Title: Cardiac Defense Response, Cognitive Flexibility and Adaptive Emotion Regulation Strategies

Running Head: CDR and Cognitive Flexibility in Emotion Regulation

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Funding: This work was supported by Universitat Jaume I

Abstract

Cardiac Defense Response (CDR) is a dynamic pattern of cardiac reactivity in response to aversive, intense and unexpected stimuli consisting of two successive accelerating/decelerating components, whose psychological significance is attributed to both attentional and motivational/emotional processes. According to the defense cascade model, the CDR second accelerative component indicates the activation of the aversive-defensive motivational system. This research aimed to analyze the relationship between CDR, levels of anxiety/depression, emotional regulation, and cognitive flexibility in a sample of healthy participants (N = 120). The experimental task consisted in the presentation of an intense aversive sound (500 ms, 105 dB) after an 8-minute rest period to prompt the CDR. A battery of questionnaires (HADS, CERQ, and CFI) was administered before the task began. Preliminary analyses did show no differences in the CDR pattern between subjects scoring high vs. low in anxiety and depression. We divided the sample into accelerators and decelerators according to their change scores obtained in the CDR second accelerative component. Significant differences were found in specific emotion regulation strategies and cognitive flexibility, with accelerators scoring significantly less in both self-report measures. Our findings suggest that cognitive flexibility and adaptive emotional regulation strategies might have an impact on cardiac defense response. It is possible that difficulties in regulating ongoing emotions might be associated with low cognitive flexibility, being key in the association of CDR and disease previously reported in the literature.

Keywords: CDR, Cognitive flexibility, Emotion Regulation, Anxiety, Depression

1. Introduction

Defensive response is a concept associated with the physiological reactions to fear or anxiety that have a protective function, preparing the organism either for flight or fight (Vila et al., 2007). However, if this defensive reactivity is maintained over time it can have negative consequences for the organism (Lovallo & Gerin, 2003). This is due, among other things, to an increase in the activity of the organs involved in these processes which seriously damage health (Kyrou, Chrousos, & Tsigos, 2006; Lang, Davis & Ohman, 2000). In fact, excessive physiological reactivity is for many authors the mechanism by which stress and illness might lead to psychopathology and health problems (Norte et al., 2019).

In laboratory settings, Vila and Fernández (1989) proposed a simple psychophysiological reactivity test, where the presentation of an aversive, discrete, intense, and unexpected (usually auditory) stimulus after a rest period ranging between 6 and 10 minutes prompts a specific phasic cardiac pattern known as *Cardiac Defense Response* (CDR). The CDR is a physiological measure of defense activation consisting of two distinct accelerative components followed by decelerative components after each acceleration –relative to the pre-stimulation baseline (usually 15 seconds),– and lasts for 80 seconds after stimulus presentation (Ramírez, Sánchez, Fernández, Lipp & Vila, 2005; Fernández, 1986; Vila, Fernández & Godoy, 1992). This cardiac reactivity pattern is interpreted as a sequence of heart rate changes with both accelerative and decelerative components, with both parasympathetic and sympathetic mediating mechanisms (Fernández & Vila, 1989), and with both attentional (first acceleration/deceleration component) and emotional (second accelerative component) significance. In addition, the habituation to the stimulus is generally very fast, especially for the second accelerative pattern (Vila et

al., 2007; Norte et al., 2019).

More specifically, the first acceleration (A1) reaches its peak at 2-3 seconds, being followed by a rapid deceleration (D1) until 13 seconds, then a more sustained acceleration (A2) which reaches the peak of maximum response between 20-45 seconds, and a final deceleration (D2) returning to the baseline and ending at 80 seconds. In particular, the first acceleration/deceleration is controlled by the parasympathetic system (inhibition during the acceleration and activation during the deceleration). On the other hand, the second acceleration/deceleration is controlled by both sympathetic and parasympathetic systems, with sympathetic activation and parasympathetic inhibition observed during the second acceleration, and sympathetic inhibition and parasympathetic activation during the second deceleration (Fuentes-Sanchez, Jaén, Cifre, Pastor, 2019).

This pattern seems to reflect the transition from attention to action (Vila et al., 2003). According to the *defense cascade model* proposed by Lang (1995), the defensive reactions follow a sequential pattern with initial attentional phases, which detect and analyze the possible threat, and posterior motivational/emotional phases which facilitate the defensive responses of fight or flight (Vila et al., 2009). The second acceleration of the CDR has been the most recently studied because it is controlled primarily by the sympathetic nervous system, which seems to be closely related to aversive motivational system and coping to face threatening stimuli (López, Poy, Pastor, Segarra, & Moltó, 2016).

