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Dizziness and fear of falling:

A behavioural and physiological approach to

”Phobic Postural Vertigo”

Johan Holmberg

LUNDS
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The Faculty of Medicine, Lund University

Lund 2006

“To dare is to lose one's footing momentarily. Not to dare is to lose oneself.”

Soren Kierkegaard

The horizontal lines on the front page are parallel. A discrepancy between actual visual sensory input and our expectation of physical reality creates an optical illusion.

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TABLE OF CONTENTS

1 LIST OF PUBLICATIONS **5**

2 ABBREVIATIONS **6**

3 INTRODUCTION **7**

3.1 Background **7**

3.2 Balance physiology and dizziness **8**

3.2.1 Visual information 8

3.2.2 Somatosensory information 9

3.2.3 Vestibular information 9

3.2.4 Sensory mismatch 9

3.2.5 Interactions of emotions and postural behaviours 10

3.2.6 Associations between balance control, anxiety, and orientation 11

3.3 Diagnostic concepts **12**

3.3.1 Psychogenic Dizziness 12

3.3.2 Cervical Vertigo 13

3.3.3 Panic Disorder and Agoraphobia 14

3.3.4 Space and Motion Phobia 16

3.3.5 Phobic Postural Vertigo (PPV) 17

3.3.6 Visual Vertigo 19

3.3.7 Chronic Subjective Dizziness 20

3.4 Treatments **21**

3.4.1 Pharmacological treatment 21

3.4.2 Vestibular rehabilitation 21

3.4.3 Cognitive behavioural therapy 22

4 AIMS **24**

5 STUDIES **25**

5.1 Populations and procedure **25**

5.2 Methods **28**

5.2.1 Questionnaires (paper II, III, V) 28

5.2.2 Posturography (paper I) 30

5.2.3 Cognitive behavioural therapy 31
5.3 Results **34**
5.3.2 Paper I 34
5.3.2 Paper II 34
5.3.3 Paper III 35
5.3.3 Paper IV 35
5.3.5 Paper V 36
6 GENERAL DISCUSSION **37**
6.1 Methodological considerations **37**
6.2 PPV versus other concepts **38**
6.3 Benefits and disadvantages with the concept of PPV **42**
6.4 Possibilities and limitations with CBT for PPV **43**
6.5 Hypothetical mechanism of PPV **45**
6.6 Conclusions and clinical implications **46**
7 POPULÄRVETENSKAPLIG SVENSK SAMMANFATTNING **49**
8 ACKNOWLEDGEMENT **52**
9 REFERENCES **53**
10 APPENDIX: PATIENT INSTRUCTIONS **63**
11 APPENDIX: PAPERS **65**

1 LIST OF PUBLICATIONS

I. Holmberg J, Karlberg M, Fransson PA, Magnusson M. Phobic postural vertigo: body sway during vibratory proprioceptive stimulation. Neuroreport. 2003;14:1007-1011.

II. Holmberg J, Karlberg M, Harlacher U, Magnusson M. Experience of handicap and anxiety in phobic postural vertigo. Acta Otolaryngol. (Stockh). 2005;125:270-275.

III. 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. 2005 Dec 19; [Epub ahead of print].

IV. Holmberg J, Harlacher U, Karlberg M, Magnusson M. Phobic postural vertigo-clinical experiences gathered during cognitive behavioural therapy. Manuscript.

V. Holmberg J, Karlberg M, Harlacher U, Magnusson M. No long term effect of cognitive behavioural therapy for phobic postural vertigo. Manuscript.

2 ABBREVIATIONS

CBT	Cognitive Behavioural Therapy
DHI	Dizziness Handicap Inventory
DSM	Diagnostic Statistical Manual
HAD	Hospital Anxiety and Depression scale.
PPV	Phobic Postural Vertigo
SSRI	Selective Seretonin Reuptake Inhibitor
VHQ	Vertigo Handicap Questionnaire
VSS	Vertigo Symptom Scale

3 INTRODUCTION

3.1 Background

The experience of 'self', consciousness, or this very moment called now, is to a high extent defined by our sensory organs and the interaction between the body and its surrounding. The interaction between body and gravitational forces is called balance and this act is a prerequisite of any goal directed behaviour. Balance is based on the integration in the central nervous system of sensory information and continuously ongoing motor responses towards the surrounding, which gives new sensory feedback and so forth. In people that perceive imbalance, dizziness or vertigo, this feedback process is somehow disturbed. Sometimes it is possible to identify a specific vestibular pathology, such as vestibular neuritis, or benign paroxysmal positioning vertigo (BPPV) as a cause for this disturbance and thereby the patients perception of dizziness. Humans spend much effort and have an enormous capacity to control suffering. To understand reasons for suffering, we categorize and label pathological processes. Vestibular neuritis and BPPV are highlights of this ability.

As for other somatic problems such as pain, there is only a weak relationship between the degree of measurable pathology and the degree of suffering or handicap. For vestibular neuritis and dizziness overall, handicap is best predicted by emotional and cognitive factors independent of pathology in the vestibular organs [1, 2]. The perception of dizziness and compensation for vestibular disorder involve sensory factors as well as cognitive and emotional factors. For remaining symptoms of dizziness, despite lack of pathological processes, it is difficult to create a definite nosology. Different concepts have been developed: psychogenic dizziness c.f. [3], cervical dizziness, phobic postural vertigo (PPV) [3], space and motion phobia [4], visual vertigo [5], and chronic subjective dizziness [6]. Those concepts reflect a still ongoing scientific process to which these thesis is supposed to contribute.

Dizziness affects a large part of the population. Studies have shown that about 20% of an unselected Scottish population have restricted daily life activities due to dizziness [7].

However, only 23% of those people visit their doctor because of their suffering, and dizziness seems to be an underestimated reason for handicap [8]. Treatments for this population have been sparsely investigated. Therefore there is a need for the development of structured treatment programs for patients with dizziness.

3.2 Balance physiology and dizziness

The Balance system is constantly active and is a substantial component for most behaviour. The ability to maintain balance is achieved by integration in the central nervous system of sensory input from the eyes, the vestibular system in the inner ear and from sensory receptors in the skin and muscles.

3.2.1 Visual information

Stabilization is related to movements of objects on the retina and to the visual information in relation to movements of the head and the vestibular system [9]. This is especially true for low frequency body sway (0.01-0.1 Hz) [10]. Vision also provides the brain with information about up-coming postural threats, which enables the organism to adjust their balance control strategy in anticipation of postural threat.

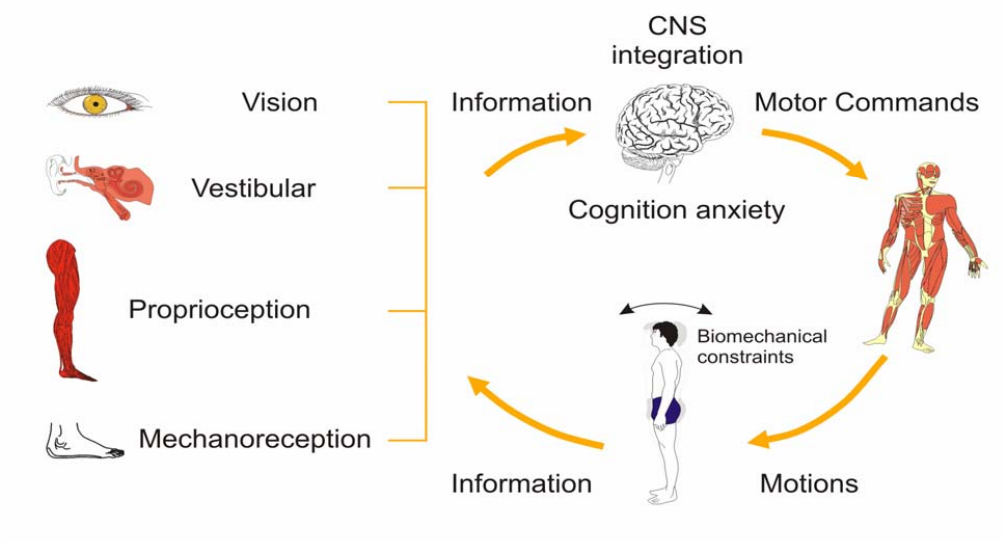

Figure 1: Overview of the balance system

3.2.2 Somatosensory information

Pressure receptors in the skin (mechanoreceptors) provide information about the location of the body in relation to gravitational forces. Sensory information from pressure receptors in the feet is the most important cue for balance control while standing [11]. Receptors in muscle spindles, in tendons and in joints provide information about the relative position of the body parts with respect to each other (proprioception) [9]. The proprioceptors of the neck also seem to be specifically important for location of the position of the head in relation to trunk [12].

3.2.3 Vestibular information

The vestibular receptors found in the inner ear, consist of five different organs: three semicircular canals and two otolith organs. Semicircular canals detect angular motion and otolith organs report linear movements and accelerations. From the vestibular organ, sensory information enters the brain stem through the 8th cranial nerve (the vestibulo-cochlear nerve). The vestibular nucleus consists of four regions that serve different functions in communicating vestibular information to other parts of the central nervous system. The vestibulo-ocular reflex is responsible for stabilizing an observed object on the retina while moving the head, which is critical in order to properly fixate gaze [9]. By action of the vestibulo-collic reflex the head is stabilized in relation to the body, and the vestibulo-spinal reflexes act on the trunk and the limbs [9].

3.2.4 Sensory mismatch

Symptoms of vertigo and dizziness are thought to be caused by conflicting sensory inputs from the different sensory organs. This is often referred to as sensory mismatch. This occurs for example when there is a sudden lesion of the vestibular organ on one side. The input from the dysfunctional side is not congruent with input from the unaffected side resulting in vertigo. If an individual receives conflicting information from different sensory modalities, as for example being under deck on a boat, visual information conflicts with vestibular information and this can create motion sickness [13]. Furthermore, the sensitivity of the sensory organs changes continuously as a result of aging or lesion for which the central nervous system compensates continuously.

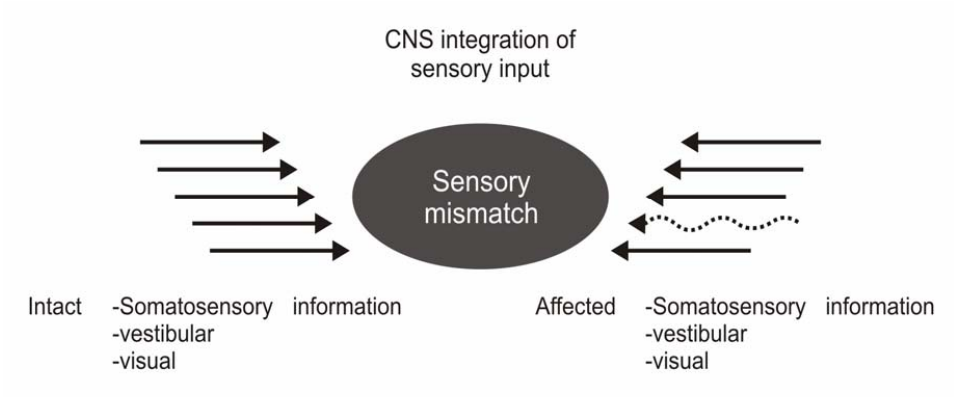

Figure 2. Sensory mismatch.

