

Management of the patient with chronic dizziness

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Abstract. In this review we present a pragmatic approach to the patient with chronic vestibular symptoms. Even in the chronic patient a retrospective diagnosis should be attempted, in order to establish how the patient reached the current situation. Simple questions are likely to establish if the chronic dizzy symptoms started as benign paroxysmal positional vertigo (BPPV), vestibular neuritis, vestibular migraine, Meniere's disease or as a brainstem stroke. Then it is important to establish if the original symptoms are still present, in which case they need to be treated (e.g. repositioning manoeuvres for BPPV, migraine prophylaxis) or if you are only dealing with chronic dizzy symptoms. In addition the doctor or physiotherapist needs to establish if the process of central vestibular compensation has been impeded due to additional clinical problems, e.g. visual problems (squints, cataract operation), proprioceptive deficit (neuropathy due to diabetes or alcohol), additional neurological or orthopaedic problems, lack of mobility or confidence, such as fear of falling or psychological disorders. A general neurological examination should also be conducted, amongst other reasons to make sure your patient's 'chronic dizziness' is not due to a neurological gait disorder. Treatment of the syndrome of chronic dizziness is multidisciplinary but rehabilitation and simple counselling should be available to all patients. In contrast, vestibular suppressants or tranquilisers should be reduced or, if possible, stopped.

1. Introduction

Perhaps the most dreaded of all patients with balance or vestibular disorders is the one with longstanding, continuous dizziness. If you are an ENT doctor this patient may have already seen a neurologist. If you are a neurologist your patient has definitely seen an ENT. If you are a neuro-otologist the patient is likely to have seen both an ENT and a neurologist. The patient almost certainly has had many sophisticated tests, including brain scans, audiograms, neck x-rays and maybe vestibular function tests, so there seems to be little room for manoeuvre for the doctor seeing this patient for the first time. Here, we intend to present our personal approach to this problem (see Bronstein and Lempert 2007) and organise a plan of action. It is therefore useful to ask sequential questions, what do

patients with chronic dizziness have? and, then, what can we do for them?

2. The origin of chronic dizziness

Patients with chronic symptoms of dizziness and unsteadiness describe their problem in many ways. They can feel dizzy or giddy in the head, including sometimes mild rotational feelings, a bit 'drunk', detached, slightly off balance or unsteady, that they seem to veer to one or both sides whilst walking, that they feel more steady if they touch or hold on to furniture, that they feel as if they were walking on a mattress or on cotton wool. These symptoms are fairly constant but there may be minor fluctuations, 'bad and good days'. We feel that the description of the *current symptom* is important but perhaps not as useful as the description of the *history of the symptom*. We feel that there are essentially three types of history, the patient who started with one or more attacks of rotational vertigo, the pa-

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tient with a progressive history of disequilibrium and the patient with neither of these.

3. The patient with a past history of vertigo

Patients in this category report that at some point in the preceding months or years they suffered one or many vertigo attacks. Detailed history taking should let you reach a retrospective diagnosis of vestibular neuritis, BPPV, vestibular migraine (migraine-related dizziness), Meniere's disease or other conditions. Essentially, the patient with vestibular neuritis will remember a single but disabling attack of vertigo lasting for about a week; the patient with BPPV will have suffered brief episodes (lasting seconds) of rotational vertigo on looking up or lying down/turning over in bed. Patients with migraine or Meniere's disease suffer recurrent attacks of vertigo lasting anything from minutes to a few days; in the case of migraine typical migraineous features (headaches, photophobia, phonophobia, visual auras) may accompany the vertigo whereas in Meniere's disease there is inevitable progression of deafness and tinnitus, as well as aural fullness due to the raised endolymphatic pressure (Lempert and Neuhauser 2005; Sajjadi and Paparella 2008).

In chronic stages patients do not experience much disabling rotational vertigo but rather a constant sensation of vague dizziness or subjective unsteadiness. If patients keep on suffering from episodic vertigo attacks (e.g. BPPV, Meniere's disease, migraine) they can easily distinguish between the acute recurrence and the chronic dizziness (Waterston, 2004). In this case, the chronic dizziness tends to increase with each vertiginous episode.

Chronic dizziness is reported by 0.3 percent of the adult population (12 months prevalence) (Neuhauser et al., 2008). However, in specialised clinics these patients are extremely common, accounting for about 11% of referrals, with a female preponderance of 2:1 (Odman and Maire, 2008)

A patient who started with vestibular neuritis will describe that, say, 10 months ago he (/she) had the 'flu' and then severe vertigo nausea and imbalance for a week. He saw a GP/ENT/neurologist at that point and was told that he had a 'viral infection of the inner ear', that the symptoms will progressively go away and that he will return to normal or near normal within 2–3 months. Textbooks state that the majority of patients recover fully within a few months but specific research shows that up to 50% of vestibular neuritis patients con-

tinue with long term residual dizziness and unsteadiness (Kammerlind et al., 2005; Palla et al., 2008). Unfortunately, because acute and recurrent vertigo are so prevalent in the general population, these patients fill up specialised dizziness clinics and experience considerable psychosocial and working disability.

Why do some patients not recover fully after one or more episodes of vertigo? We suspect that long term symptoms are the consequence of some difficulty in the process of vestibular compensation. It is difficult to ascertain in each individual patient what may have interfered with the fine tuning required to achieve full vestibular compensation. Possible causes are sometimes apparent, for instance if there are associated visual, peripheral nerve (proprioceptive) or central neurological problems, reduced mobility or advanced age, which has all of the above. Often, however, the cause is not apparent and, in patients with anxiety or depression, one is tempted to attribute the chronic dizzy symptoms to a psychological problem. Indeed, pre-morbid psychological trends of anxiety and depression do contribute to long term symptoms and disability (Godemann et al., 2004, Odman and Maire, 2008) As with any other chronic disease, psychological adjustment to protracted or recurrent vestibular dysfunction may play a critical role (de Ridder et al., 2008). At presentation, 80% have psychiatric comorbidity (Odman and Maire, 2008). At the moment, there is no definitive conclusion as to the exact mechanisms leading to chronic dizziness in patients with previous vestibular vertigo but there is agreement that the problem is multifactorial and the solution multidisciplinary. Some specific syndromes of patients with chronic dizziness have been described and, although the interpretation of these syndromes is still somewhat controversial, the reader will benefit from being familiar with the different modes of presentation in the clinic.

3.1. Visual vertigo

These are patients with chronic dizziness who report that their symptoms worsen in certain 'visually busy' surroundings. The syndrome is given different names, such as visual vertigo (Bronstein, 1995; Guerraz et al., 2001), visuo-vestibular mismatch (Longridge et al., 2002) and space and motion discomfort (Furman and Jacob, 2001). Frequently reported trigger situations are, walking through shelves in supermarket aisles (sometimes called the supermarket syndrome), viewing movement of large visual objects such as moving clouds, wind-swept trees, rivers flow, disco lights, mov-

