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Comparing real-life and laboratory-induced stress reactivity on cardio-respiratory parameters: Differentiation of a tonic and a phasic component

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ABSTRACT

To recreate stress in laboratory conditions, the nature of the elicited physiological reactions to the presentation of mental tasks has been extensively studied. However, whether this experimental response is equivalent to real-life stress reactivity is still under debate. We investigated cardio-respiratory reactivity to a sequential protocol of different mental tasks of varying difficulties, some of them involving emotional material, and repeated the measures in a baseline and in a real-life stress situation. R-R interval (RRI), breathing frequency and volumes, and respiratory sinus arrhythmia (RSA) were computed. Baseline results showed a superior sensitivity of respiratory parameters to mental task load over RRI and RSA, no effect of task difficulty or emotional material, and a habituation response of all parameters along the protocol. Stress results showed a dual effect: first, a decreased RRI and RSA in rest values, and second, a decreased reactivity in RRI in response to mental tasks. These findings are discussed through the interaction of activation, considered to be a tonic variable, and arousal, as a phasic response.

1. Introduction

Knowledge about cardiovascular reactivity to mental tasks mainly stems from stress research. Indeed, to recreate stress in laboratory conditions for reactivity research purposes, mental tasks like arithmetics or the Stroop tasks have been among the most widely applied paradigms [1]. Investigation of the nature of these reactions instigated the conceptualization of *reactivity*, defined as "a deviation of a cardiovascular response parameter from a comparison or control value that results from an individual's response to a discrete environmental stimulus" [2], or summarized as "an acute and relatively rapid change in a cardiovascular parameter as a function of the presentation of a stressor" [1]. Further research on the nature of this reactivity response focused on the clinical relevance of its predictive value with regard to hypertension and coronary heart disease. The reactivity hypothesis [3] provided the framework for this link between reactivity and pathology. Excessive sympathetic nervous system (SNS) activation was indeed frequently suggested to be the pathophysiological mediator between responses to behavioural stressors and essential hypertension. This distinction of a predictive behavioural pattern implied a relevant interindividual difference in reactivity, which is studied through cardiovascular parameters. In cardiovascular physiology, these differences in individual responses have not been treated as noise, since one of the main goals of this research was to single out predictors identifying people at risk of coronary heart disease. Three methods for identification of hyperreactive individuals have been described [4]. First, individuals producing the largest deviation from baseline when exposed to a single unrepeated stressor (e.g. a mental task, cold pressor test or a speech stressor) would be considered hyperreactive. The ecological validity of these single, unrepeated laboratory-based measures obviously is low. Second, repeatedly re-exposing participants to the same stressor, would indeed provide a measure for the reliability of the results, although generalizability of the reactivity to different stressors still could not reliably be assessed. Third, measuring the reactivity to repeated and differing stressors, would allow to obtain both test-retest reliability and a measure for the generalizability of the hyperreactive response pattern. Whereas it has widely been considered that individual differences in cardiovascular reactivity were consistent over time, and over types of stressors (e.g. between mental arithmetics and exercise) [1], some sources [5] emphasized inconsistent findings in cardiovascular reactivity research. The relevance of the magnitude of the evoked cardiovascular responses as a meaningful index in

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studies relating cardiovascular reactivity and family history of hypertension has been emphasized, at least partly explaining the inconsistent findings [6]. A positive family history would not provoke an exaggerated reactivity to stress, but rather a less discriminant reactivity. Despite its obvious relevance, few studies examined the relation between laboratory-induced reactivity and reactivity to real-life situations.

The Stroop colour-word task has repeatedly been used in the investigation of autonomic reactivity to mental stress [7–10]. Some investigators used this mental stress test, involving sensory rejection, to model defence reactions in humans [11,12]. Others [13] used the contrast of congruent and incongruent Stroop stimuli to investigate effortless and effortful mental task conditions, the latter involving response competition or inhibition. The Stroop interference has also been used on the premises that it was known to probe heart rate variability (HRV) and to involve the response inhibitory function of the anterior cingulate cortex (ACC), and showed that autonomic nervous system (ANS) modulation by the ACC is closely related to the cognitive processing function of this structure [14]. Despite methodological variations in the presentation modalities of the Stroop task, this body of research has generated an overall reactivity trend when comparing rest values to values during the Stroop task: a steady and reproducible increase in heart rate (HR) and systolic blood pressure, increased SNS activity as indexed by plasmatic epinephrine and norepinephrine concentrations or muscle sympathetic nerve activity (MSNA), and a decrease in parasympathetic nervous system (PNS) activity as measured by the high frequency component of HRV and/or the transfer magnitude between instantaneous lung volume and HR. However, since few studies assessed the PNS component, PNS results are not as robust as SNS findings [12,14].