An acute sensory loss, for example unilateral vestibular loss because of vestibular neuritis, is an extreme challenge to this adaptive ability. The process of compensation occurs at different sites in the central nervous system, by reorganization of sensory input, changes in reflex patterns and behavioural changes of different patterns of movements [13]. CNS compensation of acute unilateral vestibular sensory loss is facilitated by motion, movements and provocation of dizziness. Thus, anxiety and avoidance of dizziness obstruct central compensation and are predictors of long-term problems [1, 2, 14].

3.2.5 Interactions of emotions and postural behaviours

Experiments with dual-task design show that postural performance and cognitive performance affect each other, suggesting that they share circuits in the central nervous system [15]. Fear of falling might be induced by letting test subjects stand on a raised platform during posturography. Using this method Carpenter and colleagues have shown that the induced fear of falling leads to a stiffening of posture [16]. Changes in stiffness were also related to a backward shift of preferred stance, away from the postural threat. Such a fear of falling avoiding behaviour also modifies anticipatory postural control and changes the magnitude of adjustments of postural control [17]. By including rotations of the raised surface during posturography, Carpenter has also shown that changes in postural performance with increased anxiety include changes in muscular and kinematic responses and timing of those responses [18].

Arousal provoked by a cognitive task during posturography is associated with leaning forward and increased activity in the tibialis anterior muscle [19]. Anxiety also appears to influence the contribution of visual sensory input to balance control [19, 20]. Based on posturography on healthy students divided into a high-anxiety group and a low-anxiety group, Wada conclude that anxiety influences the interaction between vestibular, visual and somatosensory input [21]. The effect on postural behaviours of a broader spectrum of emotions as measured with self rating questionnaires has been investigated [22]. From those results it is concluded that anxiety alters vestibular sensory input. Low moods affect balance performance directly through the somatosensory or visual system [22].

3.2.6 Associations between balance control, anxiety, and orientation

Besides the links between postural performance and emotions, there are several possible interactions between anxiety and balance control. Vestibular stimulation affects autonomic regulation (i.e. respiratory [23], and cardiovascular control [24]) and cardiovascular sympathetic nerve activity [25]. Mental activity, anxiety and arousal also influence the gain of the vestibular ocular reflex [26].

The intricate connections and interactions between affective states and balance control occurring in the brainstem have been summarized by Balaban mainly by using data from experiments on rats and rabbits [27-29]. The parabrachial nucleus receives direct projections from the vestibular nuclei, while it is also a centre in the brainstem for interoceptive signals and regulation of anxiety and avoidance behaviour. The vestibular nuclei also receive noradrenergic projections from the locus coeruleus [29, 30]. Balaban also stresses the existence of serotonergic projections to the vestibular nuclei as a link to affective responses [27]

By differentiating two groups of students by level of trait anxiety it was revealed that people with higher degrees of anxiety relied more on direct sensory input. Individuals with lower degrees of anxiety seemed to create a memory-based representation of themselves in space [31]. The anxiety-influenced strategy is labelled egocentric while the memory based is called allocentric. It is suggested that people with a higher degree of anxiety are more sensitive overall to conflicting stimuli due to an egocentric strategy of orientation [32]. It

might be that anxious individuals have a lack of flexibility in using sensory information rather than being specifically sensitive for a specific sensory stimulation [33].

Higher cognitive function is an active component in the process of orientation [34]. It has been shown that bilaterally labyrinthectomized rats lose spatial ability measured by lack of the ability to find their way in a labyrinth and also exhibit agoraphobic-like behaviours [35, 36]. Bilateral vestibular loss is associated with atrophy of the hippocampus and such process reduces spatial navigation as a function of reduced memory capacity in humans [37]. Only few studies have investigated spatial ability in agoraphobic humans and it may be possible that they have deficits in spatial orientation ability [38]. Thus, there are extensive interactions between emotional factors and orientation in the central nervous system.

3.3 Diagnostic concepts

The following concepts reflect efforts to understand patients' perception of dizziness that cannot be explained by specific pathological processes. Concepts are presented in the order that they have occurred in the scientific discussion and clinical literature.

3.3.1 Psychogenic Dizziness

Psychogenic dizziness is probably the oldest and least empirically defined concept. The term "psychogenic dizziness" has at least three meanings. In clinical literature it is often used for all conditions where no pathological processes can be found to explain the patient's dizziness. It is also used in scientific literature to describe dizziness that is either directly related with/or secondary to a psychiatric condition such as panic disorder (see, e.g., [39]).

In clinical literature, psychogenic dizziness can also be used to describe symptoms that in psychiatric terminology are best understood as dissociative or conversion disorders. This later way of looking at dizziness seems more closely related to a psychoanalytic diagnostic tradition where symptoms are supposed to have an unconscious meaning for the patient.

Malingering or imagination of symptoms also seems to be notions used with a similar interpretation.

Brandt has made an effort to discriminate by specific criteria such patients as "psychogenic disorder of stance and gait" from other dizzy populations [40]:

1. *Momentary fluctuations of stance and gait, often in response to suggestion.*
2. *Excessive slowness or hesitation of locomotion incompatible with neurological disease*
3. *"Psychogenic" Romberg test with a build-up of sway amplitudes after silent latency or with improvement by distraction.*
4. *Uneconomic posture with wastage of muscular energy.*
5. *The "walking on ice pattern" which is characterized by small cautious steps with fixed ankle joints.*
6. *Sudden buckling of the knees, usually without falls.*

3.3.2 Cervical Vertigo

Experimental evidence suggests that proprioception of the neck influences balance control [41]. Experimentally induced muscular pain and muscular tension [42] sensitise muscle spindles [43]. Patients with cervico-brachial pain problems, with and without dizziness, exhibit larger body sway than healthy subjects do in response to neck and calf muscle vibration [44, 45]. Clinically, vertigo can be accompanied by cervical pain, and those problems are often treated by physiotherapy [13]. Such a therapy affects postural performance, dizziness and neck pain [44, 45]. According to Brandt, however, there is not enough evidence to propose such a diagnostic concept, mainly because of the difficulties in separating those symptoms from other pathological mechanisms and difficulties in identifying laboratory tests for the condition. It is also still unclear why some patients with severe neck pain have no vertigo and others with moderate neck pain have handicapping dizziness [46]. This concept is closely related to a concept used only in Sweden, namely tension dizziness (spänningsyrsel), that describes muscular tension as a cause of dizziness parallel to tension headache [47].

3.3.3 Panic Disorder and Agoraphobia

According to the diagnostic and statistical manual of mental disorders (DSM-IV) [48] a panic attack is defined by at least four of the following 13 symptoms of somatic and cognitive character; heart palpitations, sweating, trembling/shaking, shortness of breath, feelings of choking, chest pain, nausea/abdominal distress, dizziness, unsteadiness, light-headedness, derealization, depersonalization, fear of fainting, losing control or going crazy, fear of dying, paresthesias and chills or hot flushes [48].

For panic disorder, it is stated that there should be recurrent, unexpected panic attacks, persistent concerns about having an additional attack, worry about implications of attacks and change in behaviours related to attacks. Agoraphobia implies anxiety about being in places or situations from which escape might be difficult (or embarrassing) or in which help may not be at hand if a panic attack or panic-like symptoms occur [48]. Agoraphobic fears are provoked by a characteristic cluster of situations: being outside the home alone, being in a crowd or standing in a line; being on a bridge; and travelling in a bus, train or car. The situations are avoided or else endured but with marked distress or with anxiety about having a panic attack or panic-like symptoms or the requirement of a companion.

Panic disorder and agoraphobia are closely related and those conditions are diagnosed in relation to each other as: *Panic Disorder without Agoraphobia*, *Panic Disorder with Agoraphobia* and *Agoraphobia without history of Panic Disorder.* Panic disorder with agoraphobia simply adds agoraphobia to the criteria of panic disorder [48]. For agoraphobia without history of panic disorder with an associated medical condition, it is stated that the fear should clearly exceed that what is usually associated with this medical condition. It is also stated that patients might have attacks with limited symptoms, which implies that a single symptom might be explained as a panic attack, although the threshold of four symptoms is not reached.

The dominating two, and widely equivalent, psychological theories for panic disorder can be summarized as follows: panic attacks are built up as a consequence of normal autonomic arousal (increased heart rate, sweating): those responses elicit interpretations of coming catastrophe such as fainting, dying, and going crazy. In order to prevent these

catastrophes, avoidance behaviours for physical sensations are developed. Agoraphobic avoidance is explained by the cognitive anticipation of such responses in specific threatening surroundings [49, 50]. Panic disorder seems to be over-represented in different medical conditions such as heart and pulmonary problems [51, 52]. It is hypothesized that increased sensory stimulation increases the risk for the occurrence of physical symptoms and thereby increased risk of misinterpretation of those, resulting in a panic attack as previously described [51]. Although panic disorder with/without agoraphobia includes a wide range of symptoms, it is of special interest that this condition often includes dizziness that appears in situations that also involve particular sensory stimulation.

Population based studies suggest that agoraphobia without panic disorder (5.3% lifetime prevalence) is a more common phenomenon than panic disorder with agoraphobia (3.5% lifetime prevalence) [50, 53]. Since agoraphobia without panic disorder appears to be a non-existing phenomenon in psychiatric practice, the diagnosis has been debated [54].

The interface between panic disorder and dizziness has been subject of increasing amount of research. The most obvious connection between dizziness and panic disorder is hyperventilation during panic attacks, which results in increased heart rate, dizziness and derealization [55]. It has been shown that postural control deteriorates during hyperventilation [56] and that hyperventilation can induce nystagmus among patients with vestibular lesions [57].

Numerous studies have investigated the degree of psychiatric problems among patients with dizziness. Asmundson proposed that there is a high psychiatric co-morbidity especially with panic disorder with/without agoraphobia among patients investigated at vestibular clinics [58]. Conversely, psychiatric populations with panic disorder with/without agoraphobia also have elevated levels of non-diagnostic abnormal findings in vestibular tests [58-61], and this is especially true for those patients with panic disorder with agoraphobia and dizziness between panic attacks [62]. Among unselected patients with dizziness there is an over representation of patients with panic disorder and here the degree of agoraphobic avoidance predicts balance function deficits along with occupational restrictions [63].

