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How effortful is cognitive control? Insights from a novel method measuring single-trial evoked beta-adrenergic cardiac reactivity

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Abstract

The ability to adjust attentional focus to varying levels of task demands depends on the adaptive recruitment of cognitive control processes. The present study investigated for the first time whether the mobilization of cognitive control during response-conflict trials in a flanker task is associated with effort-related sympathetic activity as measured by changes in the RZ-interval at a single-trial level, thus providing an alternative to the pre-ejection period (PEP) which can only be reliably measured in ensemble-averaged data. We predicted that response conflict leads to a physiological orienting response (i.e. heart rate slowing) and increases in effort as reflected by changes in myocardial beta-adrenergic activity (i.e. decreased RZ interval). Our results indeed showed that response conflict led to cardiac deceleration and decreased RZ interval. However, the temporal overlap of the observed heart rate and RZ interval changes suggests that the effect on the latter reflects a change in cardiac pre-load (Frank-Starling mechanism). Our study was thus unable to provide evidence for the expected link between cognitive control and cardiovascular effort. However, it demonstrated that our single-trial analysis enables the assessment of transient changes in cardiac sympathetic activity, thus providing a promising tool for future studies that aim to investigate effort at a single-trial level.

Introduction

In daily life, there are many situations in which we have to maintain focus without being distracted, so that inappropriate responses do not occur. The ability to flexibly adapt behavior to current task demands is generally considered to be an important aspect of cognitive control (Kahneman, 1973). Cognitive control processes are typically measured in response inhibition tasks, such as the flanker task (Eriksen & Eriksen, 1974; for an overview see Eriksen, 1995), in which the amount of conflict can be manipulated. According to the conflict-monitoring theory (Botvinick, Braver, Barch, Carter, & Cohen, 2001) cognitive control is adaptively mobilized when response conflict is detected during a trial. This adaptation to conflict improves subsequent performance and is thought to reflect transient enhancements in cognitive control. In addition, when the proportion of conflict trials across a task block is high, these adaptations result in an overall reduction in the behavioral susceptibility to conflict, suggesting improved sustained cognitive control during a high-conflict task block (Botvinick et al., 2001; Gratton, Coles, & Donchin, 1992; Purmann, Badde, Luna-Rodriguez, & Wendt, 2011).

In the present study we investigated whether the transient and sustained mobilization of cognitive control is also associated with physiological responses typically interpreted as reflecting effort mobilization. Although cognitive control has often been characterized as a process requiring effort (Hasher & Zacks, 1979; Kahneman, 1973; Mulder, 1986; Schneider & Shiffrin, 1977; Westbrook & Braver, 2015; see also Rothbart, Ellis, Rueda, & Posner, 2003) there is little empirical evidence to support this notion. Only a few studies have established a link between cognitive control and effort based on demonstrating an increase in pupil dilation in response to conflict trials in cognitive control paradigms (Brown et al., 1999; Laeng, Ørbo, Holmlund, & Miozzo, 2011; Rondeel, van Steenbergen, Holland, & van Knippenberg, 2015;

Siegle, Ichikawa, & Steinhauer, 2008; Siegle, Steinhauer, & Thase, 2004; van Bochove, van der Haegen, Notebaert, & Verguts, 2013; van Steenbergen & Band, 2013; van Steenbergen, Band, & Hommel, 2015; Wendt, Kiesel, Geringswald, Purmann, & Fischer, 2014). However, although increased pupil dilation has been argued to reflect increased effort (Kahneman, 1973), it might simply reflect an increase in physiological arousal non-specific to effort mobilization (Bradley, Miccoli, Escrig, & Lang, 2008). The same issue might apply to studies that have interpreted increased effort based on observed skin conductance changes in response to conflict trials (Kobayashi, Yoshino, Takahashi, & Nomura, 2007; Naccache et al., 2005; Stennett, 1957; cf. Schacht, Dimigen, & Sommer, 2010).

