The continuum of consciousness in cardiovascular stress research : an experimental expedition

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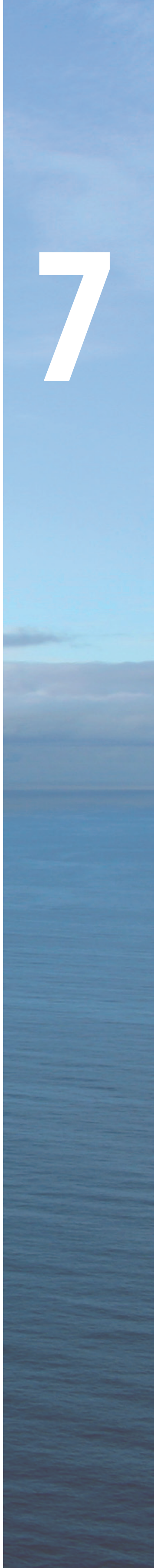
Chapter 7



Automatic vigilance is associated with impaired cardiovascular recovery from recalling emotional memories

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Abstract

Self-report outcomes insufficiently explain the relationship between psychological stress and cardiovascular (CV) health. Implicit measures may provide new tools to assess the individuals' affective state beyond self-report and contribute to the understanding of processes outside of awareness in psychosomatic research. We tested whether an emotional Lexical Decision-making Task (LDT), as implicit measure of automatic vigilance, was related to slow CV recovery after a stress induction. Participants performed an angry (n = 24) or happy recall task (n = 30), followed by a self-report of their affective state and the LDT. This generated an index of automatic vigilance for negative (AVI-N) and positive information (AVI-P). CV activity was measured throughout the experiment. Lower self-reported happiness and a higher AVI-N were found in the anger recall condition compared with the happy recall condition. There were no differences in CV activity between conditions and the LDT subscales were not related to CV reactivity. However, irrespective of condition, higher AVI-N levels were associated with a generally higher diastolic blood pressure during recovery and lower AVI-P levels were associated with slower recovery of systolic blood pressure, heart rate, and total peripheral resistance. Importantly, self-reported affect was not related to CV reactivity or recovery. Thus, automatic vigilance for negative information is increased by an anger recall task and related to diastolic blood pressure recovery after emotional recall. Using the LDT as implicit measure of psychological stress could advance research on the relationship between psychological stress and CV disease by addressing processes outside of awareness.

Psychological stress, such as work stress, marital discourse, or worrying, has been related to an increased risk of the development or worsening of CV disease (e.g., 3,5,6,9,12,93,115, 263-266). Despite the abundance of research on this topic, the relationship remains poorly understood (e.g., 8,9). This may, partly, be a result of the methods to assess psychological stress, which are usually self-report measures of for example work stress, worry, anxiety, or negative affect (22-25,292,293). More specifically, self-report measures are likely to be insufficient since individuals may not (always) be capable of reflecting on their psychological state (e.g., 28,37). The amount of explained variance of CV responses to stressors based on self-report measures remains unsatisfactory (22-29,293). Moreover, part of the psychological stress response may occur outside of awareness, that is, beyond self-report, which could activate physiological responses that may lead to adverse consequences for one's health. This is referred to as unconscious stress (26,27). Thus, regarding the detrimental effects of psychological stress on CV health, the explanatory potential of measures beyond self-report has yet to be evaluated.

In various fields of psychology, measures have been developed to assess constructs beyond self-report, referred to as implicit measures. These measures assess psychological constructs that do not require deliberate processing by the individual (74), such as implicit stereotyping (66), affective evaluation (47), decision making (42), and job-related attitudes (81). In a previous study, we have use an implicit measure of affect, the Implicit Positive And Negative Affect Test (IPANAT; 28), which assesses affect through the process of affect infusion (see also 83,86). We found that the IPANAT subscales were related to the systolic blood pressure (SBP), heart rate variability (HRV), and total peripheral resistance (TPR) reactivity, and SBP and diastolic blood pressure (DBP) recovery, in addition to the self-report measure of affect after a mental arithmetic task (202). Although the IPANAT seems to be appropriate as implicit measure of affect, changes induced by psychological stress may be present at other levels of psychological processing as well, such as processing of emotional stimuli as shown in subliminal priming studies (e.g., 132,203). Here, we conducted a study with a different implicit measure, the Lexical Decision-making Task (LDT), which is believed to assess the cognitive activation of information of the construct measured (66,81,89). The LDT could be an appropriate measure of psychological stress outside of awareness, as it taps into the cognitive processes that are active at the moment of assessment, in other words, it should be able to assess negative affectivity induced by a stressor. Measuring the activation of this stress-related cognition and relating the outcomes to CV indices would not only indicate that implicit measures can provide additional information on psychological stress relative to CV activity, but would also support the idea of unconscious stress.

In a LDT, participants have to indicate whether a string of letters is a 'word' or a 'nonword' (81,89). Based on the construct of interest, construct-congruent and incongruent responses are expressed in indexes of reaction times (RTs). Here, we used an emotional LDT in which the 'words' are negative, positive, or neutral. The accurate responses are averaged for all categories. The negative and positive average RTs are corrected for the neutral RTs, resulting in an automatic vigilance index for negative (AVI-N) and positive (AVI-P) information, respectively (90,314,315). Faster responses to one of these categories are thought to indicate greater neural accessibility (66). During a negative psychological state, such as psychological stress, the representation of negative information is activated. Consequently, accessibility of the negative concepts is enhanced, which would lead to a quicker perception and processing of the negative stimuli (e.g., 81). This has been found in relation to negative affective states such as anxiety and depression (316-321). Moreover, the LDT has been previously used as implicit measure of performance-related cognition after a cognitive challenging task (90) and was found to be related to the recovery of heart rate (HR), but not HRV. In that particular study, the word categories of the LDT were related to intelligence and positive characteristics, rather than to negative affectivity, and BP was not studied. Here, we will induce psychological stress to instigate the cognitive processing of negative information and relate the outcomes of the emotional LDT (i.e., using negative and positive words) with BP, HR, HRV, and TPR.

We used an anger recall task to induce psychological stress and measured the concurrent CV responses before, during, and after the recall. The procedure was based on a study by Gerin and colleagues (23) who found impaired CV recovery after the anger recall in participants with high levels of trait rumination. In general, anger recall procedures have been used to study the effect of psychological stress on CV activity (e.g., 93,322,323). However, these studies have not included a control for the anger induction part of the procedure and we cannot state with certainty that previous results are mainly due to the anger inducing aspect of the studies, rather than more general procedural (and perhaps also stressful) characteristics such as providing a stranger with personal information. Thus, we included a happy recall condition to control for these latter effects. Furthermore, impaired recovery from a stressor is thought to be most detrimental for health (e.g., 13,88). Therefore, we have focused on the course of the CV activity after the stressor. We expected an increase in SBP, DBP, HR, TPR, and a decrease in HRV during the task and an impaired recovery to baseline of these outcomes. Regarding the implicit measure, we expected that a higher AVI for negative affective information and a lower AVI for positive affective information would occur in the anger recall group, relative to the happy recall group, and that these responses would be related to increased CV reactivity and slower CV recovery, and that this relationship would be, at least partly, independent of self-reported affect.