Fernandez & Vila (1989) observed that participants could be differentiated as decelerators and accelerators depending on the presence or absence or the second accelerative component of the CDR. Subsequent works have shown individual differences that are associated with the acceleration or deceleration of this

component (Pérez, Fernández, García, Turpin & Vila,1998; Fuentes-Sánchez, Jaén, García, Cifre & Pastor, 2018; Vila et al., 2009). To this extent, accelerators seem to be closely related to mental health conditions, such as anxiety disorders (McTeague & Lang, 2012), depression (Fuentes-Sánchez et al., 2019), or post-traumatic stress disorder (Gillie & Thayer 2014), or more specific features of these conditions, such as excessive worry or emotional instability (Pérez et al., 1998). In addition, differences have been also found in relevant biological factors such as gender or the menstrual cycle (Vila et al., 2009). However, there are differences in the CDR pattern across disorders. Whereas people with generalized anxiety disorder or with higher scores on excessive worry tend to show an accelerative pattern, individuals with specific phobias, subclinical depression or subclinical anxiety are characterized by a decelerative pattern (Fuentes-Sanchez et al., 2019).

In recent decades, emotion regulation strategies have been widely studied mainly due to its clinical implications, as they might help to reduce some of these symptoms, such as excessive worry. The emotion regulation strategies that are considered *adaptive* (such as perspective, reappraisal, acceptance, behavioral activation, positive refocusing, social support, planning, and benefit finding) are associated with the reduction of anxious and depressive symptoms, while those that are considered *nonadaptive* (such as rumination, self-blame, other-blame, substance use, expressive suppression, emotional suppression, denial, and nonsuicidal self-injury) are associated with an exacerbation of these symptoms (Khakpoor, Saed & Armani-Kian, 2019; Mazaheri, Roohafza, Mohammadi & Afshar, 2016). Previous studies have shown that acceptance and reappraisal strategies are exceptionally useful to reduce these symptoms, especially in patients with chronic pain (Denson, Creswell, Terides & Blundell, 2014; Mazaheri, et al., 2016).

In accordance to the *Extended Process Model of Emotion Regulation* (Gross, 2015), emotion regulation strategies depend on three stages: the identification of the conflict, the selection of specific regulatory strategy to be used, and its implementation (Gross, 2015). For the selection of this strategy, it seems essential to consider how to implement the emotion regulation strategies, but also when they should be used (Tull & Aldao, 2015). To this extent, *cognitive flexibility* is defined as the ability to effectively and flexibly adapt cognitive processing and behavior to changes in the environment with the goal of coping with them in the best possible way. This requires attentional resources and the action of executive control to be able to process new information and inhibit previous information that is irrelevant (Alba,Vila, Rey, Montoya & Muñoz, 2019). Some studies have reported that people with social anxiety do not have difficulties in carrying out emotion regulation strategies when they are given the instructions to do so, but they have difficulties in using them correctly in their daily lives. Therefore, the difficulty of this type of patient is not so much the inefficiency of the regulatory strategies themselves but a problem of cognitive flexibility (Tull & Aldao, 2015),

The neurovisceral integration model (Thayer and Lane, 2009) postulates that there is a direct relationship between cognitive flexibility and the cardiovascular system through autonomous vagal tone in tasks involving attention, working memory, and inhibition (Alba, et al., 2019). Although these results have not been tested with respect to defensive responses, Otero, Muñoz, Fernández-Santaella, Verdejo-García & Sánchez-Barrera (2020) found that cognitive flexibility does have a direct effect on the first acceleration of the cardiac defense response, which has greater vagal control, in a sample of healthy women. However, although the second acceleration of CDR is mainly controlled by the sympathetic system, it is true that the

parasympathetic system also has an influence on this component, and thus cognitive flexibility might also have a certain impact on this second accelerative component.

Accordingly, the current study aimed to investigate whether individual differences in cognitive flexibility and emotion regulation strategies (measured through self-reports) would be associated with the differences in the second acceleration of the CDR, a peripheral physiological response related to the activation of the aversive-defensive motivational system, and the involvement of the parasympathetic system through autonomous vagal tone. In addition, we wanted to explore the plausible involvement of anxiety and depression symptoms in this cardiac response. More specifically, we expected to replicate the CDR pattern and the individual differences associated with the second accelerative component, dividing the overall sample into accelerators and decelerators, according to prior literature. Thus, we expected that participants with an accelerative pattern would score less on cognitive flexibility, depression and adaptive emotion regulation strategies, whereas they would score higher on anxiety and maladaptive emotion regulation strategies, in comparison to decelerators.