The present study used cardiac physiological measures as an alternative to pupil dilation and skin conductance. In particular, we focused on myocardial sympathetic activity as the operational definition of effort mobilization (Wright, 1996). Previous use of cardiovascular measures to index effort has typically analyzed cardiovascular responses at the block of trials level, thus aggregating the cardiovascular response over several minutes of task performance. For example, Richter and colleagues (Richter, Friedrich, & Gendolla, 2008) demonstrated increases in mean heart rate of a 72 trials block in proportion to experienced task difficulty. However, given that the cardiovascular system is controlled by both branches of the autonomic nervous system (Berntson, Quigley, & Lozano, 2007), heart rate can only be regarded as a measure of effort if the sympathetic activation (i.e. increase in heart rate) outweighs the parasympathetic activity (i.e. decrease in heart rate), and it is impossible to disentangle these influences using a noninvasive methodology.

A more promising measure of effort mobilization is the pre-ejection period (PEP) (Gendolla, Wright, & Richter, 2012; Kelsey, 2012; Richter, Friedrich, & Gendolla, 2008). PEP is

defined as the period between the onset of left ventricular contraction and aortic valve opening (Weissler, 1977) and has been considered to be a useful indicator of the contractile state of the heart (Kelsey, 2012; Sherwood et al., 1990). Consistent with our definition of effort mobilization (Kelsey, 2012), PEP is thought to reflect the sympathetic effects on the heart, mediated by its beta-adrenergic receptors, and has been shown to respond proportionally to task engagement (Richter et al., 2008). Further, research has shown that PEP becomes progressively shorter in response to increasing task difficulty (Richter et al., 2008; Silvestrini & Gendolla, 2013). It is important to note that these effects of decreased PEP were observed in the absence of a decrease in heart rate. This is important because heart rate slowing is associated with greater ventricular filling (cardiac pre-load) which automatically leads to increased contractility and decreased PEP via the Frank-Starling mechanism. Thus, heart rate deceleration influences PEP independently of sympathetic influences (Sherwood et al., 1990).

Some studies have also investigated the effect on cardiac reactivity at the level of single trials. To the best of our knowledge, however, this approach has yet only been used for heart rate measures. For example, heart rate slowing has been observed following attention regulation (Somsen, van der Molen, Jennings, & van Beek, 2000), error monitoring (Hajcak, McDonald, & Simons, 2003), mental transformations (Jennings, van der Molen, & Debski, 2003), and response conflict (Fiehler, Ullsperger, Grigutsch, & von Cramon, 2004; Jennings, van der Molen, Brock, & Somsen, 1991; Schacht et al., 2010; cf. Spapé & Ravaja, 2016). This transient deceleration of heart rate after stimulus onset has been interpreted to reflect an orienting response, mediated by the parasympathetic system, that helps to prepare organisms to deal effectively with task-relevant stimuli (Graham & Clifton, 1966; Jennings et al., 1991; Lynn, 1966; van der Molen, 2000).

The goal of the present study is to examine whether conditions that require increased cognitive control lead to effort mobilization as measured at a single-trial level. To this aim we developed a – to the best of our knowledge – novel method that provides an alternative measure of beta-adrenergic sympathetic impact on the heart at a single-trial level. The standard approach to measure PEP requires ensemble-averaged ICG data across many R-peaks in which PEP is typically defined as the time interval between the Q point and the B point. Given the complexity of this scoring method, guidelines have been developed to standardize visual inspection and correction (Sherwood et al., 1990). However, this method is not suitable to be applied at the single-trial level because the Q and B points are both considerably susceptible to noise and distortion. Fortunately, it has been shown that for signals ensemble-averaged over 1 minute epochs, PEP can be closely approximated by measuring the interval between the R-peak and the Z (dZ/dt_{max}) points (Lozano et al., 2007), which are fairly simple to extract, even for single QRS cycles. Given this close relationship between PEP and the RZ interval (henceforth abbreviated as RZ), the method introduced here capitalizes on this finding and will measure effort-related beta-adrenergic sympathetic impact on the heart by calculating an evoked response at trial level based on an interpolated continuous RZ signal.

