Technische Universität München

Fakultät für Sport- und Gesundheitswissenschaften

Psychopathological Precursors and Sequelae of Functional Vertigo and Dizziness Disorders

From Diagnostics to Therapy

Dipl.-Psych. Univ. Katharina Radziej

Vollständiger Abdruck der von der Fakultät für Sport- und Gesundheitswissenschaften der Technischen Universität Müchen zur Erlangung des Grades eines

Doktors der Philosophie (Dr. phil.)

genehmigten Dissertation.

Vorsitzender:	Prof. Dr. Thomas Horstmann
Prüfer der Dissertation:	1. Prof. Dr. Jürgen Beckmann
	2. Prof. Dr. Claas Lahmann
	3. Prof. Dr. Anna Buchheim

Die Dissertation wurde am 03.04.2018 bei der Technischen Universität München eingereicht und durch die Fakultät für Sport- und Gesundheitswissenschaften am 15.02.2019 angenommen.

Acknowledgements

I would like to express my sincere gratitude to Professor Claas Lahmann for giving me the opportunity to dive deep into the research field of functional vertigo and dizziness and providing me with supervision and guidance throughout the past years.

I would also like to express my appreciation to Professor Jürgen Beckmann for being the advisor of this dissertation and Professor Thomas Horstmann for serving as chairman of the examination committee.

Further, I would like to thank my present and former colleagues and co-authors – Karina Limburg, Gabriele Schmid-Mühlbauer and Thomas Probst in particular – for the inspiring exchange and support.

Finally, I thank Max who persistently helps me keep my personal balance.

Table of contents

List of abbreviations 4

List of figures 5

List of tables 5

INTRODUCTION 6

THEORETICAL BACKGROUND 7

Vertigo and dizziness 7

Out of balance – Is it organic or functional or both? 7

PPV, CSD, PPPD – Functional VD has many names 9

Disease models of functional VD 11

Psychiatric disorders – Predisposition, cause or sequelae of VD 14

Treatment of functional VD 15

Research gaps and research questions 16

Study I: Psychological traumatization as precursor to VD disorders 16

Study II: Psychopathology in the course of VD disorders 17

Study III: Tailored group psychotherapy for functional VD 19

METHODOLOGY 21

The bigger picture: Overview of the two overall study phases 21

Diagnostic study: The Munich Diagnostic and Predictor Study of Somatoform Dizziness 21

Therapy study: Tailored Care for Functional Dizziness 24

A closer view: Study designs and measures 25

Diagnostic work-up across studies 25

Determination of measures 26

Operationalization of research questions 28

PUBLICATIONS 30

Study I – Abstract and individual contribution **30**

Study II – Abstract and individual contribution **32**

Study III – Abstract and individual contribution **33**

DISCUSSION 36

Theoretical contribution **36**

The role of psychological traumatization 36

The role of symptom-related fears and cognitions 38

Prospects of tailored group psychotherapy in functional VD 39

At a glance – Integration of results in an extended disease model 40

Clinical implications and conclusions **42**

REFERENCES 44

APPENDIX 54

Manuscript I **54**

Manuscript II **62**

Manuscript III **72**

List of abbreviations

ACQ	Agoraphobic Cognitions Questionnaire
BAI	Beck Anxiety Inventory
BDD	Bodily Distress Disorder
BDI-II	Beck Depression Inventory – revised version
BSQ	Body Sensations Questionnaire
CBT	Cognitive behavioral therapy
CSD	Chronic subjective dizziness
CTQ	Childhood Trauma Questionnaire
DSM	Diagnostic and Statistical Manual of Mental Disorders
FVS	Functional vestibular symptoms
ICD	International Classification of Diseases
ICVD	International Classification of Vestibular Disorders
IES	Impact of Event Scale
OVS	Organically explained vestibular symptoms
PDS	Posttraumatic Diagnostic Scale
PHQ	Patient Health Questionnaire
PPPD	Persistent postural-perceptual dizziness
PPV	Phobic postural vertigo
RCT	Randomized controlled trial
SCID-I	Structured Clinical Interview for DSM-IV-Axis I Disorders
SVD	Somatoform vertigo/dizziness
VD	Vertigo/dizziness
VHQ	Vertigo Handicap Questionnaire
VSS	Vertigo Symptom Scale
WHO	World Health Organization

List of figures

Figure 1 Primary and secondary functional VD

Figure 2 Cognitive-behavioral model of functional VD

Figure 3 Studies I and II in light of the cognitive-behavioral model of functional VD

Figure 4 Flow chart of study participation (diagnostic study)

Figure 5 Extended cognitive-behavioral model of functional VD

List of tables

Table 1 Overview of self-rated instruments administered in the respective Studies I-III

Introduction

Vertigo and dizziness are common complaints that can be deeply unsettling and hard to communicate. They frequently result in both body and mind being *out of equilibrium*. Often not clearly attributable to one single and treatable cause, symptoms tend to take a chronic course and have been termed a "heart-sink" complaint by general practitioners.

This dissertation project adds to identifying and evaluating psychological variables in terms of their descriptive and predictive validity in patients presenting with vestibular symptoms. The focus is set on psychological traumatization as a precursor to the frequent occurrence of functional vertigo/dizziness and psychopathological sequelae that develop secondary to the experience of these symptoms.

It is part of a research project that investigated clinical subgroups, correlates and predictors of functional vertigo/dizziness and aimed at linking results from a diagnostic study to the implementation of tailored psychotherapeutic care for patients in this severely burdened and therapeutically underserved population.

Theoretical background

Vertigo and dizziness

With a lifetime prevalence of approximately 30 % vertigo and dizziness (VD) rank among the most common somatic complaints in adults (Neuhauser, 2007; Tusa, 2009). The underlying causes of those symptoms vary widely, ranging from peripheral and central vestibular disorders or other organic causes to somatoform disorders and all combinations thereof. Therefore, the various causes of VD symptoms are scattered across a wide range of medical disciplines including neurology, otorhinolaryngology, psychosomatic and internal medicine.

Irrespective of their etiology, VD symptoms are often experienced as severely distressing. They are associated with handicap in daily and working lives (Lahmann, Henningsen, Brandt, et al., 2015; Neuhauser et al., 2008; Yardley, Masson, Verschuur, Haacke, & Luxon, 1992) as well as a significant reduction of both mental and physical health-related quality of life (Weidt et al., 2014). Yet a large part of patients presenting with VD in primary care do not receive a specific diagnosis (Kruschinski et al., 2008) making it less likely to receive adequate treatment in a timely manner. Efforts at the *Integrated Center for Research and Treatment of Vertigo, Balance and Ocular Motor Disorders* at the University Hospital Munich, have tried to tackle that problem over the past decade (Brandt, Zwergal, Jahn, & Strupp, 2009).

Out of balance – Is it organic or functional or both?

Previous approaches have tried to differentiate the various VD syndromes according to organic and somatoform (or functional) manifestations. In an epidemiological study that

included more than 4000 patients of an outpatient dizziness unit, the most frequent organic diagnoses included benign peripheral paroxysmal positional vertigo (BPPV; 18,8 %), central vertigo (13,2 %), vestibular migraine (9,1 %), vestibular neuritis (7,9 %), Meniere's disease (7,4 %), bilateral vestibulopathy (3,6 %) and vestibular paroxysmia (2,7 %; Strupp et al., 2003). Notably, in the same study, functional forms of VD constituted the single most frequent syndrome with a relative frequency of 19,5 %. Adding to this, Eckhardt-Henn et al. (2003) found that in up to 50 % of patients examined at a neurologic outpatient clinic, VD complaints were *not sufficiently explained* by a vestibular deficit or a defined organic illness. Among these patients a primary type of functional VD which occurs without prior organic vestibular disorder can be differentiated from a secondary type subsequent to an organically explained VD disorder (Eckhardt-Henn et al., 2008; see figure 1).

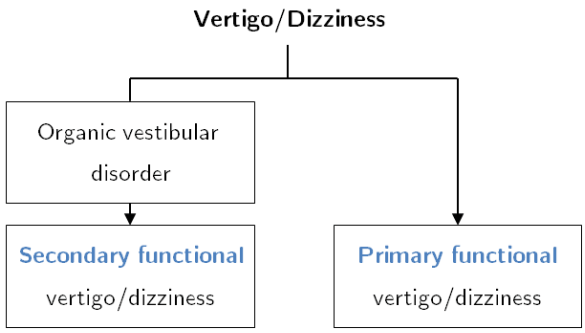


Figure 1. Primary and secondary functional VD

While plausible from a theoretical viewpoint, the individual decision to consider a symptom *organically explained* can be highly unreliable; the more so as it often cannot be stated in retrospect whether an organic illness preceded a VD manifestation with no signs of current organic pathology. The implied mind-body dualism is in line with former formulations of the category of somatoform disorders in the widely used *Diagnostic and Statistical Manual of*

Mental Disorders, 4th edition (DSM-IV-TR; American Psychiatric Association, 2000). Claims were made that the separation of mind and body should not be a part of any diagnostic nomenclature (Martin, 1999). The 5th edition of the DSM (DSM-5; American Psychiatric Association, 2013) addressed this problem: The various somatoform diagnoses (i.e., somatization disorder, undifferentiated somatoform disorder, hypochondriasis) were removed in favor of a more inclusive term, *Somatic Symptom Disorder* (SSD). In practical terms, this means that somatic symptoms, in our case VD, may or may not be associated with an identifiable organic condition. In order to qualify for an SSD diagnosis, positive criteria with regard to psychological concomitants of somatic symptoms have to be met, i.e., individual patterns of thoughts and feelings related to these symptoms. They may include disproportionate thoughts about the symptoms' seriousness, a persisting high level of anxiety about health or symptoms, and excessive time and energy devoted to these symptoms or health concerns (DSM-5 SSD C*riterion B;* American Psychiatric Association, 2013). This new diagnostic category has been shown to be highly prevalent in patients with VD disorders (Limburg, Sattel, Radziej, & Lahmann, 2016).

The beta draft of the upcoming revision of the *International Classification of Diseases* (ICD-11) includes *Bodily Distress Disorder* (BDD; WHO, 2015a) corresponding to the DSM-5 SSD. More importantly, it will also recognize a new and separate diagnostic category with regard to functional VD: *Persistent Postural-Perceptual Dizziness* (PPPD; WHO, 2015b).

PPV, CSD, PPPD – Functional VD has many names

Over roughly the past 30 years the varied presentations of functional VD inspired a number of (partly overlapping) nosological entities. In 1986, *Phobic Postural Vertigo* (PPV) was described as a syndrome of postural dizziness and fluctuating unsteadiness despite normal

clinical balance tests; VD attacks were described as being triggered by perceptual or social stimuli and frequently co-occurring with anxiety; the authors also observed obsessive-compulsive personality traits in the affected patients (Brandt, 1996; Brandt & Dieterich, 1986). In the further course, two variants of VD that could be triggered by exposure to visually rich or moving environments (such as passing traffic or moving through crowds of people) were proposed: *Space-Motion Discomfort* (Jacob, Lilienfeld, Furman, Durrant, & Turner, 1989; Jacob, Redfern, & Furman, 2009) and *Visual Vertigo* (Bronstein, 1995). In the early 21st century, Staab and colleagues added to the terminological variety when they introduced and further elaborated the concept of *Chronic Subjective Dizziness* (CSD; Staab et al., 2004; Staab & Ruckenstein, 2007; Ruckenstein & Staab, 2009). Mainly in accordance with the previous conceptualizations, it refers to persistent sensations of non-vertiginous dizziness and a heightened sensitivity to motion of self or objects in the environment. Finally, all of the above informed the diagnostic criteria of yet another diagnostic entity: Only recently did a subcommittee of the *Bárány Society* reach a consensus on the criteria of the aforementioned *PPPD* to be included in the *International Classification of Vestibular Disorders* (ICVD; Staab et al., 2017). Three main symptom clusters characterize the condition:

- Persistent dizziness, unsteadiness or non-spinning vertigo lasting more than 3 months
- Symptom exacerbation by upright posture, active or passive motion and exposure to moving visual stimuli or complex visual patterns
- Triggered by an event that caused acute vestibular symptoms (including but not limited to vestibular or neurological disorders and psychological distress)

PPPD will most likely be incorporated in the ICD-11 with all the earlier diagnostic labels being listed as synonyms (WHO, 2015b). A detailed specification of their overlapping features

as well as distinguishing characteristics is beyond the scope of this thesis, but can be found elsewhere (Staab et al., 2017).

The changing and at times vague terminology has likely slowed down research during the past decades and complicated concerted efforts in order to foster a better understanding and management of the conditions themselves. In the thesis at hand the term "functional VD" is used as an umbrella term of the various conditions described above.

Disease models of functional VD

With regard to the development and maintenance of medically unexplained or functional symptoms in general, a wide range of multi-component models have been proposed (e.g. Henningsen et al., 2007; Brown, 2004; Rief & Barsky, 2005; Rief & Broadbent, 2007; Deary et al., 2007). They describe a complex interplay of cognitive, emotional, behavioral, temperamental, physiological and social factors. Although functional somatic syndromes do show substantial overlap, research efforts have often been confined to specific syndromes (such as *Irritable Bowel Syndrome, Fibromyalgia* or *Chronic Fatigue Syndrome*). There is an on-going debate on whether there is essentially only one general functional somatic syndrome (Wessely & White, 2004). White, the opposer to this hypothesis, argues pragmatically that "*more progress has been made through splitting illnesses rather than lumping them together*" in understanding illness, etiology, treatment and outcome (p. 96).

On that note, Whalley and Cane (2017) elaborated a disease model that is specific to functional VD. Drawing heavily upon previous work by Staab (2012) and following the cognitive-behavioral tradition, (1) predisposing (*vulnerability*), (2) precipitating (*trigger*) and (3) perpetuating variables are factored into their model of pathogenesis.

Ad (1): Vulnerability factors include a heightened sensitivity to internal sensations, high trait anxiety, low trait extraversion, illness history and beliefs about one's own coping ability. They mainly come into effect in the way the affected person appraises and thinks about bodily symptoms like VD (see figure 2).

Ad (2): An acute vestibular crisis (e.g. an episode of BPPV or vestibular neuritis) or panic attack with focus on balance sensations may serve as dizzying triggers that lead to a balance control system dysfunction resulting in balance symptoms in the initial phase of the VD disorder. At this stage, *static compensation* (i.e., balance organ function is restored, e.g. when neurological processes in a vestibular migraine attack resolve or an individual recovers from an acute experience of panic) and *dynamic compensation* (e.g., unilateral vestibular dysfunction is compensated for centrally) may occur and alleviate symptoms. A range of psychological processes will impede this desired course, as described below.

Ad (3): First and foremost, the way people think about their illness and how they interpret body-related symptoms will shape their emotional and behavioral response. *Unhelpful appraisals* (e.g., "I'm having a stroke", "I'll never get better", "Everybody will think I'm drunk") will cause the patient to be worried, anxious or ashamed. Subsequently, behavioral and attentional adaptation strategies come into action. They include

- *avoidance behaviors* (e.g. avoidance of movements or triggering situations),
- *safety behaviors* (e.g. holding on for safety) and
- *attentional strategies* (e.g. constantly checking for signs of dysbalance, vigilance to potential threats in the environment, and 'up-weighting' of visual or somatosensory cues).

While being effective in keeping the individual safe during an acute phase of VD, an extended or generalized deployment of these strategies will contribute to the perpetuation of dizziness

and hypersensitivity to motion and visual stimuli. Beyond that, the updating of dysfunctional appraisals is prevented, thereby further upholding this vicious cycle. Figure 2 provides an overview of the mechanisms described above.

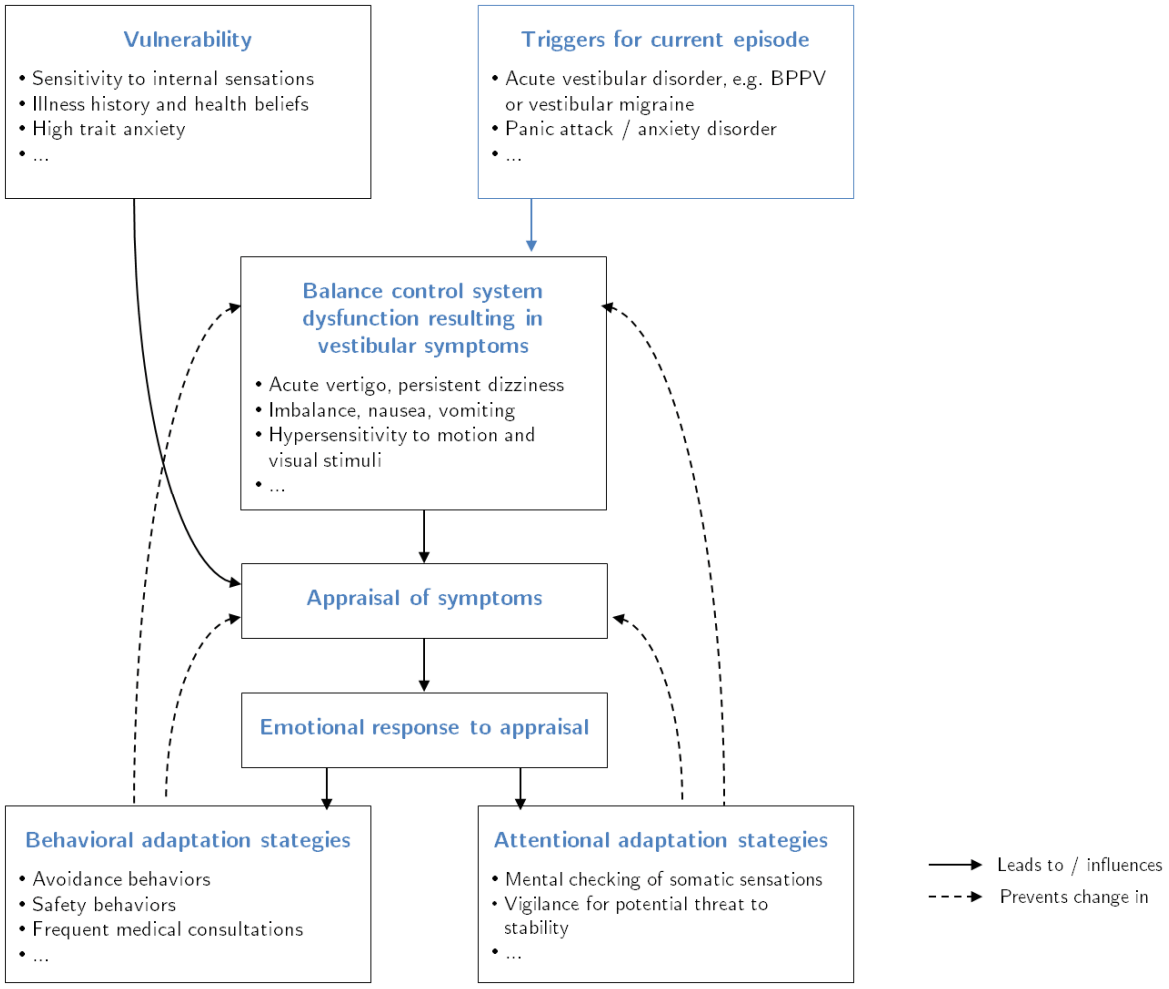


Figure 2. Cognitive-behavioral model of functional VD (modified after Whalley and Cane, 2017)

While this disease model is a VD-specific conceptualization it shares transdiagnostic mechanisms with earlier formulations of health anxiety (Salkovskis & Warwick, 1986) and cognitive models of panic disorder (Clark, 1986). Worthy of note, the model can be applied to

both primary and secondary forms of functional VD. It is also in line with the DSM-5 notion that SSD can result from an organic or non-organic cause.

Psychiatric disorders – Predisposition, cause or sequelae of VD

More often than not complaints of VD go hand in hand with elevated levels of psychopathology – a link that has frequently been described, even though the direction of the association remains difficult to ascertain. Thus, vestibular symptoms can occur as a manifestation of an underlying psychiatric disorder (*psychosomatic mechanism*; e.g. Eckhardt-Henn et al., 2009). Then again, vestibular disorders frequently cause, trigger or exacerbate secondary psychiatric comorbidities in the course of their disease (*somatopsychic mechanism*; e.g. Best et al., 2009a; Bigelow et al., 2016). Strictly speaking, vestibular and psychiatric illnesses may also co-occur independently.

A number of studies provide data on the frequent comorbidity of (both functional and organically explained) VD and psychiatric disorders. Almost half of the N=547 patients presenting with VD in a German tertiary care setting suffered from at least one psychiatric disorder which was associated with severe psychosocial impairment (Lahmann, Henningsen, Brandt, et al., 2015). The highest rates of psychiatric comorbidity have been reported for vestibular migraine, Meniere's disease, vestibular paroxysmia, and functional VD (Best et al., 2009a; Eckhardt-Henn et al., 2008; Lahmann, Henningsen, Brandt, et al., 2015); most common associations were found with anxiety and phobic (29–46 %), (other) somatoform (25–41 %) or depressive disorders (13–19 %). In line with this, a large cross-sectional representative survey of the US population found patients with VD disorders to have threefold increased odds of depression, anxiety and panic disorder (Bigelow et al., 2016).

Eckhardt-Henn et al. (2003) were the first to describe clinical subgroups of VD disorders according to their psychiatric concomitants. They argue that recognizing differences in psychopathological presentations of VD disorders – irrespective of initial etiology – is of importance in order to customize specific interdisciplinary treatment options for patients.

Treatment of functional VD

Despite the high prevalence and clinical relevance of functional VD disorders studies towards treatment options are still rare. Preliminary evidence indicates that selective serotonin reuptake inhibitors (SSRI) and serotonin norepinephrine reuptake inhibitors (SNRI) may alleviate symptom burden in chronic dizziness (Staab, 2012). Up till now, there is yet a lack of randomized controlled trials to support these treatments.

Psychotherapeutic options have yielded more promising results. A systematic review showed that there is some evidence on the effectiveness of cognitive-behavioural short-term therapy in combination with relaxation techniques or vestibular rehabilitation showing moderate effect sizes (Schmid, Henningsen, Dieterich, Sattel, & Lahmann, 2011). Yet these preliminary findings were based on only two RCTs (Andersson, Asmundson, Denev, Nilsson, & Larsen, 2006; Johansson, Akerlund, Larsen, & Andersson, 2001) and one controlled study (Holmberg, Karlberg, Harlacher, Rivano-Fischer, & Magnusson, 2006), all of which had methodological shortcomings, such as small sample sizes and lack of follow-up. None of the interventions ensured long-term improvements or an improvement of the associated psychopathology, most of all anxiety and depression.

More recent research efforts demonstrated that a psychoeducative group program, enriched by relaxation and balancing exercises, could lastingly decrease dysfunctional illness

representations in VD patients (Tschan et al., 2012). Another research group showed that a brief 3-session CBT intervention produced significant improvements in dizziness and symptom-related handicap, though again not in associated psychopathology (Edelman, Mahoney, & Cremer, 2012), that were maintained in the long-term (Mahoney, Edelman, & Cremer, 2013). Moreover, two uncontrolled studies reported positive effects of multimodal inpatient treatments (including mindfulness and cognitive-behavioral techniques in combination with vestibular and balance rehabilitation exercises) in patients with complex vestibular disorders; they reached a reduction in vertiginous symptoms as well as improvements in mood, functioning and mental health-related quality of life (Naber et al., 2011; Schaaf & Hesse, 2015).

Research gaps and research questions

Study I: Psychological traumatization as precursor to VD disorders

Psychiatric disorders, most commonly anxiety/panic disorder, depression or somatoform disorder, can take effect as the single and primary cause of vestibular symptoms. They account for approximately 8–10% of all patients presenting with a chief complaint of VD in tertiary care centers (Dieterich & Staab, 2017); though, more than one factor contributing to symptoms is rather the rule than the exception (Kroenke et al., 1992).

In particular, the link between anxiety and balance disorders has been the focus of extensive research efforts (Beidel & Horak, 2001; Furman, Balaban, Jacob, & Marcus, 2005; Jacob et al., 2009; Simon, Pollack, Tuby, & Stern, 1998; Sklare, Konrad, Maser, & Jacob, 2001; Staab & Ruckenstein, 2003; Wiltink et al., 2009). Somewhat surprising is the almost total lack of investigations of VD disorders with regard to the experience of posttraumatic stress.

Functional syndromes have very frequently been linked to childhood and lifetime traumatization (Andreski, Chilcoat, & Breslau, 1998; Kinzl, Traweger, & Biebl, 1995; Lahmann, Henningsen, Noll-Hussong, & Dinkel, 2010; Sack, Lahmann, Jaeger, & Henningsen, 2007; van der Kolk et al., 1996).

Despite this well-established association between trauma exposure and somatization, no study had reported evidence of this association in functional VD. Therefore, *Study I* was conducted in order to answer the following research questions:

(1) *Do functional and organic VD disorders differ in terms of their frequency of traumatizing childhood and lifetime experiences and severity of posttraumatic stress symptoms?*

(2) *Do trauma-related variables contribute to the severity of vertigo-related symptoms and anxiety as well as the physical and psychosocial handicap they entail?*

Study II: Psychopathology in the course of VD disorders

While psychiatric disorders can cause VD, psychological distress has been found to be even more prevalent *after* than *before* the onset of VD disorders (Eagger, Luxon, Davies, Coelho, & Ron, 1992). When VD and elevated levels of psychopathology co-occur, their intertwined relationship likely complicates the course of disease. Data from a tertiary care balance center showed that organic neurotologic conditions exacerbated preexisting psychiatric illnesses in roughly one third of patients; equally often, new depressive and anxiety disorders were triggered after the onset of the VD disorder (Staab & Ruckenstein, 2003). In another study including N=54 patients with objective evidence of a peripheral vestibular disorder, even two thirds of patients reported depression and/or anxiety since the onset of the vestibular symptoms; the majority of them scored above the cut-off point for significant psychiatric

morbidity (Eagger et al., 1992). Among nonclinical populations, dizziness also represented a risk factor for the subsequent development of depressive symptoms in a 1-year follow-up study (Nakao & Yano, 2006). Patients with a history of psychiatric disorder showed a higher risk of developing another psychiatric disorder after vestibular VD syndromes (Best et al., 2009a). In addition, patients who develop an anxiety or affective disorder in the course of their vestibular disorder frequently show poorer improvement and a high persistency of their vestibular symptoms (Best, Eckhardt-Henn, Tschan, & Dieterich, 2009b; Roh, Kim, Kim, & Son, 2018). The amount of vestibular deficit seems not to be associated with the amount of secondary psychiatric disorders (Best et al., 2009a).

In conclusion, there is consensus on the fact that psychological distress develops frequently after VD disorders, thereby further complicating balance symptoms and worsening both mental and physical health-related quality of life. Despite its clinical relevance, there is only limited understanding of the mechanisms fostering that development. Against this background and drawing upon a cognitive theory framework, *Study II* sought to explore the following research question:

(3) *Is the longitudinal link between VD symptoms and levels of depression/anxiety mediated by symptom-related fears and cognitions?*

For illustration purposes figure 3 depicts how *Studies I* and *II* relate to the cognitive-behavioral disease model of functional VD described above.

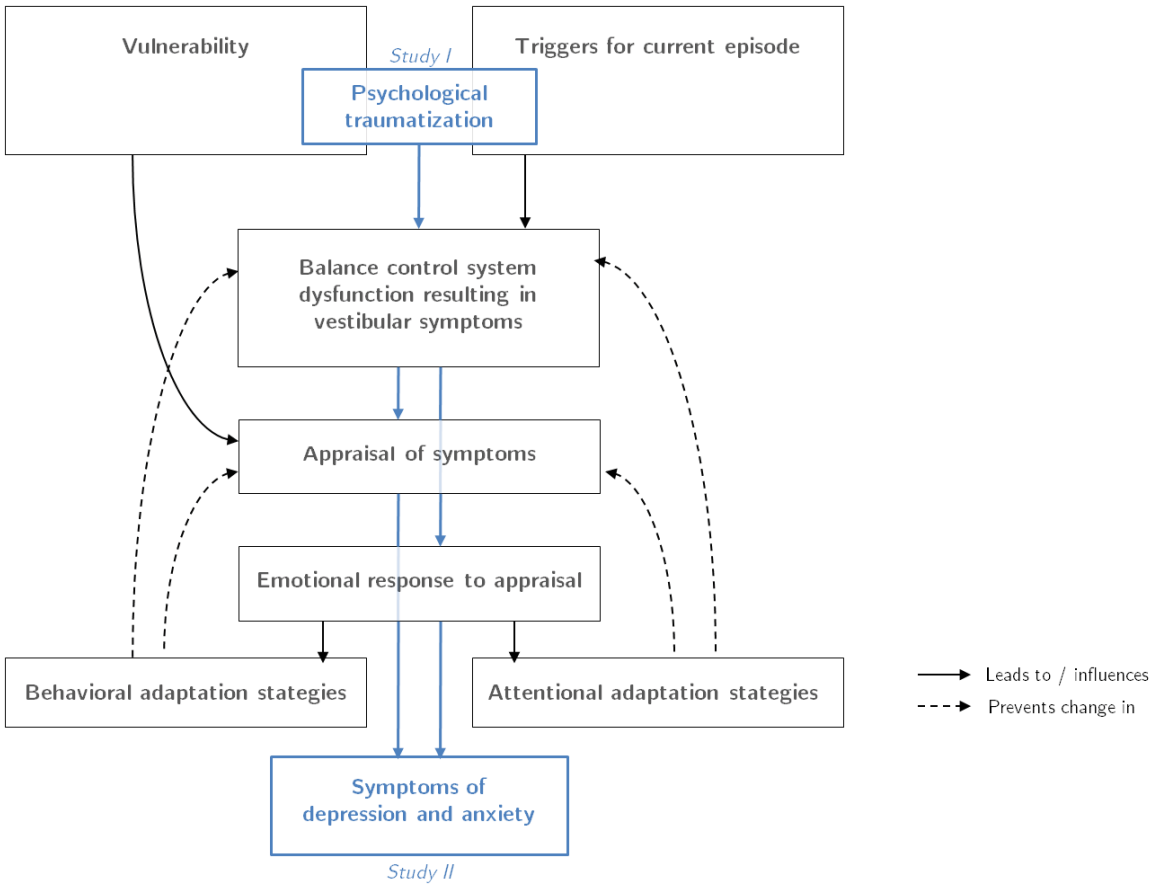


Figure 3. Studies I and II in light of the cognitive-behavioral model of functional VD

With regard to *Study I* there was no hypothesis whether previous trauma served as precipitator, i.e. trigger of VD symptoms, or vulnerability factor affecting symptom severity – hence its placement in figure 3, overlapping these two components within the model.

