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The impact of cognitions on the development of panic and somatoform disorders: a prospective study in patients with vestibular neuritis

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ABSTRACT

Background. There is a high incidence of panic and somatoform disorders after vestibular neuritis. However, the occurrence of psychiatric disorders has been shown to be unrelated to persistent functional vestibular impairment. The aim of the present study was to examine the role played by cognitions in the development of panic and somatoform disorders.

Method. In a 2-year prospective study, cognitions were recorded at various stages using the Agoraphobic Cognitions Questionnaire and Body Sensations Questionnaire. Our analysis focused on whether body-related anxiety or panic-related thoughts can predict the development of a panic or somatoform disorder.

Results. Fear arising on the first day of an acute vestibular episode did not predict the development of panic or somatoform disorders. One week after the dysfunction, however, the fear of vertigo was a significant predictor, explaining 20% of the variance in the development of either disorder. After 6 weeks, persistent fear of vertigo or vomiting predicted approximately 30% of the variance, and after 6 months panic-related thoughts predicted 40% of the variance and, with the inclusion of body-related fears, as much as 60% of the variance in the development of panic or somatoform disorders.

Conclusion. Our data confirm prospectively a number of fundamental assumptions of cognitive theory concerning the development of anxiety disorders. Subjects who experience vertigo as particularly alarming focus more intensely than other patients on the negative symptoms they perceive as being related to the disorder. Patients with panic-related cognitions were prone to develop panic or somatoform disorders.

INTRODUCTION

Panic disorder was first included as a separate diagnostic category in the third edition of the *Diagnostic and Statistical Manual* (DSM-III), published by the American Psychiatric Association in 1980. As part of these changes, the authors of the DSM also defined the symptoms of a panic attack, as well as its typical course (i.e. a rapid rise in anxiety, short duration). The fear of repeated panic attacks was introduced as an additional cognitive characteristic. This anxiety is also referred to as phobic anxiety. A number of cognitive symptoms are also associated with panic attacks. In addition to physical symptoms, such as accelerated heart rate or shortness of breath, patients have reported the fear of losing control, going crazy and/or choking to death. Indeed, these symptoms have come to be recognized as the most important defining

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characteristics of a panic attack (Clark, 1986; Aronson & Logue, 1988).

Many studies have shown that patients with panic disorders report catastrophic anxieties more often, react to changes in their own bodies more sensitively, and misinterpret these changes more frequently than the norm population (Casey *et al.* 2004; Hoehn-Saric *et al.* 2004). A variety of questionnaires have been used to record the various forms of anxiety, including, for example, the Agoraphobic Cognitions Questionnaire (ACQ; Chambless *et al.* 1984). In an earlier study, we found indications that the degree to which specific thoughts are developed in somatic diseases depends to no small extent on individual biographical experiences (Godemann *et al.* 2001). Many investigators assume that these fearful thoughts are not just a manifestation of anxiety, but that they also contribute to its development in a causal manner (Keyl & Eaton, 1990; Ehlers, 1993; Cox *et al.* 1994, 1995; Cox, 1995, 1996; Ehlers & Breuer, 1996; Middleton, 1998; Moor & Zebb, 1999; Chambless *et al.* 2000).

The assumption that fearful thoughts play a causative role in the development of anxiety is explained in a variety of ways in the literature. A number of authors point, for example, to cognitive therapy, which attempts to change catastrophic thoughts and phobic anxiety and has frequently shown that it can lead to the successful treatment of panic disorder (Salkovskis *et al.* 1991; Gelder *et al.* 1993; Clark *et al.* 1994, 1999; Brown *et al.* 1997). The conclusion might, therefore, be drawn that this successful form of therapy affects aetiopathogenetically relevant points – in this case cognitions that may play a role in the development of panic disorder. This is countered by the argument that cognitions are only an expression of anxiety and do not contribute to it in any separate or independent manner.

