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RUNNING HEAD: SIGH RATE AND RESPIRATORY VARIABILITY

Sigh rate and respiratory variability during mental load

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Abstract

Spontaneous breathing consists of substantial correlated variability. Using a dynamic system perspective on breathing regulation, negative emotional states are hypothesized to reduce correlated variability whereas sustained psychological states are expected to induce a lack of total respiratory variability. Both states are predicted to evoke sighing. To test these predictions, respiratory variability and sighing were assessed during stressful mental arithmetic, a non-stressful sustained attention task and a baseline and recovery period. A reduction in total variability was found during the sustained attention task, whereas correlated variation was reduced during stressful mental arithmetic. Sigh rates were elevated during the mental arithmetic task and during recovery from the non-stressful attention task. It is concluded that effects of mental stress and task-related attention can be differentiated by respiratory variability and sigh rate.

Keywords: mental arithmetic, sighing, respiratory variability

Sigh rate and respiratory variability during mental load

Emotional state affects breathing in a variety of parameters (see Boiten, Frijda, and Wientjes (1994) for a review). Whereas intense aversive affective states, such as fear, threat, pain and noxious stimulation evoke more deep breathing, mildly demanding challenges in stressful laboratory tasks, such as mental arithmetic, reaction time tasks and memory tasks typically elicit rapid shallow breathing (Wientjes, Grossman, & Gaillard, 1998). However, it is not clear whether the component of sustained attention or the stress component elicits this rapid shallow breathing pattern (Boiten, Frijda, & Wientjes, 1994). Furthermore, little is known about the effect of attentional and emotional manipulations on more complex variables, such as respiratory variability, and the few existing studies show inconsistent results. On the one hand, spontaneous breathing during rest in healthy subjects shows considerable variability (Bruce & Daubenspeck, 1995; Donaldson, 1992; Hughson, Yamamoto, & Fortrat, 1995; Small, Judd, Lowe, & Stick, 1999; Tobin, Yang, Jubran, & Lodato, 1995; Wysocki, Fiamma, Straus, Poon, & Similowski, 2006), and some findings suggest a reduction of respiratory variability during anxiety and negative affect. For example, healthy persons imagining anxious scripts show reduced respiratory variability, as well as healthy subjects scoring high on trait negative affectivity (Van Diest, Thayer, Vandeputte, Van de Woestijne, & Van den Bergh, 2006). On the other hand, negative emotional states are characterized by increased respiratory variability, labeled as breathing irregularity. For example, in patients with bronchial asthma and co-morbid anxiety disorder, more variable breathing is found during anger and resentment, and during guilt and sorrow (Stevenson & Ripley, 1952). In healthy subjects, more variable breathing patterns have been found during both painful stimulation by a cold pressor task (Boiten, 1998) and noxious stimulation by a light stimulus (Mador & Tobin, 1991).

In order to integrate these conflicting findings, we propose to distinguish between various types of variability: non-random and random variability. In a previous study we have argued how both fractions of variability contribute to adequate regulation of a healthy respiratory system that is able to maintain a homeostatic balance in the context of constantly changing internal and external demands (Vlemincx, Van Diest, Lehrer, Aubert, & Van den Bergh, in press-b). Occasional perturbations (e.g. through behavioral inputs) activate counterregulating control processes that return the system to its dynamic steady state by adjusting breaths depending on (determined by or correlated with) previous breaths. As a result, healthy breathing patterns are characterized by considerable chaos, i.e. structured, time-dependent, deterministic, correlated, non-random variability (Bruce & Daubenspeck, 1995; Donaldson, 1992; Hughson, Yamamoto, & Fortrat, 1995; Small et al., 1999; Tobin et al., 1995; Wysocki et al., 2006) which represents the homeostatic capacity of the respiratory system. Occasional random noise causes non-random variability (Small et al., 1999), improving system sensitivity and adaptibility: a system occasionally exposed to noise is trained to respond flexibly to changing demands (Giardino, Lehrer, & Feldman, 2000). However, too much noise may disregulate the system, as persistent stressors continuously disrupt homeostasis and prohibit the system to return to its dynamic steady state. In this view, healthy breathing consists of a balance between structured respiratory variability representing homeostatic capacity and respiratory stability, and random variability enhancing respiratory sensitivity and adaptability.