Study II introduces secondary psychiatric symptoms into the disease model and relates to symptom appraisal (*cognitions*) and emotional response (*fears*) as possible mediators.

Study III: Tailored group psychotherapy for functional VD

In summary, evidence on therapeutic options in functional VD remain unsatisfactory, especially when the need for therapeutic options tailored to the frequently associated psychiatric comorbidities is considered.

It was hypothesized that specific therapy modules tailored to the associated subgroups of mental disorders in patients with functional VD could most likely improve this situation. An intervention protocol was developed that combined proven therapeutic strategies from previous studies in a 16-session group therapy approach. The group setting was chosen since it had shown promising results in patients suffering from somatoform disorders before (Naber et al., 2011; Rief, Bleichhardt, & Timmer, 2002; Tschan et al., 2012). As one of the basic components balance rehabilitation exercises were included; their parallels to interoceptive exposure to feared sensations in CBT have been outlined before (Staab, 2011). Over and above these, the treatment concept comprises strategies in order to reduce avoidance and safety behaviors, explore and update appraisals on the cause and at times threatening experience of VD symptoms, as well as relaxation, mindfulness and acceptance techniques. More importantly, it includes therapeutic modules specifically adapted to the three most relevant psychiatric comorbidities in functional VD: anxiety/panic, depression and somatoform disorders. Eventually, *Study III* was conducted in order to answer the following research questions:

(4) *Will the integrative group psychotherapy program result in long-term clinically relevant improvements in VD-related quality of life?*

(5) *Will the integrative group psychotherapy program also alleviate associated psychopathology, namely symptoms of depression, anxiety and somatisation, as well as VD symptoms themselves?*

Study III also served as preparatory work for an on-going RCT (Lahmann, Henningsen, Dieterich, Radziej, & Schmid, 2015).

Methodology

This dissertation was conducted within the scope of a research project supported by funding from the German Federal Ministry of Education and Research within the framework of the *Integrated Research and Treatment Centers* program.

First, this chapter will give an overview of the research framework program comprised of two study phases covering diagnostic and therapeutic approaches to VD disorders. Second, a closer look will be taken at the methodology of the respective studies I-III of this dissertation.

The bigger picture: Overview of the two overall study phases

During the past decade joint research efforts of the *Department of Psychosomatic Medicine and Psychotherapy* (Klinikum rechts der Isar, Technical University of Munich) and the *German Center for Vertigo and Balance Disorders* (University Hospital Munich, Großhadern) – an interdisciplinary center for research and treatment of vertigo, balance and ocular motor disorders – have been directed towards a better understanding of the causes and course of functional VD disorders in order to improve diagnosis and treatment options in this understudied clinical population.

Diagnostic study: The Munich Diagnostic and Predictor Study of Somatoform Dizziness

The *Munich Diagnostic and Predictor Study of Somatoform Dizziness* which has been described in detail elsewhere (Lahmann et al., 2012) was conducted between May 2010 and June 2012. It incorporates a prospective study design with baseline and two follow-up assessments after six and twelve months. The study aimed at investigating diagnostic

subgroups, correlates and predictors of functional dizziness. Patients were recruited via routine care appointments at the *German Centre for Vertigo and Balance Disorders*. A total of 686 out of 860 patients screened for eligibility had given their consent to participate in the study. Figure 4 provides a flow chart of study participation including information on exclusion criteria, reasons for refusal to participate and organizational reasons why full data could not be obtained.

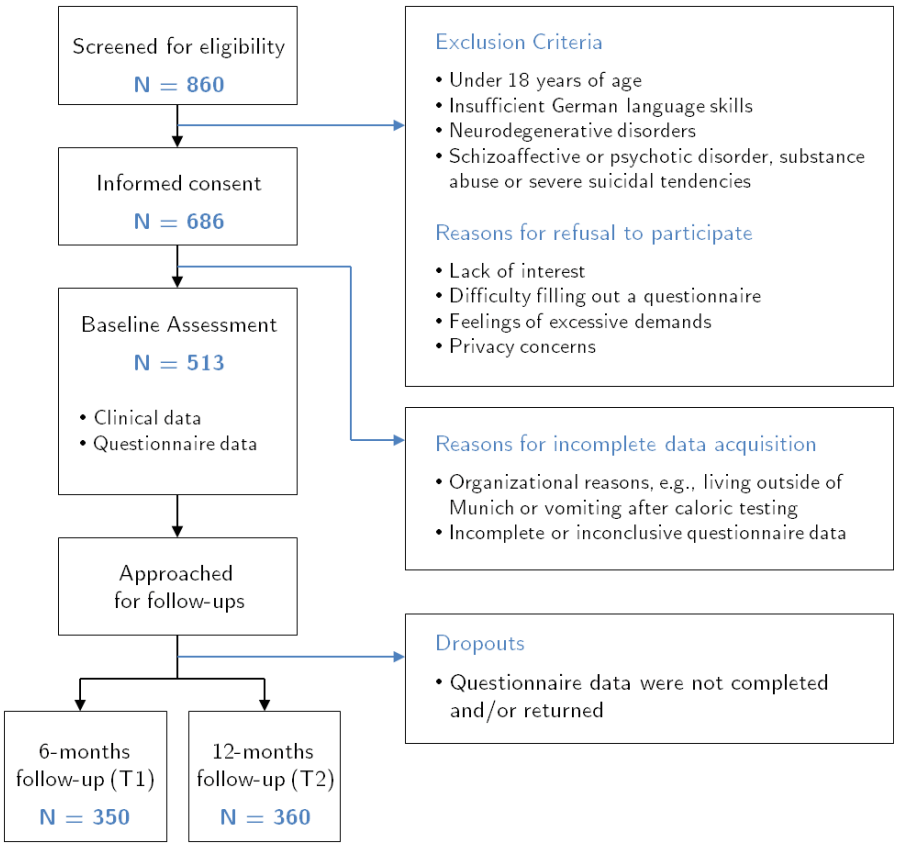


Figure 4. Flow chart of study participation (diagnostic study)

At the baseline assessment point, all participants completed a test battery of self-rated questionnaires measuring the severity of VD symptoms and handicap, depression, anxiety, dissociative and traumatic experiences, somatic factors, body- and illness-related cognitions,

attachment, quality of life and experiences with the health care system. A condensed version of this test battery was applied at follow-up assessments. Additionally, at baseline a structured clinical interview (*SCID-I*; Wittchen et al., 1997) was conducted to assess mental disorders. Neurologists documented somatic diagnoses and completed questionnaires regarding the subjective doctor-patient relationship.

Studies I and *II* of this thesis were conducted within the context of the *Munich Diagnostic and Predictor Study of Somatoform Dizziness*. Beyond that, the following research issues were addressed and published:

- Psychiatric comorbidity and psychosocial impairment among patients with VD (Lahmann, Henningsen, Brandt, et al., 2015)
- DSM-5 Somatic Symptom Disorder in patients with VD (Limburg et al., 2016)
- Course and predictors of DSM-5 Somatic Symptom Disorder in patients with VD (Limburg et al., 2017)
- Psychological distress as the link between VD symptoms and VD-related handicap (Probst et al., 2017)

Funding. Parts of this project were supported by funding from the German Federal Ministry of Education and Research (Grant Code 01 EO 0901).

Ethics approval. The study was approved by the Ethics Committee of the University of Munich (ref. 108-10).

Therapy study: Tailored Care for Functional Dizziness

Based on results from the diagnostic study a clinical trial was planned in order to evaluate a manualised short-term integrative psychotherapy taking place in a group setting that was tailored to clinical subgroups of functional VD (as described in the previous section of the manuscript at hand). The study was designed as a two-armed single-center randomized controlled open clinical superiority trial with a parallel active control intervention and balanced randomisation (for a detailed description of the study design see Lahmann, Henningsen, Dieterich, et al., 2015). Within the scope of this RCT, the therapeutic intervention will be compared to a moderated self-help group on several psychometric self-report measures assessing social and physical handicap due to VD (*primary endpoint*), severity of VD symptoms, physical and mental health-related quality of life, as well as severity of depression, anxiety and somatisation (*secondary endpoints*). Assessment points include pre-treatment (T0), post-treatment (T1) and 12-months follow-up (T2).

A pilot run (N = 12) of the treatment protocol was conducted in order to ensure acceptance and feasibility of the trial and refine therapeutic strategies if needed. Results from this pilot trial are reported in *Study III*. The manuscript outlines the therapy concept and gives further details on the specific treatment elements.

Funding. This project is supported by funds from the German Federal Ministry of Education and Research (Grant Code 01 EO 1401).

Ethics approval. This study was approved by the Ethics Committee of the University of Munich (ref: 319-14).

A closer view: Study designs and measures

This chapter provides an overview of the way the research questions of each study were operationalized and tested for. More detailed accounts of the applied measures and statistical procedures are given in the three published manuscripts.

Diagnostic work-up across studies

All participants were recruited via routine care appointments at the *German Centre for Vertigo and Balance Disorders* and gave written informed consent. They underwent structured history-taking and a systematic and standardized physical examination. The diagnostic work-up included complete neurological, neuro-otological and neuro-ophthalmological examinations. Vestibular testing comprised the video head impulse test, measurement of the subjective visual vertical and ocular torsion, as well as video-oculography with caloric irrigation. Clinical diagnoses were based on established criteria (e.g., Brandt et al., 2013), more specifically so with regard to vestibular migraine (Radtke, Neuhauser, von Brevern, Hottenrott, & Lempert, 2011), Meniere's disease (American Academy of Otolaryngology Head and Neck Surgery, 1995), and vestibular paroxysmia (Brandt & Dieterich, 1994). Patients were classified as having functional VD symptoms if either no structural dysfunction could explain their symptoms (i.e., *not organically explained*) or if symptoms went beyond what is to be expected from a current structural dysfunction (i.e., *not sufficiently organically explained*). The co-occurrence of multiple VD diagnoses, including the combination of both organic and functional diagnoses, was possible if appropriate.

Determination of measures

Due to a lack of biological markers other validation criteria for the severity of experienced functional VD symptoms were needed. In order to assess the magnitude of VD symptoms, the patient-rated *Vertigo Symptom Scale* (VSS; German version by Tschan et al., 2008) was applied. In addition, the *Vertigo Handicap Questionnaire* (VHQ; German version by Tschan et al., 2010) addressed physical and psychosocial impairments, covering restrictions of activity, social anxieties, and fears associated with VD. This approach is in line with the aforementioned diagnostic criteria redefinitions in DSM-5; herein SSD diagnosis requires somatic symptoms to be "*distressing or result in significant disruption of daily life*" (DSM-5 SSD, *Criterion A*; American Psychiatric Association, 2013).

In the therapy study a VHQ sum score above 45 points was adopted as inclusion criterion in order to ensure a sample of patients with sufficiently severe impairment (i.e., identifying those who need medical care). VHQ scores were also applied as primary outcome variable. This was chosen according to the primary goal of the psychotherapeutic intervention, which was to help patients cope with symptoms and improve overall functioning and only secondly to alleviate specific somatic symptoms such as VD.

Beyond VSS and VHQ, a battery of questionnaires, including information on key demographic characteristics, was administered to patients across studies. All of the self-report inventories are standardized instruments proven to be sufficiently reliable and valid and are widely used in clinical practice. Table 1 gives an overview of all the questionnaire-based measures that were applied across the studies.

Table 1. Overview of self-rated instruments administered in the respective Studies I-III

Instrument	**Construct**	**Reliability / Validity** (Reference)	**Study I**	**Study II**	**Study III**
Vertigo Symptom Scale (VSS)	Vertigo/dizziness symptoms	Good / moderate (Tschan et al., 2008)	✓	✓	✓
Vertigo Handicap Questionnaire (VHQ)	Handicap due to vertigo/dizziness symptoms	High / moderate (Tschan et al., 2010)	✓	-	✓
Childhood Trauma Questionnaire (CTQ)	Retrospective assessment of childhood abuse	Good to high / acceptable to moderate (Bader et al., 2009)	✓	-	-
Posttraumatic Diagnostic Scale (PDS)	Assessment of traumatic life events	High / high (Foa et al., 1997)	✓	-	-
Impact of Event Scale (IES)	Severity of post-traumatic stress symptoms	Good to high / moderate to high (Ferring & Filipp, 1994; Horowitz, Wilner, & Alvarez, 1979)	✓	-	-
Body Sensations Questionnaire (BSQ)	Somatic symptom-related fears	Good / acceptable to moderate (Ehlers & Margraf, 2001)	-	✓	-
Agoraphobic Cognitions Questionnaire (ACQ)	Somatic symptom-related cognitions	Acceptable to good / acceptable to moderate (Ehlers & Margraf, 2001)	-	✓	-
Beck Depression Inventory (BDI-II)	Depression	Good to high / moderate to high (Kühner et al., 2007)	-	✓	✓
Beck Anxiety Inventory (BAI)	Anxiety	Good to high / moderate to high (Margraf & Ehlers, 2007)	-	✓	✓
Patient Health Questionnaire (PHQ-15)	Somatic complaints	Acceptable to good / moderate (Gräfe et al., 2004)	-	-	✓

Operationalization of research questions

The respective research questions were operationalized as follows. Details on statistical analyses are provided in the respective manuscripts.

Study I

Research Questions

(1) *Do functional and organic VD disorders differ in terms of their frequency of traumatizing childhood and lifetime experiences and severity of posttraumatic stress symptoms?*

(2) *Do trauma-related variables contribute to the severity of vertigo-related symptoms and anxiety as well as the physical and psychosocial handicap they entail?*

Study I takes a cross-sectional approach to exploring the role of psychological traumatization and adverse life events in patients with organic and functional VD symptoms. Based on a thorough diagnostic assessment as described above, patients were categorized into the group of (A) *organic VD* symptoms or (B) *functional VD* symptoms. Regarding research question (1) both groups were compared on self-report measures evaluating the occurrence of childhood trauma and abuse (CTQ), the experience of other traumatic life events (PDS), and the impact and severity of traumatic disorders (IES; *difference hypotheses*). In order to test the *correlation hypotheses* of research question (2), simple and multiple linear regression analyses were computed. Trauma-related variables (PDS, CTQ and IES) served as independent variables; separate univariate regression analyses were performed for each dependent variable, namely VSS and VHQ subscales, assessing VD symptoms and the handicap due to VD symptoms respectively.

Study II

Research Question

(3) *Is the longitudinal link between VD symptoms and levels of depression/anxiety mediated by symptom-related fears and cognitions?*

Study II takes a longitudinal approach with baseline (T0), 6-months (T1) and 12-months (T2) follow-up measurements. Regression-based mediation analyses (Preacher & Hayes, 2008) were computed to test the hypothesis that fear of bodily sensations (as indicated by BSQ scores at T1) and cognitions about these symptoms (as indicated by ACQ sum and subscale scores at T1) would longitudinally mediate the effect initial VD symptoms (VSS at T0) have on the development of anxiety (BAI) and depression (BDI-II) after one year (T2).

Study III

Research Questions

(4) *Will the integrative group psychotherapy program result in long-term clinically relevant improvements in VD-related quality of life?*

(5) *Will the integrative group psychotherapy program also alleviate associated psychopathology, namely symptoms of depression, anxiety and somatisation, as well as VD symptoms themselves?*

In the therapeutic pilot trial patients received integrative group psychotherapy and were assessed before the start of treatment (T0), after the end of treatment (T1) and at 1-year follow-up (T2). With regard to research question (4), improvements on scores of VD-related handicap (VHQ) served as primary endpoint. Short-term (pre – post) and long-term (pre – follow-up) changes on measures of depression (BDI-II), anxiety (BAI) and somatisation (PHQ-15) served as secondary endpoints relating to research question (5).

Publications

This present work is submitted as a cumulative thesis and is based on three research papers which have been accepted for publication in peer-reviewed scientific journals. This chapter gives an overview of the three studies including the abstracts and individual contributions of the PhD candidate to each manuscript. Published manuscripts I-III can be found in the Appendix of this thesis.

Study I – Abstract and individual contribution

Title:	**Psychological traumatization and adverse life events in patients with organic and functional vestibular symptoms**
Authors:	Radziej, Katharina (first author) Schmid, Gabriele Dinkel, Andreas Zwergal, Andreas Lahmann, Claas
Publication status:	Published in *Journal of Psychosomatic Research*, Volume 79, pp. 123-129 (2015)

Abstract

Objective. A relationship has frequently been found between a history of traumatisation and the existence of somatoform symptoms. The objective of this study was to examine whether this relationship is also observed for functional, i.e. medically not sufficiently explained, vestibular symptoms (FVS). It was tested whether patients with FVS and organically explained vestibular symptoms (OVS) differ with regard to frequencies of previous traumatic

experiences and posttraumatic stress symptoms. The study also explored whether the impact of previous trauma was associated with characteristics of vestibular symptoms and handicap.

Methods. Patients with a diagnosis of OVS (N=185) or FVS (N=158) completed questionnaires about potentially traumatizing experiences (e.g., Childhood Trauma Questionnaire, Impact of Event Scale) and vertigo-related symptoms and handicap (Vertigo Symptom Scale, Vertigo Handicap Questionnaire).

Results. No differences were found between the two patient groups with regard to number or impact of traumatic life events. However, regression analyses across groups revealed that, regardless of their diagnosis, prior traumatic experiences and the presence of posttraumatic stress symptoms including avoidance and intrusion predicted to some extent higher overall VS-related balance symptoms and autonomic symptoms of vertigo-related anxiety.

Conclusion. Exposure to trauma and symptoms of posttraumatic stress can contribute to symptom severity and handicap experienced by patients with vestibular symptoms irrespective of their original cause, most likely serving as predisposing, modulating or perpetuating factors.

Individual contribution

The PhD candidate (KR) is the main author of this paper. KR conceived the research question in agreement with Prof. Claas Lahmann and developed the theoretical framework for the reanalysis of the data derived from the large diagnostic study (as described in the Methodology section). KR was not in charge of the data collection itself, but was responsible for data management, carried out the statistical analyses of the data and wrote this manuscript independently in agreement with and integrating feedback from the co-authors.

KR was also responsible for the whole submission process by integrating reviewers' feedback (including major revisions and further analyses) into the paper.

Study II – Abstract and individual contribution

Title:	**The longitudinal effect of vertigo and dizziness symptoms on psychological distress: Symptom-related fears and beliefs as mediators**
Authors:	Radziej, Katharina (first author) Probst, Thomas Limburg, Karina Dinkel, Andreas Dieterich, Marianne Lahmann, Claas
Publication status:	Published in *Journal of Nervous and Mental Disease*, [Epub ahead of print] (2018)

Abstract

In spite of the frequent observation that vertigo/dizziness (VD) disorders may trigger or exacerbate secondary psychiatric comorbidities there is limited understanding of the mechanisms underlying this development. To address this gap, it was investigated whether symptom-related fears and cognitions as indicated by questionnaire-based measures are mediators of the longitudinal effect of VD symptoms on anxiety and depression after one year. Data from a large study with patients of a treatment centre specialized in vertigo were analysed (N=210). Simple and multiple parallel mediation models strengthened our hypothesis that fear of bodily sensations as well as cognitions about these symptoms play a mediating role in the relationship between VD symptoms and psychopathology at follow-up

after baseline scores of the outcome were controlled for. Results are discussed within a cognitive theory framework and point to the potential benefits of interventions that modify symptom-related beliefs and fears via cognitive psychotherapy in this therapeutically underserved population.

Individual contribution

The PhD candidate (KR) is the main author of this paper. KR was responsible for the development of the research idea and developed the theoretical framework for the reanalysis of the data derived from the large diagnostic study (as described in the Methodology section). KR was not in charge of the data collection itself, but was responsible for data management, carried out the statistical analyses of the data (in liaison with the co-author Thomas Probst) and wrote this manuscript independently in agreement with and integrating feedback from the co-authors. KR was also responsible for the whole submission process by integrating reviewers' feedback (including major revisions and further analyses) into the paper.

Study III – Abstract and individual contribution

Title:	**[Tailored care for functional vertigo/dizziness – an integrative group psychotherapy approach]**
Authors:	Radziej, Katharina (first author) Schmid-Mühlbauer, Gabriele Limburg, Karina Lahmann, Claas
Publication status:	Published in *Psychotherapie - Psychosomatik - Medizinische Psychologie*, Volume 67, pp. 245-251 (2017)

Abstract

Functional vertigo and dizziness (VD) symptoms are highly prevalent and usually accompanied by a strong impairment of quality in everyday and working life. The complaints are often associated with various psychiatric disorders, particularly phobic and other anxiety disorders, depressive or somatoform disorders. Despite this clinical relevance of VD symptoms, studies towards psychotherapeutic treatment options for patients with functional VD are still rare. Thus, the manuscript outlines a manual for outpatient group psychotherapy for patients with functional VD symptoms. The approach aims to assist patients in developing an understanding of the interaction between bodily complaints and psychosocial factors while detracting from a purely symptom-based focus. The integrative-psychotherapeutic treatment program consists of 16 weekly sessions and includes a regular vestibular rehabilitation training as well as disorder-oriented modules towards anxiety and panic disorder, depression, and somatisation. Currently a randomised controlled trial is being conducted in order to evaluate the short- and long-term effectiveness of the program in reducing VD and further bodily symptoms, vertigo-related social and physical handicap as well as anxiety and depressive complaints. A previously completed pilot trial revealed small to large pre-post-effects on primary and secondary outcome measures. Conclusions regarding acceptance and feasibility of the therapy concept are discussed in the manuscript.

Individual contribution

The PhD candidate (KR) is the main author of this paper. Together with co-author Karina Limburg she is responsible for the overall study coordination of the RCT within the therapeutic study phase (as described in the Methodology section) having contributed to grant application and since. Building on preliminary work of co-author Gabriele Schmid-

Mühlbauer, KR substantially contributed to the treatment manual that is outlined in the published manuscript and was subject to evaluation within the pilot study described herein as well as the ongoing RCT. KR was in charge of the data management, carried out the statistical analyses of the data and wrote this manuscript independently in agreement with and integrating feedback from co-authors. KR was also responsible for the whole submission process by integrating reviewers' feedback (including minor revisions) into the paper.

Discussion

Persistent vestibular symptoms are complex phenomena unlikely to be explained or maintained by any single cause. They are closely intertwined with psychopathological factors contributing to every step of pathogenesis. This dissertation aimed to shed light on some of the various remaining research gaps with respect to the cause and course of functional VD. First, the following section will summarize the main results and reflect on answered and unanswered research questions. Second, the focus will be set on clinical implications and recommendations arising from the study results. Methodological shortcomings and further limitations are specified in detail in the respective manuscripts.

Theoretical contribution

The role of psychological traumatization

In a nutshell:

Research Questions and (preliminary) Answers

(1) *Do functional and organic VD disorders differ in terms of their frequency of traumatizing childhood and lifetime experiences and severity of posttraumatic stress symptoms?*
Answer: Results from Study I suggest otherwise.

(2) *Do trauma-related variables contribute to the severity of vertigo-related symptoms and anxiety as well as the physical and psychosocial handicap they entail?*
Answer: To some extent. Symptoms of posttraumatic stress explained limited variance in VD symptoms and VD-related anxiety.

Results from *Study I* revealed that adverse childhood and lifetime experiences are equally common among patients suffering from functional compared to organically explained VD

disorders. Groups also did not differ with regard to the severity of current posttraumatic stress symptoms. This stands in contrast to previous accounts of the frequently observed association between psychological trauma and functional syndromes (e.g., Sack et al., 2007), pseudo-neurological symptoms in particular (Wahlström, Michelsen, Schulman, Backheden, & Keskinen-Rosenqvist, 2013). Yet, with regard to functional VD in the investigated tertiary care sample, it seems highly unlikely that psychological traumatization was a common precipitator. Thus, a history of childhood trauma or adulthood adversity does not hold diagnostic validity with regard to functional VD disorders.

Study results revealed further that the experience of posttraumatic stress symptoms contributed to higher overall balance symptoms and autonomic symptoms of VD-related anxiety across diagnostic groups. Due to the cross-sectional study design, no conclusion can be drawn about the predictive validity of this association. Still, it raises questions about the mechanisms underlying this observation. With regard to childhood adversities, Martin et al. (2016) recently suggested that their longitudinal relationship with symptom burden in SSD was mediated by emotion regulation skills. As a result, psychological traumatization could be a vulnerability factor for a particularly strong and dysfunctional emotional response to somatic symptoms. Other research efforts emphasized that previous trauma could shape the way bodily symptoms are perceived, e.g. as very intense or threatening, and stressed the role of attentional allocation processes (Brown, 2004; Duddu, Isaac, & Chaturvedi, 2006).

The role of symptom-related fears and cognitions

In a nutshell:

Research Question and (preliminary) Answer

(3) *Is the longitudinal link between VD symptoms and levels of depression/anxiety mediated by symptom-related fears and cognitions?*
Answer: Partially yes, with differential effects for anxiety and depression, respectively.

Unhelpful appraisals have repeatedly been linked to the perpetuation of VD after an initial vestibular disorder (Godemann et al., 2005; Heinrichs, Edler, Eskens, Mielczarek, & Moschner, 2007). *Study II* suggested that the impact of such appraisals, including a perceived high likelihood of threat or perceived low ability to cope, goes beyond that: If somatic symptoms are appraised as threatening and elicit a strong sense of fear patients will be more prone to develop or exacerbate secondary symptoms of depression and anxiety disorders. Results indicated somatic symptom-related fears and cognitions to be a mediating link between VD symptoms and the psychopathology they often entail. Most importantly, this finding was not limited to functional VD disorders but found across a wide range of diagnostic categories.

Of note, effect sizes were small. Therefore the question remains open as to which other processes contribute to the frequent occurrence of secondary psychiatric symptoms after VD disorders. One conceivable option: Maladaptive illness beliefs and emotional responses have common behavioral implications, e.g. frequent medical care seeking, safety and avoidance behaviors (Creed & Barsky, 2004; Petrie, Jago, & Devcich, 2007; Prior & Bond, 2013) that in turn could also provoke or maintain symptoms of depression and anxiety. Up to this point, there is a scarcity of investigations with regard to dysfunctional illness behaviors in VD

disorders. Previous studies have almost exclusively focused on patients suffering from Meniere's disease (Savastano, Maron, Mangialaio, Longhi, & Rizzardo, 1996).

Prospects of tailored group psychotherapy in functional VD

In a nutshell:

Research Questions and (preliminary) Answers

(1) *Will the integrative group psychotherapy program result in long-term clinically relevant improvements in VD-related quality of life?*
Answer: Preliminary results from the pilot trial are promising to that effect.

(2) *Will the integrative group psychotherapy program also alleviate associated psychopathology, namely symptoms of depression, anxiety and somatisation, as well as VD symptoms themselves?*
Answer: Effect sizes indicated small to large improvements on secondary endpoint measures, Small sample size and uncontrolled study design do not allow for reliable conclusions to be drawn yet.

The overall aim of *study III* was to evaluate the acceptance, feasibility and likely effectiveness of a manualised integrative psychotherapy approach taking place in an outpatient group setting. The outlined concept adds specific therapy modules on anxiety, depression and somatization as a novel element to previous intervention strategies. Indeed, in contrast to former studies (Holmberg, Karlberg, Harlacher, & Magnusson, 2007; Schmid et al., 2011; Tschan et al., 2012) it yielded long-term improvements with regard to both VD symptoms and VD-related handicap as well as associated psychopathology. It should be emphasized that due to major limitations regarding sample size and study design no further conclusions can be drawn from the pilot trial. It does give rise to cautious optimism with respect to an on-going RCT (Lahmann, Henningsen, Dieterich, et al., 2015).

