter 5 was
designed to demonstrate increased stress-related autonomous physiological reactivity in individuals with a repressive emotional coping style, and to explore a relationship between a repressive emotional coping style and reduced habituation of autonomous physiological responses after repeated emotional stimulation.

However, a 'mirror' group of individuals may also be defined. These individuals suffer from functional somatic symptoms or somatization disorder (i.e., a medical explanation cannot be found; see Wesseley, Nimnuan, & Sharpe, 1999; Ursin, 1997). A relationship between functional somatic symptoms and the psychological trait negative affectivity (which is related to trait anxiety) has frequently been demonstrated (see Watson & Pennebaker, 1989; Wientjes & Grossman, 1994). Thus, in contrast to individuals characterized by a repressive emotional coping style, individuals belonging to this mirror group overtly demonstrate their negative affect, both verbally and with somatization. Chapter 6 describes a study on the autonomous physiological reactivity to mental stress in a group of individuals with numerous functional somatic symptoms.

The studies described in chapters 5 and 6 were designed to examine whether individual differences in cognitive avoidance of (stress-related) negative emotions are reflected in individual differences in (peripheral) autonomous physiological responses to mental stress. However, individual differences in cognitive avoidance of (stress-related) negative emotions may also be reflected in physiological differences at the level of the central nervous system. The final study described in chapter 7 compares alexithymic individuals and controls in a central physiological (i.e., EEG-coherence) response to mental stress.

The general discussion (chapter 8) summarizes the review and the results of the 5 studies of this thesis, attempts a synthesis of the main results, and makes methodological recommendations for further research.

References