Only one study so far [12] assessed the respiratory responses, and observed a marked reactivity (increase of respiratory frequency (F_resp) and tidal volume (TV) from baseline to task). Despite numerous publications emphasizing the need to either control respiration, or treat it as a variable when reporting respiratory sinus arrhythmia (RSA), the majority of reports in cardiovascular psychophysiology still does not account for the respiratory component. As the focus historically relied on cardiovascular reactivity and its predictive potential for hypertension and coronary heart disease, the cardiac-vascular coupling seemed more relevant than the cardiac-respiratory coupling. However, several studies in respiratory physiology do describe reactivity in respiratory variables between rest and task [15,16], including a decrease in TV, an increase in F_resp, a decrease in RSA, and modified respiratory timings (increased ratio inspiratory time over total breath cycle; Ti/Ttot) and mechanics (increased involvement of thoracic versus abdominal breathing during task).

To further characterize the reactivity with regard to cardio-respiratory interactions, we performed a first experiment investigating the cardio-respiratory response to a sequence of cognitive tasks of varying difficulty, some of which included emotional material. We hypothesized that reactivity to this sequence of mental tasks would show the pattern previously described (i.e. an increase in HR, a PNS withdrawal as indexed by decreased RSA, and an increase in TV and F_resp), and would vary according to emotional load and task difficulty.

In a second experiment we replicated these investigations in a repeated measures design, once in a baseline condition, and once in a known real-life stressful situation. We thus focused on the difference in reactivity to mental tasks between a baseline condition and a stress condition, whereas previous studies mainly treated reactivity as a stable dimension. The difference between rest values for baseline and stress recordings was compared to the reactivity identified in the first experiment, and an eventual interaction between mental task reactivity and stress activation was explored.

2. Method

2.1. Experiment 1

2.1.1. Subjects

Student pilots ($N = 20$) from the EAT (Ecole d'Aviation de Transport) of the French Air Force, ranging in age from 20 to 25 (mean: 22.3 years) participated. All participants were medically fit to fly according to military standards, therefore showing no significant medical antecedents, and had normal or corrected-to-normal vision. They all completed their basic flight training (corresponding to approximately 65 flights) and were in the second third of the advanced flight training.

2.1.2. Procedure

Prior to cognitive testing, participants were equipped with the LifeShirt system. After a rest recording period of 5 min, each session began with on-screen instructions, followed by a series of 7 cognitive tests in the following order: a Stroop colour-word task with neutral words (S1-N) among the colour words on a white background, a Stroop task including emotional words (S1-E) among the colour words on a white background, a similar Stroop task with neutral words (S2-N) on a black background and a Stroop task with emotional words (S2-E) on a black background. Subsequently, two recognition tasks (Rec1 and Rec2) were presented, each including neutral and emotional words from the lists presented in the four previous tasks, as well as new words. The stimuli were counterbalanced between tasks, to control for potential order effects. The last test in the sequence was a numerical Stroop task (Num). The behavioural results of the recognition tasks will not be discussed in the present paper. After the testing protocol, participants were asked to rate task difficulty for each subtest on a ten-point scale. All experimental sessions were run at the same location and the same time of day, starting between 08.30 and 10.30. During the testing, subjects wore a headset to minimize possible noise disturbance.