At a glance – Integration of results in an extended disease model

The main results of the respective *studies I-III* can be incorporated into the cognitive-behavioral model that has been introduced in the theoretical background section of this dissertation (see figure 5).

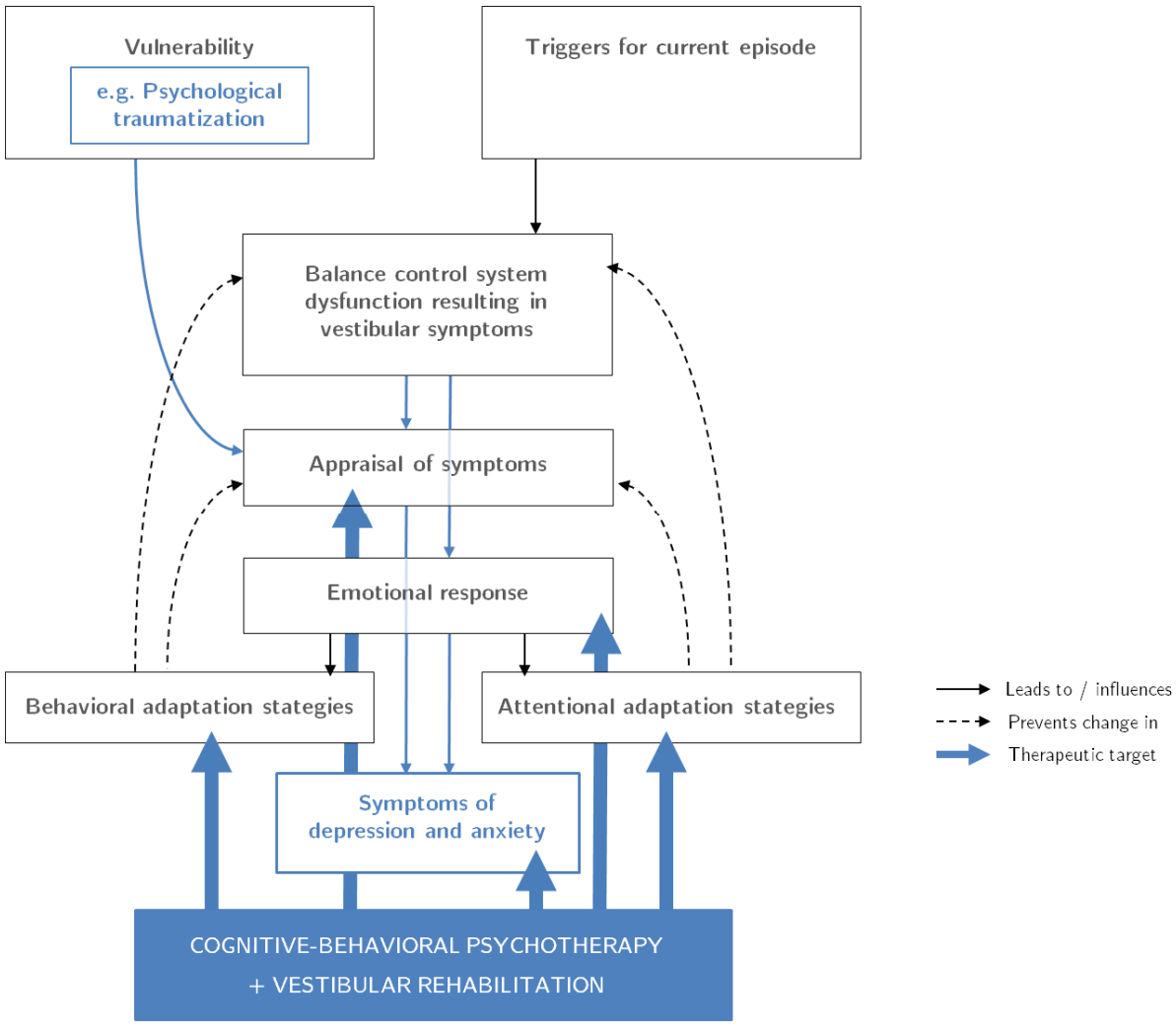


Figure 5. Extended cognitive-behavioral model of functional VD

While prior psychological traumatization had not been part of the original conceptualization by Whalley and Cane (2017), it may add to the vulnerability that impacts on how VD symptoms are appraised by an individual. Patients with a history of trauma have been

suggested to be more vulnerable to processes of *somatosensory amplification* (Barsky, 1992; Duddu et al., 2006). In this light, hypervigilance and a heightened state of bodily awareness that come with posttraumatic stress might also contribute to the attentional adaptation strategies described in the model. Above that, childhood adversities were found to impact on the emotional response to somatic symptoms via the mediating link of emotion regulation skills (Martin et al., 2016). In line with this model, the experience of posttraumatic stress symptoms was associated with higher symptom burden irrespective of the initial trigger of a VD episode (*study I*).

Secondary symptoms of anxiety and depression have been added to the model since they often contribute substantially to overall symptomatology and handicap, including social and occupational difficulties. While *study II* provided some evidence for the mediating role of symptom appraisal and symptom-related fears, this unlikely is the only mechanism behind the frequent development of anxiety and depression following VD disorders. The disease model is consistent with the finding that the extent of an initial vestibular dysfunction had no impact on the development of secondary psychopathology (Best et al., 2009a; Eagger et al., 1992; Pérez, Martin, & Garcia-Tapia, 2003). Yet, it fails to answer the question why psychiatric comorbidities tend to be more common in some vestibular disorders compared to others. It has been suggested that prognosis may be worse in patients who suffer from episodic VD conditions with relapsing attacks, such as vestibular migraine and vestibular paroxysmia (Lahmann, Henningsen, Brandt, et al., 2015); patients may experience these as particularly uncontrollable and alarming thereby increasing the likelihood of the psychopathological processes such appraisals may entail. In turn, secondary psychopathology could have a reciprocal effect on health behaviors (Wiltink et al., 2009) among other components of the disease model, hence contributing further to the perpetuation of functional VD. After all, symptoms of anxiety and depression should all the more be taken into account

for treatment planning since current psychotherapeutic efforts mostly failed to achieve clinically relevant improvements regarding these psychological concomitants (Schmid et al., 2011).

For that reason psychiatric comorbidity was explicitly accounted for as a therapeutic target in the integrative group psychotherapy concept (*study III*). Further targets for change via therapeutic strategies are depicted in figure 5. Previous findings support the idea that the effectiveness of interventions (including psychoeducation, cognitive-behavioral strategies and vestibular rehabilitation exercises) is not limited to persistent VD in the absence of an initial organic cause; patients across vestibular diagnostic groups may benefit (Andersson et al., 2006; Edelman et al., 2012; Johansson et al., 2001; Mahoney et al., 2013; Yardley et al., 2012; Yardley & Kirby, 2006).

Clinical implications and conclusions

Despite its high prevalence within VD disorders, functional VD is often not included in the early differential diagnosis resulting in subsequent delays in treatment initiation (Dieterich & Eckhardt-Henn, 2004) and incurring high health care costs (Barsky, Orav, & Bates, 2005). The studies of this dissertation add emphasis to the formerly stated claim for routine psychological assessments, specifically screening for unhelpful appraisals as well as maladaptive behavioral and attentional strategies in patients presenting with vestibular symptoms. Indicators of psychological traumatization do not seem to hold diagnostic validity regarding functional VD disorders but may contribute to symptom severity across diagnostic groups.

In line with previous and upcoming changes in the common diagnostic manuals (DSM-5, ICD-11), dichotomous thinking with regard to the organic causation of somatic symptoms such as VD should be abandoned since it has limited predictive validity. There remains the need for a more coordinated approach to the collection of information with respect to somatic symptoms' psychological concomitants; concerning this matter the SSD and BDD criteria – most likely point of reference in everyday clinical practice – retain an unavoidable arbitrariness, since no norms exist for "appropriate" levels of emotional distress, illness worry or illness behavior in patients presenting with somatic symptoms.

Irrespective of initial etiology the aforementioned psychological mechanisms can come into effect in the perpetuation of dizziness and the development of psychopathological sequelae. On a positive note, they can be subject to change in psychotherapeutic, mainly cognitive-behavioral, and vestibular rehabilitation strategies. The outlined disease model of functional VD by Whalley and Cane (2017) appears to be a convenient working model informing both the need for future research and treatment planning. In cases of vestibular disorders with a clear and treatable organic cause, early preventive strategies targeting dysfunctional appraisals and illness behaviors might interfere with pathogenic processes early on. Provided that the integrative psychotherapeutic concept proves effective in RCTs it may contribute to the implementation of better treatment options in the therapeutically underserved population of patients suffering from functional VD.

References

American Academy of Otolaryngology Head and Neck Surgery. (1995). Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. *Otolaryngology - Head and Neck Surgery*, *113*(3), 181–185.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (4th ed., text rev.)*. Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Washington, DC: Author.

Andersson, G., Asmundson, G. J. G., Denev, J., Nilsson, J., & Larsen, H. C. (2006). A controlled trial of cognitive-behavior therapy combined with vestibular rehabilitation in the treatment of dizziness. *Behaviour Research and Therapy*, *44*(9), 1265–1273.

Andreski, P., Chilcoat, H., & Breslau, N. (1998). Post-traumatic stress disorder and somatization symptoms: A prospective study. *Psychiatry Research*, *79*(2), 131–138.

Bader, K., Hänny, C., Schäfer, V., Neuckel, A., & Kuhl, C. (2009). Childhood Trauma Questionnaire – Psychometrische Eigenschaften einer deutschsprachigen Version. *Zeitschrift Für Klinische Psychologie Und Psychotherapie*, *38*(4), 223–230.

Barsky, A. J. (1992). Amplification, Somatization, and the Somatoform Disorders. *Psychosomatics*, *33*(1), 28–34.

Barsky, A. J., Orav, E. J., & Bates, D. W. (2005). Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Archives of General Psychiatry*, *62*(8), 903–910.

Beidel, D. C., & Horak, F. B. (2001). Behavior therapy for vestibular rehabilitation. *Journal of Anxiety Disorders*, *15*(1–2), 121–130.

Best, C., Eckhardt-Henn, A., Tschan, R., & Dieterich, M. (2009a). Psychiatric morbidity and comorbidity in different vestibular vertigo syndromes. Results of a prospective longitudinal study over one year. *Journal of Neurology*, *256*(1), 58–65.

Best, C., Eckhardt-Henn, A., Tschan, R., & Dieterich, M. (2009b). Why do subjective vertigo

and dizziness persist over one year after a vestibular vertigo syndrome? *Annals of the New York Academy of Sciences*, *1164*, 334–337.

Bigelow, R. T., Semenov, Y. R., Du Lac, S., Hoffman, H. J., & Agrawal, Y. (2016). Vestibular vertigo and comorbid cognitive and psychiatric impairment: The 2008 National Health Interview Survey. *Journal of Neurology, Neurosurgery and Psychiatry*, *87*(4), 367–372.

Brandt, T. (1996). Phobic postural vertigo. *Neurology*, *46*(6), 1515–1519.

Brandt, T., & Dieterich, M. (1986). Phobischer Attacken Schwankschwindel, ein neues Syndrom? *Münchener Medizinische Wochenschrift*, *28*, 247–250.

Brandt, T., & Dieterich, M. (1994). Vestibular paroxysmia: vascular compression of the eighth nerve? *The Lancet*, *343*(8900), 798–799.

Brandt, T., Dieterich, M., & Strupp, M. (2013). *Vertigo and dizziness: Common complaints, second edition*. London: Springer.

Brandt, T., Zwergal, A., Jahn, K., & Strupp, M. (2009). [Integrated Center for Research and Treatment of Vertigo, Balance and Ocular Motor Disorders]. *Der Nervenarzt*, *80*(8), 875–886.

Bronstein, A. (1995). Visual vertigo syndrome: clinical and posturography findings. *Journal of Neurology, Neurosurgery & Psychiatry*, 472–476.

Brown, R. J. (2004). Psychological mechanisms of medically unexplained symptoms: an integrative conceptual model. *Psychological Bulletin*, *130*(5), 793–812.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy*, *24*(4), 461–470.

Creed, F., & Barsky, A. (2004). A systematic review of the epidemiology of somatisation disorder and hypochondriasis. *Journal of Psychosomatic Research*, *56*(4), 391–408.

Deary, V., Chalder, T., & Sharpe, M. (2007). The cognitive behavioural model of medically unexplained symptoms: a theoretical and empirical review. *Clinical Psychology Review*, *27*(7), 781–797.

Dieterich, M., & Eckhardt-Henn, A. (2004). [Neurological and somatoform vertigo

syndromes]. *Der Nervenarzt*, *75*(3), 281–302.

Dieterich, M., & Staab, J. P. (2017). Functional dizziness: from phobic postural vertigo and chronic subjective dizziness to persistent postural-perceptual dizziness. *Current Opinion in Neurology*, *30*(1), 107–113.

Duddu, V., Isaac, M. K., & Chaturvedi, S. K. (2006). Somatization, somatosensory amplification, attribution styles and illness behaviour: a review. *International Review of Psychiatry*, *18*(1), 25–33.

Eagger, S., Luxon, L. M., Davies, R. A., Coelho, A., & Ron, M. A. (1992). Psychiatric morbidity in patients with peripheral vestibular disorder: A clinical and neuro-otological study. *Journal of Neurology Neurosurgery and Psychiatry*, *55*(5), 383–387.

Eckhardt-Henn, A., Best, C., Bense, S., Breuer, P., Diener, G., Tschan, R., & Dieterich, M. (2008). Psychiatric comorbidity in different organic vertigo syndromes. *Journal of Neurology*, *255*(3), 420–428.

Eckhardt-Henn, A., Breuer, P., Thomalske, C., Hoffmann, S. O., & Hopf, H. C. (2003). Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. *Journal of Anxiety Disorders*, *17*(4), 369–388.

Eckhardt-Henn, A., Tschan, R., Best, C., & Dieterich, M. (2009). [Somatoform vertigo syndrome]. *Der Nervenarzt*, *80*(8), 909–917.

Edelman, S., Mahoney, A. E. J., & Cremer, P. D. (2012). Cognitive behavior therapy for chronic subjective dizziness: a randomized, controlled trial. *American Journal of Otolaryngology*, *33*(4), 395–401.

Ehlers, A., & Margraf, J. (2001). *Fragebogen zu körperbezogenen Ängsten, Kognitionen und Vermeidung (AKV)*. Göttingen: Beltz Test GmbH.

Ferring, D., & Filipp, S.-H. (1994). Teststatistische Überprüfung der Impact of Event-Skala: Befunde zu Reliabilität und Stabilität. *Diagnostica*, *40*(4), 344–362.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The posttraumatic diagnostic scale. *Psychological Assessment*, *9*(4), 445–451.

Furman, J. M., Balaban, C. D., Jacob, R. G., & Marcus, D. A. (2005). Migraine-anxiety

related dizziness: a new disorder? *Journal of Neurology, Neurosurgery & Psychiatry*, *76*, 1–8.

Godemann, F., Siefert, K., Hantschke-Brüggemann, M., Neu, P., Seidl, R., & Ströhle, A. (2005). What accounts for vertigo one year after neuritis vestibularis - Anxiety or a dysfunctional vestibular organ? *Journal of Psychiatric Research*, *39*(5), 529–534.

Gräfe, K., Zipfel, S., Herzog, W., & Löwe, B. (2004). [Screening for psychiatric disorders with the Patient Health Questionnaire (PHQ). Results from the German validation study]. *Diagnostica*, *50*(4), 171–181.

Heinrichs, N., Edler, C., Eskens, S., Mielczarek, M. M., & Moschner, C. (2007). Predicting continued dizziness after an acute peripheral vestibular disorder. *Psychosomatic Medicine*, *69*(7), 700–707.

Henningsen, P., Zipfel, S., & Herzog, W. (2007). Management of functional somatic syndromes. *Lancet*, *369*(9565), 946–955.

Holmberg, J., Karlberg, M., Harlacher, U., & Magnusson, M. (2007). One-year follow-up of cognitive behavioral therapy for phobic postural vertigo. *Journal of Neurology*, *254*(9), 1189–1192.

Holmberg, J., Karlberg, M., Harlacher, U., Rivano-Fischer, M., & Magnusson, M. (2006). Treatment of phobic postural vertigo. A controlled study of cognitive-behavioral therapy and self-controlled desensitization. *Journal of Neurology*, *253*(4), 500–506.

Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of Event Scale: a measure of subjective stress. *Psychosomatic Medicine*, *41*(3), 209–218.

Jacob, R. G., Lilienfeld, S. O., Furman, J. M. R., Durrant, J. D., & Turner, S. M. (1989). Panic disorder with vestibular dysfunction: Further clinical observations and description of space and motion phobic stimuli. *Journal of Anxiety Disorders*, *3*(2), 117–130.

Jacob, R. G., Redfern, M. S., & Furman, J. M. (2009). Space and motion discomfort and abnormal balance control in patients with anxiety disorders. *Journal of Neurology, Neurosurgery, and Psychiatry*, *80*(1), 74–78.

Johansson, M., Akerlund, D., Larsen, H. C., & Andersson, G. (2001). Randomized controlled trial of vestibular rehabilitation combined with cognitive-behavioral therapy for dizziness

in older people. *Otolaryngology - Head and Neck Surgery*, *125*(3), 151–156.

Kinzl, J. F., Traweger, C., & Biebl, W. (1995). Family background and sexual abuse associated with somatization. *Psychotherapy and Psychosomatics*, *64*(2), 82–87.

Kroenke, K., Lucas, C. A., Rosenberg, M. L., Scherokman, B., Herbers, J. E., Wehrle, P. A., & Boggi, J. O. (1992). Causes of Persistent Dizziness: A Prospective-Study of 100 Patients in Ambulatory Care. *Annals of Internal Medicine*, *117*(11), 898–904.

Kruschinski, C., Kersting, M., Breull, A., Kochen, M. M., Koschack, J., & Hummers-Pradier, E. (2008). [Frequency of dizziness-related diagnoses and prescriptions in a general practice database]. *Zeitschrift Für Evidenz, Fortbildung Und Qualität Im Gesundheitswesen*, *102*(5), 313–319.

Kühner, C., Bürger, C., Keller, F., & Hautzinger, M. (2007). [Reliability and validity of the Revised Beck Depression Inventory (BDI-II). Results from German samples]. *Der Nervenarzt*, *78*(6), 651–656.

Lahmann, C., Henningsen, P., Brandt, T., Strupp, M., Jahn, K., Dieterich, M., ... Schmid, G. (2015). Psychiatric comorbidity and psychosocial impairment among patients with vertigo and dizziness. *Journal of Neurology, Neurosurgery, and Psychiatry*, *86*(3), 302–308.

Lahmann, C., Henningsen, P., Dieterich, M., Feuerecker, R., Cyran, C. A., & Schmid, G. (2012). The Munich Diagnostic and Predictor Study of Dizziness: objectives, design, and methods. *Journal of Neurology*, *259*(4), 702–711.

Lahmann, C., Henningsen, P., Dieterich, M., Radziej, K., & Schmid, G. (2015). Tailored care for somatoform vertigo/dizziness: study protocol for a randomised controlled trial evaluating integrative group psychotherapy. *Journal of Neurology*, *262*(8), 1867–1875.

Lahmann, C., Henningsen, P., Noll-Hussong, M., & Dinkel, A. (2010). Somatoforme Störungen. *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *60*(6), 227–236.

Limburg, K., Sattel, H., Dinkel, A., Radziej, K., Becker-Bense, S., & Lahmann, C. (2017). Course and predictors of DSM-5 somatic symptom disorder in patients with vertigo and dizziness symptoms – A longitudinal study. *Comprehensive Psychiatry*, *77*, 1–11.

Limburg, K., Sattel, H., Radziej, K., & Lahmann, C. (2016). DSM-5 somatic symptom

disorder in patients with vertigo and dizziness symptoms. *Journal of Psychosomatic Research*, *91*, 26–32.

Mahoney, A. E. J., Edelman, S., & Cremer, P. D. (2013). Cognitive behavior therapy for chronic subjective dizziness: longer-term gains and predictors of disability. *American Journal of Otolaryngology*, *34*(2), 115–120.

Margraf, J., & Ehlers, A. (2007). *Das Beck Angst-Inventar (BAI) – Manual. Deutsche Bearbeitung.* Frankfurt am Main: Harcourt Test Services GmbH.

Martin, A., Bruchhauser, A., Kleinstauber, M., & Rief, W. (2016). The Relationship between Childhood Adversities, Emotion Regulation and Symptom Burden in Somatic Symptom Disorder. *International Journal of Behavioral Medicine*, *23*(Suppl. 1), S244–S245.

Martin, R. D. (1999). The somatoform conundrum: a question of nosological values. *General Hospital Psychiatry*, *21*(3), 177–186.

Naber, C. M., Water-Schmeder, O., Bohrer, P. S., Matonak, K., Bernstein, A. L., & Merchant, M. A. (2011). Interdisciplinary treatment for vestibular dysfunction: The effectiveness of mindfulness, cognitive-behavioral techniques, and vestibular rehabilitation. *Otolaryngology - Head and Neck Surgery*, *145*(1), 117–124.

Nakao, M., & Yano, E. (2006). Somatic symptoms for predicting depression: One-year follow-up study in annual health examinations. *Psychiatry and Clinical Neurosciences*, *60*, 219–225.

Neuhauser, H. K. (2007). Epidemiology of vertigo. *Current Opinion in Neurology*, *20*, 40–46.

Neuhauser, H. K., Radtke, A., von Brevern, M., Lezius, F., Feldmann, M., & Lempert, T. (2008). Burden of dizziness and vertigo in the community. *Archives of Internal Medicine*, *168*, 2118–2124.

Pérez, N., Martin, E., & Garcia-Tapia, R. (2003). Dizziness: Relating the severity of vertigo to the degree of handicap by measuring vestibular impairment. *Otolaryngology - Head and Neck Surgery*, *128*(3), 372–381.

Petrie, K. J., Jago, L. A., & Devcich, D. A. (2007). The role of illness perceptions in patients with medical conditions. *Current Opinion in Psychiatry*, *20*(2), 163–167.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing

and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*(3), 879–891.

Prior, K. N., & Bond, M. J. (2013). Somatic symptom disorders and illness behaviour: Current perspectives. *International Review of Psychiatry*, *25*(1), 5–18.

Probst, T., Dinkel, A., Schmid-Mühlbauer, G., Radziej, K., Limburg, K., Pieh, C., & Lahmann, C. (2017). Psychological distress longitudinally mediates the effect of vertigo symptoms on vertigo-related handicap. *Journal of Psychosomatic Research*, *93*, 62–68.

Radtke, A., Neuhauser, H., Von Brevern, M., Hottenrott, T., & Lempert, T. (2011). Vestibular migraine-validity of clinical diagnostic criteria. *Cephalalgia*, *31*(8), 906–913.

Rief, W., & Barsky, A. J. (2005). Psychobiological perspectives on somatoform disorders. *Psychoneuroendocrinology*, *30*(10), 996–1002.

Rief, W., Bleichhardt, G., & Timmer, B. (2002). Gruppentherapie für somatoforme Störungen - Behandlungsleitfaden, Akzeptanz und Prozessqualität. *Verhaltenstherapie*, *12*(3), 183–191.

Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms-models and mechanisms. *Clinical Psychology Review*, *27*(7), 821–841.

Roh, K. J., Kim, M. K., Kim, J. H., & Son, E. J. (2018). Role of Emotional Distress in Prolongation of Dizziness : A Cross-Sectional Study. *Journal of Audiology and Otology*, *22*(1), 6–12.

Ruckenstein, M. J., & Staab, J. P. (2009). Chronic subjective dizziness. *Otolaryngologic Clinics of North America*, *42*(1), 71–77.

Sack, M., Lahmann, C., Jaeger, B., & Henningsen, P. (2007). Trauma prevalence and somatoform symptoms: are there specific somatoform symptoms related to traumatic experiences? *The Journal of Nervous and Mental Disease*, *195*(11), 928–933.

Salkovskis, P. M., & Warwick, H. M. C. (1986). Morbid preoccupations, health anxiety and reassurance: a cognitive-behavioural approach to hypochondriasis. *Behaviour Research and Therapy*, *24*(5), 597–602.

Savastano, M., Maron, M. B., Mangialaio, M., Longhi, P., & Rizzardo, R. (1996). Illness behaviour, personality traits, anxiety, and depression in patients with Menière's disease.

The Journal of Otolaryngology, *25*(5), 329–333.

Schaaf, H., & Hesse, G. (2015). Patients with long-lasting dizziness: A follow-up after neurotological and psychotherapeutic inpatient treatment after a period of at least 1 year. *European Archives of Oto-Rhino-Laryngology*, *272*(6), 1529–1535.

Schmid, G., Henningsen, P., Dieterich, M., Sattel, H., & Lahmann, C. (2011). Psychotherapy in dizziness: a systematic review. *Journal of Neurology, Neurosurgery, and Psychiatry*, *82*(6), 601–606.

Simon, N. M., Pollack, M. H., Tuby, K. S., & Stern, T. A. (1998). Dizziness and panic disorder: a review of the association between vestibular dysfunction and anxiety. *Annals of Clinical Psychiatry : Official Journal of the American Academy of Clinical Psychiatrists*, *10*(2), 75–80.

Sklare, D. A., Konrad, H. R., Maser, J. D., & Jacob, R. G. (2001). Special issue on the interface of balance disorders and anxiety: an introduction and overview. *Journal of Anxiety Disorders*, *15*(1–2), 1–7.

Staab, J. P. (2011). Behavioral aspects of vestibular rehabilitation. *NeuroRehabilitation*, *29*, 179–183.

Staab, J. P. (2012). Chronic subjective dizziness. *CONTINUUM Lifelong Learning in Neurology*, *18*(5), 1118–1141.

Staab, J. P., Eckhardt-Henn, A., Horii, A., Jacob, R., Strupp, M., Brandt, T., & Bronstein, A. (2017). Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): Consensus document of the committee for the classification of vestibular disorders of the Barany Society. *Journal of Vestibular Research: Equilibrium and Orientation*, *27*(4), 191–208.

Staab, J. P., & Ruckenstein, M. J. (2003). Which comes first? Psychogenic dizziness versus otogenic anxiety. *The Laryngoscope*, *113*(10), 1714–1718.

Staab, J. P., & Ruckenstein, M. J. (2007). Expanding the differential diagnosis of chronic dizziness. *Archives of Otolaryngology - Head & Neck Surgery*, *133*(2), 170–176.

Staab, J. P., Ruckenstein, M. J., & Amsterdam, J. D. (2004). A prospective trial of sertraline for chronic subjective dizziness. *The Laryngoscope*, *114*(9), 1637–1641.

Strupp, M., Glaser, M., Karch, C., Rettinger, N., Dieterich, M., & Brandt, T. (2003). Häufigste Schwindelform im mittleren Alter: Phobischer Schwankschwindel. *Nervenarzt*, *74*(10), 911–914.

Tschan, R., Eckhardt-Henn, A., Scheurich, V., Best, C., Dieterich, M., & Beutel, M. E. (2012). [Steadfast – Effectiveness of a Cognitive-Behavioral Self-Management Program for Patients with Somatoform Vertigo and Dizziness]. *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *62*, 111–119.

Tschan, R., Wiltink, J., Best, C., Bense, S., Dieterich, M., Beutel, M. E., & Eckhardt-Henn, A. (2008). Validation of the German version of the Vertigo Symptom Scale (VSS) in patients with organic or somatoform dizziness and healthy controls. *Journal of Neurology*, *255*(8), 1168–1175.

Tschan, R., Wiltink, J., Best, C., Beutel, M., Dieterich, M., & Eckhardt-Henn, A. (2010). [Validation of the German version of the Vertigo Handicap Questionnaire (VHQ) in patients with vestibular vertigo syndromes or somatoform vertigo and dizziness]. *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *60*(9–10), e1-12.

Tusa, R. J. (2009). Dizziness. *The Medical Clinics of North America*, *93*(2), 263–71, vii.

van der Kolk, B. A., Pelcovitz, D., Roth, S., Mandel, F. S., McFarlane, A., & Herman, J. L. (1996). Dissociation, somatization, and affect dysregulation: the complexity of adaptation of trauma. *The American Journal of Psychiatry*, *153*(7 Suppl), 83–93.

Wahlström, L., Michelsen, H., Schulman, A., Backheden, H., & Keskinen-Rosenqvist, R. (2013). Longitudinal course of physical and psychological symptoms after a natural disaster. *European Journal of Psychotraumatology*, *4*, 21892.

Weidt, S., Bruehl, A. B., Straumann, D., Hegemann, S. C., Krautstrunk, G., & Rufer, M. (2014). Health-related quality of life and emotional distress in patients with dizziness: a cross-sectional approach to disentangle their relationship. *BMC Health Services Research*, *14*(1), 317.

