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Cardiovascular and electrocortical markers of anger and motivation during a simulated driving task

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ABSTRACT

The experience of anger may have consequences for the long-term health of the individual. The psychophysiological manifestation of anger can vary in response to the motivational context of anger provocation. The current study was designed to investigate how motivational context (challenge vs. threat) influenced the cardiovascular system and frontal EEG asymmetry. 29 male participants completed a simulated driving journey with a fixed time schedule. Anger was induced by exposing participants to traffic delays at an early (challenge) and later point (threat) on the simulated route. A number of dependent variables were recorded, including 32 channels of EEG, measures of cardiovascular impedance, blood pressure and fEMG activity from the corrugator supercilii. The results indicated that traffic delays significantly increased blood pressure, heart rate, TPR and corrugator activity whilst reducing the relative level of left frontal activation in the EEG. However, there was little evidence for a consistent distinction between the early (challenge) and late (threat) introduction of traffic delay. The consequences of these findings for capturing the cardiovascular and electrocortical responses to anger induction are discussed.

1. Introduction

The experience of anger is associated with specific cognitions (e.g. perceived injustice), increased cardiovascular activation and a behavioural disposition towards aggression (Al'Absi and Bongard, 2006). There is evidence that repeated episodes of anger have consequences for the health of the individual, particularly with respect to the development of coronary heart disease (CHD) (Everson-Rose and Lewis, 2005) and hypertension (Everson et al., 1998), although this relationship is mediated by trait variables (Davis et al., 2000) and expressive style (Vella and Friedman, 2009).

The autonomic manifestation of anger is characterised by sympathetic activation in conjunction with a higher respiration rate (Kreibig, 2010). This autonomic pattern has been confirmed using several protocols designed to induce anger in the laboratory. Autobiographical recall of anger-provoking events (Hamer et al., 2007; Prkachin et al., 2001; Sinha et al., 1992) produced a pattern of increased blood pressure in combination with accelerated heart rate (HR), greater cardiac output (CO) and higher total peripheral resistance (TPR); these changes coincided with a decline of both left ventricular ejection time (LVET) and pre-ejection period (PEP). This pattern is characterised by alpha- and beta-adrenergically mediated changes in the cardiovascular system (Kreibig, 2010; Stemmler et al., 2007).

The actual provocation of anger in the laboratory presents a number of methodological and ethical challenges, but this approach is associated with greater ecological validity compared to introspective methods. For example, Stemmler et al. (2001) exposed participants to a demanding task where a high rate of failure was combined with a provocation from the experimenter. These authors reported significant shortening of PEP (i.e. the time interval between onset of ventricular depolarisation and opening of aortic valve) and LVET (i.e. the time from the opening to the closing of the aortic valve) in combination with increased diastolic blood pressure (DBP), but with no accompanying changes in TPR. Herrald and Tomarka (2002) had participants endure an interview with an experimenter who made several demeaning remarks and gestures designed to elicit anger. They reported a pattern of increased heart rate and contractility in combination with relatively low vascular resistance. Therefore, anger may be expressed via divergent patterns of cardiovascular activity across different protocols for anger induction.

Variability with respect to the autonomic correlates of anger may originate from the influence of non-emotional factors within the evaluative context of anger provocation. Stemmler et al. (2007) have argued that the psychophysiological measurement of emotion represents an aggregation of three components: (1) a non-psychological contribution to the physiological response (e.g. motor activity,

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temperature), (2) an emotion-specific component associated with somatovisceral adaptation, and (3) a component associated with those cognitive and motivational requirements associated with the emotion context. With respect to the latter, Stemmler et al. (2007) studied the influence of motivational disposition (i.e. approach vs. avoidance) on psychophysiological measures of anger. A comparison between anger in the context of approach and avoidance revealed that anger/approach was associated with higher HR and shorter PEP and LVET compared to the anger/avoidance condition. In addition, EMG activity in the corrugator supercilii muscle, which has been captured as marker of negative affect (Larsen et al., 2003), was significantly higher in the anger/approach scenario, i.e. the implicit expression of anger via facial musculature was higher in the context of motivational approach.

The influence of motivational disposition on the experience of anger has been studied with respect to frontal EEG asymmetry; see Harmon-Jones et al. (2010) for recent review. Previous EEG research revealed greater activation of the left frontal hemisphere during induction of angry mood (Wacker et al., 2003), exposure to insult (Harmon-Jones and Sigelmann, 2001) and social rejection (Harmon-Jones et al., 2009), however, there is evidence to suggest that this relationship is strongly moderated by motivational context. Harmon-Jones (2003) reported an association between left frontal activation and anger where recourse to action was available; however, Zinner et al. (2008) found greater right frontal activation in a social context where anger expression was socially undesirable.

