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Emotional anticipation rather than processing is altered in patients with vasovagal syncope ☆

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HIGHLIGHTS

- Syncopal patients show reduced cortical negativity in anticipation of unpleasant pictures.
- Syncopal patients and healthy individuals show similar electrocortical responses to arousing stimuli.
- Syncopal patients possibly engage in cognitive distraction to down-regulate emotional anticipation.

ABSTRACT

Objective: To investigate whether the electrocortical activity underlying the anticipation and processing of emotional stimuli is enhanced in individuals with recurrent episodes of vasovagal syncope (VVS).
Methods: Fifteen fainters and 15 age-matched healthy controls were presented a S1–S2 task, where the content of high-arousal pleasant and unpleasant, and neutral pictures (S2) was forecasted by word cues (S1). Stimulus Preceding Negativity (SPN) amplitude during the S1–S2 interval was computed as a measure of affective anticipation. The event-related potentials (ERPs) to S1 and S2 were measured to assess the processing of emotional warning stimuli and pictures.
Results: Relative to controls, fainters showed smaller P300 to warning cues anticipating emotional (and, particularly, unpleasant) pictures, and smaller SPN during anticipation of unpleasant pictures. No differences between groups were found with regard to ERP amplitudes during picture processing.
Conclusions: These results suggest that the anticipation, rather than the processing, of aversive stimuli is altered in syncopal patients.
Significance: The reduced cortical anticipation in fainters might reflect the use of non-adaptive emotion regulation strategies for reducing the impact of upcoming highly arousing (and, particularly, of unpleasant) events.

1. Introduction

Neurally mediated or vasovagal syncope (VVS) is a syndrome characterized by the co-occurrence of vasodilatation ("vaso") and vagally-mediated bradycardia ("vagal"), leading to hypotension and transient loss of consciousness and postural tone as a result of cerebral hypoperfusion (e.g., Van Lieshout et al., 1997; Benditt and Goldstein, 2002). VVS is the most common cause of fainting, accounting for up to 40% of syncopal events evaluated in the outpatient setting (Fenton et al., 2000).

VVS ensues when the balance between the mechanisms regulating blood pressure is temporarily disrupted, i.e., excessive venous pooling to the lower extremities due to decreased peripheral resistance is not counterbalanced by a compensatory rise in cardiac output. Vasodilation and bradycardia, instead of compensatory vasoconstriction and tachycardia, lead to hypotension and loss of consciousness (e.g., Van Lieshout et al., 1997; Fenton et al., 2000; Hainsworth, 2004).

Recurrent syncopal events of vasovagal nature may occur in otherwise healthy individuals in response to a variety of determinants. Although the proposed classifications may vary, it has been recognized that the potential triggers of VVS can be distinguished into physical factors, i.e., orthostatic stress and specific stimulation

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of sensory or visceral afferents, and psychological determinants, such as emotional distress, intense fear, sudden and severe painful experiences, or the sight of blood (see Mosqueda-Garcia et al., 2000; Moya et al., 2009). Because emotional factors are often pre-eminent, VVS is often referred to as "emotional fainting" (see Hamer and Bray, 2005). A central, rather than peripheral, neural pathway is believed to play a key role in the activation of emotionally-mediated syncope. This pathway would originate in neocortical and limbic structures of the central nervous system implicated in the evaluation and interpretation of emotional information, and descend from the cortico-hypothalamic centers to the brainstem autonomic nuclei that regulate cardiac and vasomotor activity through sympathetic and parasympathetic efferents (Kaufmann and Hainsworth, 2001; Kinsella and Tuckey, 2001; Diehl, 2005).

At least three different, but partially overlapping, interpretations of the psychological mechanisms involved in VVS have been put forward. According to Graham et al. (1961), VVS is a response to events which are likely to elicit anxiety. In the presence of a threatening situation, an emotional response involving subjective anxiety and increases in heart rate and blood pressure is initially elicited. With cessation of the threat, relief from anxiety is accompanied by a sudden and dramatic drop in both cardiovascular parameters, and fainting may ensue as a consequence.

Drawing from animal models, Engel (1978) ascribed the breakdown of the homeostatic mechanisms that normally maintain the reciprocal relationships between cardiovascular processes and somatic needs to the presence of an irresolvable conflict between contrasting action tendencies, namely fight–flight and conservation–withdrawal. Indeed, VVS typically ensues in situations characterized by uncertainty as to whether active escape or giving up is the most adaptive response to an impending (real or fantasized) threat or danger.

Sledge (1978) examined the psychological antecedents of VVS, and showed that fear, anxiety and feelings of helplessness precede fainting episodes when a threat to the body (e.g., receiving an injection or other medical procedures) or psychological harm (i.e., public ridicule and mortification) was expected in social situations. In all cases, the subject's interpretation of the social context, and the feeling that "giving in" to the perceived threat is necessary since active countermeasures would be socially inappropriate, highlight the role played by real or fantasized social pressures in the conflict between preparation for action and helpless resignation to the threat.