2. Method

2.1. Participants

A total of 130 participants (89 Female) aged between 19 and 46 years (Mean age = 21.48, SD = 3.72) took part in this experiment. For the statistical analyses ten participants were excluded due to technical problems during physiological data acquisition or excessive artifacts. As a result, statistical analyses reported here were performed with a total of 120 participants. Ethical approval from the Deontological Committee at Universitat Jaume I was obtained. Before the experiment began, all participants signed an informed consent form, and were rewarded with a financial

compensation of 10 euros or with credits for their participation in order to ensure they were properly engaged in the experimental task.

2.2. Materials and design

2.2.1. Self-reported measures

Cognitive Emotion Regulation Questionnaire [CERQ] (Garnefski & Kraaij, 2007); Spanish version validated by Domínguez-Sánchez, Lasa-Aristu, Amor & Holgado-Tello, 2011. This questionnaire comprises 36 items that evaluate nine cognitive strategies: rumination [CERQ-RU]; catastrophizing (CERQ-CA]; self-blame [CERQ-SB]; other-blame [CERQ-BO]; putting into perspective [CERQ-PP]; acceptance [CERQ-A]; positive refocusing [CERQ-PR]; positive reappraisal [CERQ-PRL]; and refocus on planning [CERQ-RP]. Answers are evaluated on a five-point Likert scale from 1 (Almost never) to 5 (Almost always). This questionnaire was validated for a sample aged 16-58 years with values Cronbach's α between .60 and .89.

Emotion Regulation Questionnaire [ERQ] (Gross & John, 2003); Spanish version validated by Cabello, Salguero, Fernández-Berrocal & Gross, 2013. This self-report questionnaire assesses two emotion regulation strategies: cognitive reappraisal [ERQ-R] (6 items) and expressive suppression (ERQ-S) [4 items]. Thus, the scale consists of 10 items and participants respond using a 7-point Likert scale (1 = strongly disagree,7= strongly agree). This questionnaire was validated with values Crombach's $\alpha = 0.79$ with expressive suppression and $\alpha = 0.42$ with cognitive reappraisal.

Anxiety and Depression Scale [HADS] (Zigmond & Snaith, 1983); Spanish version validated by Herrero et al., 2003. The HADS is a 14-item self-report screening scale that was originally developed to indicate the possible presence of

anxiety [HADS-A] and depression [HADS-D] states in the setting of a medical non psychiatric outpatient clinic. HADS consists of a 7-item anxiety subscale and a 7-item depression subscale. Each item scores on a 4-point Likert scale (e.g., as much as I always do [0]; not quite so much [1]; definitely not so much [2]; and not at all [3]), giving maximum subscale scores of 21 for depression and anxiety, respectively. The questionnaire assesses symptoms over the preceding week. This questionnaire was validated with values Cronbach's α 0.90.

The Cognitive Flexibility Inventory [CFI] (Dennis & Vander Wal, 2010). The CFI is a brief 20-item self-report instrument designed to measure the aspects of cognitive flexibility that enables individuals to challenge and successfully replace the maladaptive thoughts with more balanced thinking. It was originally developed to measure three aspects of cognitive flexibility: a) the tendency to perceive difficult situations as controllable; b) the ability to perceive multiple alternative explanations for life occurrences and human behaviors; c) the ability to generate multiple alternative solutions to difficult situations but it ended in two factors and demonstrated adequate levels of validity, reliability and internal consistency. It is composed of two subscales, one that refers to the subject's tendency to perceive difficult situations as controllable [CFI-C] and another that refers to the ability to generate multiple alternative solutions to difficult situations [CFI-A]. Cronbach's alphas for the CFI, Control and Alternatives subscales were 0.91, 0.84, and 0.91, respectively. The seven-week test-retest reliability coefficients for the CFI, Control and Alternatives subscales were 0.81, 0.77, and 0.75, respectively.

2.2.2. Physiological reactivity test

The physiological reactivity test to prompt the Cardiac Defense Response (CDR) consisted in the presentation of an intense and unexpected aversive auditory

stimulus after an 8-minutes resting baseline period. The auditory stimulus used in this task was a white noise of 105dB, 500ms duration and instantaneous rise time.

Participants were sitting in a comfortable chair and the areas where the electrodes for electrocardiogram (ECG) acquisition had to be attached were softly cleaned with cotton soaked in alcohol. Participants were instructed to remain still for the duration of this period of baseline recording, and the psychophysiological reactivity test was conducted. In addition, the temperature and light of the room were conditioned appropriately and were fitted with noise-canceling headphones (Sennheiser HD 205). They were told that the experiment was going to start and introduced to an E-Prime presentation consisting of a black screen to facilitate their relaxation.

FIGURE 1 AROUND HERE

2.3 Psychophysiological data acquisition and reduction

Stimuli control and physiological data acquisition were accomplished using Biopac MP36R. The software used for data acquisition and preprocessing was Acqknowledge 4.4.

