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The genesis and presentation of anxiety in disorders of autonomic overexcitation☆

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ABSTRACT

Introduction: We investigated the genesis and presentation of previously–reported anxiety in disorders of autonomic overexcitation in relation to interoception, body vigilance and trauma to test our hypothesis that patients with the postural tachycardia syndrome (PoTS), vasovagal syncope (VVS) and essential hyperhidrosis (EH) represent atypical anxiety phenotypes in whom affective symptoms are more related to apprehension and vigilance of physiological (interoceptive) feedback than neurotic or trauma-related factors.
Methods: The Anxiety Sensitivity Index, Body Vigilance Scale, Self-consciousness Scale, Childhood Traumatic Events Scale and heartbeat tracking tasks were completed by 23 healthy controls, 21 PoTS, 20 EH and 20 VVS patients. Interoceptive accuracy (IA) was assessed during supine rest (9 min), isometric exercise (3 min), cold pressor (90 s) and head up tilt (HUT) (9 min).
Results: In comparison to controls, PoTS, VVS and EH patients reported increased symptoms of somatic anxiety but not of social anxiety/self-consciousness or trauma. Autonomic patients' IA was diminished and consistently underestimated even during autonomic arousal compared to controls. Controls and EH IA negatively correlated with somatic anxiety/hypervigilance, whereas PoTS and VVS IA and somatic anxiety/vigilance positively correlated.
Conclusions: Affective symptoms in PoTS, VVS and EH appear to be driven by anxiety and vigilance of physical sensations/symptoms, rather than trauma or neurosis. Increased somatic vigilance/anxiety in PoTS and VVS may be due to interoception being anxiogenic in these cohorts. Diminished interoception may be due to a common central dysregulation, as both sudomotor and cardiovascular forms of autonomic dysfunction had comparable IA deficits. These findings provide a possible therapeutic pathway for psychological symptoms in PoTS, VVS and EH.

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1. Introduction

Postural tachycardia syndrome (PoTS), vasovagal syncope (VVS) and essential hyperhidrosis (EH) are forms of intermittent dysautonomia; a set of conditions characterised by temporary dysregulation of normative autonomic function, in which sympathetic and/or parasympathetic responses evoked by day-to-day physiological challenges exceed what is required to maintain homeostasis and cause functional impairments that impact quality of life (QoL). Intermittent dysautonomia is typically expressed as orthostatic intolerance (OI) or thermoregulatory dysfunction (Mathias and Bannister, 2013). Postural tachycardia syndrome (PoTS) is defined by an excessive increase in heart rate (HR) (+30 BPM or HR >120 BPM) with palpitations and dizziness occurring within 10 min of standing upright (orthostasis) or head-up tilt (HUT), but without orthostatic hypotension (systolic blood pressure [SBP] fall of >20 mm Hg or >10 mm Hg diastolic blood pressure [DBP]) (Freeman et al., 2011). Vasovagal syncope (VVS) is the most common (~40%) form of syncope (Fenton et al., 2000), caused by excessive postural vasodilatation and/or bradycardia. VVS can be provoked both by physiological challenges, including injury, prolonged standing, dehydration or heat stress, and by psychological/emotional

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challenges. Essential hyperhidrosis (EH) is defined by excessive sweating, typically on the palms of the hands, soles of feet and axillae. EH can also be provoked both by mild exertion, mild heat stress and psychological/emotional challenges (Lai et al., 2014).

Autonomic symptoms are commonly experienced by patients with psychiatric diagnoses. These symptoms may be comparable to PoTS, VVS and EH, including sweating, faintness or palpitations. However, clinical diagnostic criteria of an autonomic disorder are rarely met (Ruchinskas et al., 2002; Lkhagvasuren et al., 2011). Conversely, comorbid, typically sub-clinical psychological (cognitive-affective) symptoms are common in patients with PoTS, VVS and EH (Giada et al., 2005; Gracie et al., 2006; Ruchinskas, 2007; D'Antono et al., 2009; Rios-Martinez et al., 2009). Patients with PoTS are more likely to report anxiety and panic symptoms. Both PoTS and panic disorder share psychological and physiological symptoms (Esler et al., 2004) but PoTS symptoms are attributable to a breakdown of peripheral autonomic reflexes (Masuki et al., 2007), in contrast to panic disorder's psychogenic sympathoexcitation (Coupland et al., 2003). Many patients with PoTS express maladaptive cognitive errors, including catastrophizing, which can add to functional disability and reinforces anxiety and somatic hypervigilance (Benrud-Larson et al., 2003). Alongside comorbid anxiety and depressive symptoms, patients with PoTS often describe poor sleep quality, fatigue (Bagai et al., 2011) and 'brain fog' (Ocon, 2013; Ross et al., 2013). Psychometry may reveal deficits in cognitive functioning, including impaired attention, short-term memory and recall abilities (Raj et al., 2009; Anderson et al., 2014). However, the psychological morbidity in patients with PoTS appears secondary to the primary autonomic pathology (Khurana, 2006; Masuki et al., 2007; Raj et al., 2009).