The only way to determine which argument is correct is to perform prospective studies in psychiatrically healthy people. In a 3-year follow-up with college students, Maller & Reis (1992) found that the Anxiety Sensitivity Index, which measures the fear of arousal symptoms, predicted the intensity and frequency of panic attacks during the observation period. Schmidt *et al.* (1997) also reported that anxiety sensitivity is a risk factor for panic attacks. Although these findings strongly indicate that cognitions play an important role in the development of panic attacks, neither Maller & Reis nor Schmidt *et al.* were able to clarify whether the same applies to the development of panic disorders. Furthermore, it is conceivable that some members of their study groups suffered from a panic disorder at the beginning of the observation period, which would have led to an overestimation of the prospective meaning of the Anxiety Sensitivity Index.

Panic disorders are related in some respects to post-traumatic stress disorders (PTSD). The latter are also characterized by frequent panic attacks, and there are a variety of strategies available to avoid future adverse events. Because PTSD results from a clearly defined traumatic event, it is possible to carry out prospective longitudinal studies in these patients. Indeed, several of these studies have shown that cognitive variables can predict the development of PTSD (Dunmore *et al.* 2001). Steil & Ehlers (2000) found that the cognitions measured by the ACQ did not predict PTSD; they thus concluded that the idiosyncratic meaning of cognitive variables determines whether post-traumatic symptomatology is experienced as distressing.

Another approach to panic disorders is to chemically induce panic attacks in test subjects (Gorman *et al.* 1990; Goetz *et al.* 1993; Griez & Schruers, 1997; Strohle, 2003). It has been shown that patients with panic disorders observe their own bodies more closely and exhibit more anxiety in their interpretation of any changes they perceive compared to healthy subjects, who interpret neurovegetative changes in a more objective manner (Griez *et al.* 1987; Bradwejn *et al.* 1998). In another study, healthy test subjects who experienced their first panic attack under cholecystokinin showed dysfunctional fearful cognitions in the run-up to the test (Aluoja *et al.* 1997). However, it is unclear whether these induced panic attacks are truly related to naturally occurring ones. During chemically induced panic attacks, the test subjects are always aware of the relationship between the administration of the chemical substance and any physical changes, which allows them to react cognitively to the experience. Indeed, the suspicion that induced panic attacks may represent an entirely different phenomenon

is supported by the fact that there have been no reported cases of panic disorder subsequent to an induced attack to date.

Consequently there is evidence that primarily catastrophic or dysfunctional cognitions may play a major – perhaps even decisive – role in the aetiology of panic disorder. However, none of these previous studies analysed the specific role played by cognitions in a prospective manner. This is presumably due to the fact that panic disorder – although not a rare psychiatric illness by any means – nevertheless does not have the level of incidence required to answer the complex questions posed by prospective studies of population cohorts (Hayward *et al.* 2003; Jacobi *et al.* 2004). Indeed, the only prospective study on panic disorders to date has been that by Keyl & Eaton (1990), in which female gender and youth were reported to be risk factors.

In the present study, we attempt to address this deficit in the literature. The basic idea was to identify a somatic risk condition for the development of panic disorders. In vestibular neuritis, the literature assumes an increased incidence of panic attacks. In their retrospective investigation, Eagger *et al.* (1992) presented data showing that 28% of patients developed a panic disorder after experiencing dysfunction of this organ of equilibrium. This seems plausible, as the experience during acute vestibular dysfunction closely resembles that of a panic attack. Vestibular failure occurs suddenly (i.e. within minutes or hours) and is accompanied by extremely aggressive rotary vertigo and vomiting, which means that patients can only move about with the assistance of others. During the acute phase, which usually subsides within 14 days, patients lose control over themselves to a certain extent (Dix & Hallpike, 1952; Bont & Früh, 1995; Böhmer *et al.* 1996; Scherer, 1996). Not surprisingly, this experience has been associated with extreme fear (Pollak *et al.* 2003; Godemann *et al.* 2004*b*).

Although the aetiology of vestibular neuritis is unclear, infections and blood circulation disorders are seen as potential culprits (Schuknecht & Kitamura, 1981; Hopf, 1987; Büchele & Brandt, 1988; Karlberg *et al.* 2004). Nevertheless, affected patients are currently still left without any clear explanation of why they suffer from acute rotary vertigo. At the very least, the diagnosis can be confirmed with a warm and cold douche of both ears (i.e. caloric testing).

