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Chronic subjective dizziness: Analysis of underlying personality factors

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Abstract.
BACKGROUND: Chronic subjective dizziness (CSD) is characterized by persistent dizziness, unsteadiness, and hypersensitivity to one's own motion or exposure to complex visual stimuli. CSD may be triggered, in predisposed individuals with specific personality traits, by acute vestibular diseases. CSD is also thought to arise from failure to re-establish normal balance strategies after resolution of acute vestibular events which may be modulated by diathesis to develop anxiety and depression.
OBJECTIVE: To confirm the role of personality traits linked to anxiety and depression (i.e., neuroticism, introversion, low openness) as predisposing factors for CSD and to evaluate how individual differences in these personality traits are associated with CSD severity.
METHODS: We compared 19 CSD patients with 24 individuals who had suffered from periferal vestibular disorders (PVD) (i.e., Benign Paroxysmal Postural Vertigo or Vestibular Neuritis) but had not developed CSD as well as with 25 healthy controls (HC) in terms of personality traits, assessed via the NEO-PI-R questionnaire.
RESULTS: CSD patients, relative to PVD patients and HCs, scored higher on the anxiety facet of neuroticism. Total neuroticism scores were also significantly associated with dizziness severity in CSD patients but not PVD patients.
CONCLUSIONS: Pre-existing anxiety-related personality traits may promote and sustain the initial etiophatogenetic mechanisms linked with the development of CSD. Targeting anxiety-related mechanisms in CSD may be therefore a promising way to reduce the disability associated with CSD.

Keywords: Chronic dizziness, CSD, anxiety, introversion, openness, neuroticism, vestibular

1. Introduction

In the last twenty years there has been a renewed interest in studying the interaction between vestibular and anxiety disorders. In this context, Brandt and Dieterich [1] firstly described the Phobic Postural Vertigo (PPV) syndrome based on clinical observations that some patients have unexplainable forms of dizziness which do not fall into any of the classic neuro-otological diagnostic categories. PPV was therefore conceptualized as a set of symptoms including unexplained postural dizziness and fluctuating unsteadiness that are worsened by environmental and/or social stimuli. PPV may be triggered by vestibular disorders, medical illnesses, or psychological distress. Brandt and Dieterich [1] also indicated in the definition of PPV the presence of obsessive-

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compulsive personality traits, labile affect, as well as depression, anxiety, and autonomic disturbances. Successively, Staab [20] and Ruckenstein [16] proposed the term of Chronic Subjective Dizziness (CSD) to highlight the relationship between unexplained dizziness and the presence of pre-existing psychological and personality factors. Therefore, although PPV and CSD are broadly similar at the conceptual level, the CSD construct emphasizes more the importance of pre-existing personality traits as proneness to depression and anxiety in the pathophysiology of a form of dizziness that cannot be explained by other known causes. In contrast to PPV, anxiety-related personality traits are considered to be comorbid in CSD. Other researchers have also studied how personality traits may protect from the development of chronic dizziness. For example, Tschan et al. [25] found that subjective well-being (resiliency, sense of coherence, subjective quality of life) can reduce the risk of CSD. The same authors also suggested that patients' dispositional psychopathology and subjective well-being play a major predictive role in the long-term prognosis of dizziness, so patients should be screened for risk as well as protective factors, and offered psychotherapeutic treatment in case of insufficient coping capacity.

CSD is defined by three core symptoms [21]: 1) persistent dizziness that lasts at least three months; 2) hypersensitivity to moving stimuli, including self-motion, and 3) difficulty in visual precision tasks (e.g., using a computer) or exacerbation of symptoms in settings with complex visual stimuli (e.g. shopping malls). Another key clinical aspect of CSD is that spontaneous recovery is uncommon [21]. CSD may be triggered by otological, psychiatric, and other medical conditions that cause acute vertigo, unsteadiness, or dizziness. In particular, three common mechanisms have been reported in CSD: [20] 1) Psychogenic CSD: patients without history of vestibular or physical disorders, but with a pre-existing psychiatric illness, (e.g., panic attacks disorder), may experience dizziness during the course of their primary anxiety disorder, which precipitates the development of CSD; 2) Otogenic CSD: patients with no history of anxiety-related disorders may develope an acute vestibular disease (e.g., Benign Paroxysmal Postural Vertigo or vestibular neuritis) that in turn may induce anxiety and sustain CSD in predisposed individuals, and 3) Interactive CSD: Patients with history of anxiety disorder may develop CSD after a transient and acute episode of vertigo or dizziness triggered by any medical condition (e.g., pre-syncope, heart disease, etc.) including otological causes.