2.1.3. Apparatus

Cardio-respiratory parameters were recorded through the LifeShirt system; data were analyzed on a personal computer with the VivoLogic software (Vivonoetics, Inc). A standard single lead ECG was recorded at 200 Hz and was later digitally upsampled to 1000 Hz for R-wave detection. Respiratory movements were measured by respiratory inductive plethysmography; abdominal and ribcage excursions were recorded at 50 Hz. The mechanical activity of the heart was measured through thoracocardiography (TCG), through a single inductive plethysmography sensor band at the level of the xiphoid process. Suppression of respiratory movements with digital filtering and ECG-triggered ensemble averaging (implemented in VivoLogic 2.9.3), yielded a signal validated as a surrogate measurement for the ventricular volume curve [17]. All data were visually inspected for artefacts; ectopic beats or erroneous R-wave detections were manually corrected (removal of erroneous detection/artefact followed by a cubic spline interpolation; corrections $<1\%$).

Through a derivative based algorithm R-waves were detected, and RR intervals calculated. F_resp, TV, and RSA (peak-valley method) were computed. As variation in TV across experimental conditions was more relevant than absolute volume values per se, the Qualitative Diagnostic Calibration (VivoLogic 2.9) was applied to individual data-files. The stroke volume (SV) was calculated as the maximum-minimum of the ventricular volume curve for each cardiac cycle.

Through customised Matlab routines, the TCG signal was used to calculate the Heather Index (HI), obtained by dividing the ejection velocity by the time interval between the onset of the electrical systole and the peak of the ejection velocity, also known as the Q-Z interval [18]. This required the detection of the ejection peak on the first time derivative of the TCG curve.

2.2. Experiment 2

2.2.1. Subjects

Student pilots ($N=12$) from the Belgian Air Force in their basic flight training, aged 19 to 25 years (mean = 22.5), all medically fit to fly and free of significant medical antecedents, with normal vision, participated.

2.2.2. Procedure and apparatus

Replication of Experiment 1, in a repeated measures design: the baseline recording took place after approximately one third of the flight training, the recording under stressful conditions just before the Progress Test General Flying (PTGF), the major evaluation flight known for its stress load for student pilots. Both recording sessions were separated by a minimum of 2 and a maximum of 5 months.

3. Results

3.1. Experiment 1

3.1.1. Task ratings

The recognition tasks were rated as the most difficult ones (*M*: 6.53; *SD*: 1.46 and *M*: 6.50; *SD*: 1.53 for Rec1 and Rec2 respectively) as compared to the Stroop tasks (*M*: 2.78; *SD*: 1.24 and *M*: 2.55; *SD*: 1.04 for S1-N/S1-E and S2-N/S2-E respectively); the numerical Stroop task was rated as the easiest one (*M*: 2.00; *SD*: 0.90).

3.1.2. Physiological results

Sequences of 2 min were extracted from the recordings of the rest period and the different subtests (rest, S1-N, S1-E, S2-N, S2-E, Rec1, Rec2). This choice of 2 min sequences was made in accordance to the guidelines defined for the psychophysiological use of heart rate variability [19], i.e. a sequence long enough to allow for the computing of all frequency components, and as short as possible to minimize non-stationarity.

The difference rest-task revealed a decreased RRI, an increased *F*_resp, an increased Ti/Ttot, as well as a decrease in RSA. After this initial reactivity, however, all parameters evolved towards rest values. No effect of task switching, nor of additional task difficulty was observed. Fig. 1 delineates the concurrent variation of the different parameters, depicted as proportion of rest values. The initial reactivity, and the lack of any additive effects from task switching, task difficulty, or the presentation of emotional words, is manifest. Despite the absence of significant differences between subtests, there is a tendency in the data to return towards rest values after the initial reactivity.

A repeated measures MANOVA, with Test as the within-subject factor, showed a significant effect of Test [$F(7,148)=15.83$; $p<0.001$; $\eta^2=0.463$]. Univariate ANOVAs of the different parameters only showed significance for F_resp [$F(7,148)=10.93$; $p<0.001$; $\eta^2=0.341$], Ti/Ttot [$F(7,148)=2.52$; $p=0.018$; $\eta^2=0.106$] and RSA [$F(7,148)=5.92$; $p<0,001$; $\eta^2=0.219$], but not for RRI [$F(7,148)=1.64$; $p=0,128$], nor for TV [$F<1$]. Subsequent contrast analysis for *F*_resp, Ti/Ttot and RSA confirmed significance between baseline recordings and test recordings [*F*_resp ($p<0,001$); RSA ($p<0.001$); Ti/Ttot ($p=0.006$)]. None of the differences between cognitive tests reached significance. The reactivity response, described as the activation between rest and the first test, thus singled out as a major effect. To further investigate this reactivity, a separate MANOVA was performed on the first two sequences (rest and S1-N). The significant effect of Test was confirmed [$F(1,38)=7,77$; $p<0.001$; $\eta^2=0.629$], with significance for RRI [$F(1,38)=5.04$; $p=0.031$; $\eta^2=0.117$], *F*_resp [$F(1,38)=43.43$; $p<0.001$; $\eta^2=0,533$], Ti/Ttot [$F(1,38)=5.2$; $p=0,028$; $\eta^2=0.120$] and RSA [$F(1,38)=11.57$; $p=0.002$; $\eta^2=0.233$], but not for TV [$F<1$], SV [$F(1,38)=1.62$; $p>0,1$] or HI [$F(1,38)=1.75$; $p>0,1$]. The data for RRI, *F*_resp and RSA are summarized in Table 1.

