Psychological and physiological responses to stress
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General Discussion

The main research question of this thesis was whether individual differences in cognitive avoidance of (stress-related) negative emotions are reflected in individual differences in physiological responses to mental stress. An emphasis was placed on sympathetic, cardiac vagal, and central physiological responses. A literature review and two studies were performed to be able to improve on the measurement of autonomous physiological responses to mental stress. Three studies were then performed, each with a specific group of participants who were selected on an emotional coping style that has been related (in the literature) to deviant autonomous physiological responses to mental stress. For all studies, mental stress has been experimentally induced in the laboratory. The conclusions of the review and the main results of all 5 studies described in this thesis are summarized below. First, theoretical and methodological issues regarding some measures of autonomous physiological response to mental stress are discussed. Secondly, the studies regarding the relationship between psychological traits and physiological responses to mental stress are discussed. Thirdly, a reasonable synthesis of the main results is attempted. This synthesis emphasizes cognitive avoidance of negative emotions and its reflection in the peripheral versus central physiological responses to mental stress. Finally, some recommendations are made to improve the methodology of future studies on SNS, SAM, HPA, and PNS responses to mental stress.

Methodological and theoretical issues in studying physiological responses to stress

The review in chapter 2 discussed the evidence for differential sensitivity of the SAM and HPA axes to specific psychological characteristics of a stress situation. A dualistic model has been put forward in the literature in which mental effort induces a rapid increase of catecholamine levels mediated through the SAM axis, while (perceived) distress triggers (additional) cortisol responses mediated through the HPA axis (e.g., see Lovallo & 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). Notwithstanding the fact that the dualistic model of SAM and HPA reactivity has been an accepted standpoint for more than two decades (i.e., starting from Henry & Stephens, 1977), it is concluded that several
theoretical and methodological problems exist for the human studies that have been performed to demonstrate this model. Chapter 2 presented a more parsimonious model for the observed results of these human studies, which is more in line with theoretical notions about the central and peripheral coupling of the SAM and HPA axes. This alternative model explains the observed SAM and HPA response patterns by variations in the intensity of mental stress, concurrent muscular activation, and the occupation of glucocorticoid receptors (GR) for cortisol. This model no longer needs the assumption that different psychological dimensions of a stress situation influence the pattern of SAM and HPA responses. However, it does allow for the possibility that only stress situations of high intensity (i.e., surpassing the threshold for reactivity of the HPA axis) lead to high cortisol levels. It also allows for the possibility that previous stress or continuation of the stress situation (i.e., leading to high GR occupation) influences the balance in reactivity of the SAM and HPA axes. It is not unreasonable to suggest that specifically intense or lengthy stress situations would be labeled as ‘distressful’. In practice, therefore, the predictions of the alternative model may not differ strongly from those of the classic dualistic model. However, the new model is believed to be more theoretically robust than the dualistic model.

Although SAM and HPA responses to mental stress have been studied intensively in the past, reactivity of the PNS had, until recently, not been a prime research target. However, during the writing of this thesis, there was a marked increase in the number of publications in the field of psychophysiology and psychosomatic medicine that have used RSA as an index of cardiac vagal tone (e.g., see Berntson et al., 1997; Spalding, Jeffers, Porges, & Hatfield, 2000; Watkins, Grossman, Krishnan, & Sherwood, 1998). Unfortunately, the interpretation of the results of some of these studies may have been clouded by some methodological problems in the use of RSA as an index of cardiac vagal tone. RSA may be estimated by computing the high frequency (i.e., in the respiratory band) heart period power (Berntson et al., 1997). However, some frequency domain heart period power computation methods assume that the data are (at least weakly) stationary, which may not always be the case. The study described in chapter 3 addressed the first main issue in the estimation of cardiac vagal tone by means of RSA: the influence of non-stationarity in the estimation of RSA by means of spectral analyses of heart period data. The error that is made by using discrete Fourier transformation (i.e., a method with an assumption of stationarity) was quantified by comparing heart period power values computed by discrete Fourier transformation with those computed by discrete Wavelet transformation (i.e., a time-frequency method without an assumption of stationarity). This comparison yielded surprisingly small differences (i.e., < 1%) between the power values computed by both methods in conditions of relaxation and mental stress. The differences were only slightly larger for heart period segments classified as non-stationary as compared to segments classified as stationary. It is concluded that the estimation of heart period power values by
frequency analyses based on the Fourier transform is adequate for most psychophysiological designs. The modern time-frequency method of discrete Wavelet transformation is clearly superior (for estimating heart period power values) when additional analyses in the time-frequency domain are required (e.g., in situations where the development of spectral powers in various frequency bands needs to be tracked continuously over time).