Using this novel method, we tested the primary hypothesis that conflicting flanker task trials do not only decrease heart rate, but also increase transient effects on compensatory effort, as reflected by a lowering of evoked RZ following stimulus onset. Physiological data was acquired in the context of a conflict tasks in which participant had to respond to conflict and no-conflict flanker trials presented in random order. In addition, the proportion of conflict trials across a task block was manipulated, using low-conflict (75% no-conflict and 25% conflict trials) and high-conflict (25% no-conflict and 75% conflict trials) task blocks that were presented

in alternating order. On the basis of the known temporal dynamics of beta-adrenergic influence on the heart (Mokrane & Nadeau, 1998; Ng, Brack, & Coote, 2001), it is expected that the effects of trial conflict on RZ only emerge after 1 to 3 seconds following stimulus onset. On the other hand, based on earlier studies it is expected that the effect of trial conflict on cardiac deceleration emerges approximately 1 second after stimulus onset (i.e., the first interbeat interval following stimulus onset) and lasts for about 1 second (Fiehler et al., 2004; Jennings et al., 1991; Spapé & Ravaja, 2016). In addition, we investigated the effect of the overall proportion of conflict in the task blocks. Given previous findings showing increased behavioral interference in blocks in which the proportion of conflict trials is low (e.g. Gratton et al., 1992; Purmann et al., 2011), we expected that low-conflict compared to high-conflict blocks 1) leads to more pronounced transient enhancements of effort, reflected by a larger effect of conflict on RZ following stimulus onset; and, 2) might be associated with reduced sustained effort, reflected by an increased RZ during the pre-stimulus baseline period.

Method

Participants

Forty-eight students at Leiden University (age mean = 19.06 years, *SD* = 1.34 years; 7 males; 8 left-handed) participated as part of gaining course credit. All participants were native Dutch speakers and signed informed consent prior to their inclusion in the study. The research protocol for this study was approved by the Psychology Research Ethics Committee at Leiden University. Participants were required to meet the following inclusion criteria: 1) 18-30 years of age, 2) no previous meditation experience, 3) absence of any cardiovascular problems or psychiatric disorders, and 4) no use of medication known to influence cognition or cardiovascular responses (e.g. antipsychotics or antidepressants) at the moment of inclusion and

during the whole study. Three participants were excluded after screening of the physiological data. For two participants, the ICG signal was too noisy to analyze. One other participant was excluded because their physiological data demonstrated frequent ventricular ectopic beats across the experimental session.

Flanker task

Participants performed a modified version of the Eriksen flanker task (Eriksen & Eriksen, 1974) that included no-conflict (congruent) and conflict (incongruent) trials. We presented an arrow target stimulus that pointed to the left or to the right. This arrow target was surrounded by two arrows at either side that pointed to the same (congruent), or the opposite (incongruent) direction as the target arrow. Participants had to respond as fast and accurately as possible to the direction of the central arrow by using the "q" or "p" key on a standard keyboard. The stimuli (sized about 2.45° width x 0.25° height) were presented in black color on a gray background on a 17" monitor at a distance of about 70 cm from the participants' eyes. The flanker task was conducted using E-prime software version 2.0.10.356 (Psychology Software Tools, Pittsburgh, PA) and took about 15 minutes to complete.

After 16 practice trials that included performance feedback, participants performed six blocks of low proportion conflict (75% no-conflict and 25% conflict trials) and six blocks of high proportion conflict (25% no-conflict and 75% conflict trials) trials in alternating order. Each block comprised 40 trials. Block order was counterbalanced such that half of the participants started with a low-conflict block and the other half of the participants started with a high-conflict block. In each trial the stimulus was presented for 500 ms (or until a response was given) and was followed by a randomized inter-trial interval of 800, 900, or 1000 ms.[1] Blocks were separated by a self-paced rest period.