Depression, anxiety and blood-injection-injury phobia are common in VVS (Graham, 1961; Mcgrady et al., 2001; Luborsky et al., 1973; Karaca et al., 2007) and anxiety has been associated with increased risk of syncope during HUT (Cohen et al., 2000) and greater syncope burden (Lerma et al., 2013). VVS patients who do not respond to treatment are more anxious and depressed, report more negative thoughts regarding physical harm or death, and experience increased avoidance/protection coping behaviour and rumination (Gracie et al., 2006).

Among patients with EH, rates of anxiety are higher and underlying deficits in emotional processing, characterised by increased levels of alexithymia (inability to identify and describe emotions) are noted (Ak et al., 2013). Patients with EH report poorer QoL and increased social anxiety compared to patients with other dermatological diagnoses (Lessa et al., 2014). Thoracic sympathectomy can cause compensatory hyperhidrosis but may still improve subjective symptoms and QoL (Ramos et al., 2006). In summary, affective/emotional and sudomotor factors can be difficult to dissociate in EH.

Influential theories acknowledge the key contribution of peripheral physiological changes in the experience of emotions (James, 1894; Damasio, 1994). For example, 'somatic markers', such as a racing heart and breathlessness, enhance anxiety symptoms. The signalling and processing of internal bodily sensations, particularly those relayed by viscerosensory afferent nerves conveying autonomic state, are termed 'interoception'. Interoception contributes to homeostatic control through autonomic reflexes (e.g. baroreflex) and/or behavioural change. Correspondingly, the degree to which a person is sensitive to interoceptive signals is linked to emotional experience: e.g., people better at laboratory tests of heartbeat detection (judging when one's heart is beating) may experience emotions, notably anxiety, with greater intensity (Schandry, 1981; Wiens et al., 2000; Critchley et al., 2004). An individual's interoceptive accuracy (IA) moderates emotional and motivational behaviour (Damasio, 1999; Gray et al., 2012).

IA is a potential vulnerability factor for anxiety disorders (Dunn et al., 2010a), however, a more comprehensive account incorporates notions of attribution, expectation and prediction about bodily arousal. Discrepancies between expected and actual homeostatic signals (interoceptive prediction errors) contribute to anxious feelings. This effect can be exacerbated by imprecise interoceptive predictions and prior beliefs (Paulus and Stein, 2006). For example, an interoceptive prediction error occurs that is large-enough to reach conscious awareness when we feel dizzy, tachycardic or too hot, especially in a situation in which we consider these autonomic responses as inappropriate, based on previous experience and environmental information, such as observing others in the same environment but in an apparently dissimilar autonomic state. The interoceptive prediction error signals a disruption of homeostasis, creating a central high-order response, e.g., anxiety or behaviour modification (Garfinkel et al., 2014; Owens et al., under review; Ondobaka et al., 2015). Consistent with this proposal are observations that patients with autonomic failure, who cannot generate centrally driven states of autonomic arousal, show attenuation of some high-order emotional responses (Chauhan et al., 2008; Heims et al., 2006). Correspondingly, in patients with PoTS, VVS and EH, autonomic hyperactivity and interoceptive signals can interact in the over-expression of mood and anxiety symptoms (Eccles et al., 2015). One small study of patients with PoTS examined interoceptive ability but did not show differences from controls in cardiac IA (Khurana, 2014). However, patients with PoTS described more experience of different types of palpitations during testing, suggesting increased attention to cardiothoracic symptoms.

In this study, we investigated individual factors underlying the genesis and presentation of anxiety symptoms in disorders of transient autonomic overexcitation. Specifically, we examined how, interoceptive accuracy (IA), body vigilance and trauma interact with the expression of exaggerated autonomic responses in patients with PoTS, VVS and EH. Our central hypothesis was that apprehension and vigilance of interoceptive feedback provide better explanatory power for affective symptoms in these patients, than neurotic or trauma-related factors. We measured individual differences in IA using heartbeat detection tasks, both at rest and during autonomic arousal, and we assessed anxiety, body vigilance and history of trauma using questionnaires.

2. Methods

2.1. Participants

All experimental procedures were ethically approved by University College London Healthcare Trust Research and Design Office and the Imperial College London Research Ethics Committee. We recruited twenty-one patients with an established diagnosis of PoTS (19 female, mean age 36 years), twenty patients with diagnosis of VVS (13 female, mean age 37 years, 19 vasodepressor, 1 cardioinhibitory) and twenty patients with diagnosis of EH (5 female, mean age 46 years) alongside twenty healthy controls (13 females, mean age 35 years). Autonomic diagnoses were made after investigation at the Autonomic Unit, National Hospital for Neurology and Neurosurgery (University College London Hospitals) or the Autonomic and Neurovascular Medicine Unit, St Mary's Hospital (Imperial College Healthcare Trust). Written informed consent was provided by all participants prior to participation.

2.2. Interoception protocol

Ambient temperature of the treatment room was maintained at 20 °C throughout testing for all participants. Heart rate (HR) was recorded continuously (PowerLab 16/30, AD Instruments, Oxford, United Kingdom) and analysed offline.

During the 3rd, 6th and 9th minutes of supine baseline, participants carried out a heartbeat tracking task (Schandry, 1981), silently counting each heartbeat during an epoch of pseudorandom duration (21, 26, 36, 25, 35 or 45 s). Epoch length was taken from previous studies that have identifying optimum task windows (Pollatos et al., 2009; Dunn et al., 2010b). Participants were instructed to not manually take or touch their pulse and to declare that they could not feel their pulse against

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