The data on the course of the disease are contradictory as well. Although it has been postulated that a majority of patients recover vestibular function or that central compensation of the failure occurs within a few weeks of the episode in question, there are indications that more than 30% of patients continue to suffer from persistent vertigo (Imate & Sekitani, 1993; Okinaka *et al.* 1993; Bergenius & Perols, 1999; Halmagyi & Cremer, 2000). This suggests that a large number of patients will experience sustained functional impairment. However, in a previous study we were not able to observe any connection between vestibular function, as measured by posturography, and vertigo. Nearly all patients recovered functionally after the acute loss of equilibrium, and the prevalence of subclinical vestibular disorders was no greater among patients with persistent vertigo (Godemann *et al.* 2004*a*). Thus, it appears likely that we are dealing with a persistent vertigo that can be explained in terms of a psychiatric disorder (i.e. a panic or somatoform disorder). In fact, the standardized psychiatric diagnostic showed that in 12·9% of our subjects a panic or somatoform disorder had reappeared within 2 years. At the same time, the description of the clinical symptoms of both of these diagnoses were very similar (persistent vertigo accompanied by avoidance behaviour distinctly impairing social interaction, repeated visits to the doctor, the constant fear of worsening vertigo). In contrast to patients with panic disorder, however, patients with somatoform disorder did not report any panic attacks and denied experiencing anxiety.

In conclusion, patients who experience an episode of vestibular neuritis are suitable subjects for performing prospective, longitudinal research into current cognitive theories on the development of panic disorders. In the present study, we hypothesized that patients who would later go on to developed a panic disorder would react at an early stage to an acute physical experience in a cognitively dysfunctional way. We also expected that they would observe their own bodies more anxiously and exhibit greater fear of losing control over themselves. In addition to the patients' reaction to the acute incident, we were also interested in whether

their cognitions changed over time. The theoretical underpinning of these assumptions is that specific cognitions tend to become generalized in the course of certain psychiatric illnesses, developing into non-specific body-related anxiety.

METHOD

Design and sample

Over a period of 3 years, patients with vestibular neuritis from eight clinics in Berlin were included in our prospective study†. Clinically, they were required to have the symptom triad of rotary vertigo, spontaneous nystagmus, and nausea/vomiting. On the day after being admitted to hospital, all patients were contacted by a psychologist who was informed of each case by the participating clinics. At the same time, patients were asked to provide written informed consent to take part in the study. The diagnosis of vestibular neuritis was confirmed in all patients using caloric testing, during which the lack of thermal sensitivity in the ipsilateral semicircular canal was established. Patients were excluded from the study if they had already experienced central vestibular vertigo, reduced hearing, tinnitus, or an equilibrium disorder at any point in the past. A total of eight patients refused to take part in the study. In several additional cases, the psychologist received the information of a patient's admission to hospital too late to interview the patient within the specified time period. Because these patients were not interviewed, we are unable to provide any information on them.

Over the following 2 years, patients were interviewed and examined a total of five times. The first interview took place within 24 hours of admission; at this point, all patients were lying in bed and were severely impaired by nausea and vertigo. The second examination was carried out in hospital between 7 and 12 days after admission. Six weeks after the onset of the acute vestibular disorder, patients were interviewed again as part of a hospital outpatient appointment. Then, 6 months after admission, a questionnaire was sent to each patient's residence by standard mail. The final examination was conducted 2 years after admission and in the context of a hospital outpatient appointment. All patients were informed that the scientific study was concerned with the development of vertigo and patients' mental condition following an acute vestibular episode. The study protocol was approved by the local ethics committee.

A total of 113 patients met the inclusion criteria of the study. Ten patients were excluded from further evaluation because they did not attend all of the appointments set out in the study protocol, and two further patients were excluded because they were over 80 years of age. An additional eight patients were excluded from the study because a pre-existing panic ($n=4$) or somatoform disorder ($n=4$) was diagnosed at the second appointment. As a result, a total of 93 patients were included in the final evaluation. This random sample consisted of 50 women (53·8%) and 43 men (46·2%) with an average age of 50·3 years ($\pm 12{\cdot}6$). Of these 93 patients, seven developed a panic disorder (with or without agoraphobia) and five a somatoform disorder over the 2 years following the acute vestibular episode. This represents an incidence of 12·9% (panic disorder 7·5%, somatoform disorder 5·4%).