Procedure

The experiment took place in a laboratory based in the Faculty of Social and Behavioral Sciences at Leiden University. The data from this experiment was acquired after participants had completed an experiment in an unrelated study that involved computer-based cognitive and motor tasks under the assumption that flanker task performance and related cardiac events would not be differentially influenced by the preceding tasks. After providing written informed consent, participants were seated in front of a computer monitor after which the experimenter attached electrodes for electrocardiography (ECG) and ICG as described below.

Cardiac acquisition

Throughout the experimental session we continuously measured ECG and ICG using a BIOPAC MP150 system (BIOPAC Systems Inc., Goleta, CA, USA). Stimulus onset markers were conveyed from the flanker task program via a parallel port and saved into an event marker channel. Data was stored using AcqKnowledge software (BIOPAC Systems, Goleta, CA, USA).

In order to analyze heart rate offline, we sampled the ECG signal at 1000 Hz with a BIOPAC BioNomadix BN-ECG-2 module using three pre-gelled Ag/AgCl spot electrodes placed on skin sites prepared by abrasion and cleaning with alcohol. The first electrode was placed under the right clavicle at the mid-clavicular line, the second electrode was placed on the lower left abdomen within the rib cage frame and the third, ground, electrode and was placed at the lower right abdomen.

The ICG signal was sampled at 1000 Hz using a BIOPAC BioNomadix BN-NICO module interfaced with four sets of Ag/AgCl spot electrodes. After skin preparation, two pairs of electrodes were positioned 5 cm apart on each side of the neck and two pairs of electrodes were positioned 5 cm apart on each side of the abdomen. The distance between the lower neck

electrodes and the upper abdominal electrodes was approximately 30 cm. Within each pair of electrodes, one electrode emitted a 400 μA alternating current and the other measured the voltage developed through the thorax volume. Through the NICO100c, the ICG signal provided measures of basal impedance (Z0) and the rate of change in impedance (dZ/dt) which, in combination with the ECG signal, was used to derive RZ.

Offline cardiac analyses

To generate interbeat intervals (IBI) data, to index heart rate, and RZ data, to index effort, suitable for grand averaging and time series analysis, the ECG and dZ/dt signals were first processed in MATLAB Release 2012b (The MathWorks, Inc., Natick, MA, USA). First, the raw ECG signal was low-pass filtered at 50 Hz to remove high frequency noise and high-pass filtered at 2 Hz to detrend it, both times using zero-phase forward and reverse digital filters. Automatic R-peak detection was then performed and at each R-peak the corresponding IBI, defined as the time difference in ms relative to the preceding R-peak, was calculated. The filtered ECG signal superimposed with the automatically detected R-peaks and IBI periods was then presented to trained reviewers blind to the experimental conditions for visual inspection, allowing them to reject incorrectly detected R-peaks and IBIs. Only accepted data were used in subsequent calculations (proportion accepted R-peaks: range = 93.9 – 98.7%, mean = 97.4%).

Because the dZ/dt signals were smooth and did not require detrending, no further filtering was applied. The dZ/dt_{max} points were automatically detected by searching for the highest peak in the dZ/dt signal between each R-peak and 300 ms thereafter. In a similar fashion to the ECG data, the dZ/dt signal and detected dZ/dt_{max} points were manually inspected and corrected (proportion accepted dZ/dt_{max} points given accepted R-peak: range = 91.4 – 99.7%, mean = 98.3%). Subsequently, at each accepted dZ/dt_{max} point, the interval between each R-peak and its

corresponding Z (dZ/dt_{max}) was calculated, forming the RZ time series. At this point, the RZ and IBI time series only contained values at their respective discrete events (i.e. accepted R-peaks and accepted dZ/dt_{max} points), which were located at the R-peaks and dZ/dt_{max} points for each heartbeat, and thus arbitrarily positioned relative to each stimulus at its onset. To be able to calculate the average evoked response curves across trials, the IBI and RZ time series were transformed into continuous equidistantly spaced signals by means of linear interpolation, performed at the original acquisition sampling rate of 1000 Hz. The resulting RZ and IBI signals, together with the marker channel, were then saved as 32-bit floating point binary data.