The motivational context of anger induction is an important influence on the psychophysiological manifestation of anger at the cortex as well as the cardiovascular system. This conceptual division between approach/avoidance bears some correspondence to the biopsychosocial model of challenge and threat proposed by Blascovich and colleagues (Blascovich et al., 1999; Blascovich and Mendes, 2000). According to Blascovich et al. (1999), a state of challenge stimulates sympathetic-adrenomedullary activity so myocardial contractility is elevated leading to increased cardiac output in combination with vasodilation, hence blood pressure remains unchanged. An experience of threat is associated with pituitary-adrenocortical activity, inhibiting the release of adrenaline and producing a pattern where blood pressure increases whilst peripheral resistance declines. This model has been the subject of some debate (Blascovich et al., 2003; Wright and Kirkby, 2003), however, a recent study into the psychophysiology of social support/rejection (Koslov et al., 2011) supported the model using measures of cardiovascular impedance in combination with frontal EEG asymmetry. These authors reported that baseline levels of frontal EEG asymmetry predicted cardiovascular reactivity in response to anger during the experience of social rejection. If participants had greater left hemispheric activation at baseline, they tended to respond to social rejection as a challenge, i.e. increased cardiac output but no change in blood pressure. However, those participants who exhibited greater right hemispheric activation at baseline perceived social rejection as a threat, i.e. cardiac output was stable but blood pressure increased.

The current study was conducted to investigate the relationship between motivational context and the psychophysiological manifestation of anger within a simulated driving task. Participants were systematically frustrated by exposure to traffic delays strategically placed in the simulated journey to induce state of anger/approach/challenge and anger/avoidance/threat. Our primary hypothesis was to investigate how measures of cardiovascular reactivity and frontal EEG asymmetry responded to both challenge and threat. Earlier research was used to generate specific predictions, i.e. challenge would lead to greater left frontal activation in conjunction with increased cardiac output and no change in blood pressure, whereas threat would be associated greater right frontal activation in combination with increased blood pressure. In addition, we wish to investigate whether motivational markers of approach/avoidance were correlated across cardiovascular and electrocortical measures as indicated by Koslov et al. (2011).

2. Method

2.1. Participants

Twenty-nine male participants (mean age = 25.5 yrs., s.d. = 7.5 yrs.) were recruited via advertisements posted on the campus. Participants had no history of psychiatric illness or cardiovascular problems, were not currently taking any medication, and scored below the 80th population percentile on the Trait Anger Expression Inventory of the STAXI 2 (Spielberger, 1999). The latter restriction was included to reduce the likelihood of the researcher being exposed to aggressive or abusive behaviour during the study. All procedures for participant recruitment and data collection were approved by the University Ethical Committee prior to commencement of the study.

2.2. Simulated driving task

A simulated car journey was prepared using a STI SIM Driving Simulator software (STI Inc.). This PC-based software allowed interaction via a steering wheel/pedals console and the driving scene was projected onto a large screen (approximately 3.66 m × 4.57 m), yielding a visual angle of approximately 80°. The simulated journey consisted of a two-lane roadway passing through countryside and urban settings. The route took approximately 10 min to complete if participants travelled at the maximum speed that was permitted.

A clock was visible next to the simulated scene and participants were instructed to complete the journey within 15 min in order to earn the £20 (approximately $32/22€) payment for participation in the study. The deadline was presented to the participants within the context of a scenario, they were told that the purpose of the journey was to collect a child from school. The precedence of completing the journey on time was reinforced by providing feedback of journey progression via three instances of pre-recorded verbal messages, e.g. “five miles remaining,” “three miles remaining” and “one mile remaining.” If the driver crashed the vehicle more than twice, they were told they would lose their total participant payment. In addition, speeding warning were in operation and participants were informed that they would be fined by £1 if they broke the speed limit or committed a driving violation such as overtaking where lane marking indicated that passing other vehicles was prohibited.