The psychiatric diagnosis was made using data from the Diagnostic Interview of Psychiatric Disorders (DIPS), which was conducted both 7–12 days after hospital admission and during the final examination after 2 years. The DIPS (Margraf *et al.* 1994) is a structured interview and represents a modified version of the American 'Anxiety Disorders Interview Schedule – Revised Version'. It is based on DSM-III-R (DiNardo *et al.* 1993). The psychiatric interviews during the final examination were performed by the director of the study, who is a specialist in psychiatry and psychotherapy. At the time of the interviews, he had no knowledge of the information patients had provided on the individual scales used to measure cognitions in the study.

Materials and procedure

Cognition data were recorded at a number of different stages. The Acute Vertigo Appraisal (AVA; Godemann *et al.* 2004*a*) was used once

† The clinics were: Departments of Otolaryngology at the Free University of Berlin, St. Gertrauden Hospital, St. Hedwig Hospital, 'Im Friedrichshain' Hospital, 'Prenzlauer Berg' Hospital, 'Neukölln' Hospital, Berlin Trauma Hospital, and the Department of Neurology at the Charité University Medical Centre.

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Table 1. *Overview of patient assessment instruments over a period of 2 years after acute vestibular episode*

Period after admission	On day of admission	7–12 days	6 weeks	6 months	2 years
Measurement instrument	AVA	DIPS ACQ BSQ STAI (state and trait version)	ACQ BSQ STAI (only state version)	ACQ	DIPS

AVA, Acute Vertigo Appraisal; DIPS, Diagnostic Interview of Psychiatric Disorders; ACQ, Agoraphobic Cognitions Questionnaire; BSQ, Body Sensations Questionnaire; STAI, State-Trait Anxiety Inventory.

at the beginning of the study, the Body Sensations Questionnaire (BSQ) was applied twice (7–12 days after admission and 6 weeks after admission), and the ACQ on three occasions (7–12 days, 6 weeks, and 6 months after admission). An overview of the study design is provided in Table 1.

The AVA is a 5-point Likert scale consisting of five items. It is an instrument for recording a patient's immediate reaction to acute vestibular dysfunction. No other instruments were used on the day after admission, as these would have been too complicated considering the patients' severely impaired state; most patients were only able to open their eyes for a short time. The AVA was not able to predict the acute course of anxiety in the first 6 weeks, as almost all of the patients initially feared that something terrible was happening to them (Godemann *et al*. 2004*b*).

The questionnaire on panic-related Anxieties, Cognitions and Avoidance (ACA) is the German version of a questionnaire series by Chambless *et al*. (1984) that includes several aspects of anxiety disorder; in our study we used the fear of physical symptoms (BSQ) and panic-related cognitions (ACQ) scales (German version by Ehlers & Margraf, 1993). The BSQ records 17 items on the intensity of fear with respect to body sensations (e.g. heartbeats, feeling of not being able to breath, vertigo). The patient indicates the extent to which he or she is worried about a particular symptom using a 5-point Likert scale that ranges from 'Not at all worried or anxious' to 'Extremely anxious'.

The ACQ records 14 items concerning typically panic-related thoughts, such as 'I'm going to have a heart attack' or 'I'm going to go crazy'. The patient assesses these on a 5-point Likert scale ranging from 'The thought never occurs to me' to 'The thought always occurs to me when I am nervous'. The ACQ also has two subscales: 'Physical crises' and 'Loss of control'. Overall, the German versions of the BSQ and ACQ scales show good internal consistency and correspond in this regard to the original American questionnaire. The values for Cronbach's α were between 0·80 and 0·95 for the BSQ, and between 0·74 and 0·87 for the ACQ. The retest reliability was likewise high for both questionnaires: 0·66 for the BSQ and 0·80 for the ACQ. Both ACQ subscales also had high retest reliability, 0·84 for physical crises, and 0·80 for loss of control.

The State-Trait Anxiety Inventory (STAI) developed by Spielberger *et al*. in 1970 assesses general and current anxiety. On the state anxiety scale, the patient describes how he or she currently feels, and on the trait anxiety scale, how he or she feels in general.