However, it is important to note that all of the events that preceed CSD (e.g., otogenic causes) are neither sufficient or necessary in themselves to provoke and/or sustain CSD. In fact, for most of the cases, the acute precipitants resolve completely and the patients fully compensate for any residual vestibular deficits without developing CSD. For some people, however, an underlying predisposing factor (e.g., personality traits or anxiety disorders) may prevent this compensation and allow the full expression and manifestation of CSD. This is why current models of CSD also theorize that the precipitating causes of CSD are associated with a persistent shift to high-threat postural and oculomotor control strategies even when the period of acute symptoms is fully resolved [10, 23].

The current cross-sectional study was designed to investigate the pre-existing personality factors that may be critical in predisposing individuals to develop CSD. To this end, we selected a group of Italian CSD patients who have had at least one otogenic event in their past medical history (i.e., Benign Paroxysmal Postural Vertigo (BPPV) or Vestibular Neuritis (VN)) and compared them with a group of age- and sex-matched individuals who had suffered from the same periferal vestibular disorders (i.e., BPPV and VN) but had not developed CSD over time (PVD group). Both these groups were also compared with healthy control individuals (HCs) with no history of any neurological, psychiatric, and vestibular disorder. Overall, our approach was analogous to that described in Staab et al. [23] who found that CSD patients tend to have higher neuroticism and lower extraversion and openness scores when compared to patients with other vestibular disorders that cause similar levels of disability. A recent study by our group [10] also revealed that high neuroticism, low extraversion, and low openness were associated with significant changes in the function of brain vestibular and anxiety networks and this reinforced the notion that these personality traits may be critical contributors to the pathophysiology of CSD. This study had two main aims: 1) to replicate the findings reported in Staab et al. [23] and confirm the fundamental role of neuroticism, introversion, and low openness as predisposing and precipitating factors for CSD; 2) to extend these results and study how variability in any of these key personality traits was associated with different levels of CSD severity. In other words, we tested the hypothesis that higher neuroticism, higher

Table 1
Neuro-psychological and demograhic variables across the groups. Data expressed as a mean ± SD. HC: Healthy Controls; CSD: patients with Chronic Subjective Dizziness; PVD: patients with peripheral vestibular disorders but not CSD; SD: Standard Deviation. GAD7: Seven-Item Generalized Anxiety Disorder; PHQ9: Nine-Item Patient Health Questionnaire; DHI: Dizziness Handicap Inventory

Neuro-psychological and demographic variables				
	HC ($n = 25$)	PVD ($n = 22$)	CSD ($n = 19$)	F/T/χ^2 (p-values)
Age	32.26 (7.11)	39.5 (11.3)	34.9 (12.6)	$F_{(2,63)} = 2.8$ (0.07)
Sex (F/M)	14/11	14/8	10/9	$\chi^2_{(2)} = 0.54$ (0.76)
Anxiety (GAD7)	6.1 (4.4)	6.2 (4.5)	9.3 (5.0)	$F_{(2,63)} = 3.2$ (0.05)
Depression (PHQ9)	4.9 (4.5)	6.1 (4.3)	7.8 (5.1)	$F_{(2,63)} = 2.14$ (0.12)
DHI	–	33.6 (21.0)	42 (22.3)	$T_{(39)} = 1.2$ (0.22)

introversion, and lower openness were linked with greater levels of dizziness in patients with CSD, relative to patients with PVD.

2. Methods

This study aimed at identifying three groups that would have been distinguished by specific personality variables (i.e., patients suffering from acute vestibular pathology that had not developed CSD, patients with past peripheral vestibular disorders who had developed CSD, and healthy controls).