Electrocardiogram (ECG) was recorded at Lead II (positive electrode on the left ankle, a neutral electrode on the right ankle and a negative electrode on the right wrist) using Ag/AgCl electrodes with electrolyte paste (8 mm in diameter). A band filter of 0.5–35 Hz and a sampling rate of 1000 Hz were used. HR was obtained online from the ECG, which measured the time interval between consecutive R waves (cardiac period), and artifact correction was performed prior to statistical analyses.

During the 10 minutes that the physiological reactivity task lasted, ECG signal was continuously recorded. To obtain the CDR pattern, change scores were computed

by subtracting HR averages second-by-second for the 80 seconds after the auditory stimulus offset from baseline (HR average for the 15 seconds before the aversive stimulus onset). Thus, 80 values were obtained for each of the 80 seconds after the auditory stimulus was presented.

In order to facilitate the ultimate statistical analyses, the HR data continuously recorded during the 80 seconds after noise offset were additionally reduced to 10 values corresponding to the medians of 10 progressively longer intervals: 2 intervals of 3 seconds, 2 intervals of 5 seconds, 3 intervals of 7 seconds and 3 intervals of 13 seconds. Additionally, change scores were computed by subtracting the 10 medians from baseline HR average before stimulus onset (15 seconds).

2.4. Procedure

Each subject participated in one laboratory session, which lasted approximately 90 minutes. First, participants read an overview of the task and signed an informed consent form. Afterward, they completed an electronic survey to collect sociodemographic data including age, gender and educational level. Then, they were provided with a battery of questionnaires to measure different psychological measures based on an online survey created with Qualtrics software that had to be electronically filled out. These psychological constructs were emotion regulation (ERQ and CERQ), anxiety and depression (HADS) and cognitive flexibility (CFI). Afterward, sensors were attached while participants reclined in a comfortable armchair and the physiological reactivity test that provokes the CDR was carried out. Upon completion of the task, participants were debriefed and rewarded for their participation either with 10€ or 0,5 credit course.

2.5. Statistical analysis

Ward's hierarchical clustering method was used to conduct a cluster analysis

on the second-by-second HR changes during the 20 to 45 seconds interval after the stimulus onset. This method served to classify the experimental sample into two groups (accelerators vs. decelerators), according to previous studies (c.f., López et al., 2009).

In order to facilitate the ultimate statistical analyses, the 80 seconds scores were reduced to 10 values corresponding to the medians of 10 progressively longer intervals seconds 1–3, 4–6, 7–11, 12–16, 17–23, 24–30, 31–37, 38–50, 51–63 and 64–76 (from this point on, M1 to M10). This procedure results in a simplified representation of the CDR without altering its characteristic pattern, with M1 reflecting the first acceleration, M2 to M4 reflecting the first deceleration, M5 to M8 reflecting the second acceleration, and M9 to M10 reflecting the second deceleration (Vila et al., 2007).

A one-factor 2 x 10 ANOVA (Group x Medians) was then performed to determine if these groups were significantly different throughout the CDR pattern. Levene's test with a significance level of 0.5 was used to evaluate the equality of variances. Also, the Wilcoxon signed rank test was used with a significance level of 0.5 when necessary.

In addition, Student's t-test for independent samples was performed to examine whether there were differences between decelerators and accelerators in the scores obtained in the subscales included in the self-report questionnaires administered in this experimental protocol (CERQ, ERQ, HADS, CFI).

Subsequently, partial correlations were performed separately for accelerators and decelerators in order to test the relationships between the CDR medians corresponding to the second accelerative component (M5-M8) and the scores on the self-reported questionnaires.

Statistical analyses were performed using JMP 15.1 software. The Greenhouse-Geisser correction was applied to control for sphericity violation in the repeated measures factors.

3. Results

3.1. CDR & Clusters: Accelerators vs. Decelerators

Visual inspection of the HR waveforms based on the 80 second-by-second change score values showed that each participant presented the typical CDR pattern. The peak of the first acceleration occurred at second 3 after stimulus presentation and deceleration returned to baseline 10 seconds after the aversive stimulus presentation. Also, second acceleration occurred around 35 seconds sequential to the second deceleration after aversive stimulus, as usual for these sequential components.

In addition, two groups of participants with distinct cardiac reactivity patterns were obtained based on the performed Cluster analysis: accelerators (N = 66), participants who showed clear heart rate acceleration during the 20-45 seconds interval, and decelerators (N = 54), participants who showed a decelerative pattern during this interval corresponding to the second CDR acceleration (see Figure 2).

FIGURE 2 AROUND HERE

The one-factor ANOVA (Group x Median) showed that there were significant differences in the 10 medians of the CDR when comparing both groups (accelerators vs. decelerators, classified according to the 20-45 means interval) (see Table 1). In this way, the CDR pattern was preserved, in line with expectations based on prior literature, with a first acceleration in M1, a deceleration from M2 to M4, a second acceleration from M5 to M8, and the second deceleration from M9 to M10.