Wessely, S., & White, P. D. (2004). There is only one functional somatic syndrome. *British Journal of Psychiatry*, *185*, 95–96.

Whalley, M. G., & Cane, D. A. (2017). A Cognitive-Behavioral Model of Persistent Postural-Perceptual Dizziness. *Cognitive and Behavioral Practice*, *24*(1), 72–89.

WHO. (2015a). International Classification of Diseases, 11th edition, beta draft. Bodily distress disorder. Retrieved February 28, 2018, from http://id.who.int/icd/entity/767044268

WHO. (2015b). International Classification of Diseases, 11th edition, beta draft. Persistent postural-perceptual dizziness. Retrieved February 28, 2018, from http://id.who.int/icd/entity/2005792829

Wiltink, J., Tschan, R., Michal, M., Subic-Wrana, C., Eckhardt-Henn, A., Dieterich, M., & Beutel, M. E. (2009). Dizziness: anxiety, health care utilization and health behavior--results from a representative German community survey. *Journal of Psychosomatic Research*, *66*(5), 417–424.

Wittchen, H., Zaudig, M., & Fydrich, T. (1997). *Strukturiertes Klinisches Interview für DSM-IV*. Göttingen: Hogrefe.

Yardley, L., Barker, F., Muller, I., Turner, D., Kirby, S., Mullee, M., ... Little, P. (2012). Clinical and cost effectiveness of booklet based vestibular rehabilitation for chronic dizziness in primary care: single blind, parallel group, pragmatic, randomised controlled trial. *BMJ*, *344*, e2237.

Yardley, L., & Kirby, S. (2006). Evaluation of booklet-based self-management of symptoms in Ménière disease: a randomized controlled trial. *Psychosomatic Medicine*, *68*(5), 762–769.

Yardley, L., Masson, E., Verschuur, C., Haacke, N., & Luxon, L. (1992). Symptoms, anxiety and handicap in dizzy patients: Development of the Vertigo symptom scale. *Journal of Psychosomatic Research*, *36*(8), 731–741.

Appendix

Manuscript I

Radziej, K., Schmid, G., Dinkel, A., Zwergal, A., & Lahmann, C. (2015). Psychological traumatization and adverse life events in patients with organic and functional vestibular symptoms. *Journal of Psychosomatic Research, 79,* 123–129.

Journal of Psychosomatic Research 79 (2015) 123–129

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

Psychological traumatization and adverse life events in patients with organic and functional vestibular symptoms

Katharina Radziej [a,b], Gabriele Schmid [a,b], Andreas Dinkel [a], Andreas Zwergal [b,c], Claas Lahmann [a,b,*]

[a] Department of Psychosomatic Medicine and Psychotherapy, Klinikum rechts der Isar, Technische Universitaet, Munich, Germany
[b] German Centre for Vertigo and Balance Disorders, University Hospital Munich, Campus Großhadern, Ludwig-Maximilians-Universitaet, Munich, Germany
[c] Department of Neurology, University Hospital Munich, Campus Großhadern, Ludwig-Maximilians-Universität, Munich, Germany

ARTICLE INFO

Article history:
Received 6 October 2014
Received in revised form 7 May 2015
Accepted 10 May 2015

Keywords:
Vestibular symptoms
Somatoform
Functional
Vertigo
Dizziness
Traumatization

ABSTRACT

Objective: . A relationship has frequently been found between a history of traumatization and the existence of somatoform symptoms. The objective of this study was to examine whether this relationship is also observed for functional, i.e. medically not sufficiently explained, vestibular symptoms (FVS). We tested whether patients with FVS and organically explained vestibular symptoms (OVS) differ with regard to frequencies of previous traumatic experiences and posttraumatic stress symptoms. We also explored whether the impact of previous trauma was associated with characteristics of vestibular symptoms and handicap.
Methods: . Patients with a diagnosis of OVS (N = 185) or FVS (N = 158) completed questionnaires about potentially traumatizing experiences (e.g., Childhood Trauma Questionnaire, Impact of Events Scale) and vertigo-related symptoms and handicap (Vertigo Symptom Scale, Vertigo Handicap Questionnaire).
Results: . We found no differences between the two patient groups with regard to number or impact of traumatic life events. However, regression analyses across groups revealed that, regardless of their diagnosis, prior traumatic experiences and the presence of posttraumatic stress symptoms including avoidance and intrusion predicted to some extent higher overall balance symptoms and autonomic symptoms of vertigo-related anxiety.
Conclusion: . Exposure to trauma and symptoms of posttraumatic stress can contribute to symptom severity and handicap experienced by patients with vestibular symptoms irrespective of their original cause, most likely serving as predisposing, modulating or perpetuating factors.

Introduction

Vestibular symptoms (VS), including vertigo, dizziness, vestibulo-visual and postural symptoms [12] rank among the symptoms most commonly presented to general practitioners and neurologists [47,48]. Up to half of patients presenting with VS in a tertiary care setting suffer from a psychiatric comorbidity which was associated with severe psychosocial impairment [41]. A substantial percentage ranging from 20 to more than 50% of disorders involving vertigo/dizziness as a cardinal symptom cannot be sufficiently explained by an identifiable medical illness and are considered somatoform or functional VS (FVS; [23,41]). Secondary and primary types of FVS can be differentiated based on whether they occur with or without a prior organic vestibular disorder, respectively [19,22]. Those patients are often severely impaired in their professional and daily activities, and their symptoms tend to be chronic [64]. They also report lower health-related quality of life [62] and experience more severe symptoms and dizziness-related anxiety compared with patients with organic vertigo [11].

Somatoform and functional symptoms, albeit not FVS in particular, have frequently been linked to childhood and lifetime traumatization [4,35,40,63]. Studies of functional somatic syndromes, such as somatization disorder, chronic fatigue, fibromyalgia, and functional gastrointestinal disorders, have consistently found associations with previous trauma [20,30,46,49,54,59]. In their comprehensive review, Roelofs and Spinhoven [52] not only found increased rates of lifetime trauma in patients with medically unexplained symptoms but also an association between trauma and symptom severity. This modulating effect of posttraumatic stress symptoms on somatic symptoms as well as overall functioning and health-related quality of life has been described for different conditions such as conversion disorder, behavioural spells and chronic pain [39,50,53].

Moreover, from the vantage point of traumatization, McFarlane et al. [44] found that patients suffering from posttraumatic stress disorder (PTSD) report more somatic symptoms than non-PTSD subjects. Although somatoform symptoms often appear to be unspecific and involve multiple organ systems [17], Wahlstrom et al. [65] report that pseudoneurological symptoms including dizziness, mental fatigue, clumsiness and headaches are more strongly associated with exposure

* Corresponding author at: Department of Psychosomatic Medicine and Psychotherapy, Klinikum rechts der Isar, Technische Universitaet, Langerstr. 3, 81675 Munich, Germany. Tel.: +49 89 4140 2031; fax: +49 89 4140 4845.
E-mail address: lahmann@tum.de (C. Lahmann).

http://dx.doi.org/10.1016/j.jpsychores.2015.05.005
0022-3999/

to natural disasters than cardiorespiratory, gastrointestinal, and musculoskeletal symptoms. This finding is in line with that of Sack et al. [54], who found a specific increase in pseudoneurological symptoms (e.g. impaired balance, loss of touch or pain sensations, seizures) in patients reporting any lifetime trauma.

Several overlapping models of the relationship between adverse life events and somatoform symptoms have been proposed. These models suggest contributions of heightened arousal, disordered information processing, and the acquisition of symptom-related mental representations [1,14,44] as well as changes in self-monitoring mechanisms such as those described in a group of patients with fibromyalgia or somatoform pain disorder [34]. Overall, three major models have emerged: dissociation, conversion, and hierarchical cognitive models [52]. The first two models, which are based in psychodynamic theory, attempt to explain the aetiology of somatoform symptoms with psychological trauma serving as a predisposing or precipitating factor. The third model, however, does not describe somatic symptoms in terms of a symbolic representation of underlying dynamics; rather, it stresses the role of attentional processes, specifically an increased awareness of bodily sensations and a recurring allocation of attention to bodily symptoms. It describes mechanisms explaining how previous trauma can contribute to symptoms in a predisposing, modulating or sustaining manner, i.e., how cognitive and behavioural factors might interact with somatic factors to produce or perpetuate symptoms, and could therefore not only be applied to primary functional but also secondary FVS as well as organically explained symptoms.

Despite the well-established association between trauma exposure and somatization, to our knowledge, there are no studies reporting evidence of this association for FVS in particular. However, symptoms of posttraumatic stress including autonomic arousal, anxiety and avoidance behaviours have been reported among patients with vestibular disorders [29,36,70] and an association of PTSD symptoms with anxiety, depression and handicap has been found in patients suffering from Ménière's disease [38].

Therefore, the purpose of the present study was twofold. First, in a tertiary care setting, we compared patients with FVS and organically explained VS (OVS) in terms of their frequency of potentially traumatizing childhood and lifetime experiences and severity of posttraumatic stress symptoms. Second, we explored whether trauma-related variables contributed to the severity of vertigo-related symptoms and anxiety as well as physical and psychosocial handicap across patient groups.

Methods

Study design and sample characteristics

This cross-sectional study was conducted between May 2010 and June 2012. Patients were recruited via routine care appointments at the German Centre for Vertigo and Balance Disorders at the University Hospital Munich, Campus Großhadern. Patients were excluded if they were under 18 years of age or had any neurodegenerative disorders (e.g., dementia), schizoaffective or psychotic disorder, substance abuse, or severe suicidal tendencies. A total of 686 out of 860 eligible patients (80% response rate) gave their consent to participate in the study. Reasons for refusal to participate were insufficient German language skills, lack of interest, difficulty filling out a questionnaire due to cognitive impairment, feelings of excessive demands, or privacy concerns. Due to organizational reasons (e.g. living outside of Munich or vomiting after caloric testing), data could only be obtained from $n = 513$ out of all 686 participants. For the analyses presented herein, patients were included if they had received a definite diagnosis at the time of data collection resulting in a final sample size of $n = 343$. Patients that were excluded from analyses within that step did not differ from included patients with regard to sex distribution or any of the questionnaire-based measures (described below). However, patients that were excluded were significantly younger (included: 56.02(16.476) yrs; excluded: 52.47(15.233) yrs; $t(511) = 2.37$, $p = .018$, 95% CI of mean difference [.604, 6.507]).

This study was part of the Munich Diagnostic and Predictor Study of Somatoform Dizziness, which is described in detail elsewhere [42]. All participants gave written informed consent, and the study was approved by the Ethics Committee of the University of Munich.

Diagnostic assessment

All patients underwent structured collection of their histories and a systematic and standardized physical examination by an expert medical scientist at the German Centre for Vertigo and Balance Disorders, including complete neurological, neurootological, and neuroophthalmological examination. Vestibular testing included the head impulse test, measurements of subjective visual vertical and ocular torsion, and video-oculography with caloric irrigation.

Patients were categorized into the group of (1) organic vestibular symptoms (OVS) if their complaints were related to a defined organic illness based on exam results and established diagnostic criteria for vestibular disorders ([13,18]; frequencies of respective diagnoses are listed in the Results section). If patients' complaints were not medically sufficiently explained by a current vestibular deficit or a defined organic illness, i.e. patients with a non-organic or non-vestibular cause of vestibular symptoms, they were categorized into the group of (2) functional vestibular symptoms (FVS). Patients with former organic vestibular diagnoses that currently did not explain vestibular symptoms, i.e. secondary FVS [19], were also assigned to this group.

Patient self-report questionnaires

A battery of self-report questionnaires, including information on age and sex as key demographic characteristics, was administered to patients.

(1) Vertigo symptoms and handicap. Dizziness-related symptoms and resulting physical and psychosocial handicap were assessed using the Vertigo Symptom Scale (VSS; [71]) and Vertigo Handicap Questionnaire (VHQ; [73]).

The VSS ([71]; German version by [61]) assesses the frequency of dizziness-related symptoms through 34 items scored on a 5-point Likert scale ranging from 0 (never) to 4 (more than once a week). Scores for two main dimensions are obtained by summing up item scores for vertigo-balance symptoms (VSS-VER; e.g., dizziness, vertigo, unsteadiness, and light-headedness) and vertigo-induced autonomic-anxiety symptoms (VSS-AA; e.g., somatic sensations, sweating, pounding heart, breathing difficulties, and fainting). The VSS shows good test–retest reliability and construct validity [61,71].

The VHQ ([73]; German version by [62]) assesses physical and psychosocial impairments due to VS. It consists of 25 statements regarding disabling consequences of vertigo scored on a 5-point Likert scale ranging from 0 (never) to 4 (always). The statements address restrictions of physical activity, interferences with social relationships, and fears about vertigo (i.e., the impact of vertigo on quality of life). Item scores are summed to give a total score ranging between 0 and 100 points. Two subscales are derived: 'handicapped activity' (VHQ-ACT) and 'anxiety' (VHQ-ANX). The VHQ has good to acceptable internal consistency, test–retest reliability, and construct validity [62].

(2) Traumatization. Traumatization, including the occurrence of childhood trauma and abuse, the experience of other traumatic life events, and the impact and severity of traumatic disorders, was assessed using the Childhood Trauma Questionnaire (CTQ; original version by [8]), Posttraumatic Diagnostic Scale (PDS; [27]; German version by [58]), and Impact of Event Scale (IES; [32]).

The CTQ retrospectively assesses the history of childhood neglect and abuse. The short-form German version ([10]; German version by [68]) used in our study consists of 28 items regarding five types of maltreatment: physical, sexual, and emotional abuse as well as physical

and emotional neglect. Items are rated on a 5-point Likert scale ranging from 1 (never true) to 5 (very often true). Likely cases of abuse are categorized into three levels of severity: mild, moderate, and severe. A minimization/denial scale is also included for detecting the underreporting of traumatic events. The CTQ has been found to be a reliable and valid screening instrument [5].

The PDS is based on Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV criteria and is widely used in clinical and research settings. It consists of 49 items measuring the severity and duration of PTSD-related symptoms and their effects on daily functioning. The PDS has been validated in a group of subjects who had experienced a wide range of trauma and shows high internal consistency and test-retest reliability [28]. In the present study, we used only the first section of the PDS, which is a 12-item checklist inquiring about experience of DSM-IV Criterion A traumatic events (namely severe accidents, natural disasters, violence by a family member or a stranger, sexual violence by a family member or a stranger, sexual contact under age, war experiences, kidnapping, torture and life-threatening illness).

The IES [26,32] is one of the most frequently used self-report questionnaires for assessing the severity of a possible posttraumatic disorder. It consists of 15 items measuring two of the DSM PTSD symptom dimensions: intrusion and avoidance. Participants were asked to complete the IES only if they reported having experienced at least one potentially traumatizing event in the PDS list. If they had experienced two or more events, they were asked to refer to the most burdensome event when completing the IES. Participants rated the frequency of subjective distress due to each symptom during the last week on a non-equidistant 4-point Likert scale: 0 (not at all), 1 (rarely), 3 (sometimes), or 5 (often). Total scores exceeding a cut-off value of 25 indicate clinical relevance. Subscales are derived for the two symptom clusters. The IES has been found to have sound psychometric properties [26,32].

Statistical analysis

To test for differences among participants and non-participants with regard to sex, age and self-report questionnaire scores, categorical variables were analysed using chi-square tests, and continuous variables were analysed using independent samples t tests.

To evaluate differences in self-report questionnaire scores among the two diagnostic subgroups (OVS, FVS), we computed descriptive statistics presented as mean and standard deviation or as frequency and percentage. On categorical dichotomous outcome variables (i.e., PDS categories of potentially traumatizing experiences) we conducted binary logistic regression analyses controlling for age and sex. The fit of the model was evaluated using the omnibus test of coefficients; odd ratios are reported as indicators of effect size.

On continuous variables (CTQ and IES scores) we performed analyses of covariance (ANCOVA) controlling for age and gender. Effect sizes (partial η^2) were computed and considered small if partial η^2 was ≥0.01 and <0.06, medium if partial η^2 was ≥0.06 and <0.14, and large if partial η^2 was ≥0.14 [16]. Levene's test was used to evaluate homogeneity of variances.

To test for an association between prior potentially traumatizing life events, a history of adverse childhood events and post-traumatic stress symptoms on the one hand and vertigo- and anxiety-related symptoms and handicap (VSS and VHQ scores) on the other hand, we computed linear regression analyses. Therefore a three-step process was applied: In the first step, to control for covariates, sex and age were entered into separate multiple regression analyses for all dependent variables (VSS and VHQ subscales). In the second step, separate univariate simple linear regression analyses were performed to determine the proportion of variance in the residuals from the first step that were explained by trauma-related predictors, namely IES and CTQ subscales as well as a dichotomous variable indicating whether participants had experienced any of the potentially traumatizing life events from the PDS checklist. In the third step, significant trauma-related predictors identified in the second step were entered into univariate multiple regression analyses to determine their relative importance in explaining variance in the VSS and VHQ subscales. All regression analyses (method: simultaneous entry) were conducted across both patient groups; standardized regression coefficient β and adjusted (multiple) R^2 values are reported.

All statistical analyses were performed using SPSS software (version 21; SPSS Inc., Chicago, IL). Significance levels were set at $p < .05$.

Results

Patient characteristics

A total of 343 included patients (59% female; 55.96 (16.491) years of age (mean(SD)), range: 20–92 years) underwent diagnostic and questionnaire-based psychometric assessment.

Diagnostic assessments revealed VS due to an organic cause in 185 (54%; OVS group) patients and functional, i.e. medically not sufficiently explained, VS in 158 (46%; FVS group) patients. Among the OVS group, benign paroxysmal positional vertigo (n = 40) and Meniere's disease (n = 39) were the most frequent diagnoses, followed by bilateral vestibulopathy (n = 30), vestibular migraine (n = 19), central vertigo (n = 19), multisensory deficit (n = 18), polyneuropathy (n = 16), vestibular neuritis (n = 10), unilateral vestibular loss (n = 10), vestibular paroxysmia (n = 5) and perilymph fistula (n = 1).

Diagnostic subgroups differed with regard to sex (OVS: 53% female; FVS: 66% female; χ^2 (1, N = 343) 5.812, p = .021) and age (OVS: 59.62(16.359) yrs; FVS: 51.67 (15.638) yrs; $t(341) = 4.575$, p < .001, 95% CI of mean difference [4.530, 11.361]). Therefore, age and sex were included as covariates in all further analyses.

History of adverse life events

Over half of patients (62.8%) had experienced at least one adverse event listed in the PDS checklist. Frequencies and percentages of every trauma type are summarized in Table 1 for the total sample and both patient groups. OVS and FVS groups did not differ in terms of frequency of most of the trauma categories after controlling for gender and age. However, "violence by a family member" ($\chi^2(1, 343) = 5.756$, $p = .016$) and "war experiences" ($\chi^2(1, 343) = 4.563$, $p = .033$) were reported more often by patients in the OVS group, while "sexual violence by a stranger" ($\chi^2(1, 343) = 4.216$, $p = .040$) was reported more often in the FVS group. Differences were statistically significant at an α level of .05, but failed to reach significance if correction for multiple testing were applied. Also, large confidence intervals indicated a low level of precision of the ORs.

History of adverse childhood events

To control for gender and age, a comparison of patient groups with regard to history of adverse childhood events was conducted by including these two variables as covariates in the ANOVA, resulting in 2 × 2 ANCOVAs performed separately for each CTQ subscale. We found no differences among groups for any of the subscales (*emotional abuse*: $F(1,298) = .036$, $p = .849$; *physical abuse*: $F(1,299) = 1.683$, $p = .196$; *sexual abuse*: $F(1,297) = .059$, $p = .809$; *emotional neglect*: $F(1,299) = .715$, $p = .399$; *physical neglect*: $F(1,300) = .991$, $p = .320$; Table 2).

Impact of events

Analogous, 2 × 2 ANCOVAs were conducted to compare the amount of posttraumatic stress symptoms between groups, controlled for gender and age. We found no differences among patient groups with regard to *IES intrusion* ($F(1,155) = .155$, $p = .694$) or *IES avoidance* scores ($F(1,155) = .006$, $p = .941$; Table 2). Within the OVS group 27 participants (14.6%) exceeded the cut-off value of 25 on the *IES total score* indicating clinical relevance compared to 28 participants (17.7%) in the FVS group.

Association between traumatization and VS-related symptoms and handicap

Because OVS and FVS groups did not differ considerably with regard to any of the CTQ or IES scores, further analyses were conducted across groups. After controlling for sex and age as covariates, univariate simple linear regression analyses revealed predictors of the VSS and VHQ subscales. Standardized regression coefficients and proportions of explained variance as indicated by *adjusted* R^2 values are summarized in Table 3.

Specifically, the occurrence of traumatic life events, i.e., having experienced any of the life events on the PDS checklist, was associated with three of the criterion variables, namely VSS-VER, VSS-AA and VHQ-ANX. Beyond that, vertigo-balance symptoms (VSS-VER) were predicted by the posttraumatic stress symptom clusters *avoidance* and *intrusion* which did also show significant associations with psychological and autonomic symptoms of anxiety (VSS-AA, VHQ-ANX). Handicapped activity (VHQ-ACT) showed only an association with one of the symptom clusters (avoidance). Among the adverse childhood events assessed with the CTQ, *emotional abuse* and *emotional neglect* showed the strongest link to VS-related variables, both being significantly associated with three out of four of the VSS and VHQ scales, in particular vertigo-induced autonomic-anxiety symptoms (VSS-AA). *Physical neglect* was also significantly associated with this subscale. It should be noted that all significant predictors indicated a positive relationship between trauma- and

Table 1
PDS trauma rates by group.

	Total sample	OVS	FVS		
Type of trauma	N (%)	N (%)	N (%)	p[a]	OR [CI]
Accident	120 (37%)	63 (36%)	57 (38%)	.402	.816 [.507, 1.314]
Natural disaster	32 (10%)	18 (10%)	14 (9%)	.877	.942 [.442, 2.009]
Violence by a family member	28 (9%)	21 (12%)	7 (5%)	.016*	3.072 [1.228, 7.683]
Violence by a stranger	28 (9%)	17 (10%)	11 (7%)	.501	1.329 [.580, 3.045]
Sexual violence by a family member	19 (6%)	8 (5%)	11 (7%)	.487	.707 [.267, 1.876]
Sexual violence by a stranger	20 (6%)	6 (3%)	14 (9%)	.040*	.348 [.127, .953]
War experiences	23 (7%)	21 (12%)	2 (1%)	.033*	5.235 [1.146, 23.908]
Sexual contact under age (<18, other + 5 yrs min)	42 (13%)	22 (13%)	20 (13%)	.898	1.045 [.533, 2.049]
Kidnapping	11 (3%)	9 (5%)	2 (1%)	.425	1.948 [.378, 10.032]
Torture	5 (2%)	3 (2%)	2 (1%)	.772	1.319 [.203, 8.555]
Life-threatening illness	73 (23%)	37 (21%)	36 (24%)	.379	.783 [.454, 1.351]
Trauma not specified above	58 (18%)	25 (14%)	33 (22%)	.117	.623 [.345, 1.126]
Any of the above	206 (63%)	110 (62%)	96 (64%)	.406	.819 [.512, 1.311]

Level of significance: *p < .05; PDS = Posttraumatic Diagnostic Scale; OVS = organic vestibular symptom group; FVS = functional vestibular symptom group; OR = odds ratio; CI = confidence interval.
[a] p Values are reported for the Wald criterion of PDS trauma type as predictor within binary logistic regression models. Overall model fit was evaluated using the omnibus test of coefficients and was significant for all analyses (all $\chi^2(3, N = 323) > 22$ and all $p < .001$).

VS-related measures. According to Cohen's [16] conventions for interpreting the f^2 value derived from the multiple correlation coefficient *R*, all effects sizes were small to medium.

Subsequent multiple regression analyses revealed that the linear combination of the selected trauma-related measures was significantly related to and accounted for 6–9% of the variance (according to *adjusted* R^2 values) in the two VSS subscales as well as the VHQ-ANX scale (Table 4). Specifically, *IES avoidance* score significantly predicted vertigo-balance symptoms ($\beta = .232$, $t(1) = 2.047$, $p = .042$) and, when combined with *IES intrusion* score, *PDS trauma* and *CTQ emotional neglect*, accounted for 8.5% of the variance. The occurrence of previous lifetime trauma ($\beta = .180$, $t(1) = 2.165$, $p = .032$) predicted autonomic-anxiety symptoms (VSS-AA), with the combined predictors explaining 6% of the variance. The overall VHQ-ANX regression model explained 6.6% of the variance to which *IES intrusion* symptoms significantly contributed ($\beta = .239$, $t(1) = 2.133$, $p = .035$). The multiple regression model predicting restrictions of physical and social activity (VHQ-ACT) failed to reach significance ($F(3,153) = 2.523$, $p = .060$, *adj.* $R^2 = .028$). Variance inflation factors (VIF) indicated that predictors were lowly to moderately correlated across multiple regression analyses (all VIF < 2.4).

Discussion

One of the main purposes of this study was to investigate the existence of a relationship between adverse childhood or lifetime traumatic experiences and the severity of vertigo-related symptoms and handicap. Patient groups suffering from organically explained and functional VS did not differ considerably with regard to number of traumatic experiences after controlling for gender and age. Also, there were no differences in scores on a history of adverse childhood events and indicators of current posttraumatic stress symptoms of intrusion and avoidance. However, both groups showed elevated rates of clinically relevant posttraumatic stress symptoms, 15% and 18% in the OVS and FVS group respectively. This is in line with previous reports of partial or full PTSD symptoms among patients with vestibular disorders ([29,36,38,70]). Regardless of whether VS could be accounted for by an organic cause, previous trauma and posttraumatic stress symptoms of avoidance and intrusion predicted to some extent the variance of balance symptoms and handicap, mainly those associated with psychological and autonomic symptoms of anxiety. Effect sizes were small for most analyses and have to be interpreted with caution against the background that no correction for inflation of α error was applied.

Table 2
Posttraumatic stress symptoms and adverse childhood events by group.

	OVS Mean (SD)	FVS Mean (SD)	p[a]	Partial η^2
Impact of Events Scale				
IES intrusion	9.93 (9.334)	9.54 (9.201)	.694	.001
IES avoidance	9.24 (9.851)	9.63 (9.449)	.941	<.001
Childhood Trauma Questionnaire				
CTQ emotional abuse	7.31 (3.379)	7.87 (4.151)	.849	<.001
CTQ physical abuse	6.23 (2.789)	5.80 (1.915)	.196	.006
CTQ sexual abuse	5.66 (2.417)	5.76 (2.596)	.809	<.001
CTQ emotional neglect	10.25 (5.003)	10.650 (5.160)	.399	.002
CTQ physical neglect	7.99 (3.315)	7.35 (2.604)	.320	.003

OVS = organic vestibular symptom group; FVS = functional vestibular symptom group; IES = Impact of Events Scale; CTQ = Childhood Trauma Questionnaire.
[a] p Values are reported for F tests of main effects of IES and CTQ subscales (ANCOVA, between-subjects effects). Levene's test indicated unequal variances between groups for all subscales except CTQ emotional neglect.

In a previous study, Best et al. [11] found no correlation between acute or chronic vestibular dysfunction and scores on vertigo-related psychometric tests (including the VSS and VHQ), suggesting that non-vestibular factors must account for variance in vertigo-related symptoms. Such variance in symptoms includes the extent to which a patient feels debilitated or distressed by his or her symptoms, feels restricted in daily activities, or experiences vertigo-related autonomic-anxiety symptoms, and could partly be accounted for by trauma-related factors. Focusing on physical and psychosocial impairments due to a symptom in order to evaluate its clinical significance is in line with recent developments in DSM-V diagnostic criteria. In particular, somatic symptoms disorder, which replaces the DSM-IV category of somatoform disorders, is characterized by somatic symptoms that are distressing or significantly disrupt daily life (DSM-V 300.82, Criterion A; [2]). Thus, within this diagnostic framework, results presented herein add to the observation that boundaries between medically explained and functional VS are, in fact, blurred in a way that they hold comparable behavioural and psychiatric comorbidity.

The results give rise to the questions what mechanisms might explain the relationship between trauma-related factors and severity of VS-related symptoms and anxiety across patient groups. Concerning this matter, previous traumatic experience might be involved in the development of symptom chronicity as suggested by Brown [15] in his "new model of medically unexplained symptoms". This model proposes that previous traumatic experiences influence the way people think about their illnesses and how they interpret body-related symptoms. This could in turn contribute to the perpetuation of not only somatoform symptoms but also organically explained symptoms. Related to this possibility, previous studies indicate that maladaptive beliefs about the consequences of VS can be even more disabling than the symptoms themselves [37] and can have a substantial influence on healthcare behaviour [51]. Specifically, patients with a history of trauma have been

Table 3
Prediction of VS-related symptoms and handicap by trauma-related variables (simple regression).