During the enrolment we took particular attention to the vestibular assessment of the CSD and PVD patients including in the CSD group only those people with resolved vestibular disorders (i.e., BPPV and VN) who were still experiencing chronic dizziness symptoms in accordance to the published criteria for CSD [21]. In these patients, the percentage of vestibular asymmetry, as assessed via the Jongkees' formula [11] (percentage of reduced vestibular response) was systematically under 18% (mean 12.6%) and the recording of Vestibular evoked miogenic potentials (VEMPs) [2, 24] was within the normal limits. On the other hand, the vestibular patients not suffering from CSD were included only after at least one month from the documented return within a normal range of values of vestibular tests, although most of the VN/BPPV were still experiencing a mild degree of dizziness, which made it possible to compare them with the CSD patients in terms of disease severity.

2.1. Participants

The population examined was enrolled from patients attending the neuro-otological clinic at the University "Magna Graecia" of Catanzaro in Italy. In total, we included 66 patients (38 female, 28 male) divided into three groups:

a) 19 patients with CSD (4 BPPV/15 VN) (CSD group)
b) 22 patients with vestibular disorders who had not developed CSD (12 BPPV and 10 VN) (PVD group)
c) 25 healthy patients with no history of any neurological, psychiatric, and vestibular disorders (HC group)

Demographic and clinical data are reported in Table 1.

2.2. Evaluation

All the patients underwent a clinical-instrumental evaluation which included: 1) medical history, 2) Dizziness Handicap Inventory (DHI) [12, 15] 3) vestibular and neuro-otological evaluation (see 2.2.1) 4) psychometric evaluations through questionnaires: Seven-Item Generalized Anxiety Disorder (GAD7) [17], Nine-Item Patient Health Questionnaire (PHQ9) [9], revised NEO Personality Inventory (NEO-PI-R) [3], 5) Brain Magnetic Resonance with gadolinium contrast enhancement to evaluate the ponto-cerebellar angle and internal acoustic canals, and exclude the presence of any neurological pathology.

2.2.1. Audio-vestibular evaluation

Pure tone audiometry, tympanometry, and acoustic reflex testing.
Vestibulospinal reflexes assessment during gait and upright standing posture.
Voluntary eye movement.
Positional test (Pagnini McClure [14] and Dix Hallpike [4] maneuvres).
Head Shaking Test [7], Head Trust Test [8]
Caloric tests (Fitzgerald Hallpike) [6]
Cervical [2] and ocular [24] VEMPs (cVEMPs, oVEMPs).

The study and analysis of eye movements were carried out using the videonystagmography (VNG) software (Ulmer©Synapsys SA, Marseille, France).

2.3. Statistical analysis

Anova tests were used to evaluate the differences across groups in demographic variables, anxiety and depression scores, NEO personality traits, and dizziness severity. The Chi-squared test was used to test for differences in sex distribution across the three groups. Pearson's correlation coefficients between dizziness severity scores (as assessed via the DHI) and personality traits were also calculated in CSD and PVD patients separately, and a group by DHI and NEO-PI scores interaction was run to test the hypotheses that personality factors would have modulated the functional impairment due to dizziness differently in CSD relative to PVD patients.

Within the six facets of each main personality factor a Bonferroni correction for multiple comparisons was applied (i.e. $p < 0.05/6$). Consequently, a difference in each of the main five traits and in each facet of each factor was considered statistically significant if the p-value was less than 0.05 and 0.008 respectively. Moreover, within the six facets of each factor, we considered as trend the p-values that were less than 0.1 (after Bonferroni correction).

All statistical analysis were carried out using MATLAB®.

3. Results

The three groups were comparable in terms of demographic variables (age and sex), while the CSD and PVD were also similar in terms of depression scores and dizziness severity. An effect approaching significance ($p = 0.05$) was observed across the three groups for anxiety levels. This difference was mainly due to greater anxiety scores in CSD patients relative to the other groups. A summary of the demographic and clinical results is reported in Table 1.