TABLE 1 AROUND HERE

FIGURE 3 AROUND HERE

3.2. Individual differences in the CDR pattern

Student's t-tests for independent samples performed to compare both experimental groups in their self-report questionnaires scores (see Table 2) showed that accelerators scored significantly lower on specific strategies, refocusing [CERQ-PR] (t = -2.30; p = 0.0116) and planning [CERQ-RP] (t = -2.31; p = 0.0115).

In addition, accelerators scored lower on the subject's tendency to perceive difficult situations as controllable [CFI-C] (t = -2.18; p = 0.0158). However, no significant differences were found between both groups regarding their scores on self-blame [CERQ-SB], acceptance [CERQ-A], rumination [CERQ-RU], positive reappraisal [CERQ-PRL], putting into perspective (CERQ-PP), catastrophizing (CERQ-CA), other-blame [CERQ-BO], cognitive reevaluation [ERQ-R], expressive suppression [ERQ-S], depression [HADS-D] and anxiety [HADS-A].

TABLE 2 AROUND HERE

Separate partial correlations for accelerators and decelerators performed to test plausible associations between CDR medians and scores in the questionnaires measured here (see Table 3) showed a significant relationship between some emotional regulation strategies and heart change values in A2. More specifically, the low planning [CERQ-RP] scores were negatively related to the CDR medians during the whole second acceleration for accelerators but not for decelerators. In addition, contrary to the predicted results, rumination strategy [CERQ-RU] was negatively related to the HR medians for accelerators in M7 and M8, but not for M5 and M6. Similarly, no significant correlations were found for decelerators. Also, contrary to our expectations, other-blame [CERQ-BO] strategy was positively correlated to HR scores for the decelerative cluster in M8, but this was not the case for M5, M6 and M7, neither for the accelerator cluster. Nevertheless, nor self-blame [CERQ-SB],

acceptance [CERQ-A], positive reappraisal [CERQ-PRL], positive refocusing [CRQ-PR], putting into perspective [CERQ-PP], catastrophizing [CERQ-CA], cognitive reevaluation [ERQ-R] nor expressive suppression [ERQ-S] seems to be lineally related for accelerators or decelerators in the second acceleration of CDR.

The analysis showed that cognitive flexibility, as expected, was related to this cardiac component. On the one hand, for decelerators, control subscale [CFI-C] was positivity related in M6 and M7, but not in M5 and M6. In addition, no significant correlations were observed for accelerators. On the other hand, alternatives subscale [CFI-A] correlated significantly both for accelerators and decelerators but in different medians and opposite directions. For accelerators, a negative relationship was observed in M8, while in the decelerator cluster was observed in M5 with a positive correlation.

Finally, scores on the depression [HADS-D] and anxiety [HADS-A] subscales do not appear to be related to the explored median change scores corresponding to the second acceleration of the CDR, neither for accelerators nor for decelerators.

TABLE 3 AROUND HERE

4. Discussion

The present study aimed to investigate individual differences associated with the second accelerative component of the cardiac defense response (CDR).
The CDR is a peripheral physiological response composed of two successive accelerative and decelerative patterns, which has been associated with fear and anxiety reactions to aversive and unexpected stimuli. To this extent, we aimed at exploring the plausible involvement of anxiety and depression symptoms in this cardiac response, more specifically in the second acceleration of CDR. In addition, we explored whether cognitive flexibility and specific emotion regulation strategies

have an influence on this component.

The current study replicated the *pattern of the CDR* found in prior studies (López et al., 2009; Fuentes-Sanchez et al., 2019; Vila et al., 2007; Vila et al., 2009). In addition, it also evidences the clear distinction of two groups (accelerators vs. decelerators) with respect to the second acceleration that occurs from t20 to 45 seconds after the presentation of an intense and aversive auditory stimulus. These clusters also have differences of psychophysiological nature, which might be associated with problems in the defensive flight-fight reactions that are controlled by the sympathetic system (Otero et al., 2020; Norte et al., 2019; López et al., 2009), being replicated in prior studies by different laboratories.