	VSS-VER[a]		VSS-AA		VHQ-ACT		VHQ-ANX	
	β	*Adj. R^2*	β	*Adj. R^2*	β	*Adj. R^2*	β	*Adj. R^2*
PDS trauma	.160**	.023	.224***	.047	.078	.003	.203***	.038
Impact of Events Scale								
IES intrusion	.236**	.063	.200*	.034	.138	.013	.258**	.060
IES avoidance	.314***	.093	.200*	.034	.189*	.030	.192*	.030
Childhood Trauma Quest.								
CTQ emotional abuse	.091	.005	.140*	.016	.142*	.017	.125*	.012
CTQ physical abuse	.108	.008	.042	−.002	.076	.003	.054	.000
CTQ sexual abuse	.075	.002	.028	−.003	.067	.001	.088	.004
CTQ emotional neglect	.116*	.010	.196**	.035	.148*	.019	.090	.005
CTQ physical neglect	.097	.006	.140*	.016	.096	.006	.024	−.003

Level of significance: *p < .05, **p < .01, ***p < .001; VS = vestibular symptoms; VSS-VER = Vertigo Symptom Scale, vertigo-balance symptoms; VSS-AA = Vertigo Symptom Scale, autonomic-anxiety symptoms; VHQ-ACT = Vertigo Handicap Questionnaire, handicapped activity; VHQ-ANX = Vertigo Handicap Questionnaire, anxiety; PDS = Posttraumatic Diagnostic Scale.

[a] The table summarizes univariate simple linear regression results after controlling for variation explained by age and gender.

suggested to be more vulnerable to processes such as somatosensory amplification [21], which has been described by Barsky [6] as an altered perception of somatic sensory stimuli. Somatosensory amplification includes a tendency to experience somatic sensations as very intense and threatening as well as hypervigilance and a heightened state of bodily awareness, which are common posttraumatic reactions and are included in the list of DSM-V PTSD criteria (Criterion E: alterations in arousal and reactivity; [2]). This is consistent with our finding that VS patients with and without previous trauma exposure showed marked differences in anxiety-related/autonomic symptoms.

If cognitive factors indeed contribute to the perpetuation of symptoms, they could be a target of psychotherapeutic intervention, or, in the case of secondary FVS, prevention. Some evidence already indicates the efficacy of cognitive-behavioural and psychoeducation strategies combined with relaxation techniques or vestibular rehabilitation in the management of FVS [31,55,60]. A brief 3-session CBT intervention produced significant improvements in dizziness and symptom-related handicap [25], that were maintained in the long-term [43]. It is important to note that the effectiveness of intervention was not limited to patients with functional dizziness syndromes but has been shown across vestibular diagnostic groups [3,25,33,43,69,72].

Furthermore, the present study add emphasis to the formerly stated claim for routine psychological assessment and screening for behavioural morbidity in patients presenting with vestibular syndromes (e.g., [24,57]), including a screening for adverse life events. Our results suggest that even the assessment with a short 12-item checklist (PDS) of potentially traumatizing life events can indicate that patients are more likely to experience more severe symptoms and dizziness-related anxiety.

To our knowledge, our study is the first to examine the association between trauma-related factors comparing functional and organic VS. However, it does have some methodological shortcomings. First, the study was conducted in a tertiary care setting (i.e., a highly specialized outpatient clinic for vertigo and balance disorders) and therefore may have involved selection bias. Thus, our results might not be generalizable to VS patients in primary or secondary care settings. Because patients must be referred to the clinic on the grounds of a primary somatic symptom, it cannot be ruled out that posttraumatic stress and related symptoms could have been underestimated in our sample of patients whose presenting problem was VS. In addition, patients that were not included in data analyses were slightly younger than included patients which might have introduced a selection bias even though they did not differ on any of the questionnaire-based measures.

Second, variances on several questionnaire-based measures varied significantly between groups; however, since ANOVA appears to be relatively robust to heterogeneity of variances when sample sizes do not differ considerably between groups, it is unlikely that it affected interpretability of results. With regard to multiple regression analyses VIF indicated low to moderate correlations among predictors. With all VIF < 2.4 it is yet unlikely that this might have introduced substantial error to coefficient estimates of individual predictors. However, since the simple and multiple regression analyses in this study were merely exploratory in nature, their results should be interpreted with caution until they can be validated in a different sample of patients.

Table 4
Prediction of VS-related symptoms and handicap by trauma-related variables (multiple regression).

	VSS-VER[a]	VSS-AA	VHQ-ACT	VHQ-ANX
	β	β	β	β
PDS trauma	.080	.180*		.150
Impact of Events Scale				
IES intrusion	.073	.111		.239*
IES avoidance	.232*	.044	.153	−.032
Childhood Trauma Quest.				
CTQ emotional abuse		−.050	.038	.038
CTQ emotional neglect	.034	.160	.088	
CTQ physical neglect		.022		
Overall model fit	$F(4,152) = 4.637$ $p = .001$** *adj.* $R^2 = .085$	$F(6,145) = 2.642$ $p = .018$ *adj.* $R^2 = .061$	$F(3,153) = 2.523$ $p = .060$ *adj.* $R^2 = .028$	$F(4,150) = 3.728$ $p = .006$ *adj.* $R^2 = .066$

Level of significance: *p < .05, **p < .01; VS = vestibular symptoms; VSS-VER = Vertigo Symptom Scale, vertigo-balance symptoms; VSS-AA = Vertigo Symptom Scale, autonomic-anxiety symptoms; VHQ-ACT = Vertigo Handicap Questionnaire, handicapped activity; VHQ-ANX = Vertigo Handicap Questionnaire, anxiety; PDS = Posttraumatic Diagnostic Scale; adj. = adjusted.

[a] The table summarizes univariate multiple linear regression results after controlling for variation explained by age and gender, including significant predictors derived from univariate simple regression analyses.

Third, the effects of the timing of adverse life events (e.g., early-onset vs. late-onset), type of trauma (e.g., interpersonal vs. non-interpersonal), and severity of trauma on VS-related symptoms were not systematically evaluated, even though type of trauma has been shown to have differential effects on PTSD symptoms and somatization [63].

Because of the cross-sectional study design no conclusions can be made as to whether trauma-related variables also show predictive validity, i.e., contribute to VS symptoms in a perpetuating manner. From a clinical viewpoint, it is important to understand whether trauma-related factors can predict the course of VS symptoms. Further longitudinal studies are needed to determine whether OVS patients with prior traumatic life events or (subclinical) posttraumatic stress symptoms are more prone to develop secondary functional symptoms or psychiatric morbidity. These studies might also shed light on the specific ways in which trauma-related factors contribute to vestibular symptoms.

Conclusions

Vestibular symptoms are complex phenomena that are unlikely to be explained by any single cause. Our study results did not reveal differences in trauma-related variables between the two study groups. It expands earlier observations of modulating effects of PTSD symptoms on somatic symptoms, associated handicap and psychiatric comorbidity (e.g. [38,39,50,53]) into vestibular disorders, regardless of whether vestibular symptoms were sufficiently explained medically or not. The precise nature of this relationship, however, remains to be elucidated. The study adds emphasis to the formerly stated claim for routine psychological screening in patients presenting with vestibular syndromes.

Acknowledgements

Funding. Parts of this project were supported by funding from the German Federal Ministry of Education and Research (01 EO 0901).

Ethics approval. This study was approved by the Ethics Committee of the University of Munich (ref. 108-10).

References

[1] Allen LA, Escobar JI, Lehrer PM, Gara MA, Woolfolk RL. Psychosocial treatments for multiple unexplained physical symptoms: a review of the literature. Psychosom Med 2002;64:939–50.
[2] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
[3] Andersson G, Asmundson GJG, Denev J, Nilsson J, Larsen HC. A controlled trial of cognitive-behavior therapy combined with vestibular rehabilitation in the treatment of dizziness. Behav Res Ther 2006;44:1265–73.
[4] Andreski P, Chilcoat H, Breslau N. Post-traumatic stress disorder and somatization symptoms: a prospective study. Psychiatry Res 1998;79:131–8.
[5] Bader K, Hänny C, Schäfer V, Neuckel A, Kuhl C. Childhood Trauma Questionnaire—psychometrische Eigenschaften einer deutschsprachigen Version. Z Kl Psych Psychoth 2009;38:223–30.
[6] Barsky AJ. Amplification, somatization, and the somatoform disorders. Psychosomatics 1992;33:28–34.
[8] Bernstein DP, Fink L. Childhood Trauma Questionnaire: a retrospective self-report questionnaire and manual. San Antonio, TX: The Psychological Corporation; 1998.
[10] Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. Child Abuse Negl 2003;27:169–90.
[11] Best C, Eckhardt-Henn A, Diener G, Bense S, Breuer P, Dieterich M. Interaction of somatoform and vestibular disorders. J Neurol Neurosurg Psychiatry 2006;77:658–64.
[12] Bisdorff A, von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: towards an international classification of vestibular disorders. J Vestib Res 2009;19:1–13.
[13] Brandt T, Dieterich M, Strupp M. Vertigo and dizziness: common complaints. 2nd ed. London: Springer; 2013.
[14] Brown RJ. Psychological mechanisms of medically unexplained symptoms: an integrative conceptual model. Psychol Bull 2004;130:793–812.
[15] Brown RJ. Medically unexplained symptoms: a new model. Psychiatry 2006;5:43–7.
[16] Cohen J. Statistical power analysis for the behavioral sciences. 2. Aufl. Hillsdale: Lawrence Erlbaum Associates; 1988.
[17] Creed F, Barsky A. A systematic review of the epidemiology of somatisation disorder and hypochondriasis. J Psychosom Res 2004;56:391–408.
[18] Dieterich M. Dizziness. Neurologist 2004;10:154–64.
[19] Dieterich M, Eckhardt-Henn A. Neurologische und somatoforme Schwindelsyndrome. Nervenarzt 2004;75:281–302.
[20] Drossman DA, Talley NJ, Leserman J, Olden KW, Barreiro MA. Sexual and physical abuse and gastrointestinal illness. Ann Intern Med 1995;10:782–94.
[21] Duddu V, Isaac MK, Chaturvedi SK. Somatization, somatosensory amplification, attribution styles and illness behaviour: a review. Int Rev Psychiatry 2006;18:25–33.
[22] Eckhardt-Henn A, Best C, Bense S, Breuer P, Diener G, Tschan R, et al. Psychiatric comorbidity in different organic vertigo syndromes. J Neurol 2008;255:420–8.
[23] Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J Anxiety Disord 2003;17:369–88.
[24] Eckhardt-Henn A, Hopf HC, Tettenborn B, Thomalske C. Psychosomatische Aspekte des Schwindels. Aktuel Neurol 1998;25:96–102.
[25] Edelman S, Mahoney AEJ, Cremer PD. Cognitive behavior therapy for chronic subjective dizziness: a randomized, controlled trial. Am J Otolaryngol 2012;33:395–401.
[26] Ferring D, Filipp SH. Teststatische Überprüfung der Impact of Event-Skala: Befunde zur Reliabilität und Stabilität. Diagnostica 1994;40:344–62.
[27] Foa EB. Posttraumatic stress diagnostic scale: manual. Minneapolis, MN: National Computer Systems; 1995.
[28] Foa E, Cashman L, Jaycox L, Perry K. The validation of a self-report measure of PTSD: the Posttraumatic Diagnostic Scale. Psychol Assess 1997;9:445–51.
[29] Furman JM, Balaban CD, Jacob RG, Marcus DA. Migraine-anxiety related dizziness: a new disorder? J Neurol Neurosurg Psychiatry 2005;76:1–8.
[30] Häuser W, Galek A, Erbslöh-Möller B, Köllner V, Kühn-Becker H, Langhorst J, et al. Posttraumatic stress disorder in fibromyalgia syndrome: prevalence, temporal relationship between posttraumatic stress and fibromyalgia symptoms, and impact on clinical outcome. Pain 2013;154:1216–23.
[31] Holmberg J, Karlberg M, Harlacher U, Rivano-Fischer M, Magnusson M. Treatment of phobic postural vertigo. A controlled study of cognitive-behavioral therapy and self-controlled desensitization. J Neurol 2006;253:500–6.
[32] Horowitz M, Wilner N, Alvarez W. Impact of event scale: a measure of subjective stress. Psychosom Med 1979;41:209–18.
[33] Johansson M, Akerlund D, Larsen HC, Andersson G. Randomized controlled trial of vestibular rehabilitation combined with cognitive-behavioral therapy for dizziness in older people. Otolaryngol Head Neck Surg 2001;125:151–6.
[34] Karst M, Rahe-Meyer N, Gueduek A, Hoy L, Borsutzky M, Passie T. Abnormality in the self-monitoring mechanism in patients with fibromyalgia and somatoform pain disorder. Psychosom Med 2005;67:111–5.
[35] Kinzl JF, Traweger C, Biebl W. Family background and sexual abuse associated with somatization. Psychother Psychosom 1995;64:82–7.
[36] Kirby SE, Yardley L. Understanding psychological distress in Meniere's disease: a systematic review. Psychol Health Med 2008;13:257–73.
[37] Kirby SE, Yardley L. Cognitions associated with anxiety in Ménière's disease. J Psychosom Res 2009;66:111–8.
[38] Kirby SE, Yardley L. The contribution of symptoms of posttraumatic stress disorder, health anxiety and intolerance of uncertainty to distress in Ménière's disease. J Nerv Ment Dis 2009;197:324–9.
[39] Koby DG, Zirakzadeh A, Staab JP, Seime R, Cha SS, Nelson CL, et al. Questioning the role of abuse in behavioral spells and epilepsy. Epilepsy Behav 2010;19:584–90.
[40] Lahmann C, Henningsen P, Noll-Hussong M, Dinkel A. Somatoform disorders. Psychother Psychosom Med Psychol 2010;60:227–33.
[41] Lahmann C, Henningsen P, Brandt T, Strupp M, Jahn K, Dieterich M, et al. Psychiatric comorbidity and psychosocial impairment among patients with vertigo and dizziness. J Neurol Neurosurg Psychiatry 2015;86:302–8.
[42] Lahmann C, Henningsen P, Dieterich M, Feuerecker R, Cyran CA, Schmid G. The Munich Diagnostic and Predictor Study of Dizziness: objectives, design, and methods. J Neurol 2012;259:702–11.
[43] Mahoney AEJ, Edelman S, Cremer PD. Cognitive behavior therapy for chronic subjective dizziness: longer-term gains and predictors of disability. Am J Otolaryngol 2013;34:115–20.
[44] Mcfarlane AC, Atchison M, Rafalowicz E, Papay P. Physical symptoms in posttraumatic stress disorder. J Psychosom Res 1994;38:715–26.
[46] Näring GWB, van Lankveld W, Geenen R. Somatoform dissociation and traumatic experiences in patients with rheumatoid arthritis and fibromyalgia. Clin Exp Rheumatol 2007;25:872–7.
[47] Neuhauser HK. Epidemiology of dizziness and vertigo. Nervenarzt 2009;80:887–94.
[48] Neuhauser HK, Radtke A, von Brevern M, Lezius F, Feldmann M, Lempert T. Burden of dizziness and vertigo in the community. Arch Intern Med 2008;168:2118–24.
[49] Noll-Hussong M, Otti A, Laeer L, Wohlschlaeger A, Zimmer C, Lahmann C, et al. Aftermath of sexual abuse history on adult patients suffering from chronic functional pain syndromes: an fMRI pilot study. J Psychosom Res 2010;68:483–7.
[50] Powers A, Fani N, Pallos A, Stevens J, Ressler KJ, Bradley B. Childhood abuse and the experience of pain in adulthood: the mediating effects of PTSD and emotion dysregulation on pain levels and pain-related functional impairment. Psychosomatics 2014;55:491–9.
[51] Rief W, Nanke A, Emmerich J, Bender A, Zech T. Causal illness attributions in somatoform disorders: associations with comorbidity and illness behavior. J Psychosom Res 2004;57:367–71.
[52] Roelofs K, Spinhoven P. Trauma and medically unexplained symptoms towards an integration of cognitive and neuro-biological accounts. Clin Psychol Rev 2007;27:798–820.
[53] Roelofs K, Spinhoven P, Sandijck P, Moene FC, Hoogduin KAL. The impact of early trauma and recent life-events on symptom severity in patients with conversion disorder. J Nerv Ment Dis 2005;193:508–14.

[54] Sack M, Lahmann C, Jaeger B, Henningsen P. Trauma prevalence and somatoform symptoms: are there specific somatoform symptoms related to traumatic experiences? J Nerv Ment Dis 2007;195:928–33.
[55] Schmid G, Henningsen P, Dieterich M, Sattel H, Lahmann C. Psychotherapy in dizziness: a systematic review. J Neurol Neurosurg Psychiatry 2011;82:601–6.
[57] Staab JP, Ruckenstein MJ. Expanding the differential diagnosis of chronic dizziness. Arch Otolaryngol Head Neck Surg 2007;133:170–6.
[58] Steil R, Ehlers A. Die Posttraumatische Diagnoseskala (PDS). Jena: Universitaet Jena, Psychologisches Institut; 2000.
[59] Taylor RR, Jason LA. Chronic fatigue, abuse-related traumatization, and psychiatric disorders in a community-based sample. Soc Sci Med 2002;55:247–56.
[60] Tschan R, Eckhardt-Henn A, Scheurich V, Best C, Dieterich M, Beutel ME. Steadfast-effectiveness of a cognitive-behavioral self-management program for patients with somatoform vertigo and dizziness. Psychother Psychosom Med Psychol 2012;62:111–9.
[61] Tschan R, Wiltink J, Best C, Bense S, Dieterich M, Beutel ME, et al. Validation of the German version of the Vertigo Symptom Scale (VSS) in patients with organic or somatoform dizziness and healthy controls. J Neurol 2008;255:1168–75.
[62] Tschan R, Wiltink J, Best C, Beutel M, Dieterich M, Eckhardt-Henn A. Validation of the German version of the Vertigo Handicap Questionnaire (VHQ) in patients with vestibular vertigo syndromes or somatoform vertigo and dizziness. Psychother Psychosom Med Psychol 2009;59:1–12.
[63] van der Kolk BA, Pelcovitz D, Roth S, Mandel FS, McFarlane A, Herman JL. Dissociation, somatization and affect dysregulation: the complexity of adaptation to trauma. Am J Psychiatry 1996;153:83–93.
[64] van Leeuwen RB, van der Zaag-Loonen HJ. Unexplained dizziness does not resolve in time. Acta Neurol Belg 2012;112:357–60.
[65] Wahlstrom L, Michelsen H, Schulman A, Backheden H, Keskinen-Rosenqvist R. Longitudinal course of physical and psychological symptoms after a natural disaster. Eur J Psychotraumatol 2013;4:21892.
[68] Wingenfeld K, Spitzer C, Mensebach C, Grabe H, Hill A, Gast U, et al. The German version of the Childhood Trauma Questionnaire (CTQ): preliminary psychometric properties. Psychother Psychosom Med Psychol 2010;60:442–50.
[69] Yardley L, Barker F, Muller I, Turner D, Kirby S, Mullee M, et al. Clinical and cost effectiveness of booklet based vestibular rehabilitation for chronic dizziness in primary care: single blind, parallel group, pragmatic, randomised controlled trial. BMJ 2012;344:e2237.
[70] Yardley L, Burgneay J, Nazareth I, Luxon L. Neuro-otological and psychiatric abnormalities in a community sample of people with dizziness: a blind, controlled investigation. J Neurol Neurosurg Psychiatry 1998;65:679–84.
[71] Yardley L, Masson E, Verschuur C, Haacke N, Luxon L. Symptoms, anxiety and handicap in dizzy patients: development of the vertigo symptom scale. J Psychosom Res 1992;36:731–41.
[72] Yardley L, Kirby S. Evaluation of booklet-based self-management of symptoms in Ménière disease: a randomized controlled trial. Psychosom Med 2006;68:762–9.
[73] Yardley L, Putman J. Quantitative analysis of factors contributing to handicap and distress in vertiginous patients: a questionnaire study. Clin Otolaryngol Allied Sci 1992;17:231–6.

Manuscript II

Radziej, K., Probst, T., Limburg, K., Dinkel, A., Dieterich, M., & Lahmann, C. (2018). The longitudinal effect of vertigo and dizziness symptoms on psychological distress: symptom-related fears and beliefs as mediators. *Journal of Nervous and Mental Disease*, [Epub ahead of print].

The Longitudinal Effect of Vertigo and Dizziness Symptoms on Psychological Distress

Symptom-Related Fears and Beliefs as Mediators

Katharina Radziej, Dipl.-Psych.,† Thomas Probst, Dr. rer. nat.,‡§*
Karina Limburg, MSc,† Andreas Dinkel, Dr. rer. nat.,**
Marianne Dieterich, Univ. Prof. Dr. med.,†|| and Claas Lahmann, Univ. Prof. Dr. med.¶

Abstract: Despite the frequent observation that vertigo and dizziness (VD) disorders may trigger or exacerbate secondary psychiatric comorbidities, there is limited understanding of the mechanisms underlying this development. To address this gap, we investigated whether symptom-related fears and cognitions as indicated by questionnaire-based measures are mediators of the longitudinal effect of VD symptoms on anxiety and depression after 1 year. We analyzed data from a large study with patients of a treatment center specialized in vertigo ($N = 210$). Simple and multiple parallel mediation models strengthened our hypothesis that fear of bodily sensations and cognitions about these symptoms play a mediating role in the relationship between VD symptoms and psychopathology at follow-up after baseline scores of the outcome were controlled for. Results are discussed within a cognitive theory framework and point to the potential benefits of interventions that modify symptom-related beliefs and fears via cognitive psychotherapy in this therapeutically underserved population.

Key Words: Dizziness, vertigo, somatoform disorder, somatic symptom disorder, symptom beliefs

(*J Nerv Ment Dis* 2018;00: 00–00)

Vertigo and dizziness (VD) rank among the most common symptoms in adults with a lifetime prevalence of approximately 30% (Neuhauser, 2007; Tusa, 2009). The underlying causes of those symptoms vary largely, ranging from peripheral and central vestibular disorders or other organic causes to somatoform disorders and all combinations thereof. Irrespective of their etiology, VD symptoms are often experienced as severely distressing. They are associated with handicap in daily and working lives (Lahmann et al., 2015; Yardley et al., 1992) as well as a significant reduction of both mental and physical health-related quality of life (Weidt et al., 2014). Subjective impairment and health care use due to their VD are further increased if patients are characterized by psychiatric comorbidity (Lahmann et al., 2015; Wiltink et al., 2009). Not only do they show a higher persistency of symptoms (Best et al., 2009b), but psychological distress also has been shown to be important in the process how VD symptoms lead to VD handicap (Probst et al., 2017). More often than not VD symptoms have been linked to such psychiatric comorbidity, regardless of whether studies were conducted from the vantage point of vestibular deficits or psychopathology (Best et al., 2009a; Bigelow et al., 2015; Staab and Ruckenstein, 2003). The highest rates of psychiatric comorbidity have been reported for vestibular migraine, Ménière's disease, vestibular paroxysmia, and functional VD (Eckhardt-Henn et al., 2008; Lahmann et al., 2015). Furman et al. (2005) suggested neuroanatomical connections between vestibular stimuli and the emotional response processing system to explain the comorbidity of VD and psychiatric disorders. However, cross-sectional studies found that emotional distress was independent of the amount of vestibular deficit (Best et al., 2006; Pérez et al., 2003). The extent of vestibular dysfunction also had no impact over the course of time on the development of secondary psychiatric disorders (Best et al., 2009a; Eagger et al., 1992). On that account, the study at hand focuses on the question as to what does act as the intermediary between VD symptoms and the development or exacerbation of psychological distress.

Cognitive theory suggests that dysfunctional cognitions contribute to psychological distress in a causal manner (*e.g.*, Beck, 2005), an association that was corroborated in a recent meta-analysis (Vîslă et al., 2016). With regard to VD, patients frequently present with individual patterns of thoughts and feelings related to their VD symptoms that fulfil the B criterion of the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition*, somatic symptom disorder (Limburg et al., 2016); these may include disproportionate thoughts about the symptoms' seriousness, a persisting high level of anxiety about health or symptoms, and excessive time and energy devoted to these symptoms or health concerns (American Psychiatric Association [APA], 2013). Cognition has been described as a potent modulator of somatic and psychological symptoms via processes such as somatosensory amplification (Barsky, 1992). This concept describes an altered perception of somatic stimuli that includes a heightened attentional focus on bodily sensations and a tendency to appraise them as intense, alarming, and signs of a threatening disease. Somatosensory amplification reflects aspects of cognitive processing rather than mere interoceptive sensitivity; it has been proposed to explain some of the variability in somatic symptomatology found among different patients with the same medical disorder and one factor mediating the link between somatic and psychological symptoms (Nakao and Barsky, 2007).

Recent theoretical models have taken a closer look at the interplay between interoceptive and self-referential cognitive processes with regard to anxiety and depression and suggested that "external cues or internal thought processes might generate an anticipation of aversive body states that […] [act] as a motivating signal for individuals to withdraw (depression) or avoid (anxiety)." (Paulus and Stein, 2010, p 456). That is, if bodily symptoms such as VD are experienced as particularly troubling, potentially uncontrollable, or persistent, this appraisal guides action planning and may thereby contribute to a negative self-view, for

*Department of Psychosomatic Medicine and Psychotherapy, Klinikum rechts der Isar, Technical University of Munich; †German Centre for Vertigo and Balance Disorders, Klinikum Großhadern, Ludwig-Maximilians-Universitaet Muenchen, Munich; ‡Department of Clinical Psychology and Psychotherapy, Georg-August-Universitaet Goettingen, Goettingen, Germany; §Department of Psychotherapy and Bio-Psycho-Social Health, Danube University Krems, Krems, Austria; ||Department of Neurology, Ludwig-Maximilians-Universitaet Muenchen, Munich; and ¶Department of Psychosomatic Medicine and Psychotherapy, Faculty of Medicine, University of Freiburg, Freiburg, Germany.

Ethical approval: The ethics committee of the Medical Department of the Ludwig-Maximilians-Universitaet, Munich, Germany, reviewed and approved this study (ref: 108–10).

Send reprint requests to Katharina Radziej, Dipl.-Psych., Klinik und Poliklinik für Psychosomatische Medizin und Psychotherapie, Klinikum rechts der Isar, Technische Universität München, Langerstr. 3/I, D-81675 München, Germany. E-mail: k.radziej@tum.de.

ISSN: 0022-3018/18/0000–0000
DOI: 10.1097/NMD.0000000000000791

example, as being helpless and less self-efficient (depression) or an increased attentional bias toward indicators of potential threat associated with avoidance behavior (anxiety). With regard to catastrophic thinking of this sort (*i.e.*, the tendency to overinterpret the likelihood or intensity of potential negative consequences of symptoms, misinterpretation of somatic sensations as signs of a serious illness, or ruminating about worst-case outcomes), there is extensive literature on its correlational and predictive value in a variety of disorders (Gellatly and Beck, 2016). However, only few studies have investigated the role of symptom-related cognitions in patients presenting with VD: in their prospective study on patients with an acute episode of vestibular neuritis, Godemann et al. found catastrophic thinking, persistent fear of vertigo, and panic-related thoughts to predict chronic vertigo (Godemann et al., 2004) and the development of secondary somatoform and panic disorder (Godemann et al., 2006). Another study demonstrated that for the development of secondary somatoform dizziness after a peripheral vestibular disorder, the fear of bodily sensations was relevant in the interaction with the initial severity of dizziness (Heinrichs et al., 2007). In Ménière's disease, maladaptive beliefs about the consequences of VD even appeared to be more disabling than symptoms themselves (Yardley et al., 2001).

Although the aforementioned studies mainly focused on the role of symptom-related fears and beliefs in the development or perpetuation of (somatoform) VD symptoms, there is, to our knowledge, a lack of investigations on how fears and cognitions contribute to the effect VD symptoms longitudinally exert on the development of anxiety and depression. Previous studies on this issue have almost exclusively focused on patients with Ménière's disease. Illness uncertainty/unpredictability and illness intrusiveness have been discussed as factors contributing to depression in these patients (Arroll et al., 2012). With regard to anxiety, Kirby and Yardley (2009) found that vertigo can lead to increased anxiety, if patients with Ménière's disease reported high intolerance of uncertainty and less understanding of their illness.

The scarcity of investigations in that area is of particular concern because current psychotherapeutic efforts may be effective in reducing VD symptoms but mostly failed to achieve clinically relevant improvements concerning the frequently associated symptoms of anxiety or depression (Schmid et al., 2011).

Aim of the study. The current study seeks to explore whether the longitudinal effect of VD symptoms (assessed at baseline) on anxiety and depression (assessed at 12-month follow-up) is mediated by symptom-related fears and cognitions (assessed at the 6-month follow-up). We therefore reanalyzed data from a large study with patients of a treatment center specialized in vertigo covering a variety of VD disorders (Lahmann et al., 2012).

Hypothesis. We hypothesized that fear of bodily sensations and cognitions about these symptoms would longitudinally mediate the effect initial VD symptoms have on the development of anxiety and depression.