In terms of differences in personality traits, we found that the three groups were significantly different in Neuroticism, and Openness (Table 2). The analyses assessing for interactions between personality traits, dizziness severity and diagnosis, evidenced that Neuroticism scores were positively correlated with dizziness severity in the CSD group ($r = 0.53$, $p = 0.02$) but less so in PVD patients ($r = 0.37$, $p = 0.09$) (Fig. 1).

4. Discussion

The recently introduced clinical concept of chronic subjective dizziness is based on the hypothesis that pre-existing personality traits predispose some individuals to develop a persistent form of dizziness that is no longer related to the initial triggering event [18, 19]. In particular, Ruckenstein and Staab [16] observed that the majority of patients with CSD may display pre-existing anxiety-related personality factors that exacerbate and sustain the typical CSD symptomatology even when the eliciting event is fully resolved and compensated. On the other hand, anxiety itself can be evoked by vestibular disorders even in non-anxious individuals and this is consistent with the mutual or bi-directional nature of the strong interactions between vestibular and anxiety systems [19].

Overall, our data, similarly to the previous study by Staab et al. [23], (which differed in the vestibular comparison group), confirmed the importance of neuroticism as a key pre-morbid personality factor underlying CSD. We also found that the anxiety facet of neuroticism was higher in CSD than PVD patients and when comparing CSD patients to healthy controls (although this latter result was only a a trend). Furthermore, and perhaps more importantly, total neuroticism scores were significantly associated with dizziness severity in CSD patients but not with similar levels of dizziness in patients with PVD. However, and in contrast with a previous study [23], we did not find evidence that intraversion and low openness were specifically linked with CSD, although lower openness scores were found in either CSD patients and PVD patients relatively to HCs. These may have depended on differences in the samples' characteristics between our and the previous study (e.g., age-range, sex, cultural effects) or may be linked with the higher dizziness, anxiety, and depression scores reported in the samples in Staab et al. [23]. Alternatively, it could reflect the fact that openness and extraversion are less important personality traits than neuroticism in predisposing people to develop CSD.

Taken together, our results support current pathophysiological models of CSD, which posit that pre-existing anxiety-related factors may exacerbate and sustain the initial mechanisms that are implicated

Table 2
NEO-PI-R T-scores of the traits and facets according to the Five factor Model of personality. Data are expressed in mean (±SD). ANOVA (analysis of variance) and *post-hoc t*-tests results are also shown. The values in bold represent the traits (and facets) which show statistically significant difference across or between groups ($p < 0.05$ for the main personality traits and $p < 0.008$ for the six facets of each trait). HC: Healthy controls; CSD: Chronic Subjective Dizziness; PVD: peripheral vestibular disorders, but not CSD; (SD): standard deviation, ns: not significant; t: trend of significance