In addition, our results indicate that *cognitive flexibility* may be a regulatory factor in the CDR components, as shown with heart rate (Thayer and Lane, 2009). Furthermore, current findings suggest that this cognitive control is reflected in the second acceleration where accelerators scored significantly lower in cognitive flexibility compared to decelerators. For decelerators, *sensation of control in difficult situations* [CFI-C] was related to the most central medians (M6 & M7) of this second accelerative component. These medians correspond to the peak (30s), which has greater sympathetic control. In turn, the subject's *tendency to perceive difficult situations as controllable* [CFI-A], which may involve increased cognitive activity, was associated with M5 and M8 for decelerators (positively) and accelerators (negatively). It is possible that these central medians could have a greater influence of the parasympathetic system compared to M5 and M8. In future research it would be important to evaluate the involvement of cognitive flexibility in other components of the CDR, such as the first acceleration. Since the comparisons revealed significant differences between both clusters, it could be that this relationship is more

dichotomous.

Regarding *emotion regulation strategies*, it has been observed that they do not have the same effect on CDR. According to the current results, only *positive refocusing* and *planning* strategies have significant differences in median change scores between accelerators and decelerators. Nevertheless, certain strategies were linearly related to this psychophysiological measure. Thus, planning showed a linear effect on CDR but only for accelerators (being negatively related), and contrary to our expectations, *rumination* showed a negative linear effect for accelerators.

On the other hand, although the literature indicates that anxiety and depression have a clear effect on the CDR, and that this interaction is most likely the mechanism by which anxiety/depression and illness are related. In this sample, however, no significant differences have been observed between both clusters in relation to their scores in anxiety and depression. Likewise, no lineal relationship has been found between these variables and the CDR second acceleration. This could be due to the fact that part of the sample (decelerators) might score in subclinical anxiety and, as we have commented before, they do not present an accelerative response in the second acceleration of the CDR (Fuentes-Sanchez et al., 2019). Further research should assess the CDR pattern in clinical samples with different anxiety problems (Norte et al., 2019). Given that, as indicated by previous studies (McTeague & Lang, 2012), not all anxiety related psychopathologies affect this pattern in the same way. It would also be interesting to find out what features differentiate among them based on differences in the CDR components.

With regard to depression, although the comparison between the two groups did not reach the significance level, the results showed that decelerators did indeed score higher in depression than accelerators, as previously pointed out by Fuentes-

Sánchez et al. (2019). These results might be explained by certain features of this psychopathology that could influence the CDR. For example, in this study we found that rumination, an essential characteristic of depression, does have an effect on the second acceleration, related to attenuation of the cardiac response.

In conclusion, cognitive flexibility and emotion regulation strategies can be key to succeed when facing aversive events with more adaptive coping. Further works should investigate whether modifying these emotional regulation strategies or enhancing cognitive flexibility through cognitive-behavioral therapies could lead to modified outcomes in the cardiac defense response. Notwithstanding, our results could be due to the fact that the influence of anxiety and depression on CDR depends on more specific aspects such as the use of nonadaptive emotion regulation strategies (e.g., rumination or excessive worry). From a clinical perspective, we believe that it is certainly important to take into account this issue for future research, trying to evaluate the symptomatology in emotional disorders by means of more specific tools for particular features of these psychopathologies.

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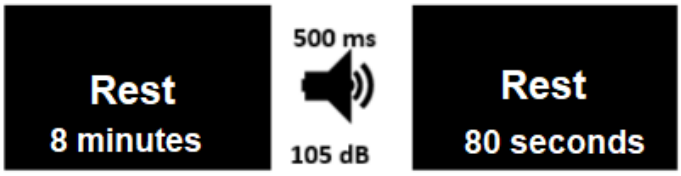

Figure 1. Physiological cardiac reactivity test for evoking the Cardiac Defense Response (CDR). Adapted from Fuentes-Sanchez et al. (2019).