Method

Participants

Participants (*N* = 61, *M* = 21.1, *SD* = 2.88) were recruited through an online registry system of Leiden University and received eight euro or course credits as a reward. Exclusion criteria were current (treatment of) psychological or/and CV health problems or/and the use of drugs that may influence CV activity. Participants were randomly assigned to the anger or happy recall condition as described below and provided informed consent before the start of the experiment. The study was approved by the Independent Ethics Committee of the Institute of Psychology of Leiden University (number 3145923676).

Instruments

As an implicit measure of psychological stress, a Dutch version of an emotional LDT (89) was provided. In the 64 trials, a string of letters was shown for 1000 ms and participants had to indicate as quickly and accurate as possible whether it constituted a 'word' or a 'nonword' on a keyboard. 'Words' consisted of eight positive (e.g., strong, intelligent), eight negative (e.g., unfair, hateful) words and 16 neutral words (e.g., sandwich, lamp), selected from the word-set of Hermans and De Houwer (255). All trials started with a fixation cross presented for 2000 ms and ended after a response. RTs and responses were recorded. For accurate responses only, outliers (>3**SD* of the overall mean RT) were excluded and averages for stimulus type (positive, negative, neutral, or nonword) were calculated. Increased activation of negative and positive information was determined by subtracting mean RT of the negative and positive trials from the neutral trials, respectively. The resulting AVI's for negative (AVI-N) and positive (AVI-P) information indicated higher activation with larger values.

As self-report measure of affect, a VAS was provided to indicate the effect of the manipulation. Participants were asked to what extent they felt a certain emotion (e.g., 'How annoyed are you at this moment?') for twelve emotions (i.e., joyful, cheerful, happy, annoyed, irritated, angry, afraid, frightened, scared, sad, gloomy, unhappy). Answers were given on a horizontal line of 10 cm at the bottom of the screen, with zero indicating 'not at all' and 100 indicating 'very much'. Scores were averaged into self-reported anger, happiness, sadness, and fear, but only self-reported anger and happiness were used in the analyses. Cronbach's α's were sufficient with .83 for both self-reported anger and happiness.

Several questionnaires on trait and personality were provided to control for any group differences. The Dutch version of the State-Trait Anxiety Inventory, Trait version (STAI-T) was provided to test the tendency to experience all situations as threatening (280). This self-report questionnaires contains 20 items rated on a 4-point Likert scale and has a good internal consistency and validity (280). In this sample the Cronbach's α was high with .90. The Dutch version of the State-Trait Anger Expression Inventory,

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trait version (324) was used to assess the tendency to display anger. The questionnaire contains 10 items rated on a 4-point Likert scale and has a good reliability and validity (324). In this sample, the Cronbach's α was high with.82. The inability or difficulty to name, and express emotions and the tendency to direct attention externally was measured with the Toronto Alexithymia Scale (TAS-20; 325). The scale has been shown to be valid (326) and in this sample the Cronbach's α was moderate with .64.

SBP and DBP (mmHg) were measured with the Portapres Model-2 (Finapres Medical Systems, Amsterdam, The Netherlands), which uses a noninvasive method to measure BP. To assess HR (bpm) the electrocardiogram was recorded with Kendall® 200 Covidien electrodes at a sample rate of 200 Hz with BIOPAC MP150 (Biopac Systems, Goleta, CA, USA). To monitor data quality during acquisition and correct artifacts Acqknowledge 3.9.1.4 was used. The data were extracted with a tailor made toolbox in Matlab R2012b, which applied a low-pass filter (20 Hz, Blackman 40 coefficients) to the BP signal, upsampled the electrocardiogram to 1000 Hz and applied a comb filter (50 Hz, Q = 5). For HRV (95) the root mean square successive differences (RMSSD; ms) were calculated from the interbeat intervals. TPR (mmHg.min/L) was calculated using an approximation of cardiac output (CO; 229,230,302) and mean arterial pressure (189). From these estimations only the outcome measure of interest, TPR, is reported. The CV outcome measures were obtained continuously and averages were calculated over the last min of baseline, the entire recall phase (M = 368.3 s, SD = 68.8), and the 15 min of recovery per min.

Procedure

After being welcomed into the lab, the participants were informed on the procedure and provided informed consent before starting with the experiment. The participant was seated in a separate room from the experimenter, who could monitor movements and behavior through a one-way mirror. First, demographics and biobehavioral variables were obtained, the CV measures were placed according to protocol, and the quality of the signals were checked. All further instructions were displayed on the monitor of a computer using E-Prime 2.0.8.90. The baseline was a five min period during which participants could read a magazine.

For the emotional recall procedure, in line with previous research (23), participants wrote down three emotional events that occurred during the previous year. In the anger recall condition, participants were asked to recall events that had upset them and made them angry. They were encouraged to choose events that were not completely solved and still evoked a lot of anger when thinking about it. In the happy recall condition, participants were asked to write down events that made them happy and cheery. The events should still elicit happiness when thinking about them. In both conditions participants had to rate the evens for the experienced anger or happiness on a 7 point Likert scale. The participants were then instructed to select one of these memories to discuss with the experimenter. The experimenter went into the room when the participants indicated they were ready. The recall took about five min, allowing participants to finish thoughts beyond the specific time frame, during which they described in detail what happened, how it made them feel at the time, and their feelings at the time of the experiment on the issue. The experimenter did not show agreement or disagreement with the participant's statements, but merely nodded and maintained eye contact to encourage further elaboration. The experimenter left the room and the recovery started with a min during which participants did not perform any tasks and were instructed to remain seated for measurement purposes. After one min of recovery, the LDT and VAS were provided. Finally, the CV measures were detached and participants were debriefed.

Statistical analyses

Baseline differences between conditions in biobehavioral variables were analyzed with *t* tests and chi-square tests. Change scores were calculated for all physiological outcome measures to represent reactivity (238). The effect of the manipulation on the AVI's and VAS subscales and CV reactivity were analyzed with two-sided *t* tests and corrected for multiple comparison using the Benjamini Hochberg procedure (275-277), for which the false discovery rate was set at 10%. Effect sizes are expressed in *r* (239). Pearson correlations were calculated for the relationship between and amongst the AVI's and VAS subscales. Hierarchical multiple regression analyses were used to assess the relationship between condition, CV *reactivity*, and the AVI's and VAS subscales. Effect size was calculated using the spreadsheet by Lakens (182).