	HC	PVD	CSD	F (*p*-values)	*Post-hoc t*-tests		
					HC-CSD	PVD-CSD	HCs-PVD
NEUROTICISM (N)	49.26 (9.9)	54.4 (7.9)	59.5 (11.7)	**5.95 (0.004)**	**3.2 (0.003)**	1.6^{ns}	2.01 (0.05)
Anxiety (N1)	51.1 (11.2)	54.7 (10.5)	63.3 (10.5)	7.1 (0.0096)	3.7 (0.036)	2.6^{t} (0.06)	1.1^{ns}
Angry Hostility (N2)	48.2 (8.2)	55.0 (9.3)	57.9 (11.9)	5.9 (0.0258)	3.2 (0.012)	0.9^{ns}	2.6^{ns}
Depression (N3)	49.9 (9.2)	55.0 (8.2)	55.6 (12.7)	2.2^{ns}	1.7^{ns}	0.2^{ns}	1.9^{ns}
Self-Consciousness(N4)	47.0 (10.4)	51.5 (8.5)	50.6 (9.9)	1.2^{ns}	1.2^{ns}	0.1^{ns}	1.4^{ns}
Impulsiveness (N5)	49.5 (9.6)	48.6 (6.9)	53.3 (11.4)	1.3^{ns}	1.2^{ns}	1.6^{ns}	0.3^{ns}
Vulnerability (N6)	50.7 (8.4)	56.0 (8.2)	61.7 (10.9)	**8.0 (0.0048)**	**3.8 (0.0024)**	1.9^{ns}	2.2^{ns}
EXTROVERSION (E)	53.7 (9.9)	51.81 (7.5)	49.2 (8.2)	1.5^{ns}	1.6^{ns}	1.1^{ns}	0.8^{ns}
Warmth (E1)	51.8 (7.4)	51.4 (10.3)	48.1 (7.3)	1.1^{ns}	1.6^{ns}	1.0^{ns}	0.3^{ns}
Gregariousness (E2)	55.4 (9.1)	56.8 (7.7)	52.3 (9.6)	1.4^{ns}	1.1^{ns}	1.7^{ns}	–0.6^{ns}
Assertiveness (E3)	53.5 (9.6)	49.7 (6.1)	50.9 (8.3)	1.2^{ns}	0.9^{ns}	–0.5^{ns}	1.5^{ns}
Activity (E4)	53.0 (8.9)	54.1 (7.3)	52.8 (9.9)	0.0^{ns}	0.1^{ns}	0.3^{ns}	–0.2^{ns}
Excitement Seeking (E5)	48.8 (9.5)	47.5 (7.3)	45.2 (10.4)	0.8^{ns}	1.2^{ns}	0.8^{ns}	0.5^{ns}
Positive emotion (E6)	52.6 (9.1)	48.8 (11.0)	47.6 (8.5)	1.8^{ns}	1.9^{ns}	0.3^{ns}	1.4^{ns}
OPENNESS (O)	55.3 (9.5)	46.55 (7.9)	46.6 (9.9)	**7.2 (0.006)**	**3.0 (0.024)**	*0.02*^{ns}	**3.4 (0.006)**
Fantasy (O1)	54.4 (11.1)	48.5 (8.3)	53.4 (10.8)	1.9^{ns}	0.3^{ns}	–1.5^{ns}	1.9^{ns}
Aesthetics (O2)	59.2 (8.1)	52,8 (7.8)	51.8 (9.4)	5.2 (0.048)	2.8 (0.042)	0.4^{ns}	2.7^{t} (0.06)
Feelings (O3)	53.9 (9.8)	47.5 (5.5)	46.5 (9.6)	5.0 (0.05)	2.5^{t} (0.06)	0.5 ^{ns}	2.6^{t} (0.06)
Actions (O4)	53.6 (12.7)	48.8 (9.5)	46.9 (11.4)	2.1^{ns}	1.8^{ns}	0.6^{ns}	1.4^{ns}
Ideas (O5)	52.8 (9.7)	45.0 (8.3)	44.4 (7.9)	6.7 (0.012)	3.1 (0.018)	0.3^{ns}	2.9 (0.03)
Values (O6)	44.5 (8.9)	41.7 (5.5)	42.3 (8.2)	0.8^{ns}	0.9^{ns}	–0.1^{ns}	1.1^{ns}
AGREEABLENESS (A)	**46.8 (7.2)**	**49.3 (9.2)**	**44.3 (8.7)**	**1.8**^{ns}	**1.1**^{ns}	**1.77**^{ns}	**1.32**^{ns}
Trust (A1)	46.6 (10.6)	45.6 (9.1)	41.4 (10.6)	1.5^{ns}	1.6^{ns}	1.3^{ns}	0.5^{ns}
Straightforwardness(A2)	46.8 (9.0)	51.8 (9.3)	45.5 (11.9)	2.2^{ns}	0.4^{ns}	1.8^{ns}	–1.8^{ns}
Altruism (A3)	52.8 (6.7)	51.6 (8.0)	49.0 (7.2)	1.5^{ns}	1.8^{ns}	0.9^{ns}	0.7^{ns}
Compliance (A4)	42.4 (8.2)	45.1 (9.9)	40.9 (13.6)	0.9^{ns}	0.4^{ns}	1.2^{ns}	–1.1^{ns}
Modesty (A5)	46.8 (9.2)	52.2 (8.6)	48.7 (10.9)	1.7^{ns}	–0.6^{ns}	1.0^{ns}	–1.9^{ns}
Tender-Mindedness(A6)	53.2 (5.4)	51.9 (9.0)	52.9 (7.6)	0.1^{ns}	0.1^{ns}	–0.3^{ns}	0.5^{ns}
CONSCIENTIOUSNESS(C)	52.8 (8.2)	49.5 (9.2)	47.6 (9.9)	1.97^{ns}	1.9^{ns}	0.6^{ns}	1.32^{ns}
Competence (C1)	49.5 (6.7)	47.8 (8.4)	44.2 (12.7)	1.9^{ns}	1.8^{ns}	1.1^{ns}	0.8^{ns}
Order (C2)	46.9 (11.4)	47.3 (8.0)	46.4 (9.6)	0.1^{ns}	0.2^{ns}	0.4^{ns}	–0.2^{ns}
Dutifulness (C3)	52.9 (8.1)	51.9 (9.4)	51.6 (12.5)	0.2^{ns}	0.4^{ns}	–0.1^{ns}	0.6^{ns}
Achievement Striving(C4)	53.5 (10.2)	48.5 (8.2)	50.2 (8.1)	2.1^{ns}	1.2^{ns}	–0.8^{ns}	2.0^{ns}
Self-discipline(C5)	52.9 (8.2)	48.3 (9.5)	48.8 (8.2)	2.5^{ns}	1.7^{ns}	–0.4^{ns}	2.0^{ns}
Deliberation (C6)	56.3 (7.3)	55.2 (7.9)	47.9 (12.8)	4.8^{t} (0.06)	2.8 (0.048)	2.2^{ns}	0.6^{ns}