Dedicated software has been developed for the estimation of stationarity and for the computation of heart period and respiratory power values by discrete Fourier and Wavelet transformation. This software also generates additional time-frequency information obtained from the Wavelet transform. This software has been written solely for the purpose of the studies reported in chapters 3, 4, and 6. The complete software package and its annotations are considered an appendix to this thesis and have been made available as freeware (download at {www.psy.vu.nl/vu-ams/software/software.ptfap.html}).

The study described in chapter 4 addressed the second main issue in the estimation of cardiac vagal tone by means of RSA: to what extent is RSA determined by factors other than cardiac vagal tone? The results of this study demonstrated that RSA does change as a consequence of stress- or exercise-induced reductions in cardiac vagal tone. However, changes in central respiratory drive, tidal volume, and respiratory rate independently lead to changes in RSA as well. An illustration of changes in RSA that were not related to reductions in cardiac vagal tone is shown in Figure 1. This Figure shows a scatter-plot of heart rate and an index of RSA (computed with discrete Fourier transformation) obtained from a participant (of the study described in chapter 3) with a deviant RSA response to mental stress. The differences in heart rate could not be explained by differences in the beta-adrenergic drive to the heart (estimated with PEP) because the PEP remained almost unchanged during the entire measurement period. Thus, differences in heart rate must have been the result of differences in cardiac vagal tone. Although the level of RSA is normally expected to decrease as cardiac vagal tone decreases, Figure 1 clearly shows two correlation patterns. More insight was obtained after these data were analyzed separately for segments classified as relatively high versus relatively low in physical activity. An index of changes in (vertical acceleration) motility values was (also) obtained from the Vrije Universiteit Ambulatory Monitoring System (VU-AMS) used for performing these measurements. Data segments with a mean motility value below 0.6 gsec were separated from data segments with a mean motility value above 0.6 gsec. This threshold value distinguished (for our VU-AMS device) segments with quiet sitting from segments with walking or turning (the participant has for example walked in between the relaxation and mental stress conditions). The expected negative correlation was found between heart rate and RSA for segments classified as relatively high in physical activity ($r=-.63$, $n=36$, $p<.001$), while a totally unexpected positive correlation was found for the majority of segments classified as low in physical activity ($r=.64$, $n=252$, $p<.001$). The segments did not
differ in the incidence of non-stationarity, and the selected high frequency interval (i.e., the respiratory band) used for the computation of the RSA values always included the respiratory frequency. Thus, the high frequency power values should have correctly estimated RSA. It is concluded that, for this particular participant, the RSA reactivity to mental stress falsely suggested increased cardiac vagal tone, where cardiac vagal tone actually must have decreased.

![Figure 1. Heart rate - RSA scatter-plot of a remarkably deviating participant.](image)

With the results of the study described in chapter 4 in mind, an increased RSA value during mental stress need not necessarily indicate increased cardiac vagal tone. It may also reflect increased arterial partial pressure of CO₂, increased tidal volume, or decreased respiratory rate. The relative contribution of these determinants to RSA is probably not fixed (i.e., it changes between mental stress, relaxation, and physical activity; see chapter 4) and it probably shows large individual differences (e.g., the participant of Figure 1 versus the bulk of participants described in chapter 4). Therefore, it is concluded that rigid interpretation of reduced RSA in terms of reduced cardiac vagal tone may sometimes be misleading.

*The relationship between psychological traits and physiological responses to stress*

The study described in chapter 5 examined autonomous physiological reactivity and habituation to emotional stimuli in individuals with a repressive emotional
coping style and controls. Although significant effects were found (for all participants) on self-reported affect, skin conductance (used to measure sympathetic reactivity), heart rate, and facial muscle responses to the emotional pictures, no differences emerged between repressors and controls. Additionally, the groups did not differ in habituation of these responses. A disposition towards cognitive avoidance of negative emotions (e.g., a repressive or alexithymic emotional coping style) has often been related in the literature to increased autonomous physiological responses to mental stress (Brown et al., 1996; Linden & Long, 1987; Sifneos, 1973; Schwartz, 1990; Ursin & Olff, 1993; Watson & Pennebaker, 1989; Weinberger, Schwartz, & Davidson, 1979). However, the results of the study described in chapter 5 did not confirm such a relationship.