In another study, Yardley failed to find definite vestibular abnormalities among patients with panic disorder with agoraphobia. However, results from posturography correlated with the patients' experiences of dizziness [64]. Jacob has described that patients with panic disorder with agoraphobia and dizziness often have signs of vestibular dysfunction and impaired balance control [60]. Jacob has similarly described that patients with panic disorder with agoraphobia who also suffer from dizziness have difficulties stabilizing themselves confronted with poor visual and proprioceptive information (being on boats and on heights) [65, 66]. He argues that vestibular dysfunction leads to a reliance on non-vestibular channels for balance control, i.e., vision and proprioception. This leads to sensitivity in situations with conflicting stimuli. Patients with this sensitivity, named *space and motion discomfort*, have poorer postural control if proprioceptive information is reduced during posturography. Such a balance strategy is labelled surface dependence [65]. Furthermore, Perna found that patients with panic disorder with abnormal posturographic test results and vestibular deficits have the highest degree of agoraphobic avoidance [67]. Treatment with selective serotonin reuptake inhibitors (SSRI) might improve postural functioning in patients with panic disorder particularly during tests with restricted visual input [68].

Godeman and colleagues have investigated whether specific cognitions involved in agoraphobia and panic disorder can predict the degree of handicap and development of panic disorder with/without agoraphobia after vestibular neuritis [69]. They found that fear of specific bodily sensations, especially vomiting, and the fear of recurrent attacks of dizziness predicted 60% of variance in the development of panic disorder or somatoform disorder.

3.3.4 Space and Motion Phobia

From the work by Jacob and Furman partly summarized above, it can be concluded that anxiety disorders might lead to psychosomatic dizziness and that vestibular disorders might lead to somatopsychic anxiety disorders. The concept of space and motion phobia concerns patients that have coping problems when confronted with dizziness. Criteria for space and motion phobia have been modified after the DSM IV, as a complement to the classification of anxiety disorders [4]. All of the following are required:

1. *Marked and persistent fear that is excessive or unreasonable, cued by anticipation or presence of situations associated with intense vestibular stimulation, visual/vestibular/ somatosensory mismatch or inadequate visual or somatosensory spatial cues.*
2. *The symptom-provoking stimuli are avoided or else endured with intense anxiety or distress.*
3. *The avoidance, anxious anticipation or distress interferes significantly with the persons normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the problem.*
4. *Duration of symptoms of months or more.*
5. *Pattern of distress is not better accounted for by an active or severe balance disorder (e.g., acute labyrinthitis, bilateral vestibular hypofunction or by a mental disorder, such as obsessive compulsive disorder or post-traumatic stress disorder.*

3.3.5 Phobic Postural Vertigo (PPV)

The concept of phobic postural vertigo (PPV) has been developed by Professor Thomas Brandt and co-workers in Munich. The concept has gained increasing interest and currently there are about 40 publications concerning the subject. The PPV condition is described by the following characteristics [3]:

1. *Dizziness and subjective disturbance of balance while standing or walking despite normal clinical balance tests such as Romberg, tandem walking, balancing on one foot, and routine posturography.*
2. *Fluctuating unsteadiness in episodes lasting for seconds to minutes or momentary perceptions of illusory body perturbations.*
3. *Although the attacks can occur spontaneously, there is usually a perceptual stimulus (bridge, staircase, and empty room, street) or social situation (department store, restaurant, concert and crowd) from where the patients have difficulty withdrawing and they recognize as provoking factor. There is a tendency for rapid conditioning, generalization and avoidance behaviour to develop*
4. *Anxiety (57%) and distressing vegetative symptoms occur during or after vertigo. Most patients have attacks both with and without anxiety.*
5. *Obsessive-compulsive type personality, labile affect or mild depression.*

6. *Onset of the condition frequently follows a period of particular emotional stress, after serious illness or following an organic vestibular disorder.*

PPV is described as a non-rotational form of dizziness that in at least 21% of cases follows an acute vestibular disorder such as benign paroxysmal positioning vertigo or vestibular neuritis. If patients with head or whiplash trauma are included the PPV population with a somatic base for symptoms gets bigger. Brandt suggests that PPV might emanate from an initial otolith dysfunction. These problems also often occur after a period of psychosocial stress or physical illness. The mean age for diagnosis is 41.5 years and men and women are equally affected [3] [70].

Brandt has suggested a hypothetical mechanism behind these symptoms. A voluntary movement is accompanied by an efference copy signal that identifies the self motion. Without this efference copy it would seem as if the surroundings are moving or as if the body is moved by external forces. A similar experience occurs if we move the eyeball with the pressure of a finger. Brandt hypothesizes that PPV patients exhibit a decoupling of the efference copy signal that leads to a sensory mismatch between anticipated and actual motion. This decoupling might be caused by a constant anxious control of balance performance [3].

Postural control among PPV patients has been investigated by Brandt and colleagues. Patients with PPV show increased sway activity in the 3.53-8 Hz frequency band during quiet stance and they are less able to stabilize their posture by visual information as compared with healthy subjects [71], [72]. However, when postural performance becomes more difficult by tandem standing on foam rubber, they exhibit a postural strategy similar to that of healthy subjects [73]. Thus, it seems as if patients with PPV, by co-contracting the flexor and extensor muscles of the legs use, even during quiescent stance, a postural strategy that is similar to that used by subjects when they are confronted with a postural threat.

PPV is a common disorder (17%) among patients investigated in a clinic for balance disorders [40]. The treatment approach suggested by Brandt and colleagues include a

complete otoneurological examination, explanation of the non-pathological but rather psychological course of problems and recommendation of repeated exposures to dizziness provoking situations. Two follow-up studies have been conducted on a PPV population after such interventions [40] [70]. In the first follow-up studies (average 2.5 years), 22% of patients were symptom free, 50% improved considerably and 28% had no improvement or experienced worse symptoms [40]. The outcome seemed to be independent of additional therapeutic interventions. There was, however, a high frequency of remaining psychological problems despite a decrease in symptoms of imbalance [74]. Persisting physical symptoms such as tinnitus and neck pain contributed to a negative course. There was a tendency for better prognosis with a shorter history of symptoms.

In a later follow-up study of the same population (average 8.5 years), 27% of patients were symptom free, 48% were improved, 22% showed no improvement and 3% got worse [70]. Males were more prone to relapse. In this population 47% received medications (SSRIs or tranquilizers), 29% underwent psychotherapy, 22% underwent physiotherapy and 17% received other types of treatments. Patients often received a combination of these treatments.

Forty two patients with PPV were subjected to structured clinical interview for the DSM III-R (SCID) [74]. It was revealed that there was a high prevalence of psychiatric disorders, mainly Panic Disorder with Agoraphobia. However, the majority of patients did not to a significant extent suffer from panic attacks or anxiety but suffered mainly from imbalance. Brandt argues that PPV differs from panic disorder with agoraphobia. It was also revealed that all 42 patients suffered from a personality disorder, mainly obsessive-compulsive personality disorder (20 patients). Furthermore, many patients had experienced serious psychological stressors.

3.3.6 Visual Vertigo

Visual vertigo is a concept developed to describe patients who experience dizziness in certain visually conflicting surroundings especially after a vestibular disorder [5]. Bronstein and co-workers suggest that this condition emerges in patients confronted with vestibular loss and those who depend on visual cues for the maintenance of balance [75].

From posturographic tests during visual stimulation, it is concluded that these patients show large postural responses to visual stimulations [39]. Those reactions are independent of signs of trait anxiety or history of motion sickness [39]. Bronstein suggests that this condition clinically most closely resembles "psychogenic vertigo". However Visual Vertigo should not be confused with this condition as patients with visual vertigo do not exhibit higher levels of anxiety and depression as compared to other patients with dizziness [76].

3.3.7 Chronic Subjective Dizziness

Staab has updated the PPV concept by postulating a broader concept named "chronic subjective dizziness". This is the most recent effort to approach those problems [77]. According to Staab those patients experience fleeting spins or tilts, unsteadiness, rocking, swaying or fullness in the head. Additionally they are highly sensitive to proprioceptive stimulation and often exhibit visual or surface dependence. They avoid complex visual stimuli and often have difficulties reading. Experiences of anxiety are not a necessity for this condition and Staab recommends a primary focus on neuro-otological evaluation rather than on anxiety. The following criteria are suggested [78]:

Physical symptoms

Subjective dizziness and imbalance: Persistent (≥ 3 months) sensations of non-vertiginous dizziness, light-headedness, heavy headedness, or subjective imbalance that are present on most days.

Hypersensitivity to motion: Chronic (≥ 3 months) hypersensitivity to one's own motion, which is not direction specific, and to the movements of objects in the environment.

Visual vertigo: Exacerbation of symptoms in settings with complex visual stimuli such as in grocery stores or shopping malls or when performing precision visual tasks (reading or using a computer)

Neuro-otological examination

History and exam: Absence of active physical neuro-otological illnesses, definite medical conditions, or medications that may cause dizziness. Past history may include episodes of true vertigo or ataxia as long as the conditions causing those symptoms have resolved.

Neuroimaging: Normal radiographic imaging of the brain.

Balance function tests: Normal or non-diagnostic findings.

3.4 Treatments

3.4.1 Pharmacological treatment

Based on clinical experience, Balaban suggests, that clonazepam, beta-blockers and antidepressants often give patients with the combined symptoms of anxiety and dizziness symptomatic relief [27]. Staab summarizes from a open trial that SSRI treatment causes substantial improvement in 84% of patients with chronic subjective dizziness [79]. He recommends SSRIs prior to vestibular suppressants or benzodiazepines. Staab has evaluated treatment with SSRIs on three different populations. One group had dizziness as a part of psychiatric diagnosis, mainly panic disorder. One group had a primary vestibular disorder with secondary anxiety, and one group had an interaction of both vestibular disorder and anxiety. Patients with a combination of vestibular pathology and anxiety did benefit less from pharmacological treatment than both patients with dizziness without vestibular disorder and those with a primary vestibular disorder [6]. The surprisingly positive results among patients with primary vestibular disorders are hypothetically explained by the possibility that SSRIs acts directly on vestibular pathways.

It has also been shown that an anxious mouse strain improve their postural performance when treated with diazepam or SSRIs [80] [81]. The mechanism of action of those agents on the balance system is not known [27]. As there are receptors that both benzodiazepine and SSRIs bind to in the vestibular nuclei, cerebellar cortex and the parabrachial nucleus network, it is possible that they act simultaneously on vestibular and anxiety-related circuits [80]. Staab suggests that for patients in the group with combined vestibular and anxiety disorders, treatment with physiotherapy and psychotherapy might also be necessary [6].