METHODS

Study Design and Sample Characteristics

This longitudinal study encompasses three assessment points: baseline (t0), first follow-up after 6 months (t1), and second follow-up after 12 months (t2). Patients were recruited via routine care appointments at the German Centre for Vertigo and Balance Disorders at the University Hospital Munich, Campus Großhadern, between May 2010 and June 2012. All patients had been referred with dizziness or imbalance as their primary complaint. Patients were excluded if they were less than 18 years of age, had no sufficient German language skills, or had a neurodegenerative disorder (*e.g.*, dementia). A total of 686 (≈80%) of 860 eligible patients gave their consent to participate in the study. There were 350 and 360 patients who took part in the first and second follow-up assessment, respectively.

For this study, only those patients for which scores on the following questionnaires could be computed were analyzed: Vertigo Symptom Scale (VSS; vertigo subscale) at t0, Body Sensations Questionnaire (BSQ), and Agoraphobic Cognitions Questionnaire (ACQ) at t1, Beck Depression Inventory (BDI–II), and Beck Anxiety Inventory (BAI) at t2. A total of 210 patients fulfilled this inclusion criterion. Patients that were excluded from the analyses did not differ from included patients with regard to sex distribution or any of the questionnaire-based measures at baseline. However, excluded patients were significantly younger (included: mean = 57.4 yrs [SD = 15.6]; excluded: mean = 54.4 yrs [SD = 16.3]; $t[685] = 2.30$, $p = 0.022$, Hedges' $g = 0.18$).

This study was part of the Munich Diagnostic and Predictor Study of Somatoform Dizziness, which is described in detail elsewhere (Lahmann et al., 2012). All participants gave written informed consent, and the study was approved by the ethics committee of the University of Munich.

Diagnostic Assessment and Patient Self-Report Questionnaires

All patients underwent structured collection of their histories and a systematic and standardized physical examination, including complete neurological, neurootological, and neuroophthalmological examination. Vestibular testing included the head impulse test, measurements of subjective visual vertical and ocular torsion, and video-oculography with caloric irrigation. Frequencies of respective diagnoses are listed in the Results section. The following self-report questionnaires were administered to patients:

(1) VD symptoms. VD symptoms were assessed using the VSS (Yardley et al., 1992). The VSS (German version by Tschan et al., 2008) consists of two scales measuring vertigo-balance symptoms (VSS-VER; *e.g.*, dizziness, unsteadiness, and light-headedness) and vertigo-induced autonomic-anxiety symptoms (VSS-AA; *e.g.*, sweating, pounding heart, and breathing difficulties). In the present study, only the VSS-VER score was analyzed. It comprises 19 items scored on a 5-point Likert scale ranging from 0 (never) to 4 (more than once a week). The VSS shows good test-retest reliability and construct validity (Yardley et al., 1992; Tschan et al., 2008). For the 210 patients of the present study, the Cronbach's alpha of the VSS-VER scale at t0 was $\alpha = 0.86$.

(2) Anxiety and depression. Psychological distress, namely, symptoms of anxiety and depressive disorders, was assessed using the BAI (German version by Margraf and Ehlers, 2007) and the revised BDI–II (German version by Hautzinger et al., 2006). The BDI–II assesses depression severity during the past 2 weeks; 21 Items are rated on a 4-point scale (0–3). It is a psychometrically sound instrument (Kühner et al., 2007) with a Cronbach's alpha of $\alpha = 0.92$ at t2 in our sample. The BAI is composed of 21 items measuring severity of anxiety symptoms during the last 7 days. Items are scored on a 4-point Likert scale ranging from 0 (not at all) to 3 (strongly). The inventory shows good reliability and validity (Margraf and Ehlers, 2007). The BAI at t2 reached a Cronbach's alpha of $\alpha = 0.93$ in our sample.

(3) Cognitions and fears with regard to bodily symptoms. To assess the fear of physical symptoms and panic-related cognitions, the BSQ and the ACQ out of a questionnaire series by Chambless et al. (1984) were applied in their German version (Ehlers and Margraf, 2001). The BSQ is composed of 17 items measuring the intensity of fear with respect to body sensations (*e.g.*, vertigo, palpitations, and feeling of not being able to breathe). On a 5-point Likert scale, patients indicate to which extent they worry about a particular symptom, ranging from 1 (not at all anxious) to 5 (extremely anxious). The ACQ is a 14-item questionnaire designed to measure maladaptive thoughts about the potential for catastrophic consequences arising from anxiety or panic. Thoughts such as "I will have a heart attack" or "I will not be able to control myself" are rated on a 5-point Likert scale (1—the thought never occurs to me; 5—the thought always occurs to me). Two subscales can be

computed: "physical crisis" (five items) and "loss of control" (seven items). Overall, the German versions of the BSQ and ACQ scales showed good internal consistency and reliability. The Cronbach's alphas of both questionnaires were high in our sample (BSQ at t1: $\alpha = 0.93$; ACQ at t1: $\alpha = 0.81$). The ACQ subscales had acceptable reliability (ACQ physical crisis at t1: $\alpha = 0.72$; ACQ loss of control at t1: $\alpha = 0.76$).

Statistical Analyses

All statistical analyses were performed using SPSS 22 statistical package. To test for differences among included versus excluded patients with regard to sex, age, and self-report questionnaire scores, categorical variables were analyzed using the chi-square tests, and continuous baseline variables were analyzed using independent samples *t*-tests. Pearson correlation coefficients were calculated to assess the correlations between the measures analyzed in the study; Bonferroni correction was applied to correct for multiple testing.

To evaluate the mediating role of symptom-related fears and cognitions (BSQ, ACQ, and its subscales) at t1 in the relationship between VD symptoms (VSS-VER) at t0 and psychological distress (BDI–II, BAI) at t2, model 4 within PROCESS (Hayes, 2013), a macro for SPSS for moderation and mediation analyses, was used. PROCESS uses bootstrapping to estimate confidence intervals (CIs) of indirect effects (IEs). For all mediation analyses in the present study, the bias-corrected bootstrap CI method was selected with 10.000 bootstrap samples. To correct for multiple testing, the confidence level for CI was set to 98.75%, taking into account that four mediation models were calculated for each outcome measure. Completely standardised indirect effects (CS) (Preacher and Kelley, 2011) were computed as a measure of effect size and considered small if $0.01 \leq CS < 0.09$, medium if $0.09 \leq CS < 0.25$, and large if $CS \geq 0.25$. The p value was set to $p < 0.05$ in all analyses. Before analyses, bivariate scatterplots of the antecedent, mediator, and consequent variables were examined to evaluate the assumption of linear relations. To test for multicollinearity, variance inflation factors (VIFs) were computed for every step in the mediation analyses; with all VIF less than 1.6, it is unlikely that this might have introduced substantial error to coefficient estimates of individual predictors.

Because the subsample the analyses are based on differed from the excluded part of the sample in terms of age, it was included as a covariate across mediation analyses. To control for baseline values of the outcome measures, the BAI and BDI–II scores at the 12-month follow-up were entered into separate univariate simple linear regression analyses with their baseline scores serving as the independent variable, respectively. Residuals from these analyses were used as outcome variable in all further mediation models.

Two simple mediation models and two multiple parallel mediation models were calculated for each of the two outcome variables (BDI–II and BAI at t2). Diagrams of the mediation models are illustrated in Figure 1. In all mediation models, the VSS vertigo score at t0 was entered as the independent variable.

First, simple mediation models evaluated symptom-related fears (BSQ; model [A]) and symptom-related cognitions (ACQ; model [B]), separately, as mediators of the effect that VD symptoms at baseline exert on the respective outcome variable. Second, both ACQ and BSQ were included in a multiple parallel mediation model [C] to evaluate whether both measures remain significant mediators when statistically controlling for the other one. Finally, model [D] contrasted the two ACQ subscales in a multiple parallel mediation model to explore whether one of the two aspects of symptom-related cognitions outweighs the other in mediating the relationship between VD symptoms and the outcome measures. We note that model [D] was exploratory in nature and did not correspond directly to the study hypothesis.

RESULTS

Characteristics and Diagnoses of the Study Sample

Of the 210 analyzed patients, 119 (57%) were female. The most frequent diagnoses were functional VD ($n = 71$), benign paroxysmal positional vertigo ($n = 37$), Ménière's disease ($n = 37$), and vestibular migraine ($n = 31$). More than one third of patients received more than one diagnosis. Descriptive statistics of all questionnaire-based measures are presented in Table 1. Across the diagnostic subgroups, 20% of patients ($n = 42$) showed moderate to severe levels of depression (BDI–II score ≥ 20) at the second follow-up, and 32% ($n = 66$) showed moderate to severe levels of anxiety according to their BAI scores (≥16) after 1 year. Table 2 gives an overview of the diagnoses and the respective descriptives of the mediator variables of interest.

Correlations Between the Measures

Pearson correlation coefficients between all the questionnaire-based measures (VSS-VER at t0, ACQ at t1, BSQ at t1, BDI–II at t0

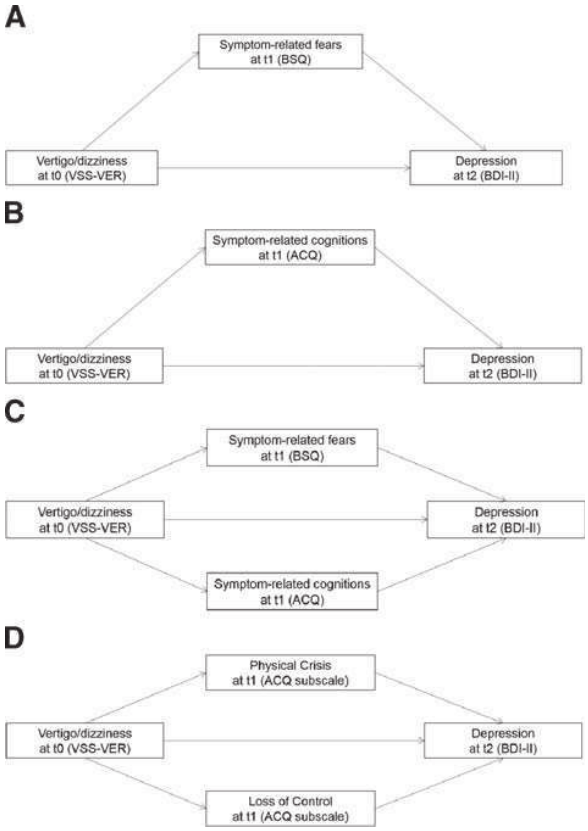


FIGURE 1. Diagrams of mediation models. (A) Simple mediation model investigating symptom-related fears as a mediator between VD symptoms and depression. (B) Simple mediation model investigating symptom-related cognitions as a mediator between VD symptoms and depression. (C) Multiple parallel mediation model investigating both symptom-related fears and cognitions as mediators between VD symptoms and depression. (D) Multiple parallel mediation model investigating physical concerns and loss of control as mediators between VD symptoms and depression. Mediation models with regard to anxiety symptoms as outcome variable (t2) were defined accordingly.

TABLE 1. Descriptive Statistics of Questionnaire-Based Measures (N = 210)

	M (SD)	Range
VD symptoms (t0)		
VSS-VER	0.96 (0.71)	0.00–3.53
Symptom-related cognitions (t1)		
ACQ	1.31 (.33)	1.00–3.07
ACQ physical crisis	1.37 (.46)	1.00–3.40
ACQ loss of control	1.30 (.40)	1.00–3.29
Symptom-related fears (t1)		
BSQ	1.88 (.66)	1.00–4.41
Depression (t2)		
BDI–II	11.90 (9.47)	0–47
Anxiety (t2)		
BAI	12.85 (11.08)	0–56

TABLE 2. ACQ and BSQ Scores by Diagnosis and Number of Diagnoses

Diagnosis	*n* (%)	ACQ (t1), *M* (SD)	BSQ (t1), *M* (SD)
Functional VD	71 (34%)	1.39 (0.400)	2.01 (0.524)
Benign paroxysmal positional vertigo	37 (18%)	1.28 (0.305)	1.81 (0.695)
Ménière's disease	37 (18%)	1.26 (0.282)	1.88 (0.693)
Vestibular migraine	31 (15%)	1.34 (0.301)	1.86 (0.649)
Bilateral vestibulopathy	24 (11%)	1.26 (0.292)	1.67 (0.524)
Multisensory deficit	16 (8%)	1.24 (0.197)	1.93 (0.719)
Central vertigo	15 (7%)	1.38 (0.332)	1.97 (0.643)
Vestibular paroxysmia	13 (6%)	1.41 (0.330)	2.00 (0.617)
Polyneuropathy	12 (6%)	1.26 (0.187)	1.91 (0.587)
Vestibular neuritis	12 (6%)	1.24 (0.302)	1.77 (0.583)
Unilateral vestibular loss	8 (4%)	1.34 (0.420)	2.02 (0.867)
Not otherwise specified	26 (12%)	1.29 (0.320)	1.88 (0.746)
No. diagnoses			
n = 1	135 (64%)	1.30 (0.340)	1.85 (0.671)
n = 2	56 (27%)	1.33 (0.332)	1.85 (0.640)
n = 3	19 (9%)	1.34 (0.278)	2.12 (0.624)

and t2, and BAI at t0 and t2) are illustrated in Table 3. Correlations were all positive and statistically significant.

Mediation Analyses Between Vertigo Symptoms and Depression

Throughout the mediation models, age did not contribute significantly to any of the regression analyses, that is, regression coefficients for age as a covariate in the prediction of the mediator or outcome variables failed to reach statistical significance. The results of the simple mediation models analyzing the IE of vertigo symptoms on depression after 1 year through symptom-related fears and cognitions (models [A] and [B]) revealed that both variables of interest at t1 functioned as significant mediators with the upper and lower levels of the CI of the IE being above zero. With regard to symptom-related fears (BSQ), the CS amounted to 0.06, indicating a small effect after baseline BDI–II scores had been netted out (IE = 0.53, Boot SE = 0.25; Boot LLCI = 0.07, Boot ULCI = 1.35). When symptom-related cognitions (ACQ) were taken into account in a simple mediation model, a CS of 0.11 indicated a medium effect (IE = 0.96, *B*oot SE = 0.32; Boot LLCI = 0.32, Boot ULCI = 1.95).

Only ACQ remained a significant mediator when both ACQ and BSQ scores were included in a multiple parallel mediation model (ACQ: IE = 0.92, Boot SE = 0.33; Boot LLCI = 0.27, Boot ULCI = 1.98; BSQ: IE = 0.07, Boot SE = 0.22; Boot LLCI = −0.45, Boot ULCI = 0.70; model [C]; see Table 4). The combination of the two variables accounted for a CS of 0.11 (BSQ: CS = 0.01; ACQ: CS = 0.10) after baseline scores of the outcome had been controlled for. In a pairwise comparison between the specific IEs, body-related cognitions (ACQ) made a significantly larger contribution than symptom-related fears (BSQ; IE contrast = 0.86, Boot SE = 0.46; Boot LLCI = 0.05, Boot ULCI = 1.87). Finally, when the two ACQ subscales were included in a multiple parallel mediation model (model [D]; see Table 5), their combination fully mediated the effect vertigo symptoms had on depression after baseline depression scores were controlled for, with a CS of 0.10 indicating a medium effect size. Only for the ACQ subscale loss of control, the CI of the IE did not include zero (ACQ loss of control: IE = 0.51, Boot SE = 0.28; Boot LLCI = 0.03, Boot ULCI = 1.47); although the IE of the ACQ subscale physical crisis was significant on a 5% alpha level, it failed to reach significance when multiple testing was corrected for (ACQ physical crisis: IE = 0.41, Boot SE = 0.22; Boot LLCI = −0.01, Boot ULCI = 1.15). In a pairwise comparison between the specific IEs, neither of the two ACQ subscales made a significantly larger contribution than the other (IE contrast = −0.10, Boot SE = 0.39; Boot LLCI = −1.00, Boot ULCI = 0.58).

TABLE 3. Pearson Correlation Coefficients Between the Measures Used in This Study (N = 210)

	VD Symptoms (VSS-VER, t0)	Symptom-Related Cognitions (ACQ, t1)	Symptom-Related Fears (BSQ, t1)	Depression (BDI–II, t2)	Depression (BDI–II, t0)	Anxiety (BAI, t2)	Anxiety (BAI, t0)
VSS-VER (t0)	–	.32*	.27*	.26*	.31*	.28*	.35*
ACQ (t1)	–	–	.58*	.62*	.54*	.56*	.55*
BSQ (t1)	–	–	–	.47*	.44*	.54*	.51*
BDI–II (t2)	–	–	–	–	.75*	.70*	.58*
BDI–II (t0)	–	–	–	–	–	.57*	.72*
BAI (t2)	–	–	–	–	–	–	.62*
BAI (t0)	–	–	–	–	–	–	–

Note: t0 indicates baseline; t1, 6-month follow-up; t2, 12-month follow-up.

*p < 0.0025 (Bonferroni correction).

TABLE 4. Symptom-Related Fears (BSQ) and Cognitions (ACQ) as Mediators of the Effect Vertigo Symptoms Exert on Depression (BDI–II) in a Multiple Mediation Model (N = 210)

	Consequent								
	M1 (BSQ at t1)			M2 (ACQ at t1)			*Y* (BDI–II at t2)[a]		
Antecedent	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***
X (vertigo symptoms at t0)	0.26	0.06	<0.001	0.14	0.03	<0.001	0.42	0.63	0.50
M1 (BSQ at t1)	–	–	–	–	–	–	0.26	0.76	0.74
M2 (ACQ at t1)	–	–	–	–	–	–	6.54	1.55	<0.001
Age	0.00	0.00	0.68	0.00	0.00	0.18	0.04	0.03	0.15
Constant	–	–	–	–	–	–	−10.96	2.48	<0.001
	R^2 = 0.08			R^2 = 0.11			R^2 = 0.13		
	$F(2,207)$ = 8.37			$F(2,207)$ = 13.00			$F(4,205)$ = 7.30		
	p < 0.001			p < 0.001			p < 0.001		
Bootstrap results for the IE	Effect	Boot SE		Boot LLCI			Boot ULCI		
IE through BSQ	0.07	0.22		−0.45			0.70		
IE through ACQ	0.92	0.33		0.27			1.98		

[a]Residuals from simple linear regression analysis were entered into mediation analyses to control for BDI–II baseline scores.

Note: *X* indicates independent variable; *Y*, outcome variable; M, mediator; Coeff., coefficient; SE, standard error; Boot, bootstrapping; LLCI, lower level of the 98.75% CI; ULCI, upper level of the 98.75% CI.

Mediation Analyses Between Vertigo Symptoms and Anxiety

The results of the simple mediation models analyzing the IE of vertigo symptoms on anxiety after 1 year through symptom-related fears and cognitions (models [A] and [B]) showed that both variables of interest at t1 functioned as significant mediators. With regard to symptom-related fears (BSQ), the CS amounted to 0.08, indicating a small effect after baseline BAI scores had been controlled for (IE = 0.96, Boot SE = 0.33; Boot LLCI = 0.34, Boot ULCI = 2.09). When symptom-related cognitions (ACQ) were taken into account in a simple mediation model, a CS of 0.09 indicated a medium effect (IE = 1.09, Boot SE = 0.40; Boot LLCI = 0.29, Boot ULCI = 2.40).

Only BSQ remained a significant mediator when both the ACQ and BSQ scores were included in a multiple parallel mediation model (ACQ: IE = 0.73, Boot SE = 0.41; Boot LLCI = −0.13, Boot ULCI = 2.08; BSQ: IE = 0.60, Boot SE = 0.29; Boot LLCI = 0.03, Boot ULCI = 1.56; model [C]; see Table 6). The combination of the two variables accounted for a CS of 0.11 (BSQ: CS = 0.05; ACQ: CS = 0.06) after BAI baseline scores had been controlled for. The CI of the contrast of the IEs did not indicate that either of them made a significantly larger contribution (IE contrast = −0.13, Boot SE = 0.57; Boot LLCI = −1.32, Boot ULCI = 0.94).

When the two ACQ subscales were contrasted in a multiple parallel mediation model (model [D]; see Table 7), only the ACQ subscale physical crisis remained a significant mediator (ACQ physical crisis:

TABLE 5. ACQ-Physical Crisis and ACQ Loss of Control as Mediators of the Effect Vertigo Symptoms Exert on Depression (BDI–II) in a Multiple Mediation Model (N = 210)

	Consequent								
	M1 (ACQ Physical Crisis at t1)			M2 (ACQ Loss of Control at t1)			*Y* (BDI–II at t2)[a]		
Antecedent	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***
X (vertigo symptoms at t0)	0.18	0.04	<0.001	0.13	0.04	0.001	0.35	0.62	0.57
M1 (ACQ physical crisis at t1)	–	–	–	–	–	–	2.45	1.02	0.02
M2 (ACQ loss of control at t1)	–	–	–	–	–	–	3.92	1.16	<0.001
Age	0.00	0.00	0.87	0.00	0.00	0.12	0.04	0.03	0.15
Constant	–	–	–	–	–	–	−10.38	2.36	<0.001
	R^2 = 0.07			R^2 = 0.08			R^2 = 0.13		
	$F(2,207)$ = 7.61			$F(2,207)$ = 8.55			$F(4,205)$ = 7.84		
	p < 0.001			p < 0.001			p < 0.001		
Bootstrap results for the IE	Effect	Boot SE		Boot LLCI			Boot ULCI		
IE through ACQ physical crisis	0.41	0.22		−0.01			1.15		
IE through ACQ loss of control	0.51	0.28		0.03			1.47		

[a]Residuals from simple linear regression analysis were entered into mediation analyses to control for BDI–II baseline scores.

Note: *X* indicates independent variable; *Y*, outcome variable; M, mediator; Coeff., coefficient; SE, standard error; Boot, bootstrapping; LLCI, lower level of the 98.75% CI; ULCI, upper level of the 98.75% CI.

TABLE 6. Symptom-Related Fears (BSQ) and Cognitions (ACQ) as Mediators of the Effect Vertigo Symptoms Exert on Anxiety (BAI) in a Multiple Mediation Model (N = 210)

	Consequent								
	M1 (BSQ at t1)			M2 (ACQ at t1)			*Y* (BAI at t2)[a]		
Antecedent	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***
X (vertigo symptoms at t0)	0.26	0.07	<0.001	0.14	0.03	<0.001	0.22	0.90	0.80
M1 (BSQ at t1)	–	–	–	–	–	–	2.29	1.08	0.03
M2 (ACQ at t1)	–	–	–	–	–	–	5.14	2.20	0.02
Age	0.00	0.00	0.67	0.00	0.00	0.22	0.05	0.04	0.17
Constant	–	–	–	–	–	–	−13.94	3.51	<0.001
	R^2 = 0.07			R^2 = 0.11			R^2 = 0.11		
	F(2,207) = 7.95			F(2,207) = 12.20			F(4,205) = 6.09		
	p < 0.001			p < 0.001			p < 0.001		
Bootstrap results for the IE	Effect	Boot SE		Boot LLCI			Boot ULCI		
IE through BSQ	0.60	0.29		0.03			1.56		
IE through ACQ	0.73	0.41		−0.13			2.08		

[a]Residuals from simple linear regression analysis were entered into mediation analyses to control for BAI baseline scores.

Note: X indicates independent variable; *Y*, outcome variable; M, mediator; Coeff., coefficient; SE, standard error; Boot, bootstrapping; LLCI, lower level of the 98.75% CI; ULCI, upper level of the 98.75% CI.

IE = 1.01, Boot SE = 0.47; Boot LLCI = 0.21, Boot ULCI = 2.60; ACQ loss of control: IE = 0.20, Boot SE = 0.26; Boot LLCI = −0.46, Boot ULCI = 1.03). Its CS amounted to 0.08, indicating a small effect. However, the descriptive difference of the IEs of the ACQ subscales did not meet criteria for statistical significance (IE contrast = 0.82, Boot SE = 0.60; Boot LLCI = −0.12, Boot ULCI = 2.31).

DISCUSSION

In this study, we examined the role of symptom-related fears and cognitions as mediators of the longitudinal effect that VD symptoms exert on symptoms of anxiety and depression after 1 year. The results across all simple mediation models support our hypothesis that their association is (at least partly) mediated by both dysfunctional apprehensions and fear of physical symptoms after baseline levels of depression and anxiety had been netted out. More precisely, reacting to physical symptoms fearfully and with anxiety-related cognitions such as fear of having a crisis or losing control over their body contributed to the process to develop psychological distress after a VD disorder. Overall, effect sizes were small to medium; because total effects were small in the first place, only a limited amount of variance in the outcome variables could be explained with the models at hand.

With regard to depression, maladaptive cognitions about bodily sensations made a significantly larger contribution than symptom-related fears. In particular, a lack of perceived level of control with regard to symptoms contributed to the development of depression after VD.

With regard to anxiety, body-related fears and cognitions accounted for a similar amount of the total effect that VD had on

TABLE 7. ACQ Physical Crisis and ACQ Loss of Control as Mediators of the Effect Vertigo Symptoms Exert on Anxiety (BAI) in a Multiple Mediation Model (N = 210)

	Consequent								
	M1 (ACQ Physical Crisis at t1)			M2 (ACQ Loss of Control at t1)			*Y* (BAI at t2)[a]		
Antecedent	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***	**Coeff.**	**SE**	***p***
X (vertigo symptoms at t0)	0.17	0.05	<0.001	0.13	0.04	0.001	0.11	0.87	0.90
M1 (ACQ physical crisis at t1)	–	–	–	–	–	–	6.11	1.44	<0.001
M2 (ACQ loss of control at t1)	–	–	–	–	–	–	1.47	1.64	0.37
Age	0.00	0.00	0.91	0.00	0.00	0.15	0.05	0.04	0.18
Constant	–	–	–	–	–	–	−13.26	3.33	<0.001
	R^2 = 0.07			R^2 = 0.07			R^2 = 0.13		
	F(2,207) = 7.09			F(2,207) = 8.07			F(4,205) = 7.50		
	p < 0.001			p < 0.001			p < 0.001		
Bootstrap results for the IE	Effect	Boot SE		Boot LLCI			Boot ULCI		
IE through ACQ physical crisis	1.01	0.47		0.21			2.60		
IE through ACQ loss of control	0.20	0.26		−0.46			1.03		

[a]Residuals from simple linear regression analysis were entered into mediation analyses to control for BAI baseline scores.

Note: *X* indicates independent variable; *Y*, outcome variable; M, mediator; Coeff., coefficient; SE, standard error; Boot, bootstrapping; LLCI, lower level of the 98.75% CI; ULCI, upper level of the 98.75% CI.

secondary anxiety symptoms. This is in line with the earlier finding that persistent fear of vertigo and panic-related thoughts predicted the development of secondary panic disorder in a subgroup of VD patients with vestibular neuritis (Godemann et al., 2006); however, in our sample, only symptom-related fears remained a significant mediator when cognitions were controlled for. When taking a closer look at the ACQ subscales, only the anticipation of a physical crisis but not loss of control was a significant mediator when statistically controlling for the other subscale, even though the comparison failed to reach statistical significance.

Although the role of somatic symptom-related fears and cognitions has been well researched in functional VD and further somatoform disorders (Martin and Rief, 2011), the results at hand suggest that they come into effect irrespective of the VD symptoms' etiology. More importantly, the observation that symptom-related fears and cognitions mediate the effect VD had on psychological distress was not limited to patients with a nonorganic cause of their symptoms, but occurred across a wide range of diagnoses. Conforming to Tschan et al. (2010), high scores on the ACQ and BSQ scales were not limited to functional VD patients; in their validation study of the German Vertigo Handicap Questionnaire, those two scales did not differentiate between patients with functional versus organic VD. In line with this, Pollak et al. (2012) showed that even in an organic VD disorder with a clear understanding of its cause and straightforward treatment, benign paroxysmal positional vertigo, there appears to be a surprising rigidity of feelings and beliefs toward the illness even after treatment.

The results give rise to the question in what way dysfunctional cognitions and fears contribute to the development of psychological distress. In their cognitive behavioral model of persistent postural-perceptual dizziness, Whalley and Cane (2017) outline psychological processes in the development and maintenance of the disorder that show similarities to cognitive models of health anxiety and panic disorder. They stress the role of a) unhelpful appraisals, b) avoidance and safety behaviors, and c) attentional strategies such as selective attention to body sensations in the maintenance of dizziness that can be triggered by both organic and nonspecific VD episodes. Our results suggest that those mechanisms might come into play not only in the perpetuation of dizziness (Heinrichs et al., 2007) but also in the development of further psychopathology.

Negative perceptions and fear of somatic symptoms may lead to the avoidance of a range of activities and physical demands (Yardley, 1994a), a mechanism that has commonly been noted in panic and phobia and inspired similar treatment strategies (Edelman et al., 2012). In fact, many patients with anxiety disorders specified VD as the most anxiety-provoking and uncomfortable sensations among the bodily sensations that may be caused by panic attacks (Asmundson et al., 1998).