This data was then imported to BrainVision Analyzer. For each individual, we segmented the IBI and RZ data for the four combinations of trial type (conflict versus no-conflict trials) and block type (low- versus high-conflict blocks), using segments from 2 seconds before to 5 seconds after stimulus onset. These were then averaged after which we subtracted the mean values during a pre-stimulus period, ranging from 2 to 1 seconds before stimulus onset. Note that we chose this pre-stimulus baseline, because the use of a later period (e.g. the last second before stimulus onset) would have been contaminated by interpolated values originating from possible heart beat changes that immediately followed stimulus onset. Note however, that our results did not critically depend on the choice of this particular baseline period because an analysis on waveforms that were not baseline-corrected revealed a pattern of results very similar to the findings reported here. The grand average of the resulting four waveforms for the IBI and RZ data are shown in Figure 1.

In order to test for the effect of conflict on transient changes both in IBI and RZ, we used two analysis steps. First, we run a standard repeated-measures ANOVA on time-series data for the pre-stimulus-corrected interval of interest (0 to 5 seconds after stimulus onset) down-sampled

to 10 Hz, to test for the effect of time point (50 points), block type (low-conflict versus high-conflict block), trial type (conflict versus no-conflict trial), and their interactions. Since these analyses revealed interactions between time point and trial type and a three-way interaction between time point, trial type and block type for both dependent variables, the subsequent analysis step tested for significant differences between conflict versus no-conflict trial types (i.e., a congruency-difference waveform) using series of paired t-tests on the pre-stimulus waveforms for each time point following stimulus onset, for the low-conflict and high-conflict blocks separately. An additional series of paired t-tests was used to test for differences between the congruency-difference waveforms of the high- and low-conflict block respectively. Because these analyses were run on the original interpolated data (sampled at 1000 Hz) they allowed identifying the temporal dynamics of the conflict effects in ms accuracy. Considering that the paired t-tests involved multiple, albeit closely dependent, time points, we only interpret results surviving a p-value < .01 threshold to protect against type 1 errors. This more conservative p-value roughly corresponds to a Bonferroni correction for multiple comparisons, taking into account that the interpolated values in the 5-second interval of interest were actually based on about 5 discrete heart beats on average.

Finally, in order to test for the effect of block type on pre-stimulus activity which might reflect sustained adaptation in effort, we also compared the effect of low-conflict blocks versus high-conflict blocks (collapsed across conflict and no-conflict trials) by submitting the average pre-stimulus interval (-2 to -1 seconds) waveforms to a repeated measures ANOVA with the factor block type (low-conflict versus high-conflict block).