3.4.2 Vestibular rehabilitation

Vestibular rehabilitation is a broad concept describing physiotherapy for patients with dizziness. However, vestibular rehabilitation exercise is also a specific program of graded voluntary movements of eyes, head and body with the aim of facilitating compensation for vestibular deficits [82]. It is thought that those movements should elicit dizziness and nausea in order to stimulate recovery, improve balance and decrease symptoms. Vestibular rehabilitation is also often combined with physical exercises in order to prevent a

passive lifestyle [83]. This method is highly effective in controlled trials for patients with dizziness of various origins [84, 85, 86]. Such an intervention has also been conducted on a population with panic disorder with agoraphobia and dizziness with encouraging results [87]. An additional effect of a customized exercise treatment program has been described by simulator-based desensitisation exposure by integration of whole body or visual environment rotators [88].

3.4.3 Cognitive behavioural therapy

Behaviourism is a learning theoretical tradition in psychology where theories of behaviours are mainly based on animal laboratory experiments, which were later extended to human behaviour. In behaviouristic models, processes within the organism are ignored and only eliciting situations, observable behaviours and their consequences are analyzed and subjected to treatment. Two basic categories of behaviours are outlined; respondent and operant. Respondent behaviours are automatic and autonomic, reflex-like responses that are elicited by relatively specific stimuli. Learning of this type ("classical conditioning") implies that new, previously neutral stimuli, become elicited autonomic reactions [89]. Operant behaviours are not restricted to the autonomic system but could comprise any type of behaviour. Learning of this type ("operant conditioning") implies that positive and negative consequences increase or decrease the frequency and type of behaviours that is performed in certain situations [90] [89].

Anxiety disorders are assumed to be developed by classical conditioning, i. e., previously neutral stimuli become anxiety-eliciting in combination with operant conditioning, i. e., flight and avoidance behaviour ("negative reinforcement"). Negative reinforcement prevents the anxious person from sufficient exposure to anxiety-eliciting situations which prohibits habituation or extinction of anxiety [91].

Treatment of phobias, based on principles of habituation and exposure, was the first clinical problem where the behaviourist tradition was shown to be highly effective. Currently about 90 % of patients with specific phobias are expected to obtain significant relief by this treatment procedure and it is often recommended as treatment of choice [92].

The behaviouristic model was extended by also taken into account processes within the organism and including interpretation of stimuli in the analysis of behaviours such as panic disorder [49]. Cognitive Behavioural Therapy (CBT) of panic disorder with or without agoraphobia includes today several techniques; re-evaluation of catastrophic beliefs, psychoeducation, controlled breathing or relaxation training, identification of safety or avoidance behaviours, exposure to bodily sensations and in vivo exposure [50].

At present there is an ongoing discussion concerning the efficacy of anxiety management methods that imply control over anxiety sensations, such as breathing retraining [93] and relaxation, mainly because those methods might be included in patients´ repertoire of avoidance behaviours [94, 95]. It is also still uncertain whether the addition of focus on catastrophic beliefs is a necessary part of successful treatment [96, 97]. However, those methods gathered under the concept of CBT, are effective treatment for panic disorder with and without agoraphobia and around 80% of patients become symptom-free after about 12 sessions [92, 98, 99].

There are a few studies that investigate the effect of CBT on dizziness. There is an effect of a combined intervention of vestibular rehabilitation exercises and CBT in a group format for elderly patients with dizziness [100]. A similar approach has recently been tested on younger patients with a documented positive effect on dizziness and handicap [101]. In a case-report a similar combined approach of CBT and vestibular rehabilitation and is described for dizziness and acrophobia [102]. The similarity between physiotherapy as vestibular rehabilitation and behaviour therapy has been highlighted in the treatment of the combined symptoms of panic disorder with agoraphobia and dizziness [103]. Staab proposes that patients with the interaction of vestibular disorders and anxiety disorders also might need vestibular rehabilitation and psychotherapy along with pharmacological treatment [6]. Several other authors also consider CBT as a possible treatment for patients with PPV [3] and dizziness [2, 58, 86]. No study has yet been conducted on CBT for a population of PPV patients.

4 AIMS

The general aim of these thesis was to explore whether PPV is a clinically valid concept. We also aimed to develop treatment programs for patients with dizziness and avoidance behaviour. The following specific aims were raised:

Study I

Does a PPV population rely more on proprioception for postural control than healthy subjects do?

Study II

Does a population with PPV perceive more anxiety and handicap than other patients with dizziness do?

Study III

Is there an additional effect of CBT compared with a self treatment program based on vestibular rehabilitation exercises for patients with PPV?

Study IV

What can behaviour analysis tell us about the PPV population?

Study V

Does CBT of PPV have a long-term effect?

Do men and women respond differently to CBT?

5 STUDIES

5.1 Populations and procedure

Patients with PPV were consecutively recruited at a tertiary referral centre for balance disorders at the Department of Otorhinolaryngology, University Hospital of Lund, Sweden. Patients were referred from other hospitals, other clinics at the hospital and from primary care. The inclusion was performed mainly by two physicians. A few patients were also referred to the study by two other physicians. All four doctors were specialists in otorhinolaryngology with a special interest in oto-neurology. The inclusion was based on a clinical neurological and oto-neurological examination according to the earlier summarized PPV criteria [3]. Patients included were between 18 and 65 years of age. They had normal caloric reactions, tests of voluntary eye movements and normal neuro-radiological examinations of the brain with computerized tomography scan (CT) or magnetic resonance imaging (MRI). Exclusion was based on signs of active vestibular or neurological disorder, polyneuropathy, substance abuse, severe psychiatric disorder or difficulties with regularly travelling to the unit for CBT.

Study I: Fourteen patients with PPV (4 men, 10 women; mean age 47 years, range 23-60 years) were investigated. We also performed posturography on 24 healthy people as a control group (14 men, 10 women; mean age 38 years, range 24-49 years). Posturography was not a routine examination at the beginning of the project, and thus we do not have posturographic data for the entire PPV population. The posturographic examinations were included in the project as we observed during treatment the high attention of postural sway and as patients appeared to stand very stable. Vibratory proprioceptive stimulation was used as patients were able to provoke dizziness by voluntary muscle co-contractions and as a propriceptive sensitivity had been described among a similar population [65].

Study II: Questionnaires were sent by letter to patients referred to the balance clinic two weeks before their balance investigation. Patients later diagnosed with PPV were excluded from the control group of unselected patients with dizziness. The population consisted of 30 men and 65 women, mean age 47 years, range 20 to 68 years. The population was

divided into three subgroups defined by the degree of vestibular pathology according to the criteria described by Grunfeld [104]: vestibular imbalance with presence of nystagmus or directional preponderance (n=10), labyrinthine disorder evidenced by canal paresis or positive results on positional testing (n=32) and absence of vestibular pathology (n=51). Two patients were not possible to classify and were excluded from this comparison. This population was compared with 34 patients with PPV (18 men and 14 women, mean age 44 years, range 23–62 years). Questionnaire data for the PPV population were collected originally as a part of Study III. Accordingly these data were collected after medical investigation in contrast to the control group where data were collected before investigation. From the entire PPV population of 39 patients one patient was excluded due to multiple sclerosis, two patients declined participation before the questionnaires were collected and two patients declined participation later in the project. Their results were thereby withdrawn from statistical evaluation in this study.

Study III, IV and V: In January 1999 we started the inclusion of patients with PPV to Study III. Patients who fulfilled the above-mentioned PPV criteria were asked if they were interested in participating in an intervention study. After providing informed consent, they were informed by the investigating physician of the mechanisms behind their dizziness based on the hypothesis developed by Brandt [3]. The necessity of self-exposure to triggering situations was explained and patients were administered written instructions to provoke dizziness modified from principles of vestibular rehabilitation exercises (see appendix).

The included patients were sent questionnaires by mail. Patients who did not return completed questionnaires were reminded by phone. Every second patient included was contacted by the psychologist and offered individual CBT. Originally, it was planned that 40 patients should be included. However the inclusion was stopped in August 2002 when 39 patients were included. From the 39 patients initially included, two patients did not want to participate after the receiving the questionnaires and another patient was excluded due to multiple sclerosis. In the CBT group, one patient denied treatment and one patient interrupted treatment. One patient in the self-treatment group had CBT and SSRIs, prescribed by another physician parallel to his self-treatment intervention and was

excluded from analysis. Two patients in the self-treatment group did not complete post-treatment measure and did not want to further participate when they were contacted by phone. Sixteen patients received the CBT as adjunct to self-treatment while fifteen underwent the self-treatment procedure. After completion of the CBT, questionnaires were simultaneously administered to both the patient who had had CBT and the patient who meanwhile just had had the self treatment procedure.

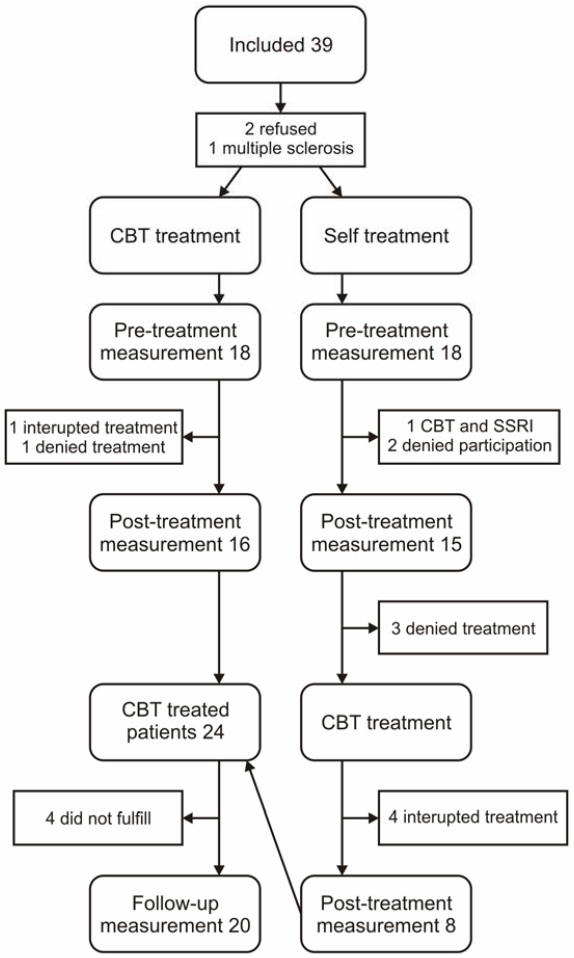

Figure 3: Flowchart of participants of studies III, IV, and V.

Two patients in the self treatment group denied participation in the project when they were offered additional CBT. Three patients denied treatment. Four patients interrupted

treatment, which left eight patients in the self treatment group who also completed the CBT. In all, 24 patients received the CBT. One year after completion of the CBT, questionnaires were re-administered. Patients who did not answer questionnaires were reminded by mail and then by phone. Twenty patients responded to this follow up.