Analytical strategy

All calculations were performed using SPSS version 11.5.1 (SPSS Inc., Chicago, IL, USA) Data are presented as mean values and standard deviation from the mean. The comparisons of mean values were carried out with the aid of a *t* test for two independent random samples. The multivariate analyses were calculated with binary logistical regressions. This procedure investigates the dependence of a dichotomous variable on other independent variables. The advantage of this method is that the influence of several explanatory variables on a dependent variable can be examined simultaneously. All variables relative to the main hypotheses and potentially confounding variables are included in the calculation. The result of the regression

Table 2. *Descriptive representation of cognitions shortly after admission, and a comparison of mean values for patients with panic and somatoform disorders and patients without these disorders*

	Without disorder ($n=70$)	Panic and somatoform disorder ($n=12$)	Significance of t test (2-sided testing)
AVA			
The vertigo came as a surprise	4·6 ± 0·95	4·83 ± 0·58	N.S.
The vertigo made me anxious	3·89 ± 1·22	4·08 ± 1·0	N.S.
I felt helpless	4·16 ± 1·18	4·83 ± 0·39	N.S.
The vertigo was embarrassing	2·11 ± 1·56	2·42 ± 1·62	N.S.
ACQ			
I'm going to go crazy	1·54 ± 1·06	1·75 ± 0·87	N.S.

AVA, Acute Vertigo Appraisal; ACQ, Agoraphobic Cognitions Questionnaire.
N.S., not significant; ACQ, all variables were tested and were not significant.

then shows which variables contribute to the variance solution.

RESULTS

The AVA was performed on the day of admission to hospital. The comparison of cognitions at this time showed no significant differences between patients who went on to develop a panic or somatoform disorder during the 2-year observation period and patients who did not go on to develop this disorder. All patients reported high scores with respect to the anxiety caused by vertigo, as well as to the feeling of helplessness and surprise (Table 2).

Ten days after the acute vestibular episode, the fear of the specific symptom of vertigo was more intense in patients who went on to develop panic or somatoform disorder than it was in patients who did not develop psychiatric disorder during the observation period. Other general body-related fears, as measured by the ACQ, did not differ between patients who went on to develop panic or somatoform disorder and those who did not. After 6 weeks, it could clearly be seen that patients' anxieties were no longer associated as strongly with the specific symptom of vertigo, but rather increasingly focused on body-related fears linked to vertigo, such as the fear of having to vomit or going crazy (Table 3).

The multivariate analysis of body-related fears contained the following items from the ACQ: 'I'm going to throw up', 'I must have a brain tumour', 'I'm going to have a stroke', 'I'm going to go crazy'. The other items on the ACQ did not show any significance. We retrospectively chose a selection of significant items for the multivariate analysis. Because of the interference with the above-mentioned fears, the ACQ mean value was not included in the multivariate analysis. The inclusion of other items or the ACQ mean value did not improve the predictive values. The results clearly show that the model's strength of evidence becomes even greater as the time between the acute vestibular episode and the interview increases. The above-mentioned items clarified the variance to only 3·9% 10 days after experiencing vertigo (Nagelkerke's R^2, χ^2 value of difference 1·49, model improvement N.S.), but this figure already increases to 31·6% after 6 weeks (Nagelkerke's R^2, χ^2 value of difference 8·80; model improvement N.S.) and rises to 43·9% after 6 months (Nagelkerke's R^2, χ^2 value of difference 11·86, significance of model improvement 0·0018**). It should be noted that patients who went on to develop a panic or somatoform disorder were diagnosed 2 years after the acute vestibular episode and not prior to this. As a result, we are unable to say exactly when during the observation period these patients first met the criteria for either disorder.