Table 1 and Fig. 1 illustrate that variations of RSA and *F*_resp are concomitant. As respiratory variations are repeatedly argued to account for major effects in RSA variations, an ANOVA for RSA was performed, introducing *F*_resp as a covariate [20]. This reduced the effect of test on RSA to only approach significance [$F(7,148)=1.96$; $p=0.064$], which corroborates the assertion that large reactivity in RSA mainly depends on breathing frequency.

Analysis of the physiological results in Experiment 1 thus revealed that reactivity between rest and first test recordings is the only factor to show significant variation, as illustrated in Fig. 1. Furthermore, this reactivity is only significant for cardio-respiratory variables, not for RSA corrected for respiration, nor for both markers of sympathetic activity.

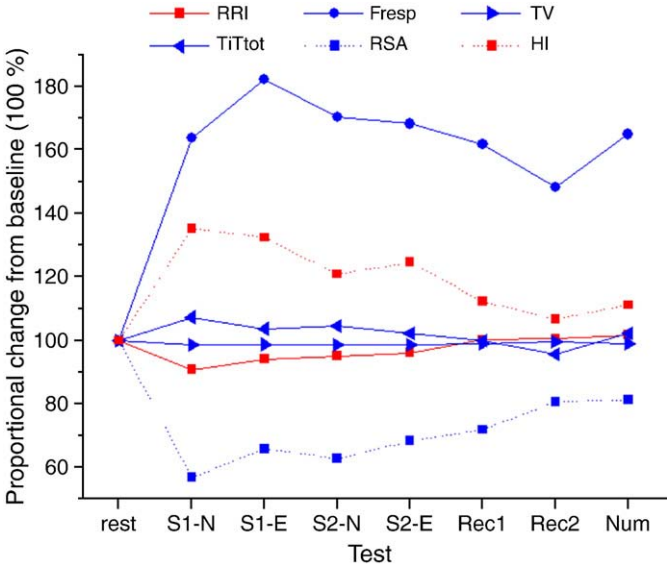

Fig. 1. Evolution of RRI, *F*_resp, TV, Ti/Ttot (full lines), and RSA and HI (dotted lines), expressed as proportional changes from baseline values (100%). This figure indicates the interdependency of the different parameters and their closely linked reactivity pattern.

3.2. Experiment 2

3.2.1. Task ratings

As in Experiment 1, the recognition tasks were rated as the most difficult ones (*M*: 6.14; *SD*: 0.69 and *M*: 5.71; *SD*: 1.11 for Rec1 and Rec2 respectively) as compared to the Stroop tasks (*M*: 2.07; *SD*: 0.84 and *M*: 2.07; *SD*: 0.84 for S1-N/S1-E and S2-N/S2-E respectively), and the numerical Stroop task (*M*: 2.50; *SD*: 0.65).

3.2.2. Physiological data

Due to inconsistencies in the TCG traces for these recordings, SV and HI data are not reported. As in Experiment 1, 2-min sequences

Table 1
Experiment 1: Summary of means and standard deviations for RR-interval (RRI), breathing frequency (*F*_resp) and respiratory sinus arrhythmia (RSA) throughout the sequence of subtests. All data were computed based on 2 min segments.