5.2 Methods

5.2.1 Questionnaires (paper II, III, V)

At the start of the study project (1998) there were no statistically explored questionnaires addressing vertigo for a Swedish population. We chose questionnaires originally developed for English speaking populations namely the Dizziness Handicap Inventory (DHI) [105], the Vertigo Symptom Scale (VSS) [106] and the Vertigo Handicap Questionnaire (VHQ) [107] as those questionnaires are most often used internationally for evaluation of these types of symptoms.

The DHI was translated into Swedish, with authorization by the author. The questionnaire consists of 25 items addressing dizziness. Perceived disability and handicap are rated on physical aspects (7 items), emotional aspects (9 items) and functional aspects (9 items). The sum of these items constitutes the total score on the DHI. Answers are scored as follows: “No” (0 points), “Sometimes” (2 points) and “Yes” (4 points). In the original English version the internal consistency for the total scale (α=.89) and the three subscales (α.72 to α.85) are satisfactory [105]. Another Swedish translation of the DHI has, since completion of our study, been statistically investigated with statistically satisfactory properties [108].

The VSS contains questions that address symptoms of balance system disorders: dizziness, vertigo, instability and falling (16 items) [106]. It addresses both the duration and frequency of those symptoms. The frequency of symptoms is rated from never (0 points) to more than once a week (4 points). It also addresses symptoms of somatic anxiety and autonomic arousal: pain, sweating, pounding heart, shortness of breath and fainting (15 items). Two scales are comprised of the sum of items; the VSS severity- and the VSS anxiety scale. The VHQ is a 25-item questionnaire that assesses handicapping

consequences of vertigo such as difficulties in performing physical activities and restrictions in social activities [107]. Answers range from 0 (never) to 4 (always) to describe how often the subject experiences each described handicap. In order to prevent response bias, 13 items are inverted (higher values indicating lower degree of handicap).

Both the VSS and VHQ have been investigated in their original versions with satisfactory statistical properties. The translations to Swedish were performed by Mendel and co-workers [109]. We found that patients systematically excluded certain items on the VSS severity scale. We asked several patients how those answers should be interpreted. On basis of those patients' answers, excluded items were scored as zero or non-symptomatic. Patients who systematically excluded items were contacted and asked to complete those.

In paper III, the Hospital Anxiety and Depression scale (HAD) was also administered. This is a 14-item self-reporting scale developed specifically to measure symptoms of anxiety and depression among physically ill people [110]. The HAD consists of 2 subscales with 7 items each, measuring anxiety and depression. The Cronbach´s α=.90 for the total scale and .0.90 for the depression subscale and .82 for the anxiety subscale [111]. The total sum of each subscale may vary from 0 to 21 points. In its original version, a cut-off level for clinical cases of depression and anxiety is made at 11 points and borderline cases from 8 to 10 points.

Statistical analysis: In the present project, parametric statistics have been applied for questionnaire data. In general no violation of the criteria for the adequacy of parametric statistics could be identified. To avoid doubts on the validity of these results non-parametric statistical tests have been performed as well, arriving to the same conclusion as parametric ones. Therefore only the results for the parametric statistics are reported. Statistical analyses of the questionnaires were performed on means of accomplished items in order to avoid influence of single, non-systematic missing items. In study IV, the subdivision of population by gender, and in analysis of patients who dropped out, subgroups become small and variance homogeneity is challenged in one variable (HAD total). Those data were investigated with non-parametric statistics. Results are however presented in means and standard deviation to make presentation of data uniform. A

difference where P<0.05 was considered statistically significant.

5.2.2 Posturography (paper I)

Body sway was recorded as antero-posterior forces and torques from the feet acting on a force platform (400x400x75 mm) equipped with strain gauges. Data were sampled at 50 Hz. The tested subjects' posture was provoked with vibratory stimulation to gastrocnemius muscles of both legs. The vibration stimulation was applied by vibrators designed as cylinders (0.06 m in length and 0.01 in diameter) and held in place by elastic straps. The vibration amplitude was 1.0 mm and the frequency 85 Hz.

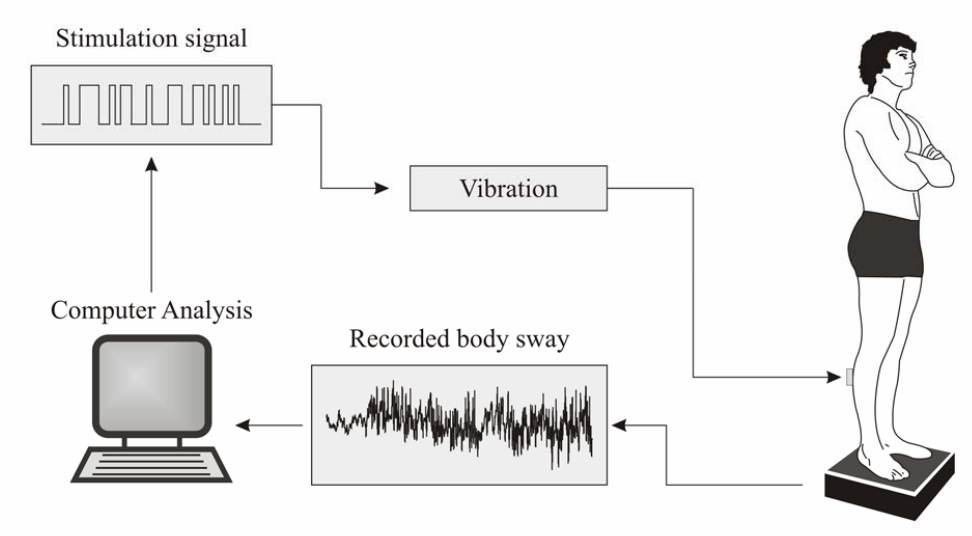

Figure 4: Posturographic recordings

The tested subject was instructed to stand erect but not at attention with the feet on the force platform at an open angle of 30° and with the arms crossed over the chest. Spontaneous body sway was recorded for 30 seconds. Thereafter the vibratory stimulation started, applied according to a pseudo-random schedule. The test subjects were exposed to stimulation for 205 sec. The test procedure was performed first with eyes closed and then with eyes open with patients focusing on a spot at a distance of about 1.5 m.

Postural performance was measured as torque variance, which reflects the mechanical

energy acting on the force platform. As this analysis is influenced by the subject's height and weight, data were normalized by squared weight and squared height. Data were multiplied by 1000 for representational purposes. Three components of the recorded body movement were quantified: total torque variance, torque variance < 0.1 Hz and torque variance > 0.1 Hz. Values were analysed for the quiet stance period and for the period with vibratory stimulation (for a full methodological description see [112]).

The normalized torque variances were used to calculate a Romberg and a vibratory quotient. The Romberg quotient reflects the degree of stability provided by vision and is calculated by eyes closed sway / eyes open sway. The vibratory quotient reflects the destabilizing effect of the vibratory proprioceptive stimulation and is calculated by vibration period sway / quiet stance period sway.

Statistical evaluation: The Mann-Whitney test was used for non-paired comparisons and the Wilcoxon test for paired comparisons of posturography data. A value of $P<0.05$ was considered to be statistically significant.

5.2.3 Cognitive behavioural therapy (CBT)

The CBT was performed at the balance clinic by the author, who is a psychologist. He was supervised by a psychologist (Uwe Harlacher) trained in CBT. Every session lasted about 60 minutes except sessions involving exposure to specific situations outside the clinic which lasted longer. The CBT was set to 10 sessions with a tolerable range for 8 to 12 sessions.

There is no psychological treatment described for patients with PPV. Accordingly we had no guidelines to follow for the psychological treatment. As Brandt had described the condition to differ from Panic Disorder with Agoraphobia, we did not want to follow treatment schedules for Panic Disorder with Agoraphobia. Our intention was to explore treatment possibilities as a pilot project for future studies. Using descriptions of the condition and the experience from treatment of a subject in a pilot project we expected the population to be diagnostically closer to those with specific phobias such as acrophobia. As one of the aims of this study was to develop treatment methods and gather clinical

experience of patients with PPV, we deliberately allowed treatment to be adjusted to individual needs as is custom in clinical practice.

Treatment started with an interview considering social and occupational status. Earlier stressful life events such as diseases or traumas were addressed along with earlier periods of psychiatric illness and treatments. Patients were informed about the nature of CBT and its focus on specific goals and relief from handicap rather than relief from specific symptoms. Patients were advised to collect information considering experiences of dizziness according to the headings often used in CBT: situation, thoughts, feelings, physiology and reactions. When the analyses of the problems were completed, patients were presented with a written summary as a basis for further interventions.

Because treatment partly was inspired by principles of exposure as described for specific phobias and agoraphobia, a hierarchy of symptom-eliciting situations was constructed for most patients. Patients were educated about the balance system. As knowledge about the population grew, this education was based on a hypothetical dizziness-maintaining vicious circle based on misinterpretation of normal body sway with increased leg muscle contraction exacerbated by anxiety (figure 5).

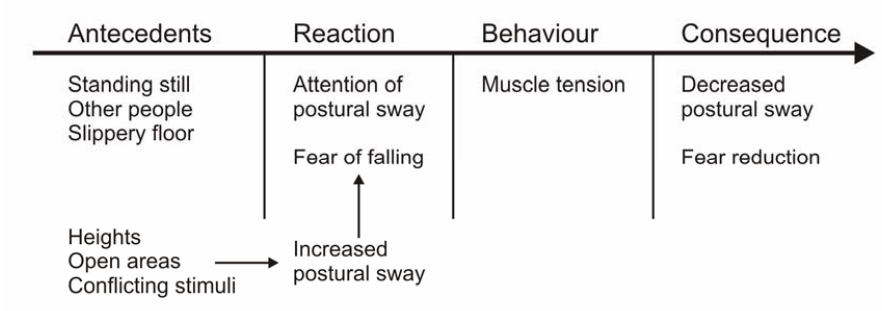

Figure 5: Hypothetical mechanism as presented during treatment.

Inspired by principles adapted from CBT of Panic Disorder with Agoraphobia, catastrophic thoughts, mainly considering falling, were identified and explored from experiences outside therapy and by behavioural experiments during standing. Avoidance or safety

seeking behaviours were identified and patients were encouraged to stop using those when exposed to dizziness-provoking situations. Patients were often instructed in controlled breathing and/or to identify and voluntarily counteract exaggerated muscle tension similar to applied relaxation [113] especially during standing. During the study it was discovered that contractions of specific muscles such as in the legs, neck, jaw and tongue induced dizziness, which is why such contractions were used as behavioural experiments in order to show patients the effect of this behaviour. Exposure to standing was initially performed at the balance clinic. Patients were then encouraged to expose themselves to specific dizziness-provoking situations or alternatively this was done together with the psychologist. Gathered behaviour changes were summarized as guidance for the coping of any future relapses.