Results

The results of the analyses on single-trial level IBI (indexing heart rate) and RZ changes relative to the pre-stimulus baseline are presented in Figure 1. For both IBI and RZ, ANOVAs revealed an interaction between trial type and time point [IBI: $F(49,2156) = 14.64$, $p < .001$, $MSE = 395.4$, $\eta_p^2 = .250$; RZ: $F(49,2156) = 7.91$, $p < .001$, $MSE = 1.2$, $\eta_p^2 = .152$] and a three-way interaction between block type, trial type, and time point [IBI: $F(49,2156) = 5.98$, $p = .001$, $MSE = 333.4$, $\eta_p^2 = .120$; $F(49,2156) = 2.83$, $p = .023$, $MSE = 1.2$, $\eta_p^2 = .061$]. In addition there was an interaction between time point and block type [IBI: $F(49,2156) = 9.32$, $p < .001$, $MSE = 181.0$, $\eta_p^2 = .175$; RZ: $F(49,2156) = 4.64$, $p = .001$, $MSE = 0.6$, $\eta_p^2 = .095$] and a main effect of time point [IBI: $F(49,2156) = 36.35$, $p < .001$, $MSE = 223.5$, $\eta_p^2 = .452$; RZ: $F(49,2156) = 72.05$, $p < .001$, $MSE = 2.0$, $\eta_p^2 = .621$]. Finally, there was a significant main effect of trial type for IBI but not for RZ [IBI: $F(1,44) = 6.71$, $p = .013$, $MSE = 1722.9$, $\eta_p^2 = .132$; RZ: $F(1,44) = 1.49$, $p = .229$, $MSE = 9.7$, $\eta_p^2 = .033$].

In order to reveal the temporal dynamics of the differences between conflict versus no-conflict trial types we subsequently used a series of paired t-tests on the waveforms for each time point following stimulus onset, for the low-conflict and high-conflict blocks separately. The lines below the x-axis in Figure 1 show samples with significant differences at $p < .01$. As predicted, and consistent with earlier observations of cardiac deceleration after response conflict (Fiehler et al., 2004), relative to no-conflict trials, conflict trials (i.e., those requiring more cognitive control in order to overcome the conflict) led to heart rate slowing (increased IBIs) about 1 second after stimulus onset, both in the high-conflict (peak at 1256 ms, $t(44) = 3.79$, $p < .001$) and in the low-conflict blocks (peak at 1576 ms, $t(44) = 6.72$, $p < .001$). Moreover, this effect was significantly stronger in the low-conflict block (peak of the differences between the congruency-difference waveforms scores at 1581 ms, $t(44) = 3.98$, $p < .001$), suggesting a stronger orienting response in

this condition, consistent with behavioral data from earlier studies that have shown increased behavioral interference scores when the proportion of conflict trials in a block is low. More importantly, we also observed an effect of conflict on RZ in the expected direction for the low-conflict block only. That is, RZ was smaller following conflict trials in this block, suggesting increased cardiac signs of effort (peak at 1615 ms, $t(44) = 5.43$, $p < .001$). This difference was also significantly stronger in comparison to the high-conflict block (peak at 1656 ms, $t(44) = 3.53$, $p < .001$). However, the temporal characteristics of this response occurred at the same time interval as the cardiac deceleration and it did not sustain for a longer period. This finding thus likely does not reflect an effort-related effect associated with beta-adrenergic influence on the heart, which should have reduced RZ around 3 seconds after stimulus onset.

In a complementary series of analyses, we also analyzed the effect of block type on the pre-stimulus values of IBI and RZ, which might reflect effects of sustained effort across the entire task block. No significant effect of block type(low-conflict versus low-conflict) was observed [IBI: M = 873.2 ms, SE = 19.6 versus M = 873.7 ms, SE = 19.3; $F(1,45) = .07$, p = .795, $MSE = 101.0$, $\eta_p^2 = .002$; RZ: M = 179.3 ms, SE = 1.9 versus M = 179.5 ms, SE = 1.9; $F(1,45) = 1.17$, p = .286, $MSE = 0.6$, $\eta_p^2 = .025$].

Discussion

Using a novel method to measure sympathetic effects on the heart, the present study examined for the first time the link between cognitive control and effort by testing whether response conflict in a flanker task (Eriksen & Eriksen, 1974) is accompanied by changes in RZ, and whether these cardiac changes are observable on a single-trial level. We hypothesized that conflicting flanker trials lead to both heart rate slowing (Fiehler et al., 2004) and increases in effort as reflected by a lowered RZ, an effect which might be more pronounced in blocks in

which the proportion of conflict trials is low, compared to blocks in which the proportion of conflict trials is high. Our findings indeed revealed an increase in IBI and decreased RZ following conflict, which, as predicted, was most pronounced during the low-conflict blocks (see Figure 1).