	Rest	S1-N	S1-E	S2-N	S2-E	Rec1	Rec2	Num
RRI (ms)	870	786	816	824	830	869	871	876
	126	110	105	110	105	115	114	110
*F*_resp (min^{-1})	12.6	20.6	20.1	19.4	19.6	18.7	17.4	19.1
	4.4	3.2	3.1	3.7	2.9	2.9	3.1	3.6
RSA (ms)	108.5	61.5	71.6	68.1	71.8	76.4	85.2	85.0
	82.7	18.9	20.9	35.0	19.2	22.8	27.0	24.6

were extracted from the recordings of the different subtests (rest, S1-N, S1-E, S2-N, S2-E, Rec1, Rec2 and Num).

Mean values and respective standard deviations for RRI, *F*_resp and RSA are reported in Table 2. As in Experiment 1, analysis of the difference between rest recordings and the first test (S1-N) revealed initial reactivity, expressed as a decreased RRI, increased *F*_resp, increased Ti/Ttot ratio, and decreased RSA. After this initial reactivity, a steady evolution is again evidenced for all parameters. The presentation of emotional words did not elicit any effect, nor did task difficulty or task switching.

The difference between baseline and pre-exam recordings substantiated through two different effects. First, the mean rest values differ, most markedly for RRI (772 ms for the pre-exam versus 851 ms for the baseline recording) and for RSA (133 ms for the pre-exam versus 188.7 ms for the baseline recording), indicating a higher activation during the pre-exam session. Second, the magnitude of the initial reactivity due to cognitive test presentation decreases in the pre-exam condition, most markedly for RRI.

A repeated measures 8 (Test) * 2 (Session) MANOVA showed a significant effect of both Test [$F(7,143)=28.73$; $p<0.001$; $\eta^2=0.584$], and Session [$F(1,143)=3.8$; $p=0{,}003$; $\eta^2=0.12$], but no interaction [$F(7,143)<1$]. The univariate ANOVAs for Test showed a significant effect for *F*_resp [$F(7,143)=17.59$; $p<0{,}001$; $\eta^2=0.463$] and for RSA [$F(7,143)=9.01$; $p<0.001$; $\eta^2=0.306$], as in Experiment 1. The univariate ANOVAs for Session showed a significant effect for RRI [$F(1,143)=11.17$; $p=0.001$; $\eta^2=0.072$] and for RSA [$F(1,143)=10.35$; $p=0.002$; $\eta^2=0.067$]. Separate MANOVAs for the baseline and pre-exam recordings showed similar effects of Test, respectively [$F(7,71)=16.97$; $p<0.001$; $\eta^2=0.626$] and [$F(7,71)=14.91$; $p<0.001$; $\eta^2=0.592$], with univariate ANOVAs for both showing significant variations for *F*_resp and RSA.

Again, the data showed no effect of presentation of emotional words, no effect of task difficulty or of task switching, neither during the baseline nor during the pre-exam session. These results thus replicated findings from Experiment 1: the main effect in the physiological data is the initial reactivity between rest recordings and recordings during presentation of the first task, with a tendency to return to rest values along time-on-task.

According to these results, Session (baseline versus pre-exam) and Test (rest versus mental task) produced different effects on physiological activation. This conflicts with literature describing cardiovascular effects of stress to be similar for long duration real-life exposure and mental test aspects. Findings from Experiments 1 and 2 showed the effect of Test expressed mainly on respiratory parameters (*F*_resp and the concomitant variation of RSA), whereas Experiment 2 showed the effect of Session to be mainly expressed on cardiac parameters (RRI and RSA).

Despite the lack of significant interaction between Session and Test, the initial reactivity in RRI shows a clear difference between baseline and pre-exam recordings. The difference in reactivity is most clearly visualised when RRI values (Table 2) are expressed proportionally, relative to rest. In baseline recordings, the initial reactivity in RRI represents a mean decrease of 10% versus a decrease of 2.3% in stressful pre-exam recordings.