Table 1. Overview of the studies

Paper	Comparison	Population	Method
I	Healthy subjects vs. PPV patients	24 healthy subjects, 14 PPV patients	Posturography
II	Unselected dizzy patients vs. PPV patients	95 dizzy patients, 34 PPV patients	Questionnaires: DHI, VSS, VHQ
III	Self treatment vs. CBT	39 patients included with PPV 16 patient had CBT 15 patients had self treatment	Questionnaires: DHI, VSS, VHQ, HAD
IV	Descriptive	24 treated patients with PPV	Summary of behaviour analysis
V	Before treatment vs. Follow up	20 treated PPV patients	Questionnaires: DHI, VSS, VHQ, HAD
V	Treated men vs. treated women	24 treated PPV patients,13 men, 11 women	Questionnaires: DHI, VSS, VHQ, HAD

5.3 Results

5.3.1 Paper I: Phobic postural vertigo: body sway during vibratory proprioceptive stimulation.

The patients with PPV exhibited larger total torque variance and torque variance in the frequency range above 0.1 Hz with eyes open. With eyes closed, those differences were significant for torque variance above 0.1 Hz. Vibratory proprioceptive stimulation made differences between the PPV group and healthy subjects larger in both the eyes open and eyes closed condition in torque variance at frequencies above 0.1 Hz while no differences in the lower frequencies were found. Torque variance was significantly lower during quiet stance with eyes open compared to eyes closed for normal subjects which was not the case for the PPV patients.

The Romberg quotient was significantly lower for the PPV patients for torque variance above 0.1 Hz. The vibratory quotient was significantly higher for PPV patients for torque variance above 0.1 Hz. The results suggest that PPV patients are more sensitive to proprioceptive disturbances than normal subjects are and less apt to use visual information to control upright stance. The PPV concept thus seems to capture a specific mechanism during standing.

5.3.2 Paper II: Experience of handicap and anxiety in phobic postural vertigo.

Results of questionnaires measuring handicap (DHI, VHQ), dizziness severity and anxiety (VSS) were compared of patients with PPV and unselected patients with dizziness. The two groups were equal in terms of age (p=.179). While there was an equal sex distribution in the PPV population (18 men, 16 women), a higher proportion of women appeared among the control group (30 men, 65 women) of unselected dizzy patients. As this difference was statistically significant (p=0.027) separate analyses were made for each gender.

Women with PPV scored significantly higher in all parameters compared to women in the control group. Men with PPV did not differ significantly from men in the control group although they tended to score higher in all parameters. A comparison between sexes within

each group revealed statistically significant differences between women and men with PPV on the DHI emotional subscale.

The control group was divided into subgroups defined by the degree of vestibular pathology. Neither of the two-subgroups exhibited gender distributions that was similar to the gender distribution of the PPV population or elevated levels in test results similar to those of the PPV-population.

It seems as if patients with PPV experience more handicap, more severe dizziness and more anxiety compared to other dizzy patients regardless of their degree of vestibular pathology. The PPV population also comprises more men than other dizzy populations and populations with anxiety disorders do.

5.3.3 Paper III: Treatment of phobic postural vertigo. A controlled study of cognitive-behavioural therapy and self-controlled desensitization.

While 15 patients underwent the self treatment only, 16 patients underwent the additional CBT. The self-treatment group and the CBT group did not differ in test results, distribution of sexes, duration and different sociodemographic parameters before treatment. The self-treatment population was however significantly older (46 years, 30-59) than the CBT group (40years, range 23-59 years) (p= .037). There was a significant decrease in scores of DHI total, DHI emotional, DHI functional, VHQ, HAD total, HAD depression and HAD anxiety for the CBT group after the treatment period. For the self-treatment group the within-group comparison revealed significant statistical reduction in the VSS severity and the VHQ. Differences between pre-treatment and post-treatment measurements were significantly larger for the CBT group compared to self treatment group for VHQ (p=.009), HAD total (p=.006), HAD depression (p=.013) and HAD anxiety scales (p=.030). There were no significant differences between groups in the VSS anxiety and in the VSS severity scales. These results show that there is an additional effect of CBT as compared to self administered vestibular rehabilitation exercises.

5.3.4 Paper IV: Phobic postural vertigo – clinical experience gathered during cognitive behavioural therapy.

Twenty four patients with PPV underwent CBT. The behaviour analyses of those

treatments were summarized. Two case reports were included.

Antecedents: Patients often experienced dizziness in stores, traffic situations and open areas and on bridges. As the experiences of the population grew, the specific sensory components of those situations were included in the analysis. The conclusion was drawn that mere standing and the awareness of spontaneous body sway elicited a fear of falling. Visual input in specific surroundings seemed to increase postural sway and thereby the perceived postural threat. However, dizziness also varied in ways that could not be explained by situational factors alone.

Reactions: Patients exhibited a wide range of behaviours that served the purpose of controlling the risk of falling. A fear of falling led to increased attention to postural control and restriction of spontaneous body sway which is assumed to parallel the findings in the posturographic study (I). Patients also experienced pain problems in their legs, backs or necks which probably was secondary to increased muscle tension. Voluntary contraction of leg, neck and jaw muscles provoked the patients' dizziness.

Treatment was developed to help patients interpret body sway as being a natural process during standing and to re-evaluate beliefs concerning poor balance ability. Patients were trained in voluntary muscle relaxation of the legs while standing. They were also exposed to dizziness provoking situations.

5.3.5 Paper V: No long term effect of cognitive behavioural therapy for phobic postural vertigo.

Twenty patients fulfilled a one-year follow up after completed CBT. Test results in the follow-up did not differ from test results collected before treatment. Among the total number of treated patients (n=24) men exhibited significantly larger reduction in HAD tot (p=.021) and HAD dep (p=.041) after the treatment period than women did.

These results indicate that the CBT developed did not give long-time relief of PPV. Moreover, women and men might benefit differently from such treatment.

6 GENERAL DISCUSSION

6.1 Methodological considerations

The dizziness-related questionnaires are not fully psychometrically evaluated for Swedish populations and in the translation of the VSS, systematic misunderstandings have been found. The Swedish versions of the VSS and the VHQ comprise additional items than the original instrument [109]. They found, using factor analysis, that the original categorization of duration and frequency in the VSS severity scale is inappropriate in the Swedish version. As the psychometric properties of those extended versions seems still uncertain, only the original items of the questionnaires, translated by Mendel et al, and the original procedure to define subscales, have been applied in the present studies. The number of different questionnaires used might increase the risk for mass significance.

As intended, treatment methods were developed and changed during the project and the CBT was based on the ability of a single therapist and a single supervisor. Such factors always restrict the possibility to make generalizations of treatment outcome and gathered clinical observations.

Considering the large number of patients investigated at our balance clinic during the period of the study (n=1218) the PPV population is surprisingly small. This is explained mainly by the fact that many of the patients investigated came from other parts of Southern Sweden and thus they could not be recruited because of difficulties to arrange transportation. A possibility is that the PPV criteria have been applied more strictly compared with the studies by Brandt [40], which might affect the degree of handicap, dizziness severity and anxiety in the population as found in paper II.

Posturography was not available for the entire PPV population. There is a gender distribution (four men and ten women) in the population of study I that do not reflect the PPV population as a whole (19 men and 19 women). The validity of posturography in identifying vestibular pathology has been questioned [114]. However, it seems as if posturographic tests show a high correlation with patients´ experience of imbalance and

handicap [115, 116, 64]. How to understand such behaviours and quantify good balance performance from bad balance performance is also still an issue of controversy. This question, which however is beyond the scope of these thesis, would be quite important for further investigations.

Different physical conditions complicated the treatment, such as fainting reactions, primary orthostatic tremor and spontaneous eye movements. These phenomena also confound the validity of the concept of PPV. It should however, be considered that this population was constituted by certain criteria applied by specialists in neuro-otology practice and this population is a clinical reality despite such aetiological confusion. The high degree of handicap and suffering in this population (II) makes it probable that our PPV population shares essential features with populations that exhibit an interaction of anxiety disorders and vestibular disorders [6] [2].

6.2 PPV versus other concepts

Is PPV a clinically valid term to use for patients with dizziness in specific situations and who experience anxiety and exhibit avoidance behaviour? Some other concepts are possible alternatives to PPV in defining this population: psychogenic dizziness, cervical vertigo, panic disorder, panic disorder without agoraphobia, agoraphobia without panic disorder, space and motion phobia, visual vertigo and chronic subjective dizziness.

The term psychogenic dizziness is ill defined and may be too unspecific. There is also a lack of empirical evidence to suggest such a category. As dizziness is a perception, regardless of origin, all types of dizziness are to some extent psychogenic in character and from this perspective the concept seems to be confusing.

Muscular factors, which are hypothesized to be the mechanisms behind cervical dizziness, merit more attention as they might parallel our findings of sensitivity to proprioceptive stimulation in PPV (I), that patients could provoke dizziness by muscle co-contraction and that they had pain problems (IV). On the other hand it seems difficult to establish the direction of causality as a PPV population is burdened with autonomic overreactions and

myofascial problems are probably over represented in any such population.

A broader category of problems as compared to PPV is defined by the concept of "space and motion phobia", which also includes sensitivity to visual stimulation along with sensitivity to proprioceptive stimulation. Jacob concludes that, "PPV fits within our diagnostic criteria for SMP" (space and motion phobia) [4]. Besides the criteria "*dizziness and subjective disturbance of balance while standing or walking"* the criteria for space and motion phobia seems to be very similar to the PPV criteria. Accordingly these concepts overlap, although there is a broader focus of sensory sensitivity in the space and motion phobia concept.

The population described by Bronstein and co workers as "visual vertigo" appears to be different from our PPV population by having lower degree of anxiety as measured by the Spielberger state and trait anxiety inventory [39]. However, as revealed by the HAD, there were patients among our PPV population that did not experience clinical levels of anxiety (IV) although they exhibited avoidance behaviours. It might be disputed for our PPV patients, as well as for the patients described by Bronstein as having no trait anxiety as measured by the Spielberger State and Trait Anxiety Inventory, whether these questionnaires capture emotional processes that are relevant for the suffering of those patients.

As defined by the criteria of PPV, patients do not always express anxiety along with dizziness. As both a PPV population and the Visual Vertigo population comprise people who seek healthcare, they obviously have had an emotional reason for seeking treatment; otherwise they would not have turned up in a hospital at all. These emotional reasons for seeking treatment may neither be captured by the Spielberger State and Trait Anxiety Inventory nor by the HAD for some of our PPV patients. As our population also seemed to be sensitive to visual stimulation as described by behaviour analysis (IV), it is still possible that a population described to have "visual vertigo" overlaps with a PPV population.

The visual vertigo population differs from PPV also in the visual sensitivity as measured by posturography (I). One might expect from our (I) and other posturographic results [71]

[72] that patients with PPV do not experience dizziness due to visual input. However, our experiences based on behaviour analysis (IV) suggest that patients with PPV experience dizziness when confronted with specific visual stimuli. Several factors might contribute to these conflicting results. A posturographic test setting where the degree of influence by vision is altered by closing the eyes is very different from a natural situation where lack of visual references might be explained by the distance to the observer. Such a mechanism has been described for healthy subjects as a reason for larger postural sway [117]. The alteration of influence by vision used in our study is also different from the visual stimulation used by Guerraz and colleagues [39].