This dynamical system perspective, distinguishing between various fractions of variability, may shed light on the inconsistent findings above that mostly relied on general measures of total variability, i.e. the standard deviation (SD) and the coefficient of variation (CV), which can be interpreted as the sum of both random and structured variability. Only few studies have used other measures to quantify structured time-dependent, deterministic, correlated respiratory variability. For example, the autocorrelation (AR), the correlation of a signal with

itself shifted a certain time lag, quantifies variations in breaths dependent upon, determined by or correlated with previous breaths. A related parameter is respiratory short term memory, which is the maximal lag for which autocorrelation is still significant (Tobin et al., 1995). Relying on general measures of total variability only, is therefore insufficiently informative. Increases in total variability during emotional states could be due to excessive random variability, indicating a lack of stability during negative emotional states involving distress. Consistent with this is the finding that panic disorder patients exhibit higher levels of baseline respiratory variability (Abelson, Weg, Nesse, & Curtis, 2001; Martinez et al., 2001; Wilhelm, Trabert, & Roth, 2001b; Yeragani, Radhakrishna, Tancer, & Uhde, 2002), but show decreased respiratory short term memory (Wilhelm, Trabert, & Roth, 2001a), suggesting that their respiration is characterized by more random and less structured variability. Also the finding that total respiratory variability is elevated during intense emotions, stress and pain is in line with this idea, assuming an increased random fraction in the total respiratory variability. In contrast, decreases in total respiratory variability during psychological states could be the result of a lack of structured variability, ensuing from sustained psychological processes supporting task-related attention or behavior and inhibiting responsiveness to environmental changes. Dynamical system theory predicts that captivity in such attentional and/or behavioral state (‘attractor’) results in reduced system flexibility and adaptability (Thayer & Lane, 2000). For example, heart rate variability decreases during sustained selective attention and working memory tasks in healthy humans (Hansen, Johnsen, & Thayer, 2003). As focused attention reduces system complexity, it may also cause a reduction of total respiratory variability (CV). Evidence that total respiratory variability is reduced during anxious imagery fits in with this reasoning.

A related aspect of respiratory variability that has largely been disregarded is sighing. Based on the perspective above, we have previously theorized that sighing acts as a resetter of

the respiratory system and helps to restore healthy variability when physiological variability becomes unbalanced, either when respiration progressively lacks variability, or when respiratory variability becomes excessively random (Vlemincx et al., in press-b).

In this perspective, the assessment of sighing and random and structured respiratory variability, in addition to basic respiratory parameters and heart rate variability could be crucial to differentiate mental stress from task-related attention. In the present study we will evaluate these various measures during both a stressful and non-stressful but attention engaging task, and compare these with a resting baseline and intermittent recovery periods. Whereas distress during the stressful task is expected to decrease structured correlated variations in breathing and to increase random variability, mere task-related attention during the non-stressful task is hypothesized to reduce total respiratory variability. Based on these hypotheses, sighing is predicted to increase during the stressful task and following the non-stressful task. Rapid shallow breathing and reduced heart rate variability are expected not to distinguish between both tasks.

Method

Participants

Forty-three healthy university students participated in the study (21 men, age 18-22, 14 psychology and 29 sports students). Because of unreliable data acquisition, respiratory and ECG data from one participant and pCO_2 data from four participants were excluded from analysis. The experiment was approved by the Ethics Committees of the Department of Psychology and of the Faculty of Medical Sciences.

Apparatus

Respiration and heart rate were measured using the LifeShirt System® (Vivometrics Inc., Ventura, CA). Breathing data were continuously collected by means of respiratory inductive

plethysmography (RIP). At the level of the rib cage and the abdomen two shielded electrical wires sewn into an elastic LifeShirt garment served as RIP transducers. These RIP transducers, together with three ECG sensors and three accelerometer sensors, were connected to the LifeShirt recorder, a small battery powered digital processing unit, including a compact flash memory card, on which data were stored. $FetCO_2$ was monitored by means of a nose canula, which was connected to a POET II capnograph (Criticare, Waukesha, WI). Surface EMG of the M. Trapezius pars descendens, pars transversalis and pars ascendens was measured using pre-gelled Ag/AgCl contact electrodes (Nikomed, Denmark), attached according to SENIAM European recommendations and wired to a digital-analog converter unit (National Instruments, Austin, TX). EMG data were collected for purposes which are beyond the scope of this paper and will be discussed elsewhere.