The study described in chapter 6 examined autonomous physiological reactivity to mental stress in a ‘mirror’ group of individuals that suffer from functional somatic symptoms or somatization disorder (i.e., a medical explanation cannot be found; see Wesseley, Nimnuan, & Sharpe, 1999; Ursin, 1997). In contrast to individuals characterized by a repressive emotional coping style, individuals belonging to this mirror group (that additionally score high on questionnaires that measure trait anxiety) overtly demonstrate their negative affect, both verbally and with somatization (see Watson & Pennebaker, 1989; Wientjes & Grossman, 1994). Self-reported somatic symptoms, experienced tenseness and anxiety, and RSA, PEP, heart rate, and respiratory responses to mental stress and (mild) physical exercise were compared between individuals with numerous functional somatic symptoms and controls. Although large effects in the differences between groups were found on self-reported anxiety and tenseness as well as on self-reported somatic symptoms, the expected group differences in autonomous physiological responses (i.e., in RSA responses) were not found.

As expected, no differences were found between repressors and controls in self-reported affective responses to emotional stimuli (see chapter 5). Repressors do not overtly report their affective responses. However, individuals with numerous functional somatic symptoms did significantly differ from controls in their self-reported affect and in somatic complaining (see chapter 6), and a high and significant correlation was found between scores on the frequency of functional somatic symptoms and trait anxiety ($r=.86, n=36, p<.001$). Thus, these individuals do overtly report their affective responses. In spite of the clear-cut difference in emotional coping, a contrast in the pattern of autonomous physiological reactivity was not found. No evidence was found for a straightforward effect of either repression or somatization on most measured autonomous physiological responses to mental stress. The only exception was a reduced PEP reactivity for individuals with numerous functional somatic symptoms. However, this unexpected effect needs replication before any valid conclusion can be drawn. Thus, individuals with numerous functional somatic symptoms are most likely not characterized by increased autonomous
physiological responses to mental stress. This is in line with previous suggestions that functional somatic symptoms are explained to a much larger degree by psychological mechanisms, as compared to (peripheral) autonomous physiological mechanism (e.g., see Troosters et al., 1999; Wientjes & grossman, 1994; Watson & Pennebaker, 1989; Sharpe & Bass, 1992).

The final study described in chapter 7 was performed to compare alexithymic individuals and controls in the responses to mental stress at the level of the central nervous system. The specific hypothesis was tested that alexithymia reflects a deficit in corticocortical interhemispheric communication (e.g., see TenHouten, Walter, Hoppe, & Bogen, 1987,1988). Differences between repressors and controls have previously been demonstrated in frontal electro-encephalogram (EEG) brain activation (Tomarken and Davidson, 1994). A relationship between individual differences in cognitive avoidance of (stress-related) negative emotions and EEG coherence patterns had, however, not yet been examined. In the study described in chapter 7, EEG-coherence patterns were measured in alexithymic individuals and controls during the presentation of emotional film excerpts. Indications were indeed found that alexithymic individuals have reduced EEG-coherence values between the right frontal lobe and the left hemisphere, independent of the content of the films. Nonetheless, it is concluded that individual differences in cognitive avoidance of negative emotions do have a measurable physiological representation at the level of the central nervous system.

**Synthesis of the main results**

Large individual variability exists in height and pattern of autonomous physiological responsiveness to mental stress situations (e.g., see Cacioppo, 1994; Brownley, Hurwitz, & Schneiderman, 2000). It has been suggested that autonomous physiological responses to mental stress are influenced by the style of emotional information processing (Brown et al., 1996; Linden & Long; 1987; Sifneos, 1973; Schwartz, 1990; Ursin & Olff, 1993; Weinberger, Schwartz, & Davidson, 1979). Nonetheless, the studies of this thesis did not demonstrate a relationship between individual differences in cognitive avoidance of (stress-related) negative emotions (i.e., a repressive emotional coping style) and individual differences in autonomous physiological responses to mental stress. Furthermore, a straightforward relationship between individual differences in functional somatic symptoms and individual differences in autonomous physiological responses to mental stress was not found.