Patients with PPV share with patients with panic disorder the essential feature of vigilance to body sensations. PPV patients exhibit vigilance to spontaneous body sway and they fear that they will fall. Patients with panic disorder are vigilant for heart palpitations that elicit a fear of having a heart attack. As described by the DSM criteria, a panic attack might include a wide array of physical and cognitive symptoms. It appears that panic disorder as a fear of panic attacks actually consists of various catastrophic interpretations of different bodily sensations which results in diverse safety-seeking behaviours [118, 119]. Among symptoms of a panic attack the following are related to dizziness and postural control: feeling dizzy, feeling unsteady, fear of fainting and fear of loosing control. Feelings of unsteadiness are mentioned in the criteria of panic attack, according to the DSM IV, but the specific fear of falling is not. Fear of loosing control, as mentioned in the DSM IV, refers to behaving in a socially unacceptable way, which often applies to PPV patients.

The fear of falling receives quite extensive attention in the research of elderly and might also be common among younger subjects [120, 121]. The fear of falling is not described in the literature examining catastrophic fears in panic disorder with and without agoraphobia [118, 119, 122]. This implies that patients with panic disorder with agoraphobia lack the fear of falling or this fear is present but it is difficult to express and thereby it is an underestimated reason for avoidance behaviours. It is not reasonable that a PPV population would differ that much from a population with panic disorder why the latter explanation seems to be most in accordance with our experience. Patients often denied a fear of falling in spite of safety seeking behaviours that clearly served to minimize the risk of falling. It is

also possible that motor safety responses that were observed (grabbing a wall, restricted limit of stability) are autonomous or reflexive in character and do not involve conscious or verbal functioning. This might also contradict our view of such behaviours as avoidant responses.

Although the cause of equal sex distribution in PPV patients remains unclear, this kind of distribution between sexes seems atypical for any anxiety disorder as such population exhibit higher percentage of women. Those differences might be explained by differences in health care seeking behaviour for men and women with dizziness, but it is still possible that there are specific aetiological factors tied to gender. Some women believed that dizziness varied with menstruation cycle but such correlations could not be established. Panic disorder with agoraphobia is a more severe condition among women [123], which might also reflect that there are gender differences tied to coping behaviours of agoraphobic avoidance.

Dizziness among patients with panic disorder might be explained by hyperventilation [55], a behaviour which also was found among two of our patients. These patients could learn to discriminate this kind of dizziness from dizziness induced by increased muscle tension. The same kind of discrimination has also been documented by Beidel and Horak [103]. The experiences of dizziness among our patients were normally not accompanied by more than four symptoms as defined by the DSM criteria for panic disorder. Attacks of dizziness did not follow the dramatic course as described for panic attacks and sometimes patients did not complain of anxiety at all. However “limited symptom attacks” as described in the DSM IV could be true for our PPV patients.

According to population-based studies agoraphobia without panic disorder is a common problem, meanwhile the problem seems to be rare in psychiatric practice [54]. One explanation for these confusing results might be that patients with agoraphobia without panic disorder do not seek psychiatric treatment but rather end up in primary care or vestibular clinics. Of the unselected patients with dizziness in study II 63% had no vestibular pathology or belonged to the intermediate group. Staab has also shown that for two thirds of patients with “psychogenic dizziness” the dizziness started with a vestibular

disorder that caused anxiety which then maintained the dizziness [124]. Only for one third of patients the origins of dizziness was a primary psychiatric disorder, mainly panic disorder with agoraphobia.

The term chronic subjective dizziness is the most recent applied to a population with dizziness without signs of active neuro-otologic disease and it seems to update the concept of PPV. The chronic subjective dizziness concept focuses on the neuro–otological investigations rather than on anxiety [78]. We agree with this development as, according to our investigations, anxiety is not always expressed by patients and as also sensory factors should be included in the analysis of problems. Staab focuses on visual factors and sensitivity to motion similarly as the concept of space and motion phobia. However, Staab does not discuss postural behaviours as a necessary factor of the understanding. A problem with chronic subjective dizziness as a concept is the word "subjective", which implies that there are types of dizziness that are objective.

6.3 Benefits and disadvantages with the concept of PPV

We see an advantage in the PPV criteria d*izziness and subjective disturbance of balance while standing or walking"* as it identifies a population of patients with fear of falling and specific postural behaviours that are not described for other populations with dizziness or anxiety reactions. However, we believe that a PPV population overlaps populations defined by the concepts of space and motion phobia, visual vertigo and chronic subjective dizziness.

PPV as a concept sprung from lack of concepts to understand this population in a balance clinic perspective. The concept of PPV (probably as well as space and motion phobia and visual vertigo) captures sensory aspects of dizziness which are assumed to be overlooked when the description and diagnosis is based on a psychiatric terminology.

By the word "phobic" in PPV we expected those reactions to be restricted to specific stimuli as compared for example with phobia of heights. However, from a psychological point of view, these problems turned out to be more complex and the group was rather

multisymtomatic with pain problems and different physical symptoms. Therefore the term phobic seems to be inappropriate and we do not suggest anxiety to constitute an exclusive diagnostic factor. PPV also include obsessive-compulsive type of personality as a criterion. This criterion is based on SCID interviews (statistical clinical interview for the DSM IV) where all included patients received an axis II diagnosis [74]. It is possible that the DSM criteria were widely applied in that study. The clinical validity of such criteria might also be questioned, as it is difficult for clinicians to appreciate, and as it might be valid for any person with longstanding suffering tied to any aversive sensation.

6.4 Possibilities and limitations with CBT for PPV

Regardless of diagnostic matters and aetiology, patients with dizziness suffer and are handicapped. CBT for such patients as developed during the project has been shown to have beneficial effect for a population with PPV. Psychoeducation around balance control, over-interpretation and vigilance or attention to postural sway, behavioural experiments by voluntary muscular co-contraction illustrating for the patient the influence of muscular control, relaxation during standing, exposure to standing and standing in dizziness-provoking situations might be useful treatment methods for this population.

Although statistically significant, the difference between patients with self treatment and those with additional CBT are relatively small and there were not differences in all applied outcome-measures. This limited effect, especially when compared with usual CBT-outcomes for other disorders such as panic disorder and specific phobias, can have several reasons of which the following seems to be most important. In the present project, the focus was not to apply known treatment-methods as effective as possible but potentially useful methods had to be developed at the first place. Established CBT-interventions could not be applied directly but rather had to be modified and served as a source of inspiration for the development of new interventions suitable for this patient group.

Additionally we found that the patients labelled as 'PPV' were not a homogenous group but presented a broad array of problems which greatly restricted the possibility to generalize procedures of problem-analysis and treatment from one patient to another.

Instead a high degree of individual analysis and individually tailored treatment was necessary. Accordingly, much of the total time of sessions was used to gather information and analyze the individual patient's problems as necessary prerequisites for the development of suitable interventions. The amount of sessions were settled to about ten based on the description of the condition of PPV as being a phobic condition and based on one patient treated before the project started. This is comparable with treatment of panic disorder, which usually is accomplished in about 12 sessions, and generally with a very good outcome [98]. However, it seems as if severe agoraphobia predicts poorer outcome [125]. Patients with severe agoraphobia might overlap with our PPV population and the difficulty to achieve treatment results for a population with severe agoraphobia illustrates that even well established CBT methods can have limited effect under certain circumstances. It might very well be that the number of sessions in the present project (III) was too low, especially considering that much of the time was used to develop and customise methods. There are good reasons to assume that the application of the developed treatment methods would result in a better outcome.

It could not be shown that the effects of CBT were stable, which is a major short-coming. There is clearly a need to further develop treatment procedures in order to achieve lasting effects. Staab has described a difficulty to achieve treatment effect of SSRI for patients with the combination of vestibular and anxiety disorders [6]. Our difficulties to achieve maintained treatment effects might be a parallel finding to those observations. Similarly as described for cervical vertigo, pain problems might influence dizziness. If this is the case for our PPV population it might also explain why our treatment did not affect experiences of dizziness. It seems unlikely that this CBT intervention would in such restricted time affect muscular problems. It is possible that remaining pain problems and dizziness symptoms contribute to relapse to previous avoidance behaviours and handicap.

The combination of anxiety and physical problems might imply a double vulnerability and proneness for relapse even after periods of reduced anxiety and handicap. Since vestibular and anxiety-problems are interacting with each other, patients with combined symptoms might have a higher vulnerability. Circumstances that elicit vestibular symptoms also induce anxiety and vice-versa.

In regular clinical work, follow up sessions ("booster-sessions") are included in treatment and patients have the possibility to return to treatment if they relapse. Especially for anxiety disorders relapse-prevention is an integral part of CBT. [126]. In the present project relapse-prevention was routinely addressed during the final sessions. However, no booster-sessions were given in the present project mainly because of difficulties to include those within the study design. It might be that relapse prevention was too sparsely applied in the CBT of this project.

6.5 Hypothetical mechanism of PPV

Cognitive and emotional processes affect postural control. Any person can recognize the difference between standing and walking on a slippery versus a stable surface. Understanding these differences is crucial for the understanding of PPV. From the results of our and others investigations, it seems probable that patients with PPV exhibit a postural behaviour similar to that exhibited by people confronted with a postural threat [73]. This hypothesis is well in line with what was found in behaviour analysis in the present project.

Patients with PPV sometimes express a fear of falling and they very often misinterpret spontaneous body sway as being a sign of disequilibrium (IV). This might result in a restricted limit of stability and in a higher frequency sway (I) [71] in situations with conflicting sensory input (standing on heights, travelling in a train or bus and standing on uneven surface) or socially demanding situations where instability might be interpreted in a stigmatizating way (appearing as being drunk or otherwise embarrassing). This higher frequency of postural sway leads to higher consumption of muscular energy, which has been shown to be associated with the fear of falling [19]. Muscular fatigue impairs postural control [127] and might also sensitize muscle spindles [42]. This mechanism could thus explain the higher sensitivity for proprioceptive stimulation found in our investigation (I) and the possibility to provoke dizziness by voluntary muscle contraction (IV). A changed postural behaviour might also explain why many of our patients also had pain problems in their legs, back, neck or head.