As interindividual differences might have blurred this effect, an issue repeatedly acknowledged in psychophysiological research (for a review, see [21]), a within-subject standardisation was applied. The range-correction procedure [22] was thus applied to RRI for initial reactivity data, allowing standardisation for individual differences in baseline and ranges. Each individual's score (A) was expressed in function of his/her own minimum value (X) and his/her own range, defined as the difference between maximal (Y) and minimal value ($Y-X$). Each score A was subsequently replaced by the value $(A-X)/(Y-X)$, expressing values for each subject in a range between 1 and 0. Fig. 2 represents these values and depicts a marked decrease in rest values during the pre-exam session, and a decreased reactivity from rest to test. A 2 (Session) * 2 (Test) repeated measures ANOVA revealed a significant effect of both Test [$F(1,10)=22.38$; $p=0.001$; $\eta^2=0.72$] and Session [$F(1,10)=4.29$; $p<0.01$; $\eta^2=0.39$], and a significant interaction between Session and Test [$F(1,10)=10.62$; $p=0.01$; $\eta^2=0.54$].

4. Discussion

In order to characterize cardio-respiratory reactivity associated with a sequential presentation of mental tasks of varying difficulty, some of which included emotionally loaded material, physiological parameters were measured in both a baseline condition, and in a known stressful real-life situation. Reactivity was evidenced to be a phasic, transient phenomenon, showing a tendency towards resetting over time-on-task (about 20 min), with no additional effect of emotional load or task difficulty. The most important effect was observed for respiratory parameters. The major decrease in RSA from rest to task seemed mainly dependent on breathing frequency, thus rather pointing towards a different cardio-respiratory coupling, than to a major vagal withdrawal. With respect to a phasic conceptualisation of reactivity, exposure to novelty has been shown to be the discriminant feature in eliciting cardiac reactivity to mental challenges, before habituation occurred [23]. In a 45 min Stroop task, energy mobilisation has been investigated, and - contrary to expectations - a decrease of blood glucose over time-on-task was observed, paired to a decrease in HR [13]. These findings endorse adaptability to varying conditions (and thus to varying demands) as the main function of the ANS. A rapid habituation in the presence of a constant stimulation suits a phasic reactivity concept, but challenges the usual interpretation of cardiac reactivity as a linear measure of workload/stress/task difficulty. This phasic conceptualisation was also supported by other authors [24], studying muscle sympathetic nerve activity (MSNA), systolic and diastolic blood pressure, and HR while performing a computerised modified Stroop task, varying over six

Table 2
Experiment 2: Summary of means and standard deviations for RR-interval (RRI), breathing frequency (*F*_resp) and respiratory sinus arrhythmia (RSA) throughout the sequence of subtests, for the baseline and the exam condition. All data were computed based on 2 min segments. Results from the exam condition are highlighted.

	Rest	S1-N	S1-E	S2-N	S2-E	Rec1	Rec2	Num
RRI (ms)	851	765	803	795	807	841	877	828
baseline	111	127	110	109	96	110	115	120
RRI (ms)	772	753	747	754	758	779	774	770
exam	111	107	98	95	98	120	103	99
F_resp (min^{-1})	9.4	19.1	17.6	17.9	17.9	15.3	16.0	17.6
baseline	2.5	3.1	3.4	4.1	3.1	2.5	2.9	2.9
F_resp (min^{-1})	10.4	19.8	18.1	17.1	17.4	16.0	15.4	17.5
exam	2.5	2.5	2.2	4.1	3.3	2.1	2.8	4.3
RSA (ms)	188.7	62.9	77.0	67.2	69.2	82.4	95.1	73.8
baseline	110.0	35.3	42.0	34.5	32.7	49.8	59.0	46.1
RSA (ms)	133.0	41.3	44.1	57.0	55.0	56.4	61.8	54.5
exam	100.2	21.1	21.3	42.5	43.4	28.3	34.8	40.3