Procedure

Participants were invited to an experiment studying the effects of mental arithmetic on physiological variables, such as respiration, heart rate and muscle tension. Upon arrival, participants were informed on the course of the experiment and signed an informed consent form. First, the EMG electrodes were attached and EMG measurement was calibrated. Second, they put on the LifeShirt garment, ECG electrodes were attached and all sensors were connected to the LifeShirt recorder. Third, the nose canula was put on and connected to the capnograph.

After having checked whether proper physiological signals were being obtained, the experimenter presented the written instructions to the participants. Participants were informed that the experiment consisted of four different tasks. A first task implied watching the documentary ‘The March of the Penguins’ during baseline and during recovery after each of the other tasks. They were ensured that no questions about the movie would be asked later on and they could relax and enjoy watching the film. A second task was a mental arithmetic task

performed under stressor conditions. A continuous series of sums of five operations with a two- or three digit number had to be performed in silence without any verbalization (e.g. 361 +7 /2 -4 *2 +13). Participants were instructed not to speak, mumble or move their lips. They used the mouse cursor to indicate the correct answer by choosing between three alternatives, after which feedback was given. The five participants who achieved the most correct answers were rewarded with a free movie ticket. The experimenter was seated next to the participant. This mental arithmetic task was stressful: task difficulty was high, feedback was given, evaluation and rewards were given in function of performance within time constraints and a near observer was present (Boiten, Frijda, & Wientjes, 1994; Gaillard & Wientjes, 1994). A third task was a non-stressful, but attention engaging task during which participants indicated the largest number of three alternatives using the mouse cursor. This attentional task required the same motor movement (indicating the correct answer with the cursor) as well as sustained focused task attention, but in contrast to the mental arithmetic, task difficulty was extremely low, no time constraints were applied and no performance rewards were given (Boiten, Frijda, & Wientjes, 1994; Gaillard & Wientjes, 1994). A fourth task was simply to sigh once. The purposes of this imposed sigh are beyond the scope of the present paper and will be discussed elsewhere. Before the experiment started, participants were explicitely instructed again not to speak, to sit comfortably, not to change posture and not to move, except for their dominant hand using the mouse cursor.

In summary, the experiment consisted of a series of seven 6-min phases, starting with a baseline, which was followed by a non-stressful attentional task (AT) and two mental arithmetic tasks (MT1 and MT2). Each of these tasks was presented in completely randomized order and followed by a 6-min recovery period (RC). The mental arithmetic task was presented twice in order to explore potential habituation effects of mental arithmetic stress. After one of the mental arithmetic tasks, the participant performed the instructed sigh

before the subsequent recovery period. Because no significant differences between the recovery phases after mental stress with and without instructed sigh were found, these two conditions have been collapsed and treated as one recovery phase in the analyses. Randomization was controlled by custom made stimulus presentation and data acquisition software Affect 4.0 (Spruyt, Clarysse, Vansteenwegen, Baeyens, & Hermans, in press).

Data analysis

Parameter extraction

Respiration

Raw respiratory data were edited using dedicated Vivologic software (Vivometrics Inc., Ventura, CA). Respiratory waveforms were calibrated in two ways. First, a qualitative diagnostic calibration was performed, consisting of the automatic detection of a 5-min period of regular breathing, during which relative gains in rib cage and abdominal RIP signals were determined. Second, the RIP sum signal was calibrated in absolute volume units by means of a fixed volume calibration using an 800-ml calibration bag. Four calibration trials, alternating sitting and standing, were run separated by 30-s pauses. In each trial, participants were asked to put on a nose clip and breathe rapidly in and out seven times filling and emptying an 800-ml calibration bag with each breath. Because the experiment was run in sitting position, the best fitting of two sitting calibration trials was chosen. Next, respiratory parameters were calculated breath-by-breath. Sighs within each phase of the experiment were defined as breaths with an inspiratory volume at least 2 times as large as the mean inspiratory volume during this phase. $FetCO_2$ values were recorded on computer by hyperterminal at a sample rate of 1 Hz and averaged across each phase.

Movement artefacts were controlled for by checking the accelerometers signals. Changes in physical activity (both motion and posture) were absent or minimal.