The infrastructure of the simulated route included a number of bends, crossroad intersections with stop lines and several sets of traffic lights. The driver encountered a low level of traffic density in both lanes with two exceptions; after approximately 2 min of the journey had elapsed, participants encountered the first traffic jam where extremely slow moving traffic in the lane was combined with high density traffic in the opposite lane, hence participants remained ‘trapped’ in the first traffic jam for 4 min. At a later point in the simulated journey (after approximately 12 min of driving), participants encountered a second traffic jam, identical to the first, that persisted for 5 min. The combined delay introduced by both traffic jams made it impossible for the participants to reach the destination within the required 15 min. There was a short two-minute section at the end of the drive when the traffic jam had cleared and participants reached the school.

It should be noted that the threats to withhold participant payment or enforce speeding fines were a deception and all participants were fully debriefed when the experiment had been completed.

2.3. Experimental design

The experiment was designed to compare psychophysiological activity during both early and the late traffic jam (TJ) sections. The early traffic jam (TJ1) occurred after only 2 min of the simulated journey and introduced a four minute delay; we assumed that most participants would feel that it was still possible to complete the journey on time at this point, hence the first traffic jam presented a *challenge* to

the goal of completing the journey on schedule. However, the timing of the late traffic jam (TJ2) in combination with the previous delay meant that participants effectively had no opportunity to complete the journey within the required schedule, therefore, the second traffic jam represented a source of *threat*. The traffic jam scenarios were also selected because they involved a minimum level of physical activity that would be comparable to the baseline condition.

2.4. Experimental measures

2.4.1. Subjective questionnaires

The state scale of the State-Trait Anger Expression Inventory 2 (STAXI 2) (Spielberger, 1999) was administered to participants before and after the simulated journey. This scale is designed to capture the subjective experience of anger. The Confidence and Perceived Control scale from the Dundee Stress State Questionnaire (DSSQ) (Matthews et al., 1997) was also completed by participants before and following the simulated journey. This sub-scale from the DSSQ measures the level of confidence (in one's own ability to perform a task adequately) and the level of perceived control over performance.

2.4.2. Cardiovascular Impedance

Cardiovascular activity was recorded using band electrodes placed on the back of the neck and the thorax (Sherwood et al., 1990). These signals were processed via the NICO100C Noninvasive Cardiac Output Module (BIOPAC Systems Inc.) in conjunction with the MP150 data recording system (BIOPAC Systems Inc.). This module delivered impedance magnitude (Zo) and derivatives (dZ/dt) at 1000 Hz. The impedance signals were analysed using an algorithm developed in our own laboratory[1] in order to detect the following measures for each cardiac cycle: Pre-Ejection Period (PEP), Cardiac Output (CO), Stroke Volume (SV), Left Ventricular Ejection Time (LVET).

2.4.3. Heart rate and blood pressure

The Inter-Beat Interval (IBI) from the heart was calculated from an ECG signal filtered between 0.5 and 0.35 Hz and sampled at 1000 Hz. This signal was collected via a two-lead electrode sensor connected to the TEL100C data capture signal (BIOPAC Systems Inc.) that also worked with the MP150 system (i.e. the ground signal for the ECG was obtained from the cardiovascular impedance apparatus). Blood pressure was measured using a CARESCAPE Vital Signs Monitor (V100) (DINAMAP Inc.) which involved placement of an inflatable cuff on the upper left arm. Readings of systolic blood pressure, diastolic blood pressure, heart rate and Mean Arterial Pressure (MAP) were all obtained using the oscillometric method (note: for the purpose of analysis, heart rate was measured from the ECG trace obtained via the MP150, not the CARESCAPE monitor). Recordings from the CARESCAPE were taken during every 2 min of the journey and coincided with the middle point of each traffic jam. Total Peripheral Resistance (TPR) at baseline and during both traffic jams was calculated by combining information from the CARESCAPE and the NICO100C, i.e. TPR = MAP/CO*80.

[1] An algorithm was developed to detect significant points (e.g. *Q, B, X*) in the impedance signals and ECG based on existing research (e.g. Sherwood et al., 1990; Lozano et al., 2007). This algorithm provided a calculation for impedance measures based on the Kubicek formula (Kubicek et al., 1966). The C point was defined as the maximum point in the dz/dt signal in a time window 60–200 ms from the R peak (Gratze et al., 1998), the X point was defined as the minimum point over the course of the cycle after the C point whereas B was set as the maximum derivative of the dz/dt signal in a time window 150–100 ms before the C point. The algorithm was validated using 10 min baseline data from the current study from 20 participants. These data were scored manually by a trained experimenter and compared to the results from algorithm. With respect to LVET times, the mean deviation between the manual and computerised scores was 82 ms (s.d. = 30 ms; range = 23–110 ms). For PEP, the mean deviation was 25 ms (s.d. = 23 ms; range = 5–79 ms). A correlation was conducted across the whole data set to assess PEP between manual scoring vs. computerised analysis, we found that scores were highly correlated (r = 0.89).