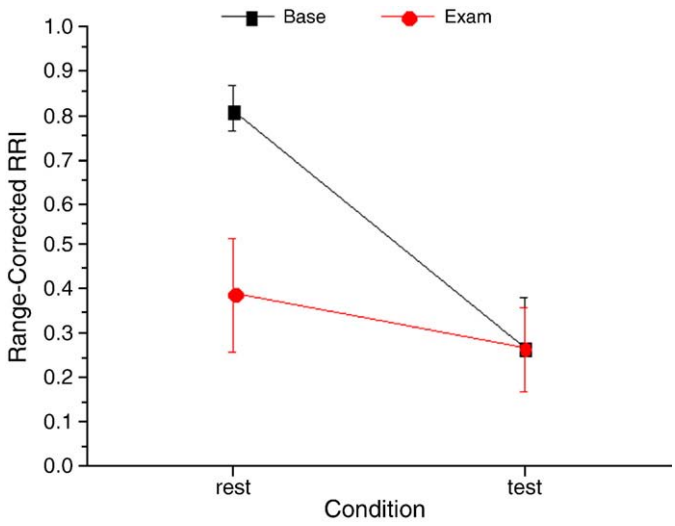

Fig. 2. Range-corrected RRI variation between base and test recordings, for baseline and pre-exam sessions. The decreased reactivity of RRI in the pre-exam sessions is clearly depicted.

levels of difficulty. Subjective ratings of perceived stress, performance decrement and systolic blood pressure showed linear increases from baseline over the increasing levels of difficulty. HR showed a significant initial reactivity from rest to the first level of task difficulty, but remained constant thereafter for 30 min (5 min of each difficulty level). In a study on cardiac and RSA responses associated with video-game performance in a threat (electric shock), and a no threat condition [25], an interaction between presentation order and threat levels on HR responses was observed. Whereas HR showed a larger reactivity in the threat condition when it was presented as the first task, the HR remained constant when subjects performed the threat condition as a second task.

Most studies linking HR variance to task difficulty are based on single reactivity measures (rest-test), to avoid confounding order or time-on-task effects. Heart period might well be sensitive in this initial reactivity, but not when the difficulty levels vary in subsequent recordings. Only one set of results [26] challenged this hypothesis, as HR showed to be both sensitive and diagnostic, showing an initial reactivity from rest to task, but also a consistent increase with task difficulty in a continuous 1 h recording during a flight simulator task.

In summary, whereas numerous reports indicated variations of HR in function of task difficulty, many failed to evidence such relationship when taking into account the dynamics of reactivity. Definition of cardiac reactivity as a phasic concept therefore proves more plausible than as a mere indicator of workload/task difficulty.

A second research question related to variation of the physiological response to the protocol in stressful conditions. An overall effect of Session on rest recordings, considered to be an effect of stress, was expressed as an increased HR and decreased RSA, supporting earlier findings (e.g. [27,28]). Interestingly, whereas reactivity of RSA to mental task emerged mainly related to breathing frequency, the differences between baseline RSA and pre-exam resting RSA recordings were not related to differences in breathing parameters; they thus reflected a true decrease of parasympathetic tone. Therefore, as our findings suggested, the relevance of respiratory parameters in reactivity research deserves specific emphasis. Indeed, breathing frequency showed to be the most sensitive parameter (more than heart period) in response to mental tasks. As this variation accounts for most of the reactivity of RSA in response to task, the literature, for decades advocating the use of RSA as a more sensitive workload indicator than HR alone [29], might just have been overlooking the main variable.

Our results clearly evidenced a differentiation in physiological responses: whereas the critical variable in response to mental task was breathing frequency, paired to heart period for the initial reactivity, the effect of real-life stress was evidenced on heart period and RSA. This conflicts with the conception of reactivity as an aspecific response, that could be elicited by any stimulation and be reliable across situations. A multidimensional concept of reactivity is not new [30]. A body of research on various behavioural demands differentiated between activation in response to challenges requiring active coping (e.g. mental tasks) and tasks requiring passive coping (e.g. the cold pressor). For instance, cold pressor responses have been distinguished [20] from physiological reactions to emotion induction, handgrip, and mental arithmetics. The stressors imposed in the present study (cognitive tasks and pre-exam stress) could qualify as requiring active coping. Pre-exam stress in this respect might be expected to act on a more tonic level, with stress as a constant throughout - but not induced by - the experimental procedure, whereas the mental task stress could be viewed as induced by the protocol, and acting on a more phasic level.

Besides the effect of stress on resting values, our results also indicated an interaction between the stress during the pre-exam session and cardiac reactivity to a mental task. According to these results, stress not only affected rest levels, but also the dynamic range of heart period reactivity. Stress research repeatedly targeted a reliable measure for cardiovascular reactivity (e.g. [8]), and data interpretations most often pointed towards a dispositional model of cardiovascular reactivity, implying reactivity to be a stable characteristic for each individual. Our results, to the contrary, showed that heart period reactivity varies as a function of the situation.