If, in addition to the above-mentioned items from the ACQ, the fear of vertigo and the total mean value of the BSQ, as well as the mean value of the STAI (state version) are included in the multivariate analysis, the variance solution increases to ~24% 10 days after the vertigo episode (Nagelkerke's R^2 24·3%, χ^2 value of difference 9·86; model improvement N.S.) and to ~58% 6 weeks after the vertigo episode (Nagelkerke's R^2 57·7%, χ^2 value of difference 17·51, significance of model improvement

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Table 3. *Comparison between the fears of patients with panic and somatoform disorders and patients without these disorders 10 days, 6 weeks, and 6 months after vertigo episode*

ACQ	I'm going to throw up	I must have a brain tumour	I'm going to have a stroke	I'm going to go crazy	Total mean value
Values after 10 days in patients without disorder after 2 years	1·40 ± 0·73	1·37 ± 0·83	1·63 ± 0·96	1·37 ± 0·81	1·38 ± 0·48
Values after 10 days in patients with disorder after 2 years	1·56 ± 1·01	1·33 ± 0·5	1·78 ± 0·97	1·22 ± 0·44	1·34 ± 0·27
t test[a] after 10 days	N.S.	N.S.	N.S.	N.S.	N.S.
Values after 6 weeks in patients without disorder after 2 years	1·28 ± 0·45	1·26 ± 0·70	1·51 ± 0·88	1·19 ± 0·55	1·27 ± 0·40
Values after 6 weeks in patients with disorder after 2 years	2·00 ± 0·89	1·67 ± 0·82	1·83 ± 0·75	2·00 ± 1·1	1·79 ± 0·47
t test[a] after 6 weeks	N.S.	N.S.	N.S.	0·005**	0·006*
Values after 6 months in patients without disorder after 2 years	1·25 ± 0·44	1·31 ± 0·62	1·61 ± 0·96	1·14 ± 0·54	1·25 ± 0·41
Values after 6 months in patients with disorder after 2 years	2·17 ± 0·98	1·33 ± 0·52	1·50 ± 0·55	1·33 ± 0·52	1·31 ± 0·17
t test[a] after 6 months	0·000***	N.S.	N.S.	N.S.	N.S.

BSQ	Fear of vertigo	Total mean value
Values after 10 days in patients without disorder after 2 years	2·95 ± 1·48	1·99 ± 0·78
Values after 10 days in patients with disorder after 2 years	4·33 ± 0·5	2·53 ± 0·52
t test[a] after 10 days	0·000***	0·049*
Values after 6 weeks in patients without disorder after 2 years	2·58 ± 1·37	1·83 ± 0·79
Values after 6 weeks in patients with disorder after 2 years	3·64 ± 1·43	2·07 ± 0·82
t test[a] after 6 weeks	0·020*	N.S.
Values after 6 months in patients without disorder after 2 years	NI	NI
Values after 6 month in patients with disorder after 2 years	NI	NI
t test[a] after 6 months	NI	NI

STAI	Trait	State Total mean value
Values after 10 days in patients without disorder after 2 years	37·06 ± 9·77	37·6 ± 10·41
Values after 10 days in patients with disorder after 2 years	40·81 ± 9·99	43·22 ± 9·96
t test[a] after 10 days	N.S.	N.S.
Values after 6 weeks in patients without disorder after 2 years	NI	35·32 ± 9·64
Values after 6 weeks in patients with disorder after 2 years	NI	42·73 ± 11·23
t test[a] after 6 weeks	NI	0·024*
Values after 6 month in patients without disorder after 2 years	NI	NI
Values after 6 month in patients with disorder after 2 years	NI	NI
t test[a] after 6 months	NI	NI

ACQ, Agoraphobic Cognitions Questionnaire (variables not reported were tested without any significance); BSQ, Body Sensations Questionnaire (variables not reported were tested without any significance); STAI, State-Trait Anxiety Inventory; With disorder, patients with panic and somatoform disorders after 2 years; Without disorder, patients without these disorders after 2 years; NI, not investigated.

[a] *t* test = *t* test significance for two independent random samples (two-sided).

* $p < 0{\cdot}05$; ** $p < 0{\cdot}01$; *** $p < 0{\cdot}001$; N.S., not significant.

0·014*). The patients were not asked to fill out the BSQ at 6 months.

DISCUSSION

This study indicates that cognitions play a key role in the development of both panic and somatoform disorders. There are at least three prospective studies that show the effect of anxious cognitions on the development of panic attacks (Maller & Reis, 1992; Schmidt *et al.* 1997). However, to our knowledge, our investigation is the first to evaluate in a prospective manner the role played by cognitions in panic and somatoform disorders.