2.4.4. Facial electromyography (fEMG)

fEMG activity was recorded from the corrugator supercilii (located just above the eyebrow) using two external electrodes (Cacioppo et al., 1990). fEMG data were sampled at 1000 Hz and filtered between 30 and 500 Hz. During post-processing, the sample rate was reduced to 512 Hz and artifacts due to eye blinks were removed (vertical EOG was recorded separately and this signal was subtracted from the corrugator trace). The resulting fEMG data was normalised using a root-mean-square (RMS) transformation.

2.4.5. Frontal EEG asymmetry

EEG was recorded monopolarly from 32 Ag-AgCl pin-type active electrodes mounted in a BioSemi stretch-lycra head cap. Electrodes were positioned using the 10–20 system and recorded activity from the following sites: frontal pole (FP1, FP2), anterior frontal (AF3, AF4), frontal (F3, Fz, F4), frontocentral (FC5, FC1, FC2, FC6), central (C3, Cz, C4), temporal (T7, T8), parietocentral (CP5, CP1, CP2, CP6), parietal (P7, P3, Pz, P4, P8), occipitoparietal (PO3, PO4) and occipital (O1, Oz, O2). Electrodes were also placed at earlobe sites (A1, A2) allowing offline re-referencing with a linked ears montage. AC differential amplifiers amplified signals at source with continuous digitization at 16384 Hz and online down sampling to 512 Hz. Offline filtering was performed using high and low pass filters of 0.05 Hz and 60 Hz respectively and a notch filter of 50 Hz. EEG was recorded continuously throughout baseline prior to the task (with eyes open) and continuously throughout the task. Analysis was performed using BESA software (MEGIS software GmbH, Gräfelfing, Germany). A linked ears montage was applied offline for asymmetry analysis and an average reference was used for analysis of alpha modulation. Automatic correction of blink artifacts and horizontal and vertical saccades was performed using detection through predefined topographies. Muscle activity over 100 μV was also excluded. An average of 5.8% of analysed data was rejected for each participant due to artifacts. Fast Fourier transforms were computed over 50% overlapped windows of 2 s (1024 points). Average power spectra were then computed for each experimental condition by averaging mean FFT results of both blocks for each level. Power spectra in μV^2 were log transformed (using the natural log) to normalise distribution.

Alpha power was measured as the average power within the 7.5–13 Hz band. Frontal asymmetry values were obtained using a linked-ears reference scheme from the following sites: AF4, F8, F4, FC2, FC6 (right hemisphere sites) AF3, F7, F3, FC1, FC5 (left hemisphere sites). Power estimates for the alpha (7.5–13 Hz) band were then used in the following formula: Ln [right total alpha power] − Ln [left total alpha power] to generate an index of asymmetry for each homologous pair of electrodes in each band. Positive values on the index indicated greater relative right alpha power and greater relative left frontal activity, while greater relative right frontal activity was indicated by negative values (Allen et al., 2004).

2.5. Procedure

Participants who responded to flyers and emails were screened via a health questionnaire and the Trait Anger Expression Inventory prior to recruitment; see exclusion criteria in the Participants section. Participants who passed this screening procedure were invited to the laboratory where they received information about the test and signed a consent form. They were subsequently provided with a short training session on the driving simulator of approximately 5 min duration. On completion of the training session, participants were fitted with cardiovascular electrodes. This equipment was tested before the EEG and fEMG electrodes were fitted to the participant. This process took approximately 40 min and the participant remained seated throughout. Baseline levels of psychophysiology were collected; participants were asked to sit and relax for 10 min; blood pressure values were collected at two minute intervals and participants eyes were open throughout

the baseline period. Data from the last 5 min of this period were averaged to yield baseline values with the exception of the blood pressure data, which was averaged from three readings taken at 2 min, 5 min and 10 min.

Participants completed two pre-test questionnaires: the State Anger Expression Inventory and the Control/Confidence scale from the DSSQ. They were subsequently presented with a set of standardised instructions, which are paraphrased as follows: "You are expected to collect a child from school in 15 minutes and therefore, you must complete the journey in this time. Please check the clock to ensure that you complete the journey on schedule. If you fail to do so, you will not be reimbursed for your time. You will receive a £1 penalty if you exceed the speed limit or overtake where the lane markings indicate that overtaking is illegal. If you crash the car more than twice, you will not be reimbursed for your time. Remember, you can withdraw from the task at any time without the need for explanation." After reading these instructions, participants performed the simulated journey as described earlier. When the participants had completed the simulated drive, they were asked to complete post-test versions of both questionnaires. Participants were then fully debriefed as to the true nature of the experimental task.