A major limitation of the studies of this thesis is that the responses of other physiological systems that react to mental stress were not measured (i.e., responses of the SAM and HPA axes). Thus, the possibility cannot be excluded that individuals with a repressive emotional coping style or individuals with
Numerous functional somatic symptoms differ from controls in the responses of the SAM and HPA axes to mental stress. Additionally, since all participants of the studies of this thesis were young first-year psychology students, these results do not exclude the possibility that in the long term some psychological styles of emotional information processing may become correlated to autonomous physiological responses to mental stress (i.e., for older people).

Assuming that the results presented in this thesis do generalize to SAM and HPA measures and also hold true in older populations, it is concluded that a repressive emotional coping style and functional somatic complaining are not characterized by deviant autonomous physiological responses to mental stress. Nonetheless, individual differences in cognitive avoidance of negative emotions are not necessarily invalidated by the ‘hard’ psychophysiological results of this thesis. Alexithymic individuals, for example, showed significantly reduced EEG-coherence values between the right frontal lobe and the left hemisphere. It is therefore speculated that individual differences in repressive emotional coping or individual differences in functional somatic symptoms complaining do not reflect individual differences in (peripheral) autonomous physiological responses to mental stress, but they do reflect individual (physiological) differences at the level of the central nervous system. For example, the central threshold for detection of somatic sensations, which according to Damasio (1996) resides mainly in the frontal lobes, might be lower in individuals with numerous functional somatic symptoms, either by disposition or as a result of increased attention to somatic sensation during stress situations. In contrast, the central threshold for detection of affective and/or somatic sensations might be impaired in repressors and alexithymic individuals (as evidenced by reduced EEG-coherence values between the right frontal lobe and the left hemisphere for alexithymic individuals).

Methodological recommendations

Discrete Fourier transformation is an appropriate method for the estimation of RSA by computing heart period power values in the respiratory frequency band. However, the modern time-frequency method of discrete Wavelet transformation is clearly superior when the development of respiratory-related heart period power (RSA) needs to be tracked continuously over time. An example of such an application (for a time-frequency method) may be found by analyzing the cardiac vagal contribution to the bi-phasic heart rate response to looking at emotional pictures. The vagal contribution to this relatively fast change in heart rate (i.e., within seconds) could not be estimated with the classic method of frequency analysis using discrete Fourier transformation. However, this information is available in the time-frequency information obtained from discrete Wavelet transformation.
Studies designed to measure changes in RSA as an index of changes in cardiac vagal tone should also measure changes in the beta-adrenergic drive to the heart (e.g., with changes in PEP), changes in central respiratory rate (e.g., with end-tidal partial pressure of CO$_2$, or with an estimation of the transcutaneous partial pressure of CO$_2$; see Garssen, Buikhuizen, Hornsveld, Klaver, & vanDoornen, 1994), changes in tidal volume (e.g., with respiratory power), and changes in respiratory frequency. To correct (changes in) RSA values with (changes in) these measures is especially recommended for studies on patient groups with respiratory-related complaints like asthma and hyperventilation.

The proposed alternative model of reactivity of the SAM and HPA axes (outlined in chapter 2) may (if it holds up) help guide future research on the physiological responses to mental stress. First, the prediction of this model should be tested that the (initial) central GR occupation (in the hippocampus, pituitary, and hypothalamus) influences the balance in reactivity of the SAM and HPA axes to mental stress. This may be experimentally demonstrated by measuring the SAM and HPA responses to mental stress after administration of a placebo versus a drug that occupies the GRs to two randomly assigned groups of healthy participants. It should then be tested whether individual differences in (chronic) GR occupation reflect individual differences in chronic (mental) stress (i.e., that the GR occupation results from chronic activation of the HPA axis). Estimating individual differences in GR occupation may be problematic. One approach would be to test individual differences in the effects of administration of a drug that occupies the GRs on the SAM and HPA responses to mental stress. These should be illustrative of the existing individual differences in (initial and/or chronic) GR occupation. Performing these studies might provide evidence for a relationship between chronic stress and specific autonomous response patterns to mental stress.

An open and intriguing question is whether (chronic) GR occupation is in any way also related to central emotional information processing. Such a relationship may ultimately provide a more sophisticated model for the relationship between psychological mechanisms and the balance in SAM and HPA responses to mental stress. For example, Newcomer and co-workers (1999) have already demonstrated that increased cortisol levels (i.e., induced by stress level cortisol treatment) impair memory performance in healthy adults. Thus, future experiments combining assessments of psychological mechanisms, central nervous system functioning, and autonomous responses can extend our understanding of the complex relationship between psychological mechanisms and physiological responses to mental stress.
References