The data from our 2-year observation period suggest that cognitions are not just a symptom of these disorders, but that they also contribute to their development. Similarly to the PTSD model by Ehlers & Clark (2000), cognitions such as the anxious assessment of one's own body or the fear of experiencing adverse body

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sensations are predictors for the development of the illness. Six weeks after the acute vestibular episode, specific anxieties and the fear of vertigo in our patients were able to explain up to 60% of the variance in the development of a panic or somatoform disorder over the next 2 years.

In addition, our study also examined cognitions as they developed over time. Whereas almost all patients felt helpless and anxious directly after the failure of vestibular function, it was only the patients who exhibited intense preoccupation with the vertigo episode who later developed a panic or somatoform disorder. In contrast, the severity of vertigo was not correlated with the later development of a panic or somatoform disorder.

Vertigo appears to be considerably more frightening for the group at risk for developing panic disorder – even when the fear of vertigo is not linked to broader (or more specific) anxieties. In the weeks following their admission to hospital, the patients who went on to develop a panic or somatoform disorder continued to be preoccupied with what they had experienced during the acute vestibular episode, continually recalling what they had experienced even after the clinical symptoms had receded. During this process, the fear of once again experiencing extreme nausea, which is typical of acute vestibular disorder, became the most important predictor for the development of a panic or somatoform disorder. In addition, the patients in question suffered, at least for a time, from the fear of insanity.

The experience of acute vestibular failure produces strong emotions and, in the acute phase, is difficult to distinguish from the symptoms of an acute stress reaction. Acute emotional distress often leads to PTSD (Fullerton *et al*. 2004). Schnyder *et al*. (2003) reported that cognitive processes contribute fundamentally to the further course of PTSD, a finding corroborated by a large number of publications on this subject (Schnyder *et al*. 2003). In the early phase of acute stress disorder, debriefing has been investigated as a means to prevent PTSD. However, the majority of studies report either negative effects or no effects at all, although it is important to note that these findings are very inconsistent (Rose *et al*. 2003).

Interestingly, our data may provide a clue as to why debriefing has led to such poor results in the treatment of PTSD. In the early phase of PTSD, many patients react with strong emotions, but this alone is not sufficient for predicting the subsequent development of symptoms. In our study, the development of a psychiatric disorder was linked to a patient's increased preoccupation with his or her own symptoms – a foundation upon which, within a matter of weeks, anxieties may then develop and persist. As a result, forgetting the traumatic event soon after it has occurred appears to have a protective effect; debriefing during this period may carry with it the danger of strengthening negative cognitive processes. Perhaps providing patients with cognitive behavioural skills can prevent the development of panic disorder by reducing anxiety sensitivity (Telch *et al*. 1993). At a later point in time, cognitive therapy may be successful because it is able to discover and change maladaptive cognitions (Clark *et al*. 1994, 1999; Ehlers & Clark, 2003). The importance of maladaptive cognitions is corroborated by the findings of Clark *et al*. (1994, 1999), which show that a successful modification of such cognitions by the end of treatment predicts a positive outcome in panic disorders at follow-up.

We have shown that dysfunctional cognitions are closely related to the development of panic and somatoform disorders. However, these variables do not allow us to characterize specifically which traits make people vulnerable to the development of these disorders. In our study, the trait of anxiety did not help explain the increased vulnerability with respect to dysfunctional cognitions. Further relevant traits may include various characteristics of personality such as a higher co-morbidity with dependent and insecure personality disorders (Marshall, 1996). Psychoanalytical theories assume that panic disorder develop as a result of a breakdown in the defence organization (De Masi, 2004) – a hypothesis that can also be investigated prospectively.

This study has some limitations. Despite the high incidence of panic and somatoform disorders, the number of subjects included in our investigation was still limited. We suggest that panic and somatoform disorders following an acute vestibular episode can be compared to panic and somatoform disorders following other traumatic events such as traffic accidents

or robbery. It should be noted that even though our patients reported having experienced all of the typical symptoms of a panic disorder (e.g. dizziness, fear of fainting, intense fear, accelerated heart rate), our findings should not be over-generalized. In particular, it is impossible to rule out that a panic disorder following vestibular neuritis is a special form of panic disorder and, therefore, the generalization of results is limited.

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DECLARATION OF INTEREST

None.

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