2.6. Ethical considerations

The decision to deceive the participants was felt to be absolutely necessary in order to accomplish the goals of the experiment, i.e. to induce genuine experience of anger. By inviting participants to the laboratory to give their time, we were obliged to compensate them financially but also needed to introduce real-world consequences associated with failure to complete the journey on schedule. Therefore, a decision was taken to give participants an impression that financial compensation was dependent on performance.

3. Results

All data were subjected to a repeated measures ANOVA analysis. Initial tests were performed on the subjective data as a manipulation check. A comparison of pre- and post-test values of the state scale from the STAXI 2 (Spielberger, 1999) revealed a significant increase of subjective anger during the simulated journey [$F(1,28) = 34.52$, $p<.01$, $\eta^2 = .55$]. The opposite trend was observed for the subjective level of confidence/control experienced by our participants [$F(1,28) = 148.64$, $p<.01$, $\eta^2 = .84$], i.e. participants experienced a significant fall in control during the simulated journey. Descriptive statistics for both subjective data are presented in Table 1.

A repeated measures ANOVA was conducted on both systolic and diastolic blood pressure for three periods: baseline, the first traffic jam (TJ1) and during the second traffic jam (TJ2). The analysis of systolic blood pressure (SBP) revealed a significant main effect [$F(2,27) = 16.71$, $p<.01$, $\eta^2 = .56$] and descriptive statistics are illustrated in Table 1. Post-hoc Tukey HSD tests revealed that SBP was significantly higher during both TJ1 and TJ2 compared to baseline ($p<0.01$). The same analysis was applied to diastolic blood pressure (DBP) and also revealed a significant main effect [$F(2,27) = 15.29$, $p<.01$, $\eta^2 = .54$]. Post-hoc Tukey tests indicated that DBP was significantly elevated during both traffic jams ($p<.01$).

The ANOVA model was applied to a number of cardiovascular variables. The analysis of heart rate revealed a significant main effect [$F(2,26) = 8.14$, $p<.01$, $\eta^{2=} = .39$], i.e. heart rate was significantly elevated during both traffic jams compared to baseline. The data record was incomplete for six participants for cardiovascular impedance analysis due to equipment failure. Analysis of PEP revealed a null effect and the analysis of LVET failed to reach significance. A main effect was found for TPR [$F(2,21) = 3.14$, $p = .05$, $\eta^2 = .24$]; post-hoc Tukey tests revealed that TPR was significantly higher during TJ1 compared to baseline ($p = .05$) whereas the increase of TPR at TJ2 did not significantly differ from baseline. Both analyses of CO and SV failed to reach significance. All descriptive statistics for cardiovascular measures are presented in Table 2.

Muscle activity from the corrugator supercilii was captured and normalised (via RMS transformation) prior to analysis. The ANOVA revealed a main effect [$F(2,27) = 9.93$, $p<.01$, $\eta^2 = .43$] and subsequent Tukey tests indicated that: (1) corrugator activity was higher during both traffic jams compared to baseline ($p<.02$), and (2) corrugator activity increased during TJ2 compared to TJ1 ($p<.01$). Frontal EEG asymmetry was captured using a measure of relative difference between left and right side at five pairs of frontal sites: AF3/4, F3/4, F7/8, FC1/2 and FC5/6. These data were subjected to individual ANOVA analyses. A significant main effect was found at F3/4 [$F(2,27) = 3.03$, $p = .05$, $\eta^2 = .16$]. A post-hoc Tukey test revealed a decrease of left hemisphere activation during TJ1 compared to baseline condition ($p = .05$). Descriptive statistics for both fEMG and EEG frontal asymmetry are presented in Table 2.

Table 1
Mean and standard deviations for subjective levels of anger and control ($N = 29$).

Subjective measure	Pre-test	Post-test
Anger	15.38 (3.75)	22.55 (6.91)
Control	24.79 (3.97)	14.72 (5.11)

Table 2
Mean and standard deviations for cardiovascular and fEMG measures during baseline and both traffic jams. Note: HR (heart rate), SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), fEMG_corr (activity of the corrugator supercilii muscle), PEP (Pre-Ejection Period), LVET (Left Ventricular Ejection Time), TPR (Total Peripheral Resistance), CO (Cardiac Output), and SV (Stroke Volume) and frontal EEG asymmetry at F3/F4. ($N = 23$). Note: data in italics is significantly different to baseline levels.