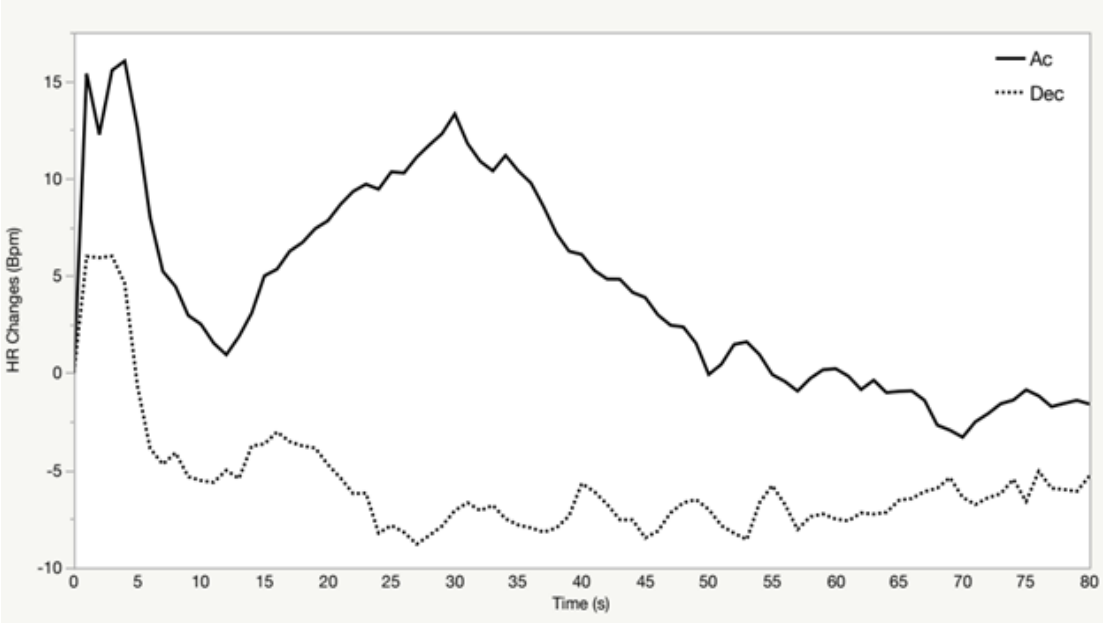

Figure 2. CDR pattern for accelerators (continuous line) and decelerators (dotted line), according to the 80 second-by-second HR averages (change scores deviated from 15s baseline). Clusters were calculated based on the second-by-second heart rate averages during the 20 to 45 seconds interval (A2).

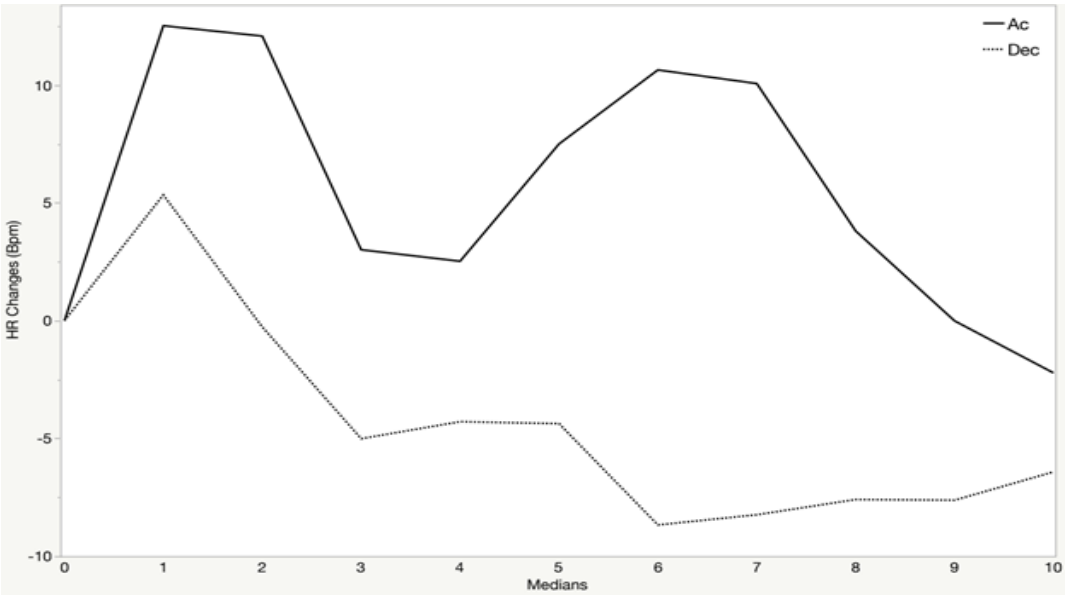

Figure 3. CDR pattern for accelerators (continuous line) and decelerators (dotted line), according to the 10 medians of HR (change scores deviated from 15s baseline). Clusters were calculated based on the M5-M8 interval corresponding to second acceleration (A2).

Table 1

Means (SD) and statistical comparison between decelerators and accelerators in the 10 medians of cardiac defense response (CDR).

	Accelerators Mean (SD)	*Decelerators* Mean (SD)	ANOVA	*T*	Levene	Wilcoxon
M1	12.52 (12.63)	5.33 (9.52)	11.92**	3.55**	$p > 0.05$	
M2	12.08 (13.64)	-0.26 (8.93)	32.74***	5.96***	$p > 0.05$	
M3	3.01 (12.13)	-5.01 (7.59)			$p = 0.01$	Z = -3.37**; ji2=16.71***
M4	2.52 (10.20)	-4.29 (7.93)	16.09***	4.11***	$p > 0.05$	
M5	7.50 (8.29)	-4.37 (7.48)	66.47***	8.24***	$p > 0.05$	
M6	10.65 (8.72)	-8.67 (8.10)			$p = 0.04$	Z = -9.19***; ji2=84.44***
M7	10.07 (9.58)	-8.24 (6.07)			$p = 0.03$	Z = -8.86***; ji2=78.63***
M8	3.81 (7,16)	-7.59 (5.97)	87.16***	9.51***	$p > 0.05$	
M9	-0.01 (6.25)	-7.62 (5.97)	45.80***	6.80***	$p > 0.05$	
M10	-2.22 (5.76)	-6.42 (6.50)	14.06**	3.70**	$p > 0.05$	

Notes: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Table 2

Student's t-test comparison between accelerators and decelerators on the self-report questionnaires.