Although the temporal effect of conflict on IBI (onset at around 1 s) is consistent with previous findings (Fiehler et al., 2004), the temporal response of the effect on RZ is not consistent with the physiological mechanism that is thought to underlie effort-related cardiac responses. Whereas the beta-adrenergic effects underlying effort mobilization are thought to start influencing RZ after a couple of seconds, we observed a much quicker response in RZ around 1 second after the flanker stimuli onset. Since heart rate deceleration was also observed in the same time period, it is likely that our observation of lowered RZ following conflict is driven by the concomitantly slowed heart rate. A reduction in heart rate may lead to increased ventricular filling (increased cardiac pre-load), which automatically increases myocardial contractility via the well-known Frank-Starling mechanism. The result is an RZ shortening that is not caused by sympathetic activity. Consequently, our data does not provide evidence for a link between cognitive control and cardiovascular effort as indexed by RZ, at least as measured in the context of the current flanker task. An interpretation of these null effects suggests that available evidence from other physiological measures of effort such as pupil dilation (e.g. van Steenbergen & Band, 2013) and skin conductance response (e.g. Naccache et al., 2005) in cognitive control tasks, could simply reflect general arousal which does not necessarily involve effort mobilization.

Whilst our study did not provide evidence for an effort-related effect of conflict on RZ, it is important to emphasize that the novel single-trial method introduced here, that measured beta-adrenergic impact on the heart using an interpolated signal of the interval between the R-peak

and Z of the dZ/dt function (Lozano et al., 2007), was sensitive to heart rate-induced changes in cardiac pre-load. This effect was observed despite the use of a short intertrial interval. This finding is in line with other studies on the skin conductance response, another slow physiological response, that have shown that differences between conditions are not affected by short intertrial intervals (Recio, Schacht, & Sommer, 2009; Schacht et al., 2010; Schacht, Nigbur, & Sommer, 2009).

Obviously this can only be done as long as conditions are presented in random order so that effects at the current trial are not confounded by carry-over effects from previous trials. In addition, we expressed the changes in RZ and IBI relative to a pre-stimulus baseline period which further reduces the effect of random noise introduced by previous trials. Nevertheless, we cannot exclude the possibility that our design was less sensitive to prolonged responses in RZ due to noise introduced by the different subsequent trials presented, which would be problematic in case it would turn out that overlapping cardiac responses do not linearly add up. In the light of evidence that the dissipation of epinephrine and norepinephrine which impact the beta-adrenergic receptors on the heart can last for about 9 seconds, it is therefore advisable that future studies use longer intertrial intervals. Notwithstanding these limitations, we believe that the method introduced here provides a promising tool to measure single-trial effort-related effects in other cognitive paradigms, such as attention-shifting (Apps, Grima, Manohar, & Husain, 2015), decision-making (Kool & Botvinick, 2014), task-switching (Kool, McGuire, Rosen, & Botvinick, 2010), and working memory paradigms (Westbrook, Kester, & Braver, 2013), as well as other paradigms that already have demonstrated PEP effects at the block level (Gendolla & Silvestrini, 2010; Richter et al., 2008; Richter & Gendolla, 2009; Silvestrini & Gendolla, 2011). It is well possible that the cardiovascular measures of effort in those tasks, that are likely more

difficult than the two-alternative forced choice flanker task used here, will be more sensitive to reveal effort-related effects in RZ.

In conclusion, our findings show that RZ and heart rate changes could be reliably assessed at a single-trial level in a flanker conflict paradigm. Although conflict did change early heart rate and RZ, associated with a physiological orienting response, it did not result in a prolonged RZ response that is associated with effort mobilization. These results suggest that the link between cognitive control and effort processes is not clear yet, and that future research is needed to gain a better understanding of the conditions under which the exertion of cognitive control does become effortful.