Although the interplay between vestibular disorders and anxiety has repeatedly been pointed out, the association with symptoms of depression seems somewhat less apparent. Nakao and Yano (2006) found that dizziness is a significant predictor of depression, with the mediating processes remaining unclear. With regard to our results, symptom-related cognitions, loss of control in particular, might play a greater role in the development of depression compared with anxiety after VD. Patients may become passive, withdrawn, or excessively help-seeking if they feel they are not in control of their symptoms and have a low perceived self-efficacy. Indeed, in their longitudinal study with patients with recurrent vertigo, Yardley (1994b) found that handicap was negatively related to internal locus of control and positively correlated with relinquishing responsibility.

On a positive note, negative beliefs about the consequences of VD can be subject to modification in cognitive psychotherapy (Yardley et al., 2001). Dysfunctional fears and beliefs related to somatic sensations can be targeted both implicitly and explicitly: the parallels between vestibular rehabilitation and interoceptive exposure to feared sensations in cognitive behavioural therapy have been outlined before (Staab, 2011). By testing the validity of fears about VD, they not only promote psychological habituation to the symptom but can also build confidence in the predictability and controllability of symptoms. In addition, beliefs about VD can be challenged explicitly via cognitive restructuring. Providing an adequate appraisal of VD symptoms might be helpful in preventing secondary depressive and anxiety symptoms from developing and exacerbating in patients with various vestibular disorders.

Focusing on protective instead of risk factors, Tschan et al. (2011) found that patients who reported resilient coping and a higher sense of coherence were indeed less likely to develop a comorbid mental disorder 1 year after vestibular disease.

Strengths and Limitations

To our knowledge, this is the first study to investigate how symptom-related fears and beliefs contribute to the effect VD symptoms longitudinally exert on the development of anxiety and depression. The large sample includes patients with a wide range of vestibular disorders, and measurements cover a follow-up period over 1 year.

However, the study does have some methodological shortcomings. First, given the correlational and nonexperimental design of the study, no causal interpretation can be made with certainty at this stage; in light of this, results should be interpreted cautiously.

Second, the study was conducted in a tertiary care setting (*i.e.*, a highly specialized outpatient clinic for vertigo and balance disorders) and therefore may have involved selection bias. In addition, because patients who were excluded from analyses due to missing data or loss at follow-up were significantly younger, the current sample is not fully representative for the patients' group of interest. However, the generalizability of the results might not be too strongly impaired as the age difference amounted to a small effect size only and age did not influence the results of the mediation models.

Third, the study focuses on somatic symptom-related fears and cognitions, not taking into account overall illness beliefs, that is, assumptions about the cause, course, and the expected response to treatment as well as controllability of the illness itself (not its symptoms). These illness perceptions may add to the way people cope with and respond emotionally to an illness; they have been shown to affect health-related quality of life and predict long-term outcome (Frostholm et al., 2007). However, we did concentrate on symptom cognitions in light of the heterogeneity of diagnoses, thereby considering that, for instance, the expectation of an illness to be long-lasting might be the result of a cognitive distortion in some patients or an unfortunately correct assumption in certain diagnostic groups such as Ménière's disease.

Fourth, it was pointed out that there is partial item overlap between the mediator and outcome measures that might have introduced bias; when analyses were repeated without respective items (specifically, items 5, 14, and 16 from the BAI), significance and effect size of the IEs did not change.

A final point of potential critique lies within the study design: measures of somatic symptom-related fears and beliefs at the first follow-up after 6 months were analyzed as mediator variables of interest. One might argue that those appraisals and fearful responses appear in closer time proximity after VD symptoms. However, the analyses yielded comparable results if baseline scores of the respective variables were entered into mediation models; the results are available from the author upon request.

CONCLUSION

Fear of bodily sensations and dysfunctional cognitions about these symptoms longitudinally contribute to the effect that initial vertigo and dizziness symptoms, irrespective of their etiology, have on the development of anxiety and depression. These findings are

compatible with cognitive-behavioral models of psychological sequelae after VD disorders and point to the potential benefits of interventions to modify specific symptom-related beliefs and fears via different kinds of psychotherapies such as cognitive behavioural therapy.

DISCLOSURE

Parts of this project were supported by funds from the German Federal Ministry of Education and Research under grant code 01 EO 0901. The authors bear full responsibility for the content of this publication.

The authors declare no conflict of interest.

REFERENCES

American Psychiatric Association [APA] (2013) *DSM-V.* Washington, DC: APA Publishing.

Arroll M, Dancey CP, Attree EA, Smith S, James T (2012) People with symptoms of Ménière's disease: The relationship between illness intrusiveness, illness uncertainty, dizziness handicap, and depression. *Otol Neurotol*. 33:816–823.

Asmundson GJ, Larsen DK, Stein MB (1998) Panic disorder and vestibular disturbance: An overview of empirical findings and clinical implications. *J Psychosom Res*. 44: 107–120.

Barsky AJ (1992) Amplification, somatization, and the somatoform disorders. *Psychosomatics*. 33:28–34.

Beck AT (2005) The current state of cognitive therapy: A 40-year retrospective. *Arch Gen Psychiatry.* 62:953–959.

Best C, Eckhardt-Henn A, Diener G, Bense S, Breuer P, Dieterich M (2006) Interaction of somatoform and vestibular disorders. *J Neurol Neurosurg Psychiatry.* 77: 658–664.

Best C, Eckhardt-Henn A, Tschan R, Dieterich M (2009a) Psychiatric morbidity and comorbidity in different vestibular vertigo syndromes. Results of a prospective longitudinal study over one year. *J. Neurol*. 256:58–65.

Best C, Eckhardt-Henn A, Tschan R, Dieterich M (2009b) Why do subjective vertigo and dizziness persist over one year after a vestibular vertigo syndrome? *Ann N Y Acad Sci*. 1164:334–337.

Bigelow RT, Semenov YR, du Lac S, Hoffman HJ, Agrawal Y, Lac S (2015) Vestibular vertigo and comorbid cognitive and psychiatric impairment: The 2008 National Health Interview Survey. *J Neurol Neurosurg Psychiatry.* 87:367–372.

Chambless DL, Caputo GC, Bright P, Gallagher R (1984) Assessment of fear of fear in agoraphobics: The Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. *J Consult Clin Psychol*. 52:1090–1097.

Eagger S, Luxon LM, Davies RA, Coelho A, Ron MA (1992) Psychiatric morbidity in patients with peripheral vestibular disorder: A clinical and neuro-otological study. *J Neurol Neurosurg Psychiatry.* 55:383–387.

Eckhardt-Henn A, Best C, Bense S, Breuer P, Diener G, Tschan R, Dieterich M (2008) Psychiatric comorbidity in different organic vertigo syndromes. *J Neurol*. 255: 420–428.

Edelman S, Mahoney AE, Cremer PD (2012) Cognitive behavior therapy for chronic subjective dizziness: A randomized, controlled trial. *Am J Otolaryngol*. 33: 395–401.

Ehlers A, Margraf J (2001) *Fragebogen zu körperbezogenen Ängsten, Kognitionen und Vermeidung (AKV)*. Göttingen: Beltz Test GmbH.

Frostholm L, Oernboel E, Christensen KS, Toft T, Olesen F, Weinman J, Fink P (2007) Do illness perceptions predict health outcomes in primary care patients? A 2-year follow-up study. *J Psychosom Res*. 62:129–138.

Furman JM, Balaban CD, Jacob RG, Marcus DA (2005) Migraine-anxiety related dizziness (MARD): A new disorder? *J Neurol Neurosurg Psychiatry.* 76:1–8.

Gellatly R, Beck AT (2016) Catastrophic thinking: A transdiagnostic process across psychiatric disorders. *Cognit Ther Res*. 40:441–452.

Godemann F, Koffroth C, Neu P, Heuser I (2004) Why does vertigo become chronic after neuropathia vestibularis? *Psychosom Med*. 66:783–787.

Godemann F, Schabowska A, Naetebusch B, Heinz A, Ströhle A (2006) The impact of cognitions on the development of panic and somatoform disorders: A prospective study in patients with vestibular neuritis. *Psychol Med*. 36:99–108.

Hautzinger M, Keller F, Kühner C (2006) *Beck Depressions-Inventar Revision (BDI–II)*. Frankfurt am Main: Harcourt Test Services.

Hayes A (2013) Introduction to mediation, moderation, and conditional process analysis (pp 3–4). New York, NY: Guilford.

Heinrichs N, Edler C, Eskens S, Mielczarek MM, Moschner C (2007) Predicting continued dizziness after an acute peripheral vestibular disorder. *Psychosom Med*. 69:700–707.

Kirby SE, Yardley L (2009) Cognitions associated with anxiety in Ménière's disease. *J Psychosom Res*. 66:111–118.

Kühner C, Bürger C, Keller F, Hautzinger M (2007) Reliability and validity of the Revised Beck Depression Inventory (BDI–II). Results from German samples. *Nervenarzt*. 78:651–656.

Lahmann C, Henningsen P, Brandt T, Strupp M, Jahn K, Dieterich M, Eckhardt-Henn A, Feuerecker R, Dinkel A, Schmid G (2015) Psychiatric comorbidity and psychosocial impairment among patients with vertigo and dizziness. *J Neurol Neurosurg Psychiatry.* 86:302–308.

Lahmann C, Henningsen P, Dieterich M, Feuerecker R, Cyran CA, Schmid G (2012) The Munich Diagnostic and Predictor Study of Dizziness: Objectives, design, and methods. *J Neurol*. 259:702–711.

Limburg K, Sattel H, Radziej K, Lahmann C (2016) *DSM-5* somatic symptom disorder in patients with vertigo and dizziness symptoms. *J Psychosom Res*. 91:26–32.

Margraf J, Ehlers A (2007) *Das Beck Angst-Inventar (BAI)–Manual*. Deutsche Bearbeitung. Frankfurt am Main: Harcourt Test Services GmbH.

Martin A, Rief W (2011) Relevance of cognitive and behavioral factors in medically unexplained syndromes and somatoform disorders. *Psychiatr Clin North Am*. 34:565–578.

Nakao M, Barsky AJ (2007) Clinical application of somatosensory amplification in psychosomatic medicine. *Biopsychosoc Med*. 1:17.

Nakao M, Yano E (2006) Somatic symptoms for predicting depression: One-year follow-up study in annual health examinations. *Psychiatry Clin Neurosci*. 60:219–225.

Neuhauser HK (2007) Epidemiology of vertigo. *Curr Opin Neurol*. 20:40–46.

Paulus MP, Stein MB (2010) Interoception in anxiety and depression. *Brain Struct Funct*. 214:451–463.

Pérez N, Martin E, Garcia-Tapia R (2003) Dizziness: Relating the severity of vertigo to the degree of handicap by measuring vestibular impairment. *Otolaryngol Head Neck Surg*. 128:372–381.

Pollak L, Segal P, Stryjer R, Stern HG (2012) Beliefs and emotional reactions in patients with benign paroxysmal positional vertigo: A longitudinal study. *Am J Otolaryngol*. 33:221–225.

Preacher KJ, Kelley K (2011) Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychol Methods*. 16:93–115.

Probst T, Dinkel A, Schmid-Mühlbauer G, Radziej K, Limburg K, Pieh C, Lahmann C (2017) Psychological distress longitudinally mediates the effect of vertigo symptoms on vertigo-related handicap. *J Psychosom Res*. 93:62–68.

Schmid G, Henningsen P, Dieterich M, Sattel H, Lahmann C (2011) Psychotherapy in dizziness: A systematic review. *J Neurol Neurosurg Psychiatry.* 82:601–606.

Staab JP (2011) Behavioral aspects of vestibular rehabilitation. *NeuroRehabilitation*. 29:179–183.

Staab JP, Ruckenstein MJ (2003) Which comes first? Psychogenic dizziness versus otogenic anxiety. *Laryngoscope*. 113:1714–1718.

Tschan R, Best C, Beutel ME, Knebel A, Wiltink J, Dieterich M, Eckhardt-Henn A (2011) Patients' psychological well-being and resilient coping protect from secondary somatoform vertigo and dizziness (SVD) 1 year after vestibular disease. *J Neurol*. 258:104–112.

Tschan R, Wiltink J, Best C, Bense S, Dieterich M, Beutel ME, Eckhardt-Henn A (2008) Validation of the German version of the Vertigo Symptom Scale (VSS)

in patients with organic or somatoform dizziness and healthy controls. *J Neurol*. 255:1168–1175.

Tschan R, Wiltink J, Best C, Beutel M, Dieterich M, Eckhardt-Henn A (2010) Validation of the German version of the Vertigo Handicap Questionnaire (VHQ) in patients with vestibular vertigo syndromes or somatoform vertigo and dizziness. *Psychother Psychosom Med Psychol*. 60:e1–e12.

Tusa RJ (2009) Dizziness. *Med Clin North Am*. 93:263–271.

Vîslă A, Flückiger C, Grosse Holtforth M, David D (2016) Irrational beliefs and psychological distress: A meta-analysis. *Psychother Psychosom*. 85:8–15.

Weidt S, Bruehl AB, Straumann D, Hegemann SC, Krautstrunk G, Rufer M (2014) Health-related quality of life and emotional distress in patients with dizziness: A cross-sectional approach to disentangle their relationship. *BMC Health Serv Res*. 14:317.

Whalley MG, Cane DA (2017) A cognitive-behavioral model of persistent postural-perceptual dizziness. *Cogn Behav Pract*. 24:72–89.

Wiltink J, Tschan R, Michal M, Subic-Wrana C, Eckhardt-Henn A, Dieterich M, Beutel MA (2009) Dizziness: Anxiety, health care utilization and health behavior–results from a representative German community survey. *J Psychosom Res*. 66:417–424.

Yardley L (1994a) Contribution of symptoms and beliefs to handicap in people with vertigo: A longitudinal study. *Br J Clin Psychol*. 33:101–113.

Yardley L (1994b) Prediction of handicap and emotional distress in patients with recurrent vertigo: Symptoms, coping strategies, control beliefs and reciprocal causation. *Soc Sci Med*. 39:573–581.

Yardley L, Beech S, Weinman J (2001) Influence of beliefs about the consequences of dizziness on handicap in people with dizziness, and the effect of therapy on beliefs. *J Psychosom Res*. 50:1–6.

Yardley L, Masson E, Verschuur C, Haacke N, Luxon L (1992) Symptoms, anxiety and handicap in dizzy patients: Development of the Vertigo Symptom Scale. *J Psychosom Res*. 36:731–741.

Manuscript III

Radziej, K., Schmid-Mühlbauer, G., Limburg, K., & Lahmann, C. (2017). Psychotherapie bei Schwindelerkrankungen - Eine störungsorientierte Gruppenbehandlung für angst-, depressions- und somatoform betonten Schwindel. *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *67,* 245–251.

Thieme

Psychotherapie bei Schwindelerkrankungen – Eine störungsorientierte Gruppenbehandlung für angst-, depressions- und somatoform betonten Schwindel

Tailored Care for Functional Vertigo/Dizziness – An Integrative Group Psychotherapy Approach

Autoren
Katharina Radziej, Gabriele Schmid-Mühlbauer, Karina Limburg, Claas Lahmann

Institut
Klinik für Psychosomatische Medizin und Psychotherapie, Klinikum rechts der Isar, Technische Universität München, München

Schlüsselwörter
Schwindel, somatoforme Störung, funktionelle Störung, Gruppenpsychotherapie, integrative Psychotherapie

Key words
dizziness, vertigo, somatoform disorder, group psychotherapy, integrative psychotherapy

eingereicht 19.10.2016
akzeptiert 21.02.2017

Bibliografie
DOI http://dx.doi.org/10.1055/s-0043-104768
Psychother Psych Med 2017; 67: 245–251

ISSN 0937-2032

Korrespondenzadresse
Dipl.-Psych. Katharina Radziej
Klinik für Psychosomatische Medizin und Psychotherapie
Klinikum rechts der Isar, Technische Universität München
Langerstraße 3
81675 München
k.radziej@tum.de

ZUSAMMENFASSUNG

Funktionelle Schwindelbeschwerden zeigen eine hohe Prävalenz und gehen nicht selten mit einer deutlichen Beeinträchtigung im Alltags- und Berufsleben einher. Oft sind sie mit einer Reihe anderer psychischer Störungen, insbesondere phobischen und weiteren Angststörungen, depressiven Störungen oder somatoformen Störungen im engeren Sinne assoziiert. Trotz der klinischen Relevanz ist die Datenlage zur Evidenzbasierung psychotherapeutischer Behandlungsansätze in dieser Patientengruppe nach wie vor dünn. Vor diesem Hintergrund wurde ein Manual zur ambulanten Gruppenpsychotherapie für Patienten mit funktionellen Schwindelerkrankungen entwickelt. Dieses zielt darauf ab, ein besseres Verständnis für die Wechselwirkungen zwischen Körper und psychosozialen Faktoren zu entwickeln und so von einer Symptomfokussierung wegzuführen. Neben regelmäßigen Gleichgewichtsübungen beinhaltet das Programm unter anderem störungsorientierte Module zu Angst- bzw. Panikstörung, Depression und Somatisierung. Das phasendefinierte, 16 wöchentliche Sitzungen umfassende integrativ-psychotherapeutische Behandlungskonzept wird an dieser Stelle inhaltlich vorgestellt und ist Gegenstand einer aktuell laufenden randomisierten, kontrollierten Effektivitätsstudie. Diese soll der Evaluation der kurz- und langfristigen Wirksamkeit im Hinblick auf Schwindelcharakteristika und weitere Körperbeschwerden, die durch den Schwindel erlebten Beeinträchtigungen sozialer und physischer Aktivitäten sowie der schwindelassoziierten Angst und depressiven Beschwerden dienen. In einer Pilotstudie zeigten sich kleine bis große Prä-Follow-up-Effekte auf den primären und sekundären Ergebnismaßen. Aus der Pilotstudie ergeben sich zudem Hinweise auf die Akzeptanz und Durchführbarkeit des Gruppentherapiekonzeptes.

ABSTRACT

Functional vertigo and dizziness (VD) symptoms are highly prevalent and usually accompanied by a strong impairment of quality in everyday and working life. The complaints are often associated with various psychiatric disorders, particularly phobic and other anxiety disorders, depressive or somatoform disorders. Despite this clinical relevance of VD symptoms, studies towards psychotherapeutic treatment options for patients with functional VD are still rare. Thus, the present study outlines a manual for outpatient group therapy for patients with functional VD symptoms. Our approach aims to assist patients in developing an understanding of the interaction between bodily complaints and psychosocial factors while detracting from a purely symptom based focus. The integrative-psychotherapeutic treatment program consists of 16 weekly sessions and includes a regular vestibular rehabilitation training as well as disorder-oriented modules towards anxiety and panic dis-

order, depression, and somatisation. We are currently conducting a randomised controlled trial in order to evaluate the short- and long-term effectiveness of the program in reducing VD and further bodily symptoms, vertigo-related social and physical handicap as well as anxiety and depressive complaints. A previously completed pilot trial has led to small to large pre-follow-up-effects on primary and secondary outcome measures. Conclusions regarding acceptance and feasibility of the therapy concept are discussed.

Hintergrund

Funktioneller Schwindel

Schwindel stellt eines der häufigsten Leitsymptome in der Allgemeinmedizin und Neurologie dar. Einem substanziellen Anteil von ca. 30–50 % der persistierenden Schwindelerkrankungen liegt dabei eine funktionelle Störung zugrunde, die nicht nur mit deutlichen Beeinträchtigungen in der Lebensführung und erlebter Lebensqualität assoziiert ist [1–3], sondern auch deshalb von hoher klinischer und wirtschaftlicher Relevanz ist, weil sie mit einem dysfunktionalen hohen Inanspruchnahmeverhalten im Gesundheitssystems einhergeht [4]. Wird dem Wunsch nach wiederholter somatischer Abklärung nachgegangen, so sieht sich der Patient in seiner somatischen Krankheitsüberzeugung bestätigt, was nicht selten zur Chronifizierungstendenz des funktionellen Schwindels beiträgt. Bis zur adäquaten Diagnosestellung vergehen zeitweise mehrere Jahre; darüber hinaus mangelt es an adäquaten Versorgungsangeboten [5].

Wiederholt wurden klinische Subgruppen funktioneller Schwindelerkrankungen mit der Akzentuierung von Angst- und Panikstörungen, Depressivität oder (im engeren Sinne) somatoformen Beschwerden beschrieben [1, 6]. Eine weitere Klassifikation in primär und sekundär somatoforme Schwindelsyndrome unterscheidet dahin gehend, ob sich der Schwindel als Folge einer organischen Schwindelerkrankung manifestiert oder ohne organische Erkrankung in der Vorgeschichte auftritt (z. B. [7]). Dabei ist das Fehlen eines organpathologischen Korrelats als diagnostisches Kriterium für somatoforme Störungen mit der letzten Überarbeitung des DSM zunehmend in den Hintergrund getreten [8]. Die Symptomatik bei Schwindelerkrankungen wird demnach Ätiologie übergreifend nicht in erster Linie durch ein quantifizierbares vestibuläres Defizit bestimmt [9], sondern unter anderem durch die begleitende Psychopathologie [2, 10, 11] sowie Persönlichkeitsdispositionen und maladaptive Verhaltensstrategien [12–14].

Psychotherapie bei Schwindel

Empirische Evidenz hinsichtlich der Behandlung somatoformer bzw. funktioneller Störungen im Allgemeinen liegt v. a. für die kognitive Verhaltenstherapie vor [15]. Wenngleich Gruppentherapie als primäres Behandlungsangebot bei funktionellen Störungen praktisch kaum untersucht worden ist, zeigten Gruppenangebote als Teil eines komplexeren, i. d. R. stationären, Behandlungsangebots vielversprechende Resultate [16].

Bezogen auf den speziellen Bereich der Schwindelbeschwerden ist die Datenlage nach wie vor dünn. Es gibt Hinweise darauf, dass kognitiv-verhaltenstherapeutische Ansätze in Kombination mit vestibulärem Rehabilitationstraining wirksam sind [17]. Eine klinisch relevante Verbesserung zeigte sich jedoch vordergründig im Hinblick auf eine Reduktion der Schwindelsymptomatik, nicht aber begleitender ängstlicher und depressiver Beschwerden. Während die Effekte einer verhaltenstherapeutischen Intervention von nur 3 wöchentlichen Sitzungen auch über das 6-Monats-Follow-up stabil blieben [18], fielen sie anderenorts nach einem Jahr auf das Ausgangsniveau zurück [19]. In dem schwindelspezifischen Gruppenschulungsprogramm „Standfest“, welches neben psychoedukativen Elementen, Gleichgewichts- und Entspannungstraining v. a. kognitiv-verhaltenstherapeutische Techniken umfasst, zeigte sich eine langfristige Verbesserung hinsichtlich dysfunktionaler Krankheitsrepräsentationen und der Krankheitsbewältigung, nicht aber der Schwindelstärke und psychopathologischen Begleitsymptomatik [20]. Inzwischen liegen auch Ergebnisse zur Wirksamkeit eines multimodalen stationären Behandlungsprogramms vor, dessen Effekte sowohl bezüglich des Schwindels als auch begleitender Angst und depressiver Symptome über ein Jahr aufrechterhalten werden konnten [21]. Für den ambulanten Bereich fehlten bis dato Behandlungsansätze, die den Fokus mehr auf die dem funktionellen Schwindel oft zugrunde liegenden psychischen Störungen legen und damit einen kurz- und langfristigen Effekt auf die begleitende Psychopathologie wahrscheinlicher machen.

Störungsorientiertes Gruppentherapiekonzept

Das im Folgenden beschriebene Behandlungskonzept bündelt therapeutische Strategien und Vorgehensweisen, die sich im Umgang mit primärem und sekundärem funktionellem Schwindel als wirksam erwiesen haben (vgl. [17, 20]), und ist damit im therapeutischen Vorgehen integrativ (Integrative Psychotherapie) und Schulen übergreifend, in der inhaltlichen Ausrichtung jedoch auch störungsorientiert. Erweitert wurden die bisherigen Behandlungsansätze insbesondere um Module zu Angst und Panik, Depression sowie Somatisierung im engeren Sinne, um die häufig bestehende psychopathologische Begleitsymptomatik in dieser Patientengruppe angemessen zu berücksichtigen.

Vor dem Hintergrund der revidierten Klassifikation somatoformer Störungen im DSM-V kann die Indikation des Programms auch auf somatische Schwindelerkrankungen mit chronisch-therapierefraktären Verläufen, die mit hohem Leidensdruck und begleitender Psychopathologie einhergehen, erweitert werden.

Behandlungsrahmen Die Therapie findet im ambulanten geschlossenen Gruppensetting über 16 wöchentliche 90-minütige Sitzungen mit einer zusätzlichen Booster-Sitzung nach 3 Monaten statt. Geleitet werden die Gruppen von je einem Therapeuten und einem Kotherapeuten bei einer Gruppengröße von 6–8 Teilnehmern. Die Gruppenintervention gliedert sich grob in 3 Therapiephasen: Initialphase (Sitzungen 1–4), Arbeitsphase (Sitzungen 5–13) und Transferphase (Sitzungen 14–16).

Phasenübergreifende Therapieelemente

Therapeutische Haltung und Gesprächsführung Da der therapeutischen Beziehung bei somatoformen Beschwerden eine besondere Rolle zukommt, soll von Anfang an durch eine aktiv-stützende Haltung des Therapeuten die Herstellung eines Arbeitsbündnisses gefördert werden. Die Realität der Schwindelbeschwerden wird anerkannt und auf intrapsychische Themenbereiche wird zunächst eher beiläufig eingegangen (tangentiale Gesprächsführung). Somatische Krankheitsmodelle der Patienten sollen nicht konfrontativ infrage gestellt oder Gegenstand einer „Entweder-oder"-Diskussion werden. Stattdessen werden beispielhaft Zusammenhänge mit auslösenden oder modulierenden Faktoren vorgeschlagen, um die individuellen Erklärungsmodelle zu erweitern und zu erarbeiten, dass eine mögliche organische Beteiligung nicht ausreicht, um das Beschwerdebild vollständig zu beschreiben („sowohl als auch"; vgl. [22]). Über den Behandlungsverlauf hinweg soll zudem – durch eine ressourcenorientierte Grundhaltung – das Vertrauen der Patienten in die eigene Handlungsfähigkeit gefördert werden.

Die Gruppe als Akteur Die Therapeuten unterstützen die Gruppe im Aufbau eines Zusammengehörigkeitsgefühls (Supportivität) und fördern die Gruppenkohäsion, indem v. a. solche Gemeinsamkeiten, die von einer Symptomzentrierung wegführen, betont werden. Zugleich erleben die Patienten in der symptomhomogenen Gruppe, dass sie mit den funktionellen Schwindelbeschwerden nicht allein sind, was die Bereitschaft fördern kann, neue Sichtweisen und Veränderungen zuzulassen. Oft gehen der Behandlung als enttäuschend erlebte Beziehungserfahrungen im Gesundheitssystem voraus, verbunden mit dem Gefühl, in der eigenen somatischen Krankheitsüberzeugung nicht ernst genommen zu werden. Vor diesem Hintergrund bildet die Gruppe einen Raum, in dem die Symptomklage entgegengenommen und validiert wird, und gibt die Möglichkeit für Selbstöffnung und interpersonelles Lernen (sensu MacKenzie [23]).

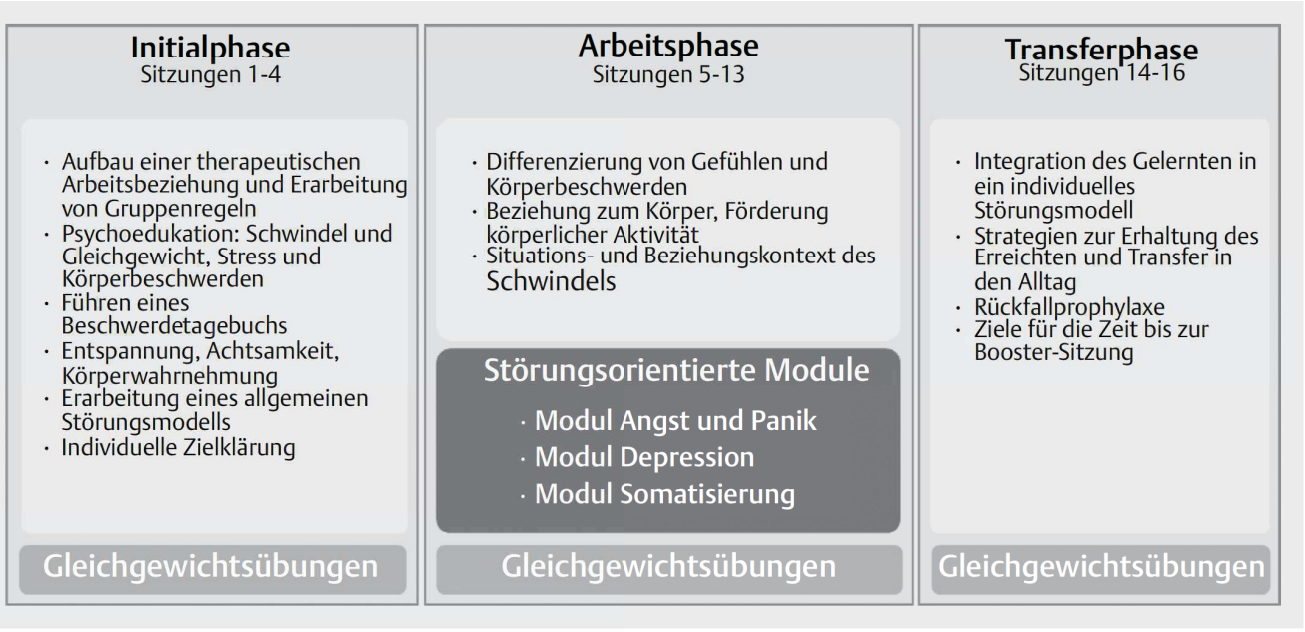
Gleichgewichtsübungen Eine besondere Rolle kommt in dem Behandlungskonzept den Gleichgewichtsübungen zu, die bereits in der zweiten Sitzung eingeführt und fortan regelmäßig zu Beginn der Sitzungen gemeinsam sowie eigenständig zu Hause von den Teilnehmern durchgeführt werden. Für ähnliche vestibuläre Rehabilitationsprogramme konnten nicht nur positive Auswirkungen auf das physische, sondern auch auf das mentale Wohlbefinden gezeigt werden [24].