To summarize the psychosocial factors accompanying or preceding emotional fainting appear to include fear of bodily or psychological harm, anxiety, helplessness, and a sense of resignation to the perceived threat. Surprisingly, while altered anticipatory processing has been recognized as a critical component of some mental disorders, such as anxiety disorders (Barlow et al., 1996), none of the above-mentioned explanations of emotional fainting specifically emphasizes the role played by anticipation. Anticipation has a perceptual connotation, in that the individual awaits the occurrence of a stimulus. Also, anticipation is an active process, because the organism might be behaviorally passive while awaiting for perceptual input, but specific brain areas are already active before its actual delivery. The functional role of anticipatory processes is therefore to organize a set of changes in the state of the organism that ultimately allow for a more efficient interaction with expected upcoming stimuli (van Boxtel and Böcker, 2004). However, when brain activity underlying anticipation of relevant stimuli is either exaggerated or abnormally low (as has been observed in individuals with anxiety disorders of several types, depending on the specific experimental conditions used to investigate anticipatory processes; e.g., Proulx and Picton, 1984; Klorman and Ryan, 1980; Kimble et al., 2004), the individual's behavior becomes no longer flexible and adjusted to future environmental demands. On this basis, it is reasonable to hypothesize that in predisposed individuals the cascade of psychological events that culminates in fainting might not only involve abnormal responding, but also altered anticipatory responses to emotionally relevant or, more specifically, unpleasant situations.

In research using the event-related potentials (ERPs) to investigate the neural correlates of psychological processes, anticipation is usually studied by means of experimental paradigms that elicit a slow cortical potential, the Contingent Negative Variation (CNV; Walter et al., 1964; Brunia and van Boxtel, 2001; van Boxtel and Böcker, 2004). The CNV develops during the time interval between a warning signal (S1) and an imperative stimulus (S2), to which the subject is required to respond (e.g., with a button press). Importantly, a motor response to S2 is a sufficient, but not a necessary condition to elicit a negative potential between S1 and S2 (Brunia, 1988; Chwilla and Brunia, 1991; van Boxtel and Brunia, 1994). Non-motor CNV is termed Stimulus-Preceding Negativity (SPN). The SPN has been observed during the anticipation of stimuli with both positive (e.g., erotic pictures; Howard et al., 1992) and negative valence (e.g., a mild electric shock, or unpleasant slides; Lumsden et al., 1986; Rockstroh et al., 1989), and is therefore considered to reflect affective-motivational anticipation (Brunia et al., 2011). Irrespective of valence, the amplitude of the SPN is significantly larger preceding high rather than low arousal pictures, indicating that the SPN is modulated by the intensity of the motivational engagement ascribed to affective stimuli (Poli et al., 2007). The SPN preceding affective stimuli in picture-anticipation tasks shows a frontal maximum, that is particularly evident when the forthcoming stimulus is negative in valence (see Takeuchi et al., 2005). Importantly, cortical negativity is larger before S2 stimuli that are expected to be followed by aversive outcomes (Regan and Howard, 1995; Amrhein et al., 2005), suggesting that it might represent a psychophysiological indicator of expectancy of negative consequences.

In the present study, we investigated emotional anticipation in individuals with recurrent episodes of VVS and healthy controls, using a paradigm where the emotional content of high-arousal pleasant and unpleasant, and neutral pictures (S2) was signaled by congruent word stimuli (S1). The amplitude of the SPN preceding the onset of S2 was measured as an index of emotional anticipation. Moreover, the amplitudes of the P300 and the following Late Positive Potential (LPP) of the ERPs to word and picture stimuli were computed as classic indices of attentional engagement with emotional stimuli (Palomba et al., 1997; Cuthbert et al., 2000; Schupp et al., 2000).

Because in daily life emotional situations can trigger fainting episodes in patients with recurrent episodes of VVS, and considering that anticipating negative consequences from emotional stimuli is reflected in larger electrocortical negativity during anticipation (Amrhein et al., 2005), the hypothesis could be formulated that SPN in anticipation of high-arousal emotional stimuli would be enhanced in syncopal patients as compared with healthy individuals. Syncopal patients were also expected to deploy more attentional resources to emotional stimuli due to their relevance as major trigger factors. This would be reflected in larger P300 amplitude to emotional word stimuli and larger P300/LPP amplitude after emotional picture onset.

2. Methods

2.1. Participants

The study group consisted in 15 consecutive patients, aged 22–53 years, with recurrent syncopal episodes, referred to the Syncope Unit of the Cardiovascular Department of the Umberto I Hospital (Venice-Mestre, Italy) for head-up tilt testing evaluation. In order to be included in the study sample, patients had to meet the