ECG

Vivologic software (Vivometrics Inc., Ventura, CA) was used to edit raw ECG, from which the time between consecutive R-peaks (RR intervals) was derived. Three controls for artifacts were performed. First, movement artefacts were kept as low as possible through explicit instruction (see above). Next, automated ectopic beat identification was based on four criteria : (1) an extremely short RR interval followed by an extremely large RR interval, surrounded by a normal interval; (2) the difference of the extreme large and small interval must exceed 100 ms; (3) the sum of the extreme large and small interval should be within 10% of the sum of the preceding and following normal intervals; (4) the values of the surrounding normal intervals should be within 20% of the value of the following normal interval. After this ectopic beat detection, these ectopic beats were linearly interpolated. Finally, the data were visually checked for additional artifacts, but none were found.

Measures

The number of sighs and respiratory variability were considered as primary measures. The coefficient of variation (CV) and autocorrelation at one breath lag (AR) of inspiratory volume (Vi), respiration rate (RR) and minute ventilation (MV) were calculated as measures of total respiratory variability and correlated respiratory variability, respectively. Both measures of respiratory variability were calculated including and excluding sighs, to find out whether differences in variability could be due to differences in sighs.

Secondary measures were mean basic respiratory parameters Vi, RR, MV, contribution of ribcage breathing to inspiratory volume (%RCi) and fractional end-tidal CO_2 ($FetCO_2$), and heart rate (HR) and measures of heart rate variability (HRV: Total Power, LF, HF and RMSSD).

Statistical analysis

Each of the above measures was calculated within each 6-min phase and subjected to a repeated measures ANOVA with 'phase' (baseline, AT, MT1, MT2, RC after AT, RC after MT) as within subject variable. In order to explore further differences between each phase, baseline and RC, post hoc contrasts were tested by means of Tukey comparisons.

Next, to further explore how mean respiratory parameters and heart rate evolved across time, averages across 2-min periods within each 6-min phase were calculated. Each of these variables was analysed in a repeated measures ANOVA with 'phase' (baseline, AT, MT1, MT2, RC after AT, RC after MT) and 'time' indicating the 2-min period within the 6-min phase (time 1, time 2, time 3) as within subject variables. Only significant post hoc Tukey differences between times within each phase will be reported here. Reported p-values are Greenhouse-Geisser corrected and ε-values are reported. Effect sizes are reported as partial eta square (η_p^2).

In addition, HRV indices were subjected as dependent variables to a mixed model analysis with 'phase' as categorical independent variable and respiration rate and tidal volume as continuous covariate variables. Since respiration has been shown to have effects on HRV independent of cardiac vagal tone, this possible confound of respiratory parameters was taken into account in the HRV analysis (Ritz, 2009). In this way, the HRV results could be interpreted in terms of cardiac vagal tone solely. In order to account for the within subject manipulation of the categorical predictor and continuous covariates in predicting a continuous outcome, a mixed model approach was chosen.

Results

Respiratory variability measures (including and excluding sighs), sighing and mean basic respiration parameters comparing baseline, AT, MT1, MT2 and RC can be found in Table 1.

Primary measures

Respiratory variability

Phase had a significant effect on all measures of respiratory variability (see Table 1). The following significant post hoc Tukey contrasts were revealed.

Inspiratory volume

Compared to baseline, CV(Vi) was significantly higher during MT1 ($p<.0001$), MT2 ($p<.0001$), RC after AT ($p<.0001$), but did not differ from CV(Vi) during AT and RC after MT. Excluding sighs reduced overall CV(Vi) (see Table 1), but did not change this pattern across phases. In line with the predicted results, total variation in Vi was increased during MT.

Whereas correlated variability was predicted to be reduced during MT, AR(Vi) appeared to be significantly lower during MT2, but not during MT1. Compared to baseline, AR(Vi) was significantly lower during MT2 ($p<.0001$) and RC after AT ($p<.001$). After excluding sighs, AR(Vi) during baseline and during RC after AT did no longer differ.

Respiration Rate

CV(RR) during AT was significantly lower compared to MT1 ($p<.0001$), MT2 ($p<.0001$), RC after AT ($p<.0001$) and marginally lower compared to baseline ($p=.07$) and RC after MT ($p=.06$). These marginally significant differences became significant when sighs were excluded ($p<.05$). Thus, as predicted, AT was marked by decreased total variability in RR.