While the sequencing of baseline and stress-condition recordings, and the specific task sequence, set the stage for unraveling the phasic and tonic components in reactivity patterns, inherent limitations are inevitable. Most studies to date tried to differentiate reactivity for different cognitive modalities using different types of presentations, comparing each task to a preceding rest, whereas we explicitly presented a sequence of tasks without resetting. In fact, if HR and HRV responses are prospected as measures of task difficulty and mental effort in applied settings (e.g. [31–33]), investigating the sensitivity of these indicators in a sequential task presentation is important. The present protocol offered this possibility to investigate the dynamics of reactivity, by introducing its evolution over time. Absence of task switching effects underpinned our presumption that the main feature of reactivity is a rather aspecific arousal response, unaffected by additive effects of different cognitive functions, varying levels of difficulty or emotional content.

Furthermore, the observation, in the pre-exam session, that RRI showed both a lower baseline value and a lower reactivity, might hint towards interpretation of these results in the framework of Law of Initial Values (LIV) [34]. The LIV concept implies that magnitude and direction of response of a physiological function depend to a large extent on its initial (pre-experimental) level. “The higher the initial value, the smaller the response to function-raising, the higher the response to function-depressing stimuli” [34]. At present, the statistical and physiological validity of LIV are still under debate. It has been argued that the frequency with which the LIV appeared in psychophysiological research can be attributed to the amount of random error present in the data [35]. However, potential validity of the LIV concept has important technical implications in psychophysiological research. If the observations of rest and test values are dependent, statistical issues may arise, and notions of floor and/or ceiling effects may be applicable. Furthermore, as previously emphasized, physiology is not linear by nature, and thus requires specific procedures to investigate initial level dependencies. A *t*-statistic testing procedure has been introduced to detect probable presence of LIV dependency in psychophysiological data sets [34]. LIV is considered to have a bearing when rest variance is larger than test

variance, resulting in a significantly positive *t*-value; lack of significance would point towards the overlap hypothesis, implying absence of any relation [34]. If, on the other hand, test variance is larger than rest variance, meaning the *t*-value is significantly negative, the Fanspread Hypothesis is accepted, also described as Reactivity [35] or anti-LIV [36].

The LIV dependency was investigated in the data from both experiments. For Experiment 1, the test revealed LIV to be applicable for RRI, *F*_resp, Ti/Ttot and RSA; the Overlap Hypothesis for TV and SV; and the Fanspread Hypothesis for HI. For Experiment 2 however, the test displayed LIV only to be applicable to Ti/Ttot and RSA, for both baseline and pre-exam recordings. The difference in reactivity between rest and test recording thus cannot be reduced to an expression of LIV.

5. Conclusion

In summary, our findings first showed respiratory parameters to be more sensitive to the presentation of mental task than cardiac reactivity, and to account for the major decrease in RSA in response to task presentation. Simultaneous assessment of respiration in HRV studies thus proves mandatory, as repeatedly acknowledged (e.g. [37]). Second, cardio-respiratory reactivity showed sensitive to mental task but not diagnostic in a sequential task presentation, a finding bearing specific implications in applied workload studies. Third, reactivity to mental task presentation and real-life stress elicited distinct response patterns. Real-life stress response was evidenced through two different effects. The mean values for the rest recordings differed, showing in the stress condition a marked decrease in heart period and RSA, unrelated to changes in respiration, and therefore indicating vagal withdrawal. Furthermore, a real-life stress context reduced the magnitude of the initial reactivity in heart period to the presentation of cognitive tasks, implying that stress not only affects rest levels, but also the dynamic range of heart period. These findings suggest two divergent concepts of reactivity to stress. Reactivity to mental task, which can be conceived as a phasic response, was mainly characterized by breathing frequency and to a lesser extent heart period, whereas the effect of pre-exam stress, which can be conceived as a tonic response, is evidenced on heart period and RSA. Moreover, heart period responses revealed an interaction between these phasic and tonic activations, herewith endorsing the conceptualisations of a phasic arousal component and a tonic activation component as systemic outcomes. These findings challenged the concept of reactivity as an aspecific response, that could be elicited by any stimulation and be reliable across situations.

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