Verschiedene Autoren beschreiben bei Patienten mit funktionellem Schwindel eine erhöhte visuelle Feldabhängigkeit, d. h. eine Verschiebung der Gewichtung von visuellen, vestibulären und propriozeptiven Reizen zur Haltungs- und Bewegungskontrolle zugunsten visueller Informationen [25]. Der Effekt ist gut vereinbar mit der häufigen Exazerbation der Beschwerden in Situationen mit komplexer visueller Informationsverarbeitung (z. B. offene Plätze, Supermärkte). Bewegung und Haltung unterliegen bei diesen Patienten verstärkt einer bewussten Kontrolle, die zum einen erhöhter Aufmerksamkeitsressourcen bedarf und sich zum anderen durch rigidere Haltungs- und Bewegungsabläufe abbildet. Dazu trägt auch die Angst zu fallen bzw. ein mangelndes Vertrauen in das eigene Gleichgewicht bei, welches mit der Vermeidung Schwindel auslösender Situationen oder Bewegungen einhergeht und dadurch aufrechterhalten wird. Hier werden die Parallelen von vestibulärem Rehabilitationstraining und Expositionsbehandlung in der KVT deutlich [26].

Beschreibung der Therapiephasen

▶ **Abb. 1** gibt einen Überblick über die Struktur des Behandlungsprogramms. Auf die konkreten Inhalte und die Ausgestaltung der Sitzungen wird im Folgenden näher eingegangen.

Initialphase Im Einzel- wie im Gruppensetting kann der psychotherapeutische Prozess dadurch erschwert sein, dass Patienten auf einer organischen Ursache ihrer Beschwerden beharren. Daher kommt der Eingangsphase zur Schaffung von Therapie-



▶ **Abb. 1** Struktur des Behandlungsablaufs.

und Änderungsmotivation eine besondere Bedeutung zu. Über die ersten Sitzungen hinweg wird von Therapeutenseite viel Struktur vorgegeben; einen wesentlichen Baustein stellen psychoedukative Elemente bezüglich des Gleichgewichtssystems, der Entstehung von Schwindel sowie der Wechselwirkungen zwischen körperlichen Beschwerden und Stresserleben dar. Die Gleichgewichtsübungen werden eingeführt und die Teilnehmer ausdrücklich dazu ermuntert, diese täglich als Routine in den Alltag zu integrieren. Kurze Achtsamkeits- und Entspannungsübungen sollen die Patienten dabei unterstützen, die Körperwahrnehmung zu schulen, den Schwindel bewertungsfrei wahrzunehmen und zunehmend positive Körperempfindungen aufzuspüren.

Während dieser Phase wird ein Beschwerdetagebuch geführt, um die Teilnehmer mehr und mehr für situative und emotionale Einflüsse auf die Symptomatik zu sensibilisieren; später kann bei der Erarbeitung eines allgemeinen Störungsmodells auf Beobachtungen aus den Schwindeltagebüchern zurückgegriffen werden. Das Störungsmodell orientiert sich an dem von Rief und Hiller [27] vorgeschlagenen Modell der somatoformen Störung, das Prozesse der Aufmerksamkeitsfokussierung, kognitiver Fehlbewertungen und störungsaufrechterhaltende Verhaltensweisen plausibel beschreibt.

Anschließend kommt dem Erarbeiten realistischer individueller Ziele eine besondere Bedeutung zu – insbesondere, da viele Patienten zwischen starken Versorgungswünschen mit Heilserwartungen einerseits und Resignation bzw. einer durch die oft bereits bestehende Chronifizierung geringen Selbstwirksamkeitserwartung andererseits schwanken. Eine Fantasiereise soll diesen Schritt erleichtern.

Arbeitsphase mit störungsorientierten Modulen In dieser Phase stehen die Beziehung zum eigenen Körper, die Einordnung der Schwindelbeschwerden in einen situativen und Beziehungskontext sowie die spezifischen Module zu Angst- bzw. Panikstörung, Depression und Somatisierung im Fokus. Die Reihenfolge der einzelnen Module wird nicht vorgegeben; auch der konkrete Sitzungsumfang kann an die Bedürfnisse der Gruppe angepasst werden.

Zur Vertiefung eines bio-psycho-sozialen Krankheitsverständnisses werden zunächst mithilfe von Verhaltensanalysen die Schwindelbeschwerden in ihrem Entstehungskontext und Verlauf auf emotionaler, körperlicher, kognitiver und Verhaltensebene rekonstruiert. Die von Patientenseite eingebrachten Erfahrungen psychosomatischer und somatopsychischer Prozesse werden als solche markiert und besprochen; oft bieten auch die Symptomtagebücher hierfür ausreichend Material. Ebenso wird die Rolle dysfunktionaler Gedanken bei der Auslösung bzw. Verstärkung (z. B. die Bewertung natürlicher Körperschwankungen als bedrohlich) und Aufrechterhaltung von Schwindel (z. B. „Der Schwindel ist Zeichen einer schweren Erkrankung".) aufgezeigt. Mithilfe der Gruppe können die Gültigkeit solcher Gedanken überprüft und alternative Gedanken entwickelt werden. Dies dient auch dazu, vereinfachte Ursachenattributionen des Schwindels zu hinterfragen.

In der Arbeitsphase wird immer wieder das Zusammenspiel von Affekten und Körper(miss)empfindungen aufgegriffen. So kann z. B. die Enttäuschung vom eigenen Körper durch fehlende Funktionsfähigkeit Thema sein. Die Patienten werden ermutigt, bei körperlicher Eigenaktivität positive Erfahrungen zu sammeln, um sich von dem Bild des eigenen Körpers als „bloßer Symptomträger" zu lösen. Oftmals haben Betroffene ähnliche Beziehungserfahrungen im Gesundheitssystem, mit Angehörigen oder Kollegen gemacht, die als invalidierend oder enttäuschend erlebt werden.

Das Modul Angst und Panik rückt den Schwindel als Angstsymptom bzw. die Angst als Folge des Schwindels (z. B. Angst vor Kontrollverlust) ins Visier. Paroxysmaler Schwindel kann Ausdruck einer panischen Angst sein, oft begleitet von weiteren Paniksymptomen wie Palpitationen oder Atemnot, ohne dass das klinische Vollbild einer Panikstörung zum Tragen kommt. Anfallsartigem oder Dauerschwindel kann aber auch eine generalisierte Angststörung zugrunde liegen, die mit einer anhaltenden Anspannung und Nervosität einhergeht. In beiden Fällen wird nicht selten die Angst einseitig als Folge der körperlichen Symptomatik erlebt. Wahrscheinlicher ist, dass sich im Sinne eines Circulus vitiosus die emotionalen und körperlichen Symptome mittels katastrophisierender Bewertungen gegenseitig aufschaukeln. Die Bewusstmachung dieses Angstkreislaufs und dessen physiologischen Hintergrunds kann bereits als entlastend erlebt werden.

Die Angst vor der Angst selbst bzw. vor dem Auftreten des Schwindels kann darin münden, dass bestimmte Situationen (z. B. U-Bahn, Rolltreppen, aber auch soziale Situationen) gänzlich vermieden werden. Das Vermeidungsverhalten wird als wesentlicher aufrechterhaltender Faktor problematisiert; die Rationale von Expositionsübungen und Habituation wird gemeinsam mit den Patienten erarbeitet, um Vermeidungsverhalten Schritt für Schritt abzubauen.

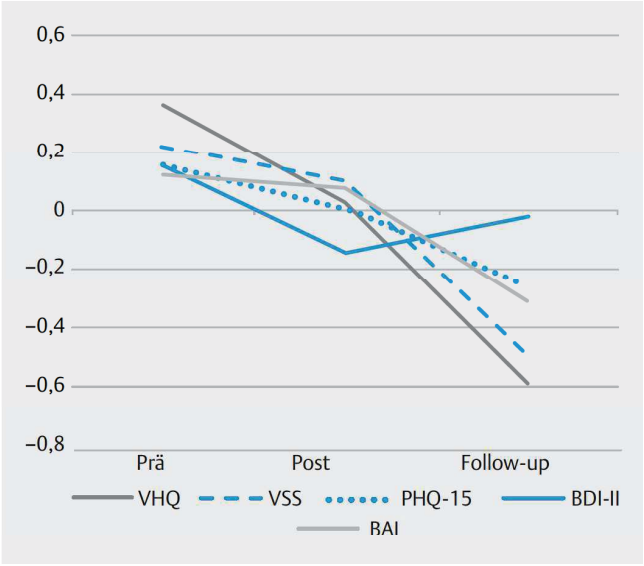
Nicht selten tritt bei Schwindelerkrankungen auch eine depressive Begleitsymptomatik auf oder der Schwindel selbst ersetzt im Sinne eines Affektäquivalents die Wahrnehmung der Niedergeschlagenheit. Im Rahmen des Moduls Depression steht eingangs die Psychoedukation zu depressiven Symptomen und deren Entstehung im Vordergrund. Wenn sich Patienten als „dem Schwindel ausgeliefert" erleben und die eigene Bewältigungskompetenz als niedrig einschätzen, werden Antriebslosigkeit und Inaktivität als aufrechterhaltende Faktoren begünstigt. Ziel ist, den Patienten aus seiner (passiven) Krankenrolle herauszuholen und in eine aktive Rolle zu versetzen, in welcher er Verantwortung für die eigene Genesung übernimmt. Dabei kommen Techniken der kognitiven Umstrukturierung zur Anwendung (z. B. Disputation des Gedankens „Wenn der Schwindel nicht organisch bedingt ist, ist er nicht behandelbar"). Besonderes Augenmerk wird auf das Aufdecken depressiver Denkfehler (sensu Beck) gerichtet, auf die Verbesserung der gegenwärtigen Kommunikationsmuster sowie die generelle Förderung selbstfürsorglichen Verhaltens.

Dies wird im anschließenden Modul Somatisierung weiter vertieft. Gemeinsam mit der Gruppe werden Verhaltensweisen problematisiert, die störungsaufrechterhaltend wirken, z. B. wiederholte somatische Abklärungen, ausgeprägtes Schonverhalten oder das permanente Beobachten und Kontrollieren der Symptome (Checking). Trotz körperlicher Beschwerden soll ein Zutrauen in die eigene körperliche Belastbarkeit gefunden und eine generelle Toleranz für alltägliche körperliche Missempfindungen entwickelt werden. Zu Letzterem trägt bei, dass allein durch die Gleichgewichtsübungen Schwindelsensationen bewusst provoziert werden und die Erfahrung gemacht werden kann, dass diese wieder abklingen. Sukzessive soll dadurch zu einem realistischen Gesundheitsbegriff gefunden werden, der körperliche Gesundheit nicht mit ab-

soluter Freiheit von Missempfindungen gleichsetzt. Die aktuellen Symptome werden vor dem Hintergrund früherer Krankheitserfahrungen und des Umgangs mit Krankheit in der Familie und anderen Beziehungskontexten eingeordnet.

Transferphase Die abschließenden 3 Sitzungen dienen der Bilanzierung über die gemeinsame therapeutische Arbeit. Nachdem das bisher Gelernte in ein individuelles Störungsmodell integriert wird, sollen Risikofaktoren und Strategien für den Umgang mit Rückschlägen erarbeitet werden (Rückfallprophylaxe). Wichtig ist dabei einzuräumen, dass auch bei erneutem oder verstärktem Auftreten von Schwindel die Patienten in ihrem Selbstexpertentum eigenständig am bisher Erreichten weiterarbeiten können. Mit Bezug auf die eingangs formulierten Therapieziele wird überprüft, welche Schritte bereits erfolgreich absolviert wurden und welche (Teil-) Ziele und Veränderungen im Alltag sich für die kommende Zeit herauskristallisieren. In dieser Abschlussphase wird auch Raum gegeben, Emotionen im Hinblick auf das Therapieende – z. B. Unsicherheit, Enttäuschung oder Stolz – zu bearbeiten.

In der Booster-Sitzung treffen die Gruppenteilnehmer erneut aufeinander. Die Sitzung dient der „Auffrischung" der Therapieinhalte und es wird besprochen, inwiefern die erarbeiteten Strategien aufrechterhalten werden konnten, welche Veränderungen und Schwierigkeiten zwischenzeitlich aufgetreten sind und – im Sinne der Ressourcenorientierung – was gut geklappt und wie die Patienten dazu beigetragen haben.



▶ **Abb. 2** Übersicht der z-standardisierten Outcome-Maße über die Messzeitpunkte hinweg.
VHQ = Vertigo Handicap Questionnaire; VSS = Vertigo Symptom Scale; BDI-II = Beck Depressionsinventar, revidierte Version; BAI = Beck Angstinventar; PHQ-15 = Patient Health Questionnaire. In die Berechnung sind die Daten aller n = 12 Gruppenteilnehmer eingegangen.

Studie zur Überprüfung des Therapiekonzepts

Zur Evaluation der kurz- und langfristigen Wirksamkeit des vorab beschriebenen Behandlungsansatzes wird eine randomisierte kontrollierte Effektivitätsstudie, gefördert vom Bundesministerium für Bildung und Forschung (01 EO 1401), durchgeführt. Das Studiendesign ist 2-armig; Als Vergleichsgruppe dient eine moderierte, störungsspezifische Selbsthilfegruppe (low level active control; vgl. [28]). Diese orientiert sich in ihren Rahmenbedingungen wie Sitzungsdauer, Sitzungsfrequenz und Gruppengröße an der Interventionsgruppe und dient der Kontrolle unspezifischer Wirkfaktoren; dazu zählen z. B. die regelmäßigen Sitzungen, Gruppenkohäsion, Zuwendung durch andere und die Möglichkeit zur Selbstöffnung im Gruppensetting (z. B. [23, 29]).

Beide Behandlungsarme (Fallzahlkalkulation N = 216) werden bzgl. der Verbesserung der Schwindelsymptomatik, der durch den Schwindel bedingten physischen und psychosozialen Beeinträchtigungen, sowie assoziierten angstbezogenen und affektiven Beschwerden verglichen. Primärer Endpunkt ist die mit dem Vertigo Handicap Questionnaire (VHQ) erfasste Verbesserung der schwindelbezogenen Lebensqualität ein Jahr nach Behandlungsende. Sekundäre Outcomeparameter sollen die Intensität der Schwindelsymptomatik (Vertigo Symptom Scale, VSS), Depressivität (Beck-Depressions-Inventar, BDI-II), Angst (Beck-Angst-Inventar, BAI) und Körperbeschwerden (Patient Health Questionnaire, PHQ-15) erfassen. Zudem werden posturografische Daten erhoben und eine Kosten-Effektivitäts-Analyse durchgeführt. Alle beschriebenen Maße werden vor der Behandlung, im Anschluss an die Behandlung sowie zum Ein-Jahres-Follow-up erhoben.

▶ **Tab. 1** Rohwerte und Effektstärken der primären und sekundären Outcome-Maße.

Outcome-Maß	Prä-Messung n = 12; M [SD]	Post-Messung n = 12; M [SD]	Follow-up n = 8; M [SD]	Prä-Post-Effekt Hedges g [95 % KI]	Prä-Follow-up-Effekt Hedges g [95 % KI]
VHQ	54,6 [15,44]	48,4 [19,30]	37,1 [18,25]	0,34 [− 0,47;1,15]	0,94 [− 0,09;1,97]
VSS	45,2 [20,90]	42,8 [22,13]	30,9 [13,71]	0,11 [− 0,69;0,91]	0,90 [− 0,13;1,93]
BDI − II	17,8 [9,41]	14,9 [9,62]	16,1 [10,91]	0,29 [− 0,52;1,09]	0,37 [− 0,61;1,36]
BAI	18.0 [9.79]	17.5 [11.25]	13.4 [11.73]	0.05 [− 0.75:0.85]	0.35 [− 0.64:1.34]
PHQ − 15	13,4 [6,22]	12,6 [4,68]	11,1 [6,27]	0,15 [− 0,65;0,95]	0,32 [− 0,66;1,31]

M = Mittelwert; SD = Standardabweichung; KI = Konfidenzintervall; VHQ = Vertigo Handicap Questionnaire; VSS = Vertigo Symptom Scale; BDI-II = Beck Depressionsinventar, revidierte Version; BAI = Beck Angstinventar; PHQ-15 = Patient Health Questionnaire. Zur Berechnung des Prä-Follow-up-Effekts wurden nur n = 8 Pat. berücksichtigt, für die Daten zu beiden Messzeitpunkten vorlagen.

Eingeschlossen werden volljährige Patienten mit einer organisch nicht ausreichend erklärbaren Schwindelerkrankung, die mit einer deutlichen Beeinträchtigung der Lebensqualität (VHQ > 45 Punkte) einhergeht. Die Rekrutierung und neurologische, neurootologische bzw. neuroophthalmologische Abklärung erfolgt über die Ambulanz am Deutschen Schwindel- und Gleichgewichtszentrum, Großhadern. Das ausführliche Studienprotokoll wurde anderenorts im Detail veröffentlicht [28].

Ergebnisse der Pilotstudie

Inzwischen liegen die im Rahmen einer Pilotierungsstudie erhobenen Daten von 2 Behandlungsgruppen mit je 6 Teilnehmern vor, die die Therapie abgeschlossen haben. Lediglich eine Person brach die Gruppentherapie nach 13 Sitzungen ab (7,7 %). Unter den Teilnehmern waren 3 Männer und 9 Frauen mit einem Durchschnittsalter von 60 ± 11 Jahren. Eingeschlossen wurden Patienten, bei denen in der o. g. Schwindelambulanz ein primär oder sekundär somatoformer Schwindel diagnostiziert worden war.

Für 8 Teilnehmer liegen neben den Prä- und Post-Daten auch Fragebogendaten zum Ein-Jahres-Follow-up vor. Die 4 Evaluations-Dropouts waren im Schnitt etwas älter (66 vs. 58 Jahre) und zeigten deskriptiv eine geringere Symptombelastung hinsichtlich Schwindel und Depressivität zu Behandlungsbeginn (statistisch nicht signifikant).

Zur Bewertung der Prä-Post- bzw. Prä-Follow-up-Veränderungen wurden für die primären und sekundären Endpunktmaße standardisierte und für kleine Stichproben adjustierte Mittelwertdifferenzen als Effektstärken berechnet (Hedges g). Dabei wurden gemäß Cohen Effekte zwischen 0,2–0,5 als klein, zwischen 0,5–0,8 als mittel und Effektstärken > 0,8 als groß eingestuft. Das in der Pilotstudie verwendete Behandlungsmanual deckt sich nicht in allen Punkten mit dem oben beschriebenen Therapieansatz, da die im Prozess gesammelten Erfahrungen für die Ausgestaltung in der zweiten Therapiephase weiter genutzt wurden. Konkret wurden die störungsorientierten Module weiter ausgearbeitet und das Programm insgesamt durch die Ergänzung von Arbeitsblättern und Wochenaufgaben verdichtet. Ein psychodynamischer Behandlungsfokus, der das jeweilige Bindungsmuster sowie aktuelle Beziehungswünsche und -konflikte besonders berücksichtigt, ist eher in den Hintergrund getreten.

Über alle Ergebnismaße hinweg zeigten sich positive Entwicklungen im Sinne einer Abnahme der Symptomatik, welche sich mit Ausnahme der depressiven Beschwerden nach Therapieende noch fortsetzte. ▶ **Abb. 2** veranschaulicht dies anhand der z-standardisierten Fragebogenmaße zu allen 3 Messzeitpunkten. ▶ **Tab. 1** zeigt die deskriptive Statistik der Rohwerte sowie die Größe der Effektstärken. Im Prä-Post-Vergleich ergaben sich kleine Effekte bezüglich des schwindelassoziierten Handicaps und der depressiven Symptome. Im Prä-Follow-up-Vergleich zeigten sich als groß einzuordnende Effekte auf dem primären Ergebnismaß (VHQ) und der Schwindelsymptomatik (VSS) sowie kleine Effekte auf allen Maßen der psychopathologischen Begleitsymptomatik. Die Belastbarkeit dieser Ergebnisse ist durch die sehr kleine Stichprobengröße klar eingeschränkt; einer Signifikanzprüfung auf einem α-Fehler-Niveau von 5 % halten die Ergebnisse nicht stand. Auch wurde in der Pilotphase noch keine Kontrollintervention durchgeführt.

Diskussion und Ausblick

Vor dem Hintergrund einer hohen Prävalenz funktioneller Schwindelbeschwerden bei zugleich unzureichender Datenlage hinsichtlich psychotherapeutischer Behandlungsangebote wurde ein integratives Gruppenpsychotherapiekonzept entwickelt. Während bisherige Studien in dieser Patientengruppe v. a. kurzfristige Effekte psychotherapeutischer Interventionen belegen konnten, steht der Nachweis einer stabilen Symptombesserung, v. a. im Hinblick auf die nahezu regelhaft bestehende psychopathologische Begleitsymptomatik, noch aus [17].

Die Ergebnisse der Pilotierungsstudie geben erste Hinweise darauf, dass die Kurzzeit-Gruppenpsychotherapie auch über das Behandlungsende hinaus wirksam sein kann. Es bleibt für eine endgültige Bewertung abzuwarten, ob sich der beschriebene Behandlungsansatz bei funktionellen Schwindelbeschwerden in der aktuellen kontrolliert-randomisierten Effektivitätsstudie [28] bewähren wird.

FAZIT FÜR DIE PRAXIS

Mit dem vorgestellten integrativen Gruppenpsychotherapiekonzept wurde ein Manual zur ambulanten Behandlung funktioneller Schwindelbeschwerden entwickelt, das jene therapeutischen Vorgehensweisen bündelt, die sich im Umgang mit dieser Patientengruppe bewährt haben. Anhand störungsorientierter Module zu Angst und Panik, Depression sowie Somatisierung im engeren Sinne wird dabei auch die häufig bestehende psychopathologische Begleitsymptomatik in dieser Patientengruppe angemessen berücksichtigt. Das Gruppensetting kann – neben den ökonomischen Vorteilen – auch den Prozess erleichtern, das initial oft somatisch orientierte Krankheitsmodell um psychosoziale Faktoren zu erweitern und zu einem realistischen Gesundheitsbegriff zu finden. Aus der Pilotstudie ergeben sich Hinweise auf die Akzeptanz und Durchführbarkeit des Therapiekonzeptes sowie positive Effekte auf schwindelassoziierte Beschwerden und das dadurch im Alltag erlebte Handicap.

Interessenkonflikt

Die Autoren geben an, dass kein Interessenkonflikt besteht.

Literatur

[1] Eckhardt-Henn A, Breuer P, Thomalske C et al. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J. Anxiety Disord. 2003; 17: 369–388

[2] Lahmann C, Henningsen P, Brandt T et al. Psychiatric comorbidity and psychosocial impairment among patients with vertigo and dizziness. J. Neurol. Neurosurg. Psychiatry. 2015; 86: 302–308

[3] Weidt S, Bruehl AB, Straumann D et al. Health-related quality of life and emotional distress in patients with dizziness: a cross-sectional approach to disentangle their relationship. BMC. Health Serv. Res. 2014; 14: 317

[4] Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. Arch. Gen. Psychiatry. 2005; 62: 903–910

[5] Wiltink J, Tschan R, Michal M et al. Dizziness: anxiety, health care utilization and health behavior–results from a representative German community survey. J. Psychosom. Res. Elsevier Inc; 2009: 66: 417–424

[6] Ronel J, Lahmann C. Vertigo und Psyche. Nervenheilkunde 2008; 9: 846–852

[7] Eckhardt-Henn A, Tschan R, Best C et al. Somatoform vertigo syndrome. Nervenarzt 2009; 80: 909–917

[8] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th. 2013. Arlington, VA: American Psychiatric Publishing; 2013

[9] Best C, Eckhardt-Henn A, Diener G et al. Interaction of somatoform and vestibular disorders. J. Neurol. Neurosurg. Psychiatry. 2006; 77: 658–664

[10] Radziej K, Schmid G, Dinkel A et al. Psychological traumatization and adverse life events in patients with organic and functional vestibular symptoms. J. Psychosom. Res. 2015; 79: 123–129

[11] Piker EG, Jacobson GP, McCaslin DL et al. Psychological comorbidities and their relationship to self-reported handicap in samples of dizzy patients. J. Am. Acad. Audiol. 2008; 19: 337–347

[12] Yardley L, Redfern MS. Psychological factors influencing recovery from balance disorders. J. Anxiety Disord. 2001; 15: 107–119

[13] Yardley L. Prediction of handicap and emotional distress in patients with recurrent vertigo: symptoms, coping strategies, control beliefs and reciprocal causation. Soc. Sci. Med. 1994; 39: 573–581

[14] Godemann F, Koffroth C, Neu P et al. Why does vertigo become chronic after neuropathia vestibularis? Psychosom. Med 2004; 66: 783–787

[15] Kroenke K. Efficacy of treatment for somatoform disorders: a review of randomized controlled trials. Psychosom. Med. 2007; 69: 881–888

[16] Rief W, Bleichhardt G, Timmer B. Gruppentherapie für somatoforme Störungen - Behandlungsleitfaden, Akzeptanz und Prozessqualität. Verhaltenstherapie 2002; 12: 183–191

[17] Schmid G, Henningsen P, Dieterich MS et al. Psychotherapy in dizziness: a systematic review. J. Neurol. Neurosurg. Psychiatry. 2011; 82: 601–606

[18] Mahoney A, Edelman S, D Cremer P. Cognitive behavior therapy for chronic subjective dizziness: longer-term gains and predictors of disability. Am. J. Otolaryngol. Elsevier Inc. 2013; 34: 115–120

[19] Holmberg J, Karlberg M, Harlacher U et al. One-year follow-up of cognitive behavioral therapy for phobic postural vertigo. J. Neurol. 2007; 254: 1189–1192

[20] Tschan R, Eckhardt-Henn A, Scheurich V et al. Steadfast – effectiveness of a cognitive-behavioral self-management program for patients with somatoform vertigo and dizziness. Psychother Psych Med 2012; 62: 111–119

[21] Schaaf H, Hesse G. Patients with long-lasting dizziness: A follow-up after neurotological and psychotherapeutic inpatient treatment after a period of at least 1 year. Eur. Arch. Oto-Rhino-Laryngology. Springer Berlin Heidelberg 2015; 272: 1529–1535

[22] Lahmann C, Henningsen P, Noll-Hussong M et al. Somatoforme Störungen. PPmP - Psychother. • Psychosom. • Medizinische Psychol 2010; 60: 227–236

[23] MacKenzie KR. Time managed group psychotherapy: Effective clinical applications. Washington: American Psychiatric Press; 2005

[24] Meli A, Zimatore G, Badaracco C et al. Effects of vestibular rehabilitation therapy on emotional aspects in chronic vestibular patients. J. Psychosom. Res. 2007; 63: 185–190

[25] Schniepp R, Wuehr M, Huth S et al. Gait characteristics of patients with phobic postural vertigo: effects of fear of falling, attention, and visual input. J. Neurol. 2014; 261: 738–746

[26] Staab JP. Behavioral aspects of vestibular rehabilitation. NeuroRehabilitation 2011; 29: 179–183

[27] Rief W, Hiller W. Somatisierungsstörung (Fortschritte der Psychotherapie Band 1). Göttingen: Hogrefe; 2011

[28] Lahmann C, Henningsen P, Dieterich M et al. Tailored care for somatoform vertigo/dizziness: study protocol for a randomised controlled trial evaluating integrative group psychotherapy. J. Neurol. 2015; 262: 1867–1875

[29] Tschuschke V. Kurzgruppenpsychotherapie - Wirksamkeit und Wirkfaktoren TT - Short-term group therapy - Efficacy and effective factors. Zeitschrift für Individ 2004; 29: 6–19