AR(RR) during baseline was significantly higher compared to MT1 ($p<.001$) and MT2 ($p<.01$), but did not differ from all other phases, suggesting that, in line with the hypotheses, correlated variability in RR was reduced during MT. Excluding sighs yielded the same pattern.

Minute Ventilation

CV(MV) was significantly lower during baseline, compared to MT1 ($p<.05$), MT2 ($p<.05$) and RC after AT ($p<.01$), but did not differ from CV(MV) during AT and RC after MT. When excluding sighs the difference between baseline and MT2 remained only marginally significant ($p=.05$). Thus, as hypothesized, total variability in MV increased during MT.

However, consistent with variability in Vi, correlated variability in MV was decreased during MT2 but not during MT1: higher AR(MV) was found during baseline and AT compared to MT2 ($p<.0001$). AR(MV) during MT1 appeared to be significantly higher compared to MT2 ($p<.0001$), RC after AT ($p<.01$). When excluding sighs, AR(MV) during MT1 was also significantly higher compared to RC after MT ($p<.05$).

Sighing

The mean number of sighs was significantly different across phases (see Table 1). Post hoc Tukey comparisons showed significantly more sighs during MT1 ($p<.01$), MT2 ($p<.001$) and RC after AT ($p<.0001$) compared to baseline. The mean number of sighs during baseline did not differ from that during AT and RC after MT. This suggests that, consistent with our predictions, sighs appeared characteristic of MT and RC following AT.

Secondary measures

Heart Rate

The effect of phase on HR was significant ($F(5,205)=43.23$, $p<.0001$, $\varepsilon=.42$, $\eta_p^2=.51$). Post hoc Tukey contrasts revealed significantly higher HR during AT, MT1 and MT2 compared to baseline ($p<.01$, $p<.0001$, $p<.001$, respectively) and RC phases (all $p<.0001$). HR during MT1 also was significantly higher compared to AT ($p<.0001$) and MT2 ($p<.0001$). During AT and MT1, HR was significantly higher at time 1 compared to time 2 ($p<.01$, $p<.0001$,

respectively) and time3 (p<.05, p<.0001, respectively). These results indicate that HR was elevated during both MT and AT, but most so during the onset of MT1.

Heart Rate Variability

Phase had a significant effect on Total Power ($F(5,205)=8.13$, $p<.0001$, $\varepsilon=.46$, $\eta_p^2=.17$), LF ($F(5,205)=6,77$, $p<.0001$, $\varepsilon=.46$, $\eta_p^2=.14$), HF ($F(5,205)=7.54$, $p<.0001$, $\varepsilon=.52$, $\eta_p^2=.16$) and RMSSD ($F(5,205)=7.33$, $p<.0001$, $\varepsilon=.42$, $\eta_p^2=.19$). Post hoc contrast showed that, compared to baseline, Total Power and LF were significantly lower during AT (all p<.0001) and MT1 (p<.05; p<.05, respectively). HF and RMSSD were significantly higher during baseline compared to AT (p<.0001, p<.001, respectively), MT1 (p<.001, p<.0001, respectively) and MT2 (both p<.05). These uncorrected HRV results suggest reduced vagal tone during AT and MT.

However, taking into account the effects of RR and Vi on these HRV indices, phase no longer had influence on Total Power ($F(5,245)=1.76$, *ns*), LF ($F(5,245)=1.42$, *ns*), and HF ($F(5,245)=1.14$, *ns*). The effect of phase on RMSSD remained significant ($F(5,245)=3.61$, $p<.05$). Post hoc Tukey comparisons revealed higher RMSSD during baseline and RC phases compared to MT1 (all p<.05). RMSSD during MT2 was marginally lower compared to baseline (p<.06). Thus, when correcting for respiration, only RMSSD results show reduced vagal tone during MT1, and to a lesser extent during MT2.

Basic respiratory parameters

The effect of phase was significant for all basic respiratory parameters except for $FetCO_2$ (see Table 1). Post hoc Tukey comparisons revealed the following patterns of results.

Inspiratory volume

Significantly higher Vi during baseline and MT1 was found compared to AT (p<.0001), MT2 (p=.05) and RC periods (p<.05). During AT and MT1, Vi appeared to be significantly

higher at time 1 compared to time 2 ($p<.01$, $p<.0001$, respecively) and time 3 ($p<.01$, $p<.0001$, respectively), suggesting that high Vi was specific to the onset of MT1.