in generating, promoting, and sustaining CSD over time [16].

Finally, it is important to emphasize that both ours and previous results may be relevant for future clinical trials aiming at preventing and limiting the burden of CSD via acting on anxiety-related personality factors. For example, a recent trial [18] showed that anti-anxiety and anti-depressant pharmacological treatments (e.g., sertraline) may be efficacious in reducing dizziness in CSD patients who do not display active neuro-otologic illnesses. However, it remains to be determined whether relevant personality factors such as neuroticism can predict the beneficial role of these drugs on CSD-related symptoms, especially in the long-term. In particular, it would be important to verify if neuroticism scores can discriminate between people who are more likely to respond, and to have a a prolonged response, to pharmacological treatments from those who are less likely to display benefits from the same therapies. Our data may also encourage future developments of psychological interventions in CSD with the aim of attenuating the strong influence of anxiety on the vestibular systems and *vice versa*. A recent psychotherapy trial by Edelman, Mahoney, and colleagues (2012, 2013) demonstrated sustained benefits of a brief cognitive behavioural therapy in patients with CSD, with improvements in physical symptoms,

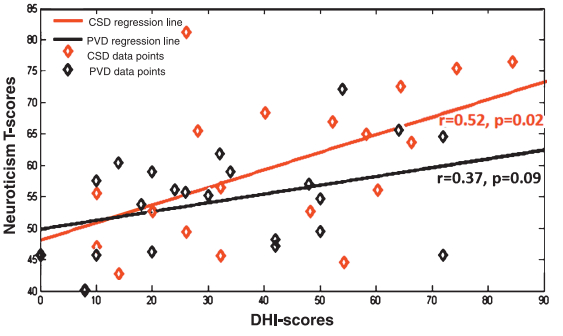

Fig. 1. Regression analysis between Neuroticism T-scores and DHI scores in the CSD and PVD groups. A positive correlation between the two variables is evident only in the CSD group (red line, $r = 0.53$, $p = 0.02$) but not in PVD (patients with peripheral vestibular disorders but not CSD) (black line, $r = 0.37$, $p = 0.09$). CSD: Chronic Subjective Dizziness; r: Pearson's correlation coefficient, p: *p*value.

disability, and functional impairment [5] which were sustained at six months post intervention [13].

All in all these data support the notion that targeting anxiety-related mechanisms in CSD may be a promising method to reduce the burden and disability associated with a common, complex but still too often overlooked chronic disorder.

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