Measure	Units	Baseline	TJ1	TJ2	Sig
HR	bpm	66.37 (12.16)	*73.75 (13.45)*	*75.20 (15.95)*	<.01
SBP	mmg/Hg	118.69 (8.53)	*126.86 (11.65)*	*129.39 (13.05)*	<.01
DBP	mmg/Hg	77.70 (7.45)	*84.75 (7.86)*	*82.89 (8.02)*	<.01
fEMG_corr	mV(RMS)	12.37 (0.79)	*16.34 (1.73)*	*18.35 (1.72)*	<.01
PEP	ms	114.5 (43.39)	112.5 (28.39)	129.6 (41.94)	n.s.
LVET	ms	276.3 (65.67)	291.6 (55.98)	295.8 (38.24)	n.s.
TPR	dyne-s*cm^{-5}	996.56 (370.10)	*1239.30 (383.72)*	1131.12 (385.38)	<.05
CO	L/min	6.06 (3.98)	7.22 (3.81)	7.32 (2.82)	n.s.
SV	ml	93.36 (49.11)	104.40 (38.11)	112.52 (33.61)	n.s.
F3/F4	Difference score	0.14 (0.22)	*−0.02 (0.37)*	0.03 (0.36)	<.05

4. Discussion

The subjective self-report variables provided a manipulation check that the simulated driving task induced feelings of anger and loss of control/confidence (Table 1), albeit on pre- and post-task basis only. SBP and DBP were both elevated during both traffic jams compared to baseline. As anticipated, HR significantly increased between baseline and the traffic jams (Table 2) but there was no equivalent effect on CO, SV or PEP. TPR increased significantly during the first traffic jam (compared to baseline) but there was no significant effect on LVET (Table 2). EMG activity from the corrugator supercilii showed a significant linear trend, indicating that negative affect peaked during the second traffic jam (Table 2). The analysis of frontal

EEG asymmetry revealed a reduction of left hemisphere activation at F3/F4 during both traffic jams (Table 1).

It was anticipated that the first traffic jam would provoke anger in the motivational context of challenge. By contrast, the anger response to the second traffic jam would occur in the context of threat/avoidance, because participants had no chance of completing the journey on schedule due to the timing of this delay. We found little evidence to support either hypothesis. The pattern of cardiovascular reaction observed during the first traffic jam suggested a state of threat as blood pressure and TPR were both elevated whilst CO remained stable. It has been argued that this pattern is associated with a reduction of adrenaline and lower levels of vasodilation (Blascovich and Mendes, 2000). The absence of any significant change in PEP indicated that observed changes in systolic blood pressure were moderated by variation in vascular resistance as opposed to a beta-adrenergic influence on the force of myocardial contraction. Changes in frontal EEG asymmetry during the first traffic jam indicated a significant reduction of left frontal activation/approach motivation when an increase of left activation had been anticipated (Harmon-Jones, 2003). The general pattern observed during the first traffic jam was broadly indicative of anger in the context of changes in vascular resistance combined with reduced approach motivation.

The second traffic jam provoked an increase of expressive anger from the corrugator muscle but no other significant differences were found between the two traffic jams. Corrugator activity is not an unambiguous measure of anger expression via facial musculature and increased activity could be associated with other concepts, such as increased mental effort or concentration; however, this seems unlikely as the mental demands of the driving task were minimal during the traffic jam. The cardiovascular pattern did not fit either state of challenge or threat (as defined by Blascovich et al., 1999) as CO remained relatively unaffected. A correlational analysis was conducted to replicate the evidence for corresponding changes in frontal EEG asymmetry and cardiovascular manifestation of anger reported by Koslov et al. (2011). We found no direct support for their findings as none of the cardiovascular variables showed any significant association with frontal EEG asymmetry.