Respiration rate

Increased RR seemed characteristic of both AT and MT. RR during AT, MT1 and MT2 was significantly higher compared to baseline ($p<.0001$) and RC periods ($p<.0001$). At time 1 of MT1, RR was significantly higher compared to time 3 ($p<.001$). During RC periods, RR was significantly lower at time 1 compared to time 3 (all $p<.05$).

Minute ventilation

MV during baseline and AT was significantly lower than MV during MT1 ($p<.0001$), but was significantly higher than MV during RC phases ($p<.001$). During AT, MT1 and MT2, MV was significantly higher at time 1 compared to time 2 ($p<.0001$, $p<.0001$, $p<.01$, respectively) and time 3 ($p<.0001$, $p<.0001$, $p<.01$, respectively). In sum, the start of MT1 was marked by high MV.

Proportion ribcage breathing to inspiratory volume

%RCi during MT1 did not differ from %RCi during baseline and MT2 ($p>.05$), but was significantly higher compared to AT ($p<.001$) and RC periods ($p<.001$), suggesting that increased %RCi was specific of MT.

Discussion

The primary aim of the present study was to compare respiratory variability and sigh frequency during mental stress elicited by a mental arithmetic task with task-related attention in an easy control task and with a baseline and intermittent recovery phases. Secondarily, basic respiratory measures and heart rate variability measures were collected to replicate effects of mental stress. These will be discussed first.

Heart rate variability measures in this study suggest reduced vagal tone during the non-stressful attentional task and during the mental stress tasks. This finding is consistent with other results reported in literature (Cacioppo et al. 1994; Delaney & Brodie, 2000; Hansen, Johnsen, & Thayer, 2003; Thayer & Lane, 2000). These results suggest that reductions in vagal tone may be an effect of sustained focused attention, rather than mental stress specifically. However, when correcting heart rate variability measures for respiratory influences, only few effects of mental stress remained, and only in the RMSSD measure. Whether respiratory variables should be controlled for when considering heart rate variability as an index of cardiac vagal tone is subject of an intense debate. Regardless of one's position in this respect, it is clear that the different tasks show strong effects on respiration and that correcting for respiration largely eliminates the effects of task characteristics on HRV.

Our results show that mental arithmetic provokes rapid shallow breathing, but this particular breathing pattern is not specific to mental stress. Also during the non-stressful attentional task breathing rate strongly increases, suggesting that a rapid shallow breathing pattern is more characteristic of sustained task attention rather than a marker of mental stress. In contrast, the percentage of ribcage breathing during inspiration was higher during the mental stress task compared to the control task. This suggests that the proportion of thoracic breathing could be considered as indicator of mental stress, which is consistent with evidence that more thoracic breathing is found during negative affect, pain, threat of pain and unpleasantness (Boiten, Frijda, & Wientjes, 1994).

Although the mental stress task could not be distinguished from the non-stressful attentional task based on average respiratory timing and volume parameters, both tasks could be clearly differentiated by measures of respiratory variability. During mental stress, total breathing variability increases, but autocorrelation is reduced, which implies that the mental stress task increased random respiratory variability. This increase in randomness is not mainly

due to increased sighing, since the results do not change when sighs are excluded. In contrast, a lack of total respiratory variability is found during the non-stressful attentional task. Together, these findings suggest that the nature of respiratory variability, rather than mean basic respiratory parameters, differentiates between sustained task engagement and mental stress. This conclusion fully applies to respiratory variability of respiration rate, but less to the volume parameter. As the timing parameter shows the most pronounced effects of mental arithmetic, it is not surprising to find more consistent results in this parameter. For volume parameters these results only apply to the second mental arithmetic task. Apparently, coping with the mental stress task changes across the repeated trials. When initially exposed to the mental stress, a particularly strong increase in inspiratory volume, minute ventilation and heart rate are found. This pattern matches physiological responses during affective states with high arousal level (Boiten, Frijda, & Wientjes, 1994). Once the initial effects have disappeared and the participants have adapted to the task requirements, only respiration rate remains high, and autocorrelation strongly decreases, suggesting that these are specifically characteristic of the mental stress task.