As main analyses, CV *recovery* was analyzed with multilevel modelling for all outcome measures separately (e.g., 279). Multilevel analyses (MLA) were used to assess the role of Condition (angry vs. happy) and the associations of AVI-N, AVI-P, VAS-anger, and VAS-happy with CV recovery throughout the 15 min of recovery (306). MLA it has various advantages over repeated measures ANOVAs when analyzing effects of time, such as a better handling of missing data and including individual slopes into the model and thus is able to consider multiple levels in the data (e.g., 279). The change in physiological responding over the 15 min was modelled with Condition, AVI-N, AVI-P, VAS-anger, and VAS-happy as predictors. CV baseline and reactivity were included as covariate in the model and were mean centered, as were AVI-N, AVI-P, VAS-anger, and VAS-happy. For each CV measure a separate model was built, but for all models Time was the level 1 variable (the measurements' course over 15 min) and Level 2 was the person level (all other predictors and covariates). Significant changes in the Akaike information criterion (AIC) and Bayesian information criterion (BIC), based on chi-square tests, were used to determine the model fit (279).

Models were built in the following order. First, the basic growth model was fit, which included the covariates, to model change over time. Here, Time was used as continuous predictor to model linear, quadratic, and cubic change (278). The best fitting covariance structure for the error variance was applied, which was either heterogeneous autoregressive, autoregressive, or diagonal, as is appropriate for fitting growth models (see for example 278). The additional value of BMI, smoking, gender, and relationship status was considered at this stage and included when it improved the model (based on the AIC and BIC). Finally, Condition was evaluated as a predictor, resulting in Model 1. Sequentially, we included AVI-N and AVI-P, and their interaction with Time and Time^2 (Model 2) to evaluate the association with CV recovery for each measure. In a similar way, the associations of CV recovery with VAS-anger and VAS-happy were evaluated (Model 3). Finally, all of these measures were added (Model 4) to evaluate the additional explanatory value of the implicit measure in addition to VAS subscales. The fit of each Model 2 and 3 was compared with Model 1. The fit of Model 4 was compared with one of the other models, depending on which had a better fit. All analyses were performed using SPSS 23.0.

Results[1]

Three participants were excluded based on the use of sympathomimetic drugs (salbutamol) and one participant was excluded due to extreme alcohol consumption (10 units in the 24 h before the experiment). In two cases, the experiment failed due to incorrect execution of the manipulation and in one case there were severe technical issues. The data for these participants were excluded from analyses. In several cases BP or HR recordings were of low quality, which led to smaller sample sizes for some

[1] We also performed a similar study, with the only difference being the implicit measure used. In this second study the LDT was replaced with the Morphing Faces Task (MFT, also known as the Facial Expression Recognition Task; 327). In this task, a series of faces morph from a neutral into a happy, angry, scared, or sad facial expression. Once participants recognize an emotion they stop the morph and have to identify the emotion. Faster responses to negative stimuli are thought to indicate an attentional bias, which has been found with the MFT in individuals with social anxiety and generalized anxiety disorders (e.g., 328,329). Antypa et al. (2011, 330) found that recent stressful events were related to earlier recognition of emotional expression of sadness and anger. It was expected that a faster recognition of negative emotion expressions and slower recognition of positive emotion expressions would occur in the angry recall condition, compared with happy recall condition, and that these responses were related to increased CV reactivity and slower recovery of CV responses in addition to self-reported affect. The final sample ($N = 48$, age $M = 21.2$, $SD = 2.29$, 79.2 % female) was randomly assigned to the angry ($n = 29$) or happy ($n = 19$) recall condition. However, the different subscales of the MFT (sad, anger, fear, happiness) were highly correlated ($r = .99$), there were no differences between conditions (ts < .50, ps> .70), and no relationship with CV reactivity or recovery was apparent (rs < .25, p > .05). This led to the conclusion that the task as executed here (see 330), was not a valid measure for this purpose and was considered inappropriate for measuring psychological stress beyond self-report.

outcome measures. Two participants showed deviating levels of RMSSD (> 3*SD) for which the data were considered to be missing at random. A square root transformation and a log transformation were applied to RMSSD and TPR, respectively. Furthermore, one outlier was found for the AVI-N for which the data were also labelled as missing and considered to be random. Finally, RT data of two participants with an accuracy below 70% were excluded. The final sample (N = 54) had a mean age of 21.1 (SD = 2.96) and consisted of 42 females (77.8 %). There were no differences between the angry (n = 24) and happy (n = 30) recall condition in baseline characteristics, see Table 1, except that in the anger recall condition more participants were in a relationship, compared with the happy recall condition. However, when checking for this variable in the main analyses it did not show any significant contribution to the model.

Self-reported affect and automatic vigilance

After the manipulation, in the anger recall condition VAS-anger (M = 19.2, SD = 19.7) was higher compared with the happy recall condition (M = 9.52, SD = 9.63), but this was not statistically significant, Mann-Whitney U = 256, Z = 1.81, p = .070, r = .25. However, VAS-happy was significantly lower in the anger recall condition (M = 64.7, SD = 15.1) compared to the happy recall condition (M = 74.4, SD = 11.6), $t(52)$ = 2.69, p = .010, r = .35. Furthermore, in the anger recall condition the AVI-N (M = 29.1, SD = 29.4) was larger compared to the happy recall condition (M = 3.34, SD = 50.0), $t(47.8)$ = -2.31, p = .025, r = .32 (corrected dfs since the equality of the variances could not be assumed). The AVI-P did not statistically differ between conditions (anger: M = 28.7, SD = 38.3; happy: M = 11.8, SD = 53.7, $t(50)$ = -1.26, p = .215, r = .18).

In addition, no statistically significant associations were found between the AVI's and the VAS subscales, rs < .30, ps > .05, and between the AVI's, $r(51)$= .27, p = .055, but a strong negative relationship was found between VAS-anger and VAS-happy, $r(54)$ = -.67, p < .001.

Cardiovascular reactivity

In both conditions changes from baseline during the manipulation were evident for SBP (ΔM = 14.0, SD = 10.7, $t(44)$ = 8.77, p < .001, r = .80), DBP (ΔM = 7.41, SD = 4.79, $t(44)$ = 10.4, p < .001, r = .84), HR (ΔM = 5.45, SD = 4.13, $t(51)$ = 9.51, p < .001, r = .80), and TPR (ΔM = -0.033, SD = 0.06, $t(44)$ = -6.20, p < .001, r = .68), but not for RMSSD (ΔM = 0.153, SD = 0.994, $t(49)$ = 1.09, p = .28, r = .15).

However, we found no statistical support for any differences between the conditions on SBP reactivity (anger recall: ΔM = 13.4, SD = 12.7; happy recall: ΔM = 14.4, SD = 9.36, $t(43)$ = -0.299, p = .77, r = .05), DBP reactivity (anger recall: ΔM = 6.82, SD = 5.09, happy recall: ΔM = 7.80, SD = 4.64, $t(43)$ = -0.669, p = .51, r = .10), HR reactivity (anger recall: ΔM = 4.81, SD = 3.32, happy recall: ΔM = 5.96, SD = 4.67, $t(50)$ = -0.992, p = .35, r = .14), RMSSD reactivity (anger recall: ΔM = 0.261, SD = 0.911, happy recall: ΔM =

0.062, *SD* = 1.07 *t*(48) = 0.701, *p* = .49, *r* = .10), and TPR reactivity (anger recall: Δ*M* = -0.030, *SD* = 0.034, happy recall: Δ*M* = -0.034, *SD* = 0.037 *t*(43) = 0.394, *p* = .70, *r* = .06).