Our protocol failed to distinguish between threat and challenge as defined by Blascovich and colleagues (Blascovich et al., 1999; Blascovich and Mendes, 2000; Blascovich and Tomaka, 1996). In addition, levels of left frontal activation in the EEG remained stable and reduced (relative to baseline) during both traffic jams. In hindsight, we wondered if the distinction between challenge/approach and threat/avoidance, as defined by the goal to complete the journey on schedule, was obfuscated as participants responded to their immediate experience of frustration during traffic jam. It is also possible that our use of financial penalties to prevent participants from subverting the time schedule manipulation could have reduced approach-related anger. Alternatively, the task scenario (to collect a child from school) may have produced an empathetic response that mitigated approach-related anger. A second drawback of the current study was the lack of counterbalancing between our challenge/approach and threat/avoidance scenarios. This is an obvious and binding limitation given the role of elapsed time and journey schedule in our methodology. It is also possible that the initial traffic jam came as an unexpected surprise, resulting in higher reactivity than anticipated, whilst experience of the second traffic jam was less novel and psychophysiological responses were subdued as a direct result. In addition, we focused on the subjective measurement of anger and control, when in hindsight, a broader approach would have been useful to capture related emotional categories (e.g. fear) that may have played a role. The study was also limited with respect to the decision to recruit male participants that was taken to preserve the homogeneity of the psychophysiological response to the anger induction; it remains to be seen whether these findings may be generalised to a female population.

From a methodological perspective, the simulated driving task proved to be a reliable method for anger induction compared to our previous study (Spiridon and Fairclough, 2009) where a combination of a computer malfunction and harassment by experimenter were used to provoke anger. The anger-inducing properties of the current methodology were derived from the presence of real-world consequences (payment) associated with failure and a resonance between the simulated traffic jam and participants' actual experience as drivers. The absence of a control condition meant that it was impossible to differentiate between the effects of anger provocation (traffic jams) and task context (journey schedule/financial penalties) on psychophysiological markers. However, the protocol does run the risk of producing domain-specific instances of anger, i.e. the expression of anger in a driving scenario may not generalise to the measurement of anger other domains of life such as home or the workplace, i.e. Bongard and Al'Absi (2005).

To summarise: the results of our study demonstrated increased cardiovascular reactivity and reduced approach motivation in response to anger in the context of a simulated traffic jam. Further studies are required to understand the connection between frontal EEG asymmetry and cardiovascular correlates of anger in order to create a broad framework for the analysis of anger in a motivational context.

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References

Al'Absi, M., Bongard, S., 2006. Neuroendocrine and behavioural mechanisms mediating the relationship between anger expression and cardiovascular risk: assessment considerations and improvements. Journal of Behavioural Medicine 29, 573–591.
Allen, J.J.B., Coan, J.A., Nazarian, M., 2004. Issues and assumptions on the road from raw signals to metrics of frontal EEG asymmetry in emotion. Biological Psychology 67, 183–218.
Blascovich, J., Mendes, W.B., 2000. Challenge and threat appraisals: the role of affective cues. In: Forgas, J.P. (Ed.), Feeling and Thinking: the role of affect in social cognition. Cambridge University Press, New York, pp. 59–82.
Blascovich, J., Tomaka, J., 1996. The biopsychosocial model of arousal regulation. Advances in Experimental Social Psychology 28, 1–51.
Blascovich, J., Berry-Mendes, W., Hunter, S.B., Salomon, K., 1999. Social "Facilitation" as challenge and threat. Journal of Personality and Social Psychology 77, 68–77.
Blascovich, J., Mendes, W.B., Tomaka, J., Salomon, K., Seery, M., 2003. The robust nature of the biopsychosocial model challenge and threat: a reply to Wright & Kirkby. Personality and Social Psychology Review 7, 234–243.
Bongard, S., Al'Absi, M., 2005. Domain-specific anger expression and blood pressure in an occupational setting. Journal of Psychosomatic Research 58, 43–49.
Cacioppo, J.T., Bush, L.K., Tassinary, L.G., 1990. Microexpressive facial actions as a function of affective stimuli: replication and extension. Personality and Social Psychology Bulletin 18, 515–526.
Davis, M.C., Matthews, K.A., McGrath, C.E., 2000. Hostile attitudes predict elevated vascular resistance during interpersonal stress in men and women. Psychosomatic Medicine 62, 17–25.
Everson, S.A., Goldberg, D.E., Kaplan, G.A., Julkunen, J., Salonen, J.T., 1998. Anger expression and incident hypertension. Psychosomatic Medicine 60, 730–735.
Everson-Rose, S.A., Lewis, T.T., 2005. Psychosocial factors and cardiovascular disease. Annual Review of Public Health 26, 469–500.
Gratze, G., Fortin, J., et al., 1998. A software package for non-invasive, real-time beat-to-beat monitoring of stroke volume, blood pressure, total peripheral resistance and for assessment of autonomic function. Computers in Biology and Medicine 28 (2), 121–141.
Hamer, M., Tanaka, G., Okamura, H., Tsuda, A., Steptoe, A., 2007. The effects of depressive symptoms on cardiovascular and catecholamine responses to induction of depressive mood. Biological Psychology 74, 20–25.
Harmon-Jones, E., 2003. Anger and the behavioural approach system. Personality and Individual Differences 35, 995–1005.
Harmon-Jones, E., Sigelmann, J.D., 2001. State anger and prefrontal brain activity: evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. Journal of Personality and Social Psychology 80, 797–803.
Harmon-Jones, E., Peterson, C.K., Harris, C.R., 2009. Jealousy: novel methods and neural correlates. Emotion 9, 113–117.