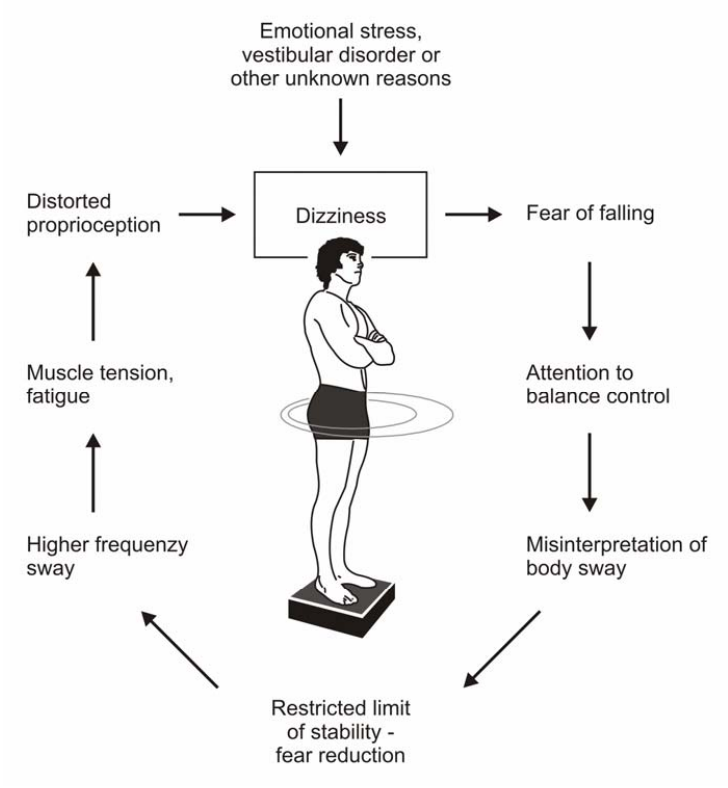

Figure 6: Hypothetical mechanism of PPV.

6.6 Conclusions and clinical implications

The concept of PPV captures a clinically relevant mechanism behind long-standing dizziness, by the criterion "*dizziness and subjective disturbance of balance while standing or walking"*. Postural behaviours might be specifically targeted during treatment but the treatment methods developed in this project need to be further investigated.

- The postural control of patients with PPV is more easily disturbed by vibratory proprioceptive stimulation.
- Patients with PPV differ from other patients with dizziness in perceived degree of handicap, symptom severity and anxiety.
- There is an additional effect of CBT as compared to a self treatment procedure

although the effect on experienced dizziness is weak.

- Patients with PPV often exhibit a fear of falling and restricted limit of stability as an avoidant response during standing.
- There is no long term effect of CBT .
- Men benefit more than women from CBT.

It is reasonable to assume that populations defined as panic disorder with and without agoraphobia, space and motion phobia, visual vertigo or chronic subjective dizziness overlap a PPV population. Despite considerable efforts to create a homogenous population, a PPV population will comprise different problems. As there are different types of dizziness, even within a single patient, we recommend a smaller focus on diagnostic matters and a focus on the degree of handicap as a clinical guidance for treatment. We claim that both cognitive and emotional factors are necessary components for the understanding of dizziness regardless of its aetiology. The aetiology for each patient of the interaction between proprioceptive, vestibular, visual, emotional and cognitive factors is probably best understood and made understandable by specialists in balance disorders. An emphasis on diagnostic matters does not encourage such an analysis.

Vestibular rehabilitation exercises have been developed to create compensation and habituation in the central nervous system for vestibular deficits. Being well evaluated for populations with dizziness of various origins the method seems to be a necessary component of treatment also for PPV [86] along with physiotherapeutic knowledge of postural and musculoskeletal problems. There is a growing body of evidence also suggesting effect of treatments with SSRIs on dizziness of different origins [6] why pharmacological treatment also might be a necessary component. A multidisciplinary analysis of problems and multidisciplinary treatment programs are recommended and should also include physiotherapeutic and pharmacological treatment.

From the outlined treatment methods and results in these thesis a structured multidisciplinary treatment approach might be evaluated on a larger sample with questionnaires addressing vertigo that are now better validated for Swedish samples such as the latest translation of the DHI [108]. Possible gender-related differences in coping

behaviour should be taken into account. The posturographic responses in the population can be further evaluated. It is also necessary to further evaluate, from a learning psychological point of view, the interaction of fear of falling and subsequent responses. How proprioceptive and visual stimulation interacts in the PPV patients also merits further investigation.

7 POPULÄRVETENSKAPLIG SVENSK SAMMANFATTNING

Yrsel är ett mycket vanligt symtom bland patienter inom olika specialiteter inom sjukvården. Orsaken bakom symtomen kan ligga i neurologiska sjukdomar, sjukdomar i innerörats balansorgan samt i psykiatriska sjukdomar, framför allt ångestsjukdomar. Medicinska undersökningar leder inte alltid till att orsaken till yrseln konstateras i en specifik sjukdom och ibland kan man konstatera att det funnits en sjukdom som läkt ut men att upplevelsen av yrsel trots det kvarstår. För sådana patienter förutses graden av lidande och handikapp bäst av graden av ångest.

Undersöker man en grupp patienter på en mottagning för yrselpatienter finns det en tydlig överrepresentation av ångestsjukdomar. Samtidigt verkar det som om patienter med panikångest har mer störningar i funktionen i innerörats balansorgan. Hur patienter med yrsel och handikapp utan påtagliga sjukdomar i balansorganens funktion ska diagnostiseras är oklart liksom hur de skall behandlas.

Ett alternativ till en psykiatrisk klassifikation är begreppet fobisk postural yrsel (PPV). PPV karakteriseras av följande:

1. Yrsel av ”ostadighetskaraktär” framförallt i stående och gående utan att fall har förekommit och utan tecken på störd balansförmåga.
2. Sekund- / minutlånga attacker av ostadighet och / eller plötsliga korta attacker med illusion av egen rörelse eller rörelse av omgivningen.
3. Undvikande beteende av situationer som utlöser yrsel.
4. Ångest vid eller efter yrselattacker (inte alltid).
5. Viss tvångsmässig personlighet
6. Debut efter en skada eller sjukdom på balansorganet, annan sjukdom, fall eller en period av stress.

Syftet med detta arbete är att undersöka fördelar och nackdelar med detta begrepp. Vi vill också undersöka om dessa patienter kan behandlas med kognitivt beteendeterapeutiska metoder företrädesvis utvecklade för behandling av ångesttillstånd. Kognitiv

beteendeterapi för ångestreaktioner bygger framför allt på utmaning av de situationer som leder till obehag.

Denna avhandling består av fem delarbeten. Resultaten av dessa kan sammanfattas enligt följande: Balansförmågan hos 14 patienter med PPV undersöktes med posturografi och jämfördes med resultaten för 24 friska personer (studie I). Patienterna hade mer högfrekvent kroppssvaj. Patienterna stördes mer av vibrationsstimulering av vadmusklerna och hade svårare att stabilisera sitt stående med hjälp av synen.

Svaren i frågeformulär som mäter grad av upplevd yrsel, handikapp och ångest för en grupp patienter med PPV jämfördes med en oselekterad grupp patienter med yrsel (studie II). I gruppen med PPV-patienter var det signifikant fler kvinnor varför resultaten analyserades separat för kvinnor och män. Patienter med PPV överlag, men specifikt kvinnor hade mer ångest och var mer handikappade än övriga patienter med yrsel.

16 patienter fick kognitiv beteendeterapi (CBT) medan 15 patienter endast fick ett självträningsprogram (studie III). De patienter som erhöll CBT förbättrades i större utsträckning. Alla behandlade patienter fyllde också i frågeformulären efter ett år som en uppföljning vilket visade att resultaten av behandlingen försvunnit och besvären var desamma som före CBT (studie V). Det verkade som om män hade större positiv effekt av behandlingen än vad kvinnor hade.

Under projektets gång utvecklades behandlingsmetoder. Dessa var framförallt inriktade på att ändra patienters övertolkning av spontant kroppssvaj som ett tecken på obalans och beteenden som syftade till att minska risken för att trilla (studie IV). Ett mycket vanligt sådant beteende inom gruppen var ökad muskelspänning vilket också antogs förklara varför många patienter hade långvariga smärttillstånd. Att muskelspänning bidrog till problemen visades med viljemässig spänning av muskler vilket provocerade patienternas yrsel. Behandlingen baserades framförallt på exponering för stående i successivt mer provocerande situationer där risken successivt ökades för pinsamma konsekvenser av yrseln t.ex. rädslan att uppfattas som berusad.

Begreppet PPV fångar en specifik rädsla för att trilla och till denna rädsla är knutet ett förändrat sätt att hålla balansen. Detta beteende verkar genom uttröttning av musklerna leda till ökad yrsel och i en ond cirkel som också inkluderar muskelsmärta. Ett sådant beteende finns inte beskrivet för andra ångesttillstånd. Det finns emellertid fler anledningar till långvarig yrsel som inte kan förklaras med PPV begreppet. Begreppet riskerar också leda till en överfokusering på ångestrelaterade faktorer, vilket kan leda till att sinnesfysiologiskt förklarbara reaktioner frånses. De utvecklade behandlingsmetoderna kan vara användbara men effekten av dessa metoder måste ytterligare undersökas. En kombinationsbehandling som också inkluderar sjukgymnastik och farmakologisk behandling kan ha ytterligare bättre effekt.

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THE END

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10 APPENDIX: PATIENT INSTRUCTIONS

Note! You will experience some dizziness when you do the exercises!

Måns Magnusson Öron-Lund 2005

A home-based Training program for dizziness

1. Sit down, fixeate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixating the object. Repeat twice a second for 15 seconds. Count one-thousand-one; one-thousand-two …one-thousand -fifteen to keep pace and time.

2. Stand up and put a finger on a stabile object (chair/table), fixate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds. Count one-thousand-one; one-thousand-two …one-thousand -fifteen to keep pace and time. .

3. Stand up without support or touching any object, fäst blicken på ett föremål 1,5 - 2 m bort. Samtidig
fixate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds.

4. Stand up and close your eyes (with and then without support) .
fixate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds.

5. Stand up , fixeate on an object 1,5 - 2 m away.
Shake your head **vertically** from side to side - still fixating the object. Repeat twice a second for 15 seconds.

6. Walk forwards fixeate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixating the object.
Repeat twice a second for 15 seconds.

A home-based trainingprogram for dizziness (cont´d)

7. Stand on a pillow from your coach in a corner of the room. Remain there fore *1 minute* then close your eyes and remain standing like this for another minute. If it is difficulte place a chair in front of you and initially you may put a fingertip on the chair.

8. Stand up holding a glass of water which is filled halfways again in a corner of the room. Remain there fore 1 minute than close your eyes and remain standing like this for another minute. If it is difficulte pu a chair in front of you and initially you may put a fingertip on the chair.

9. Take walk outdoor for at least 30 minutes. Try windowshopping which will have you turning your head from side to side when you walk.

How to execute the head movements*:*
Begin to smoothly shake your head. Increase the speed until vision gets blurred. Decrease speed to regain clear vision, then increase again. The idea is to push the limit were vision gets blurred.