TABLE 1 Biobehavioral characteristics stratified by condition

	Angry (*n* = 24)			Happy (*n* = 30)			
Measure	*M*	*SD*	*n*	*M*	*SD*	*n*	t/χ^2
Demographics							
Age, years	20.7	2.53	24	21.4	3.28	30	-0.89
Female sex[1]	18	(75)	24	24	(80)	30	0.19
BMI	22.0	3.07	24	22.2	2.65	30	-0.20
Dutch nationality[1]	24	(100)	24	30	(100)	30	n.s.
Biobehavioral variables							
Smoking[1]	4	(17)	24	2	(7)	30	1.35
Caffeine (test day)	0.21	0.51	24	0.27	0.58	30	-0.39
Alcohol use (glass/last 24h)	0.75	1.78	24	0.38	1.60	26	0.77
Relationship[1]	14	(58)	24	10	(33)	30	3.38^{+}
Cardiovascular measures							
SBP	128.0	18.1	21	124.4	14.2	28	0.77
DBP	68.7	11.0	21	67.3	10.6	28	0.46
HR	78.5	12.6	24	76.0	11.4	29	0.77
RMSSD[2]	35.4	19.7	24	39.6	22.3	27	-0.62
TPR[3]	10.0	2.81	21	10.3	2.30	28	-0.51
Personality							
Trait anxiety	38.3	8.63	24	37.1	9.96	29	0.46
Trait anger[4]	17.5	3.80	22	16.0	3.99	28	1.38
Alexithymia	47.0	7.56	22	48.1	7.19	28	-0.57

Note. There were no significant differences between conditions. *Abbreviations*: BMI = Body mass index, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR = Heart rate, RMSSD = Root mean square of successive differences, TPR = Total peripheral resistance.

[1] Displayed are the number of positive responses (percentage). The Pearson χ2 was used as test statistic.

[2] RMSSD was square root transformed. Untransformed *M*s and *SDs* are displayed.

[3] TPR was logarithmic transformed. Untransformed *M*s and *SDs* are displayed.

[4] One participant in the anger condition was excluded for the variable of trait anger, which normalized the data

CV reactivity and affect

To test the hypothesis that CV reactivity would be related to AVI-N and AVI-P, hierarchical regression analyses were conducted for each CV reactivity measure. In all the models condition was added at step 1 and VAS subscales at step 2. Since we expected that automatic vigilance would explain CV activity over and above VAS subscales, the

AVI's were added in step 3. Self-reported NA and PA were highly correlated, $r(54) = -.665$, $p < .001$, but VIF and tolerance were of acceptable levels in all tests and thus the assumption of multicollinearity was not violated (308). Adding BMI, smoking status, gender, or relationship status did not improve the model fit, ΔR^2s< .10, *p*s > .05, and models are reported without these variables. Changes in CV reactivity were not related to the AVI'S or VAS subscales, ΔR^2s <.15, *p*s > .10, see Table 2.

CV recovery

To model SBP recovery, a heterogeneous autoregressive covariance structure was applied to the error variance. The slope of Time was allowed to vary randomly between participants. Adding Condition to the Model did not improve the fit, nor did the addition of any of the covariates. Results are displayed in Table 3. There were significant associations of Time as well as Time^2, indicating that the recovery slope was composed of a linear decrease as well as a quadratic change (Model 1). The latter represented a trend with the fastest decrease at the beginning and a (small) increase in SBP towards the end of the recovery phase. The addition of AVI-N and AVI-P and their interactions with Time (Model 2) improved the model, $\Delta AIC = 183.7$ and $\Delta BIC = 167.0$, compared with Model 1. AVI-P×Time was positively associated with the recovery of SBP ($B = 0.005$, $t(41.8) = 2.13$, $p = .040$). Adding VAS-anger and VAS-happy and their interactions with Time to the model without the AVI's (Model 3) did not improve the model fit, $\Delta AIC = -1.6$ and $\Delta BIC = -19.5$, compared with Model 1, nor did a combination of the AVI's and VAS subscales (Model 4; $\Delta AIC = -2.3$ and $\Delta BIC = -20.2$) compared with Model 2. Thus, Model 2 provided the best fit to the data and indicates that a lower AVI-P was related to a slower linear decrease of SBP during recovery, that is slower recovery, but AVI-N and VAS subscales were not related to recovery of SBP.

To model DBP recovery, an autoregressive covariance structure was applied to the error variance. The slope of Time was allowed to vary randomly between participants. Adding Condition to the Model did not improve the fit, nor did the addition of any of the covariates. Results are displayed in Table 4. There was a significant association of Time, indicating that the recovery slope was composed of a linear decrease (Model 1). When adding AVI-N and AVI-P and their interactions with Time to the model (Model 2) the model fit improved, $\Delta AIC = 126.9$ and $\Delta BIC = 109.5$. Recovery of DBP was positively associated with AVI-N ($B = 0.04$, $t(37.9) = 2.75$, $p = .009$) and tended to be negatively associated with AVI-N×Time (but not statistically significantly so), $B = -0.002$, $t(42.8) = -1.95$, $p = .058$. Adding VAS-anger and VAS-happy and their interactions with Time to the model without the AVI's (Model 3) did not improve the model fit, $\Delta AIC = -5.8$ and $\Delta BIC = -23.8$, compared with Model 1, nor did a combination of the AVI's and VAS subscales compared with Model 2 (Model 4; $\Delta AIC = -6.5$ and $\Delta BIC = -24.4$). Thus, Model 2 provided the best fit to the data and indicated that, in contrast to our hypothesis,

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TABLE 2 Summary of hierarchical multiple regressions for the CV change scores during the emotion recall procedure

	SBP (mmHg)[a]			DBP (mmHg)[a]			HR (bpm)[b]			RMSSD (ms)[c]			TPR (mmHg.min/L)[a,d]		
	B	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	***β***
Constant	46.1**	14.2		21.0**	6.46		4.67	5.01		-1.46	1.25		-0.05	0.05	
Condition	-4.41	3.72	-.20	-2.38	1.70	-.24	-1.63	1.38	-.19	0.41	0.35	.21	0.01	0.03	.13
Self-reported anger	-0.22	0.15	-.30	-0.08	0.07	-.24	0.03	0.05	.12	0.008	0.01	.12	0.0002	0.001	.09
Self-reported happiness	-0.41*	0.18	-.53	-0.17	0.08	-.50	0.01	0.06	.04	0.02	0.01	.29	0.0002	0.001	.09
AVI-N	0.04	0.04	.14	0.006	0.02	.05	-0.02	0.02	-.19	-0.003	0.004	.12	-0.0001	0.0001	-.17
AVI-P	0.03	0.04	.14	0.02	0.02	.18	0.02	0.01	.23	-0.001	0.003	.07	-0.0001	0.0001	-.13
F		1.34			1.10			1.05			0.82			0.44	
R^2		.15			.13			.11			.06			.06	
ΔR^2		.04			.03			.06			.02			.05	

Note. The table shows the associations between condition, affect, and CV change scores as generated by the final model (step 3); Condition was added at step 1, self-reported NA and PA at step 2, and AVI-N and AVI-P at step 3 to indicate the additional value of the implicit measure**.** The *F* statistic refers to that of the final model. ΔR^2 is the difference in explained variance between step 2 and step 3, which represents the additional variance of the change scores explained by AVI-N and AVI-P. *Abbreviations*: AVI-N = Automatic vigilance index for negative words, AVI-P = Automatic vigilance index for positive words, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR = Heart rate, RMSSD = Root mean square of successive differences, TPR = Total peripheral resistance.

[a] $N = 43$.

[b] $N = 49$.

[c] $N = 47$, a square root transformation was applied.

[d] A logarithmic transformation was applied.

* $p < 0.05$, ** $p < 0.01$

a generally higher DBP during recovery was related to *higher* AVI-N. VAS subscales and AVI-P were not related to recovery of DBP.

To model HR recovery, an autoregressive covariance structure was applied to the error variance. The slope of Time was allowed to vary randomly between participants. Adding Condition to the Model did not improve the fit, nor did the addition of any of the covariates. Results are displayed in Table 5. There was a significant association of Time, $Time^2$, and $Time^3$ indicating that the recovery slope was composed of a general linear decrease, but also of a quadratic and cubic change (Model 1). Adding AVI-N and AVI-P and their interactions with Time (Model 2) improved the model, $\Delta AIC = 241.8$ and $\Delta BIC = 224.1$, compared with Model 1. AVI-P×Time was positively associated with the recovery of HR, $B = 0.002$, $t(47.1) = 2.36$, $p = .022$. Adding VAS-anger and VAS-happy and their interactions with Time to the model without the AVI's (Model 3) did not improve the model fit, $\Delta AIC = -0.4$ and $\Delta BIC = -18.9$, compared with Model 1, nor did a combination of the AVI's and VAS subscales (Model 4; $\Delta AIC = -3.6$ and $\Delta BIC = -21.9$) compared with Model 2. Thus, Model 2 provided the best fit to the data and indicated that a lower AVI-P was related to a slower recovery of HR during recovery, which is consistent with our hypothesis, and the VAS subscales were not related to recovery of HR.

To model recovery of RMSSD, a heterogeneous autoregressive covariance structure was applied to the error variance. The slope of Time was allowed to vary randomly between participants. Adding Condition to the Model did not improve the fit, nor did the addition of any of the covariates. Results are displayed in Table 6. There was a significant association of Time and $Time^2$, indicating that the recovery slope was composed of a general linear decrease and a quadratic change (Model 1). The addition of AVI-N and AVI-P and their interactions with Time to the model (Model 2) compared with Model 1 did improve the model fit, $\Delta AIC = 98.5$ and $\Delta BIC = 81.8$, but no individual predictors were significantly related to recovery of RMSSD. Adding VAS-anger and VAS-happy to the model without the AVI's (Model 3) did not improve model fit compared to Model 1, $\Delta AIC = -6.2$ and $BIC = -24.6$, nor did the combined addition of the AVI's and VAS subscales compared to Model 2 (Model 4: $\Delta AIC = -6.8$ and $BIC = -25$). Thus, although Model 2 provided the most optimal model fit for the data, the predictors were not significantly related to the recovery of RMSSD.

Finally, to model recovery of TPR, an autoregressive covariance structure was applied to the error variance. The slope of Time was allowed to vary randomly between participants. Adding Condition to the Model did not improve the fit, nor did the addition of any of the covariates. Results are displayed in Table 7. There was a significant association of Time and $Time^2$, indicating that the recovery slope was composed of a general linear decrease and a quadratic change (Model 1). The model fit improved when AVI-N and AVI-P and their interactions with Time were added (Model 2) compared with Model 1, $\Delta AIC = 142.4$ and $\Delta BIC = 159.8$. Recovery of TPR was positively associated with AVI-N, *B* = 0.0003, *t*(42.0) = 2.13, *p* = .039, and negatively with AVI-P×Time, *B* = -0.00002, *t*(42.9) = -2.42, *p* = .020. Adding VAS-anger and VAS-happy and their interactions with Time to the model without the AVI's (Model 3) slightly improved the model fit, Δ*AIC* = 1 and Δ*BIC* = 19.1, compared with Model 1. Recovery of TPR was positively (but not statistically significantly) associated with VAS-anger, *B* = 0.0007, *t*(43.5) = 1.68, *p* = .099. Furthermore, a combination of the AVI's and VAS subscales provided a slightly better fit compared with Model 2 (Model 4; Δ*AIC* = 0.9 and Δ*BIC* = 18.8). In this model 4, recovery of TPR was negatively related with AVI-P×Time, *B* = -0.00002, *t*(42.9) = -2.31, *p* = .026. The relationship with AVI-N was no longer statistically significant. Thus, Model 4 provided the most optimal fit to the data and indicates that participants with a lower AVI-P showed a slower recovery of TPR.

To summarize, for none of the CV outcome variables the model was improved by Condition, that is, there were no differences between the conditions in the slope of recovery as indicated by increasing, rather than decreasing, AIC's (Δ [0.31;73.9]) and BIC's (Δ [1.5;78.3]) compared to Model 1 for all variables. The basic models were most optimal when including the CV baseline and reactivity, that is, baseline and reactivity were predictive of the CV recovery slopes. Regarding the AVI's, across both conditions, lower levels of AVI-P were related to a slower recovery of, as indicated by significant AVI-P×Time interactions for SBP (*B* = 0.005, *t*(40.7) = 2.13, *p* = .040), HR (*B* = 0.002, *t*(47.1) = 2.36, *p* = .022), and TPR (*B* = -0.00002, *t*(42.9) = -2.31, *p* = .026). A higher AVI-N was only related to a generally higher DBP during recovery (*B* = 0.04, *t*(37.9) = 2.75, *p* = .009). See Figure 1 and 2. Notably, the VAS subscales were not related to CV recovery.

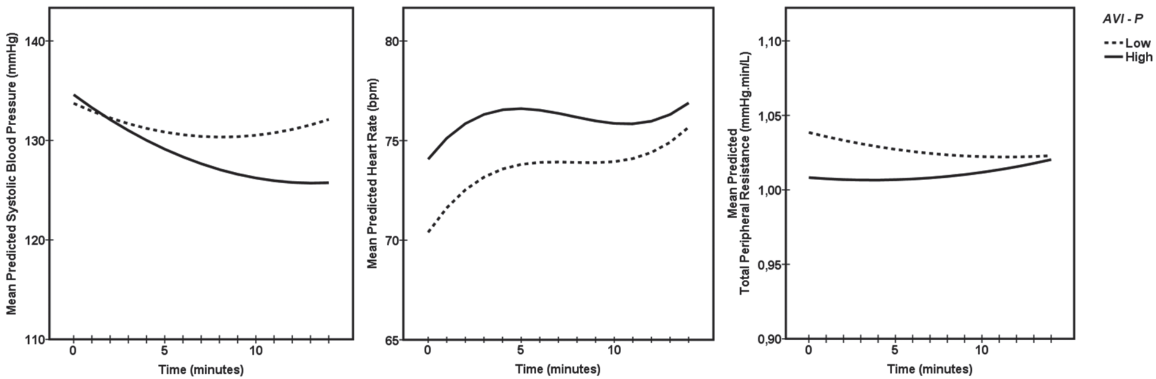

FIGURE 1 Mean predicted values of SBP, HR, and TPR over each of the 15 min of recovery from the emotion recall procedure displayed for low and high automatic vigilance for positive information (AVI-P), which was dichotomized for display purposes

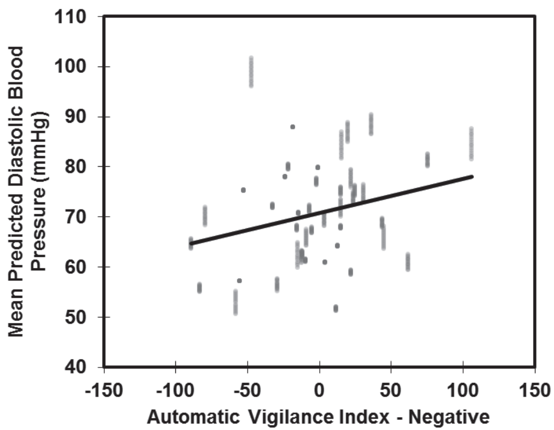

FIGURE 2 Mean predicted DBP during recovery in relationship with mean automatic vigilance for negative information (AVI-N). The grey spots display the raw values of the AVI-N to indicate the spread

TABLE 3 Summary of multilevel analysis for recovery of SBP (mmHg)

	Model 1			Model 2			Model 3			Model 4		
Predictor	*B*	*SE*	*t*	*B*	*SE*	*t*	*B*	*SE*	*t*	*B*	*SE*	*t*
Constant	135.2	1.57	86.2***	135.0	1.55	87.1***	135.4	1.48	91.7***	135.1	1.46	92.7***
Time	-1.05	0.29	-3.63***	-1.09	0.30	-3.42***	-1.10	0.29	-3.81***	-1.12	0.30	-3.76***
$Time^2$	0.05	0.02	2.71**	0.05	0.02	2.79**	0.05	0.02	2.88**	0.05	0.02	2.88**
Baseline SBP	0.80	0.08	10.7***	0.83	0.08	10.9***	0.84	0.07	11.5***	0.87	0.07	11.8***
Reactivity SBP	0.37	0.11	3.34**	0.38	0.11	3.42**	0.39	0.11	3.51**	0.41	0.11	3.68***
AVI-N				0.04	0.04	1.12				0.05	0.03	1.38
AVI-P				-0.03	0.03	-0.98				-0.03	0.03	-1.00
Time×AVI-N				-0.003	0.003	-0.95				-0.003	0.003	-1.02
Time×AVI-P				0.005	0.002	2.13*				0.005	0.002	1.87+
VAS-anger							-0.30	0.12	-2.42*	-0.25	0.12	-2.10*
VAS-happy							-0.17	0.13	-1.34	-0.07	0.12	-0.54
Time×VAS-anger							0.02	0.01	1.86+	0.01	0.01	1.42
Time×VAS-happy							0.02	0.01	1.60	0.008	0.01	0.68
AIC		4275.2			4091.5			4276.8			4093.8	
BIC		4383.6			4216.6			4403.1			4236.8	
N		24			28			28			32	

Note. Error at Level-1 was organized with a heterogeneous autoregressive first-order covariance structure. At Level-2 the covariance was unstructured. Predictors were grand mean centred. When interactions with $Time^2$ were added to Model 2 and 3, the Models did not provide a better fit and the interactions were omitted from Model 3 and 4. Model 2 shows the best fit and was used to interpret the data. *Abbreviations*: SBP = Systolic blood pressure, AVI-N = Negative automatic vigilance index, AVI-P = Positive automatic vigilance index, VAS = Visual analogue scale, AIC = Akaike information criterion, BIC = Bayesian information criterion, *N* = Number of parameters.

$^{+}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

TABLE 4 Summary of multilevel analysis for recovery of DBP (mmHg)

	Model 1			Model 2			Model 3			Model 4		
Predictor	*B*	*SE*	*t*	*B*	*SE*	*T*	*B*	*SE*	*t*	*B*	*SE*	*t*
Constant	71.5	0.62	116.0***	71.7	0.59	121.5***	71.5	0.61	117.9***	71.7	0.58	123.1***
Time	-0.09	0.05	-1.99+	-0.10	0.05	-2.16*	-0.09	0.05	-2.01+	-0.10	0.05	-2.19*
Baseline DBP	0.90	0.05	19.3***	0.88	0.05	19.0***	0.90	0.05	19.3***	0.89	0.05	19.1***
Reactivity DBP	0.51	0.11	4.85***	0.52	0.10	5.00***	0.51	0.11	4.68***	0.51	0.11	4.81***
AVI-N				0.04	0.02	2.75**				0.04	0.02	2.75**
AVI-P				-0.02	0.01	-1.29				0.01	0.01	1.00
Time×AVI-N				-0.002	0.001	-1.95+				-0.002	0.001	-1.91+
Time×AVI-P				0.001	0.001	1.49*				0.001	0.001	1.14
VAS-anger							-0.06	0.06	-1.05	-0.06	0.06	-1.08
VAS-happy							-0.07	0.06	-1.12	-0.05	0.06	-0.74
Time×VAS-anger							0.005	0.004	1.19	0.004	0.004	1.02
Time×VAS-happy							0.006	0.004	1.41	0.004	0.005	0.86
AIC		3095.4			2968.5			3101.2			2975.0	
BIC		3136.0			3026.5			3159.8			3050.9	
N		9			13			13			17	

Note. Error at Level-1 was organized with an autoregressive first-order covariance structure. At Level-2 the covariance was unstructured. Predictors were grand mean centred. Model 2 shows the best fit and was used to interpret the data. *Abbreviations*: DBP = Diastolic blood pressure, AVI-N = Negative automatic vigilance index, AVI-P = Positive automatic vigilance index, VAS = Visual analogue scale, AIC = Akaike information criterion, BIC = Bayesian information criterion, *N* = Number of parameters.

$^{+}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

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TABLE 5 Summary of multilevel analysis for recovery of HR (bpm)

	Model 1			Model 2			Model 3			Model 4		
Predictor	*B*	*SE*	*t*	*B*	*SE*	*T*	*B*	*SE*	*t*	*B*	*SE*	*t*
Constant	72.8	0.49	107.0^{***}	72.8	0.49	148.5^{***}	72.8	0.48	150.7^{***}	72.9	0.49	148.7^{***}
Time	1.30	0.23	5.58^{***}	1.32	0.24	5.48^{***}	1.30	0.23	5.59^{***}	1.31	0.24	5.47^{***}
$Time^2$	-0.18	0.04	-4.62^{***}	-0.18	0.04	-4.52^{***}	-0.18	0.04	-4.63^{***}	-0.18	0.04	-4.52^{***}
$Time^3$	0.008	0.002	4.21^{***}	0.008	0.002	4.14^{***}	0.008	0.002	4.22^{***}	0.008	0.002	4.14^{***}
Baseline HR	0.94	0.03	27.6^{***}	0.94	0.04	26.5^{***}	0.94	0.03	27.5^{***}	0.94	0.04	26.4^{***}
Reactivity HR	0.48	0.10	5.03^{***}	0.48	0.10	4.90^{***}	0.48	0.10	4.99^{***}	0.48	0.10	4.88^{***}
AVI-N				0.0009	0.01	0.10				-0.0003	0.01	-0.03
AVI-P				-0.01	0.009	-1.41				-0.01	0.01	-1.18
Time×AVI-N				0.0005	0.001	0.52				0.0003	-0.001	0.34
Time×AVI-P				0.002	0.001	2.36^{*}				0.002	-0.001	1.77^{+}
VAS-anger							-0.03	0.03	-0.80	-0.02	0.04	-0.667
VAS-happy							-0.03	0.04	-0.73	-0.02	0.04	-0.48
Time×VAS-anger							0.009	0.003	2.74^{**}	0.007	0.003	2.15^{*}
Time×VAS-happy							0.009	0.004	2.46^{*}	0.007	0.004	1.65
AIC		3991.7			3749.9			3992.1			3753.5	
BIC		4042.9			3818.8			4061.8			3840.7	
N		11			15			15			19	

Note. Error at Level-1 was organized with a heterogeneous autoregressive first-order covariance structure. At Level-2 the covariance was unstructured. Predictors were grand mean centred. When interactions with $Time^2$ and $Time^3$ were added to Model 2 and 3, the Models did not provide a better fit and the interactions were omitted from Model 3 and 4. Model 2 shows the best fit and was used to interpret the data. *Abbreviations*: HR = Heart rate, AVI-N = Negative automatic vigilance index, AVI-P = Positive automatic vigilance index, VAS = Visual analogue scale, AIC = Akaike information criterion, BIC = Bayesian information criterion, *N* = Number of parameters.

$^{+}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

TABLE 6 Summary of multilevel analysis for recovery of HRV (RMSSD, ms)

	Model 1			Model 2			Model 3			Model 4		
Predictor	*B*	*SE*	*t*	*B*	*SE*	*T*	*B*	*SE*	*t*	*B*	*SE*	*t*
Constant	6.42	0.10	63.4***	6.40	0.10	66.4***	6.42	0.10	63.3***	6.40	0.10	66.1***
Time	-0.08	0.03	-2.88**	-0.07	0.03	-2.70**	-0.08	0.03	-2.87**	-0.07	0.03	-2.68**
Time^2	0.003	0.002	1.94+	0.003	0.002	1.67+	0.003	0.002	1.92+	0.003	0.002	1.67+
Baseline HRV	0.88	0.04	20.4***	0.92	0.04	24.2***	0.88	0.04	20.6***	0.92	0.04	24.4***
Reactivity HRV	0.27	0.08	3.62***	0.35	0.07	5.19***	0.28	0.08	3.74***	0.36	0.07	5.29***
AVI-N				-0.002	0.002	-1.16				-0.002	0.002	-1.23
AVI-P				0.0003	0.002	0.21				0.0005	0.002	0.26
Time×AVI-N				0.00002	0.0003	0.11				0.00002	0.0003	0.07
Time×AVI-P				-0.0001	0.0002	-0.66				-0.00008	0.0002	-0.40
VAS-anger							-0.001	0.007	-0.20	0.002	0.007	0.25
VAS-happy							-0.002	0.008	-0.20	-0.001	0.008	-0.12
Time×VAS-anger							-0.0006	0.0008	-0.83	-0.0004	0.0008	-0.50
Time×VAS-happy							-0.0008	0.0008	-0.88	-0.0006	0.001	-0.60
AIC		1546.3			1447.8			1552.5			1454.6	
BIC		1657.0			1575.2			1681.6			1600.2	
N		24			28			28			32	

Note. Error at Level-1 was organized with a heterogeneous autoregressive first-order covariance structure. At Level-2 the covariance was unstructured. Predictors were grand mean centred. When interactions with Time^2 were added to Model 2 and 3, the Models did not provide a better fit and the interactions were omitted from Model 3 and 4. Model 2 shows the best fit and was used to interpret the data. RMSSD was square root transformed. *Abbreviations*: HRV = Heart rate variability, RMSSD = Root mean square successive differences, AVI-N = Negative automatic vigilance index, AVI-P = Positive automatic vigilance index, VAS = Visual analogue scale, AIC = Akaike information criterion, BIC = Bayesian information criterion, *N* = Number of parameters.

$^{+}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

TABLE 7 Summary of multilevel analysis for recovery of TPR (mmHg.min/L)

	Model 1			Model 2			Model 3			Model 4		
Predictor	*B*	*SE*	*t*	*B*	*SE*	*T*	*B*	*SE*	*t*	*B*	*SE*	*t*
Constant	-0.76	0.10	-7.74^{***}	-0.73	0.10	-7.53^{***}	-0.77	0.09	-8.27^{***}	-0.73	0.09	-8.07^{***}
Time	-0.002	0.001	-1.76^{+}	-0.002	0.001	-1.78^{+}	-0.002	0.001	-1.77^{+}	-0.002	0.001	-1.79^{+}
Time^2	0.0001	0.00007	1.95^{+}	0.0001	0.00007	1.86^{+}	0.0001	0.00007	1.95^{+}	0.0001	0.00007	1.85^{+}
Baseline TPR	0.89	0.05	18.4^{***}	0.87	0.05	18.2^{***}	0.89	0.05	19.2^{***}	0.88	0.05	19.4^{***}
Reactivity TPR	0.27	0.08	3.62^{***}	0.54	0.14	3.98^{***}	0.51	0.13	3.81^{***}	0.53	0.13	4.19^{***}
AVI-N				0.0003	0.0001	2.13^{*}				0.0002	0.0001	1.81^{+}
AVI-P				0.00007	0.0001	0.62				0.0001	0.0001	1.21
Time×AVI-N				-0.00001	0.00001	-1.11				-0.00001	0.00001	-0.98
Time×AVI-P				-0.00002	0.00001	-2.42^{*}				-0.00002	0.00001	-2.31^{*}
VAS-anger							0.0007	0.0004	1.68^{+}	0.0005	0.0004	1.18
VAS-happy							-0.00001	0.0005	-0.20	-0.0004	0.0005	-0.96
Time×VAS-anger							-0.00005	0.00004	-1.33	-0.00004	0.00004	-0.96
Time×VAS-happy							-0.00004	0.00004	-1.01	-0.000008	0.00004	-0.20
AIC		-2967.7			-2825.3			-2966.7			-2824.4	
BIC		-2922.6			-2762.8			-2903.5			-2744.0	
N		10			14			14			18	

Note. Error at Level-1 was organized with an autoregressive first-order covariance structure. At Level-2 the covariance was unstructured. Predictors were grand mean centred. When interactions with Time^2 were added to Model 2 and 3, the Models did not provide a better fit and the interactions were omitted from Model 3 and 4. Model 4 shows the best fit and was used to interpret the data. TPR was log transformed. *Abbreviations*: TPR = Total peripheral resistance, AVI-N = Negative automatic vigilance index, AVI-P = Positive automatic vigilance index, VAS = Visual analogue scale, AIC = Akaike information criterion, BIC = Bayesian information criterion, *N* = Number of parameters.

$^{+}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

Discussion

This study set out to test whether a measure beyond self-report of psychological stress, that is, an implicit measure, should be employed to add explanatory power in the psychological stress and CV responses relationship. We explored the potential explanatory role of the emotional LDT as implicit measure of psychological stress in CV recovery from a stressor. As the LDT is assumed to measure automatic vigilance, representing the cognitive activation of information (66,81,89), higher levels of automatic vigilance for negative information (the AVI-N index) and lower levels of automatic vigilance for positive information (the AVI-P index) would indicate psychological stress beyond self-report and relating these findings to CV activity may provide support for the unconscious stress hypothesis. The findings indicate that the CV activity during and after an anger recall procedure to induce psychological stress, as well as after a happy recall procedure as control condition, was related to AVI-N, AVI-P, and self-reported affect. During the recall task, SBP, DBP, and HR increased, and TPR decreased relatively to the baseline, but HRV did not. In other words, in contrast to the expectations there were no differences in CV reactivity between conditions. Automatic vigilance for negative, but not for positive, information was stronger in the anger recall condition compared with the happy recall condition, but the implicit measure was not related to CV reactivity. CV recovery was also similar across conditions. However, stronger automatic vigilance for negative information was found to be related to a general higher DBP during recovery, but not to change over time. It was not related to SBP, HR, HRV, or TPR. In contrast, lower automatic vigilance for positive information was related to a slower recovery of SBP, HR, and TPR, but not to recovery of DBP or HRV. Importantly, self-reported affect was not related to the CV reactivity nor to recovery, although it was related to condition in the expected direction, that is, more negative affect (albeit statistically nonsignificant) and less positive affect after the anger condition compared to the control condition. Thus, the emotional LDT appears to detect cognitive processes related to stress-induced CV recovery in addition to self-report. We will discuss these results in more detail below.

In contrast to our expectations, we did not find differences in CV reactivity and recovery between conditions. Both conditions elicited an increase in SBP, DBP, and HR, and a decrease in TPR and the level of reactivity was related to the progress of recovery. Previous studies did not include a control condition (e.g., 23,93,322,323) and based on the current findings it can now be questioned whether it was the induced anger (or reduced positive affect for that matter) or the procedure itself that elicited changes in CV reactivity. This is further stipulated by the absence of a statistical significant difference between conditions in self-reported anger. On the other hand, these findings are similar to those in our previous study (202), in which there was also an absence of CV differences between a standard anger-induction procedure (math with harassment), and a logical control (math without harassment) that previous studies using this anger provocation did not use. These findings call for more rigorous and consistent use of control conditions in psychosomatic research of the physiological effects of psychological stress.

Furthermore, we expected an increase but found a decrease of TPR throughout the experiment. An increase in TPR is assumed to represent threat, which one would expect to occur in response to a stressor (99,100). The decrease of TPR would then indicate challenge, meaning that the participants are likely to have experienced the emotional recall procedure as a challenge. One explanation is the nature of the sample, which consisted mainly of psychology students who may well be fond of discussing emotional content and could try to do this to their best effort, but it may also be that the study information provided in advance reduced the threatening nature of the manipulation. To our knowledge, TPR in relation with psychological stress has not been previously addressed with an anger recall procedure and the current findings should therefore be replicated in a similar but also in different samples. The absence of an effect on HRV reactivity is also in line with our previous study (202), which may indicate that in this healthy sample participants could regulate their affective state adequately. This is further stipulated by the relationship of AVI-P with SBP, HR, and TPR, as we will discuss below.

In line with our expectations, differences in self-reported affect was apparent in the anger recall condition compared with the happy recall condition, that is, participants reported more, statistically nonsignificant, self-reported anger and less self-reported happiness. However, these findings were not related to CV activity and may be the result of procedural characteristics of the study. Furthermore, in line with our expectations automatic vigilance for negative, but not for positive, information was higher in the anger recall condition compared with the happy recall condition. Importantly, this activation of negative information was related to elevated DBP during recovery. However, activation of positive information was found to be related to faster recovery of SBP, HR, and TPR. As there was no effect of condition on this positive subscale of the LDT, it seems likely that a general activation of AVI-P was present that resulted in adaptive CV responses to psychological stress. A similar effect of positive affectivity measured at an implicit level has been found earlier in studies with the IPANAT, where it was related to a lower cortisol excretion and faster CV recovery (85,202). All in all, these findings suggest that implicit measures, both the negative and positive indexes, explain CV recovery in addition to self-reported affect and stipulate the additional value of implicit measures in CV stress research. Implicit measures such as the LDT and IPANAT appear to able to detect parts of the *core* affect (310) that are not necessarily reflected by self-report but may ultimately be co-determinants in the etiology of CV disease.

Some limitations of this study have to be considered. First, no baseline measurement was performed for the self-report and implicit measures. Consequently, no firm statements can be made on pre-existing psychological states and their influence on the current findings. However, these measurements were intentionally left out to prevent carry-over effects on the measures and possible priming effects of the emotion-related words, but further studies on the validation of the LDT would need to include a baseline measure. Second, the duration of the LDT was on average five min, but the recovery phase lasted 15 min. This means that participants had to sit and wait quite some time for the recovery phase to be finished. Despite our efforts to make sure that the participants were comfortable, this may have led to, for example, boredom. Finally, since participants chose the situation they wanted to tell the experimenter, it could be that they did not pick out the situation that elicited the strongest emotions. In an effort to respect the privacy of the participants, the emotion recall may not have been as strong as expected.

Conclusion

The current study suggests that the LDT, measuring automatic vigilance, is related to CV recovery after an emotional recall procedure to induce psychological stress, in addition to self-reported affect. Not only do these findings emphasize the prospective explanatory capabilities of implicit measures, it also highlights the role of processes outside of awareness in CV activity which may negatively contribute to the worsening or development of CV disease and provides further evidence for the unconscious stress hypothesis.