Another marker of mental stress is sigh rate. As predicted, sigh rate strongly increased during the mental load task. In line with this, increased sigh rates are found during other aversive states, such as unpleasant thoughts (Finesinger, 1944) and pain (Keefe & Block, 1982; Keefe & Hill, 1985; Keefe, Wilkins, & Cook, 1984). However, sighing is also associated with relief of aversive states. Sighs are related to relief of dyspnea and perceived restlessness (Hirose, 2000), relief of negative affectivity and craving during smoking withdrawal (McClernon, Westman, & Rose, 2004), and relief of stress (Soltysik & Jelen, 2005; Vlemincx et al., in press-a). These findings suggest that sighing is not just a marker of relief, but actually induces relief during aversive states. This fits in with the hypothesis that sighing is a way of coping with stress by ‘resetting’ psychologically, physiologically or both.

First, sighing could be a means to deal with tension elicited by induced stress when aversiveness becomes untolerable. Second, sighing could also be used to manage physiological changes that arise by inducing stress. For example, Franco et al., 2003 has shown how sighing resets reduced parasympathetic activity in infants that died later of Sudden Infant Death Syndrome. In addition, recent evidence shows that sighs occur towards increasingly random breathing and reset structured correlated respiratory variability (Baldwin et al., 2004; Vlemincx et al., in press-b). Resetting parasympathetic dominance and deterministic respiratory variability by sighing could be associated with relief of tension built up when sympathetic activity dominates or breathing becomes too irregular.

In addition, increased sighing is found during the first period of recovery following the non-stressful attentional task, resulting in increased variability in volume during recovery from this task. As this task itself is marked by reduced total respiratory variability, this result is in line with our prediction that sighs are more likely to occur when total respiratory variability becomes reduced during sustained psychological states. A lack of respiratory variability, i.e. breathing at a constant volume, will cause a progressive collapse of alveoli (atelectasis), which will reduce lung compliance and gas exchange efficiency. Sighing is assumed to remediate atelectasis (Bendixen, Smith, & Mead, 1964; Reynolds, 1962) and to restore lung compliance (Caro, Butler, & Dubois, 1960; Ferris & Pollard, 1960; McIlroy, Butler, & Finley, 1962; Mead & Collier, 1959) and gas exchange (Cherniack, Euler, Glowgowska, & Homma, 1981). Thus, it seems plausible that the rigid breathing pattern caused by the control task elicited atelectasis. When the control task ended, participants apparently recovered by sighing, possibly in order to restore reduced lung compliance and/or gas exchange caused by progressive atelectasis.

In a broader perspective, these findings fit in with the hypothesis that sighs play an important role in the dynamics of respiratory variability and related psychological states.

Information theory posits that occasional noise enhances the system's signal to noise ratio and this way generates information to the system (Suki et al., 1998; Wiesenfield & Moss, 1995). Excessive noise, on the other hand, has the opposite effect and deprives the system of information (Suki et al., 1998). Deficient complexity in the respiratory system reflects disfunctional regulation of the respiratory system: minimal total variability or excessive random variability reflect a lack of system sensitivity or a lack of system stability, respectively. Both can be regarded as situations in which the signal to noise ratio is low. Sighs can be considered as noise factors that reset structured, time-dependent, deterministic, correlated variability and thus restore the complex nature of respiratory variability, enhancing the respiratory system's signal. Psychological states altering respiratory dynamics by either minimizing respiratory variability, or adding excessive noise, deprive the respiratory system of information and this way may evoke sighing, which would not only reset respiratory responsiveness and complexity, but may also counteract the experienced subjective state. Temporary relief of these physiological and/or psychological states by sighing may act as a negative reinforcer, promoting learning of sighing behavior as a coping response.

Although information theory proposes many other chaos and complexity measures to quantify signal-to-noise, it appears that many of those non-linear analysis techniques cannot differentiate between chaotic and random signals (Denton, & Diamond, 1991). Because our main goal is to distinguish between correlated and random variability, the reported results are limited to more straightforward analysis of the coefficient of variation and autocorrelation.

In sum, the present study shows that it is important to consider measures of respiratory variability and sighing, in addition to mean basic respiratory parameters when investigating the influence of emotion upon respiration. Stressful mental tasks not only elicit rapid shallow breathing, but also evoke random breathing. In contrast, mere task-related attention appears to be marked by a lack of respiratory variability. Finally, sighing appears to be evoked either

during mental stress characterized by random respiratory variability, either following sustained attention marked by reduced breathing variability.