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Footnotes

1. Due to a programming error, we missed the behavioral responses during the inter-trial interval (comprising about 40% of the responses) and therefore we do not report the behavioral results here. However, note that the analyses on the subset of behavioral responses that was recorded confirmed that response conflict was successfully induced, as conflict versus no-conflict trials were associated with slower responses (M = 401 ms versus M = 365 ms) and more errors (M = 22% versus M = 5%), Fs > 140, ps < .001.

Figure 1.

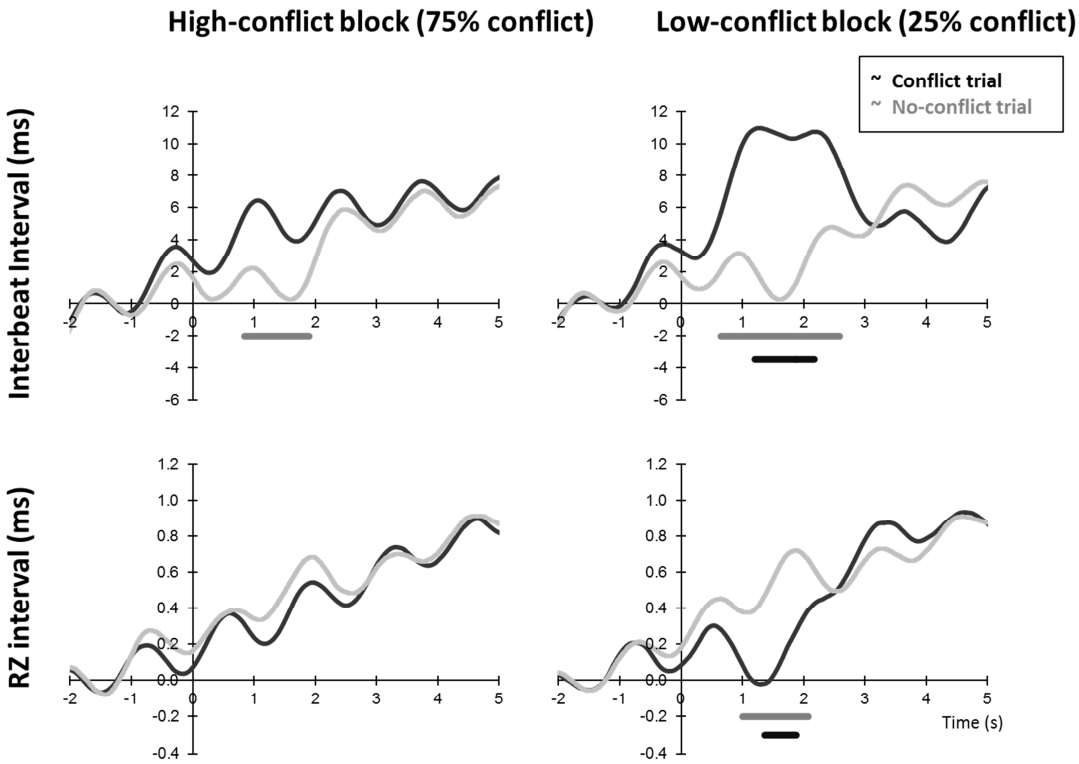

Figure 1. Effect of flanker conflict on interbeat interval (IBI; upper row) and RZ interval (lower panels) during high-conflict (left panels) and low-conflict (right panels) blocks. Waveforms show grand average across participants. Time 0 indicates stimulus onset. Gray lines below the x-axis indicate samples with a significant congruency effect (conflict versus no-conflict trials) for the paired waveforms at $p < .01$. Black lines below the x-axis in the right panels indicate samples where the difference between the congruency difference waveforms of the high- versus low-conflict block is significant at $p < .01$.