	Accelerators Mean (SD)	*Decelerators* Mean (SD)	*t*
CERQ-A	12.87 (3.21)	13.57 (2.81)	-1.25
CERQ-PR	9.15 (3.60)	10.67 (3.53)	-2.30*
CERQ-PRL	13.89 (4.57)	14.80 (3.88)	-1.15
CERQ-PP	14.15 (3.47)	14.08 (2.44)	0.10
CERQ-SB	10.82 (2.83)	10.93 (2.79)	-0.22
CERQ-CA	8.29 (3.06)	8.43 (3.06)	-0.24
CERQ-RP	14.35 (3.36)	15.71 (2.94)	-2.31 *
CERQ-BO	7.33 (1.87)	7.69 (2.03)	-1.00
CERQ-RU	13.15 (3.41)	13.95 (3.24)	-1.30
CFI-C	3.86 (1.65)	4.54 (1,75)	-2.18 *
CFI-A	72.66 (8.79)	70.86 (8.29)	-1.14
ERQ-R	28.51 (5.63)	29.51 (4.94)	-1.01
ERQ-S	12.93 (4.94)	13.54 (4.37)	-0.71
HADS-A	7.71 (4.21)	7.59 (3.82)	0.16
HADS-D	3.40 (2.75)	2.87 (2.58)	1.07

Notes: self-blame (CERQ-SB), acceptance (CERQ-A), rumination (CERQ-RU), positive reappraisal (CERQ-PRL), planning (CERQ-RP), positive refocusing (CRQ-PR), putting into perspective (CERQ-PP), catastrophizing (CERQ-CA), other-blame (CERQ-BO), cognitive reevaluation (ERQ-R), expressive suppression (ERQ-S), control (CFI-C), alternatives (CFI-A), depression (HADS-D) and anxiety (HADS-A)

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Table 3

Separate correlations for accelerators and decelerators to assess the relationships between CDR medians corresponding to the second accelerative component (M5-M8 interval) and self-report questionnaire scores

	Decelerators				*Accelerators*			
	M5	M6	M7	M8	M5	M6	M7	M8
CERQ-A	0.11	0.00	0.04	0.14	0-02	-0.12	-0.18	-0.09
CERQ-PR	-0.03	0.15	0.09	0.10	0.08	-0.11	-0.04	0.02
CERQ-PRL	0.03	0.11	0.24	0.20	-0.06	-0.09	-0.14	-0.21
CERQ-PP	0.14	0,21	0.27	0.20	0.07	-0.13	-0.11	-0,05
CERQ-SB	-0.03	-0.15	-0.08	-0.10	-0.17	-0.07	0.07	0.00
CERQ-CA	-0,09	-0.21	-0.13	-0,02	-0.04	0.04	0.00	-0.04
CERQ-RP	-0.05	-0.03	0.10	-0.01	-0.26*	-0.31*	-0.30*	-0.31*
CERQ-BO	0.08	0.16	0.25	0.28*	-0.10	-0.15	-0.04	-0.05
CERQ-RU	0,03	-0,02	0.07	0.08	-0.12	-0.20	-0.28*	-0.28*
CFI-C	-0.02	0.32*	0.32*	0.20	-0.08	-0.80	-0.20	-0.08
CFI-A	0.31*	0.08	0.08	0.02	0.03	-0.14	-0.24	-0.25*
ERQ-R	-0.12	-0.03	-0.05	-0.07	0.01	-0.1	0.05	0.02
ERQ-S	-0.05	.0.04	-0.02	0.14	-0.11	-0.03	-0.07	-0.15
HADS-A	-0.03	-0.14	-0.08	-0.02	0.15	-0.08	-0.08	-0.05
HADS-D	-0.16	-0.20	-0.24	-0.06	0.02	0.19	0.21	0.07

Notes: self-blame (CERQ-SB), acceptance (CERQ-A), rumination (CERQ-RU), positive reappraisal (CERQ-PRL), planning (CERQ-RP), positive refocusing (CRQ-PR), putting into perspective (CERQ-PP), catastrophizing (CERQ-CA), other-blame (CERQ-BO), cognitive reevaluation (ERQ-R), control (CFI-C), alternatives (CFI.-A), expressive suppression (ERQ-S), depression (HADS-D) and anxiety (HADS-A)

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$