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Table 1

Mean basic respiratory parameters (Vi (ml), RR (breaths/min), MV (l/min), $FetCO_2$ (%), RCi (%)), sigh rate (N) and variability measures (CV and AR) including and excluding sighs during the experimental phases

				Phase					
	F (5, 205) (5,190)#	ε	η_p^2	baseline	AT	MT1	MT2	RC after AT	RC after MT
Mean									
Vi	9.31***	.60	.19	375.48 $_{a}$	334.33 $_{b}$	375.31 $_{a}$	353.32 $_{a,b}$	351.47 $_{b}$	343.31 $_{b}$
RR	31.99***	0	.44	16.18 $_{a}$	18.76 $_{c}$	18.19 $_{b,c}$	17.41 $_{b}$	15.88 $_{a}$	15.92 $_{a}$
MV	30.51***	.49	.43	5.78 $_{a}$	6.00 $_{a}$	6.47 $_{b}$	5.8 $_{a}$	5.34 $_{c}$	5.22 $_{c}$
RCi	7.33***	.70	.15	40.85 $_{a,b}$	39.14 $_{b}$	42.95 $_{a}$	41.66 $_{a}$	39.02 $_{b}$	38.88 $_{b}$
$FetCO_2$#	0.43 ns	.49		5.24 $_{a}$	5.29 $_{a}$	5.24 $_{a}$	5.25 $_{a}$	5.28 $_{a}$	5.23 $_{a}$
sigh rate	6.41***	.71	.14	0.79 $_{a}$	1.38 $_{a,b}$	1.93 $_{b}$	2.05 $_{b}$	2.12 $_{b}$	1.36 $_{a,b}$
CV									
Vi	11.37***	.76	.22	24.00 $_{a}$	29.56 $_{a}$	36.35 $_{b,c}$	38.29 $_{c}$	36.61 $_{b,c}$	30.5 $_{a,b}$
Vi ex. sighs	9.07***	.67	.18	18.54 $_{a}$	20.01 $_{a,b}$	24.28 $_{d}$	23.35 $_{c,d}$	21.98 $_{b,c,d}$	21.41 $_{b,c}$
RR	5.93***	.81	.13	16.37 $_{a,b}$	13.34 $_{b}$	18.40 $_{a}$	18.24 $_{a}$	17.84 $_{a}$	16.43 $_{a,b}$
RR ex. sighs	5.22***	.80	.11	15.95 $_{a}$	12.40 $_{b}$	17.28 $_{a}$	16.86 $_{a}$	16.54 $_{a}$	15.62 $_{a}$
MV	5.76***	.67	.12	20.59 $_{a}$	21.17 $_{a}$	25.80 $_{b}$	25.52 $_{b}$	26.52 $_{b}$	23.49 $_{a,b}$
MV ex. sighs	5.86***	.74	.13	19.13 $_{a}$	19.4 $_{a}$	23.72 $_{b}$	22.28 $_{a,b}$	23.13 $_{b}$	21.39 $_{a,b}$
AR									
Vi	8.81***	.65	.18	.20 $_{a}$	.12 $_{a,b}$	.11 $_{a,b}$	.01 $_{c}$	.05 $_{b,c}$	.14 $_{a,b}$
Vi ex. sighs	4.6**	.79	.10	.24 $_{a}$	.24 $_{a}$	.22 $_{a}$	.11 $_{b}$	.18 $_{a,b}$	.21 $_{a}$
RR	6.03***	.82	.13	.26 $_{a}$	.22 $_{a,b}$	.12 $_{c}$	.13 $_{b,c}$	.21 $_{a,b}$	.22 $_{a}$
RR ex. sighs	6.04**	.82	.13	.47 $_{a}$	.46 $_{a}$	.40 $_{b}$	.39 $_{b}$	.44 $_{a,b}$	.46 $_{a}$
MV	8.4***	.75	.17	.28 $_{a}$	.25 $_{a,b}$	.30 $_{a}$	.11 $_{c}$	.18 $_{b,c}$	.23 $_{a,b}$
MV ex. sighs	7.66***	.76	.16	.31 $_{a,c}$	.32 $_{a,c}$	.37 $_{c}$	.18 $_{b}$	.23 $_{a,b}$	.26 $_{a,b}$

*(**p<.001, *** p<.0001, means with different subscripts differ at .05 using Tukey corrections)*