Harmon-Jones, E., Gable, P.A., Peterson, C.K., 2010. The role of asymmetric frontal cortical activity in emotion-related phenomena: a review and update. Biological Psychology 84, 451–462.
Herrald, M.M., Tomarka, J., 2002. Patterns of emotion-specific appraisal, coping and cardiovascular reactivity during an ongoing emotional episode. Journal of Personality and Social Psychology 83, 434–450.
Koslov, K., Mendes, W.B., Pajtas, P.E., Pizzagalli, D.A., 2011. Asymmetry in resting intracortical activity as a buffer to social threat. Psychological Science 22 (5), 641–649.
Kreibig, S.D., 2010. Autonomic nervous system activity in emotion: a review. Biological Psychology 84, 394–421.
Kubicek, W.G., Karnegis, J.N., Patterson, R.P., Witsoe, D.A., Mattson, R.H., 1966. Development and evaluation of an impedance cardiac output system. Aerospace Medicine 37, 1208–1212.
Larsen, J.T., Norris, C.J., Cacioppo, J.T., 2003. Effects of positive and negative affect on electromyographic activity over the zygomaticus major and corrugator supercilii. Psychophysiology 40, 776–785.
Lozano, D.L., Norman, G., Knox, D., Wood, B.L., Miller, B.D., Emery, C.F., Berntson, G.G., 2007. Where to B in dZ/dt. Psychophysiology 44 (1), 113–119.
Matthews, G., Joyner, L., Gilliland, K., Campbell, S., Falconer, S., Huggins, J., 1997. Validation of a comprehensive stress state questionnaire: towards a state 'Big Three'? In: Mervielde, I., Deary, I.J., De Fruyt, F., Ostendorf, F. (Eds.), Personality Psychology in Europe. Tilburg University Press, Tilburg.
Prkachin, K., Mills, D., Zwaal, C., Husted, J., 2001. Comparison of hemodynamic responses to social and nonsocial stress: evaluation of anger interview. Psychophysiology 38, 879–885.
Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., van Doornen, L.J.P., 1990. Methodological guidelines for impedance cardiography. Psychophysiology 27, 1–23.
Sinha, R., Lovallo, W.R., Parsons, O.A., 1992. Cardiovascular differentiation of emotion. Psychosomatic Medicine 54, 422–435.
Spielberger, C.D., 1999. State-Trait Anger Expression Inventory-2. Psychological Resource Inc., Odessa, Fl.
Spiridon, E., Fairclough, S.H., 2009. Cardiovascular and EEG indicators of anger and control/no control states. Psychophysiology 46 (S1), S54.
Stemmler, G., Heldmann, M., Pauls, C.A., Scherer, T., 2001. Constraints for emotion specificity in fear and anger; the context counts. Psychophysiology 38, 275–291.
Stemmler, G., Aue, T., Wacker, J., 2007. Anger and fear: separable effects of emotion and motivational direction on somatovisceral responses. International Journal of Psychophysiology 66, 141–153.
Vella, E.J., Friedman, B.H., 2009. Hostility and anger in: cardiovascular reactivity and recovery to mental arithmetic stress. International Journal of Psychophysiology 72, 253–259.
Wacker, J., Heldmann, M., Stemmler, G., 2003. Separating emotion and motivational direction in fear and anger: effects on frontal asymmetry. Emotion 2003, 167–193.
Wright, R.A., Kirkby, L.D., 2003. Cardiovascular correlates of challenge and threat appraisals: a critical examination of biopsychosocial analysis. Personality and Social Psychology Review 7, 216–233.
Zinner, L.R., Brodish, A.B., Devine, P.G., Harmon-Jones, E., 2008. Anger and asymmetrical frontal cortical activity: evidence for an anger-withdrawal relationship'. Cognition & Emotion 22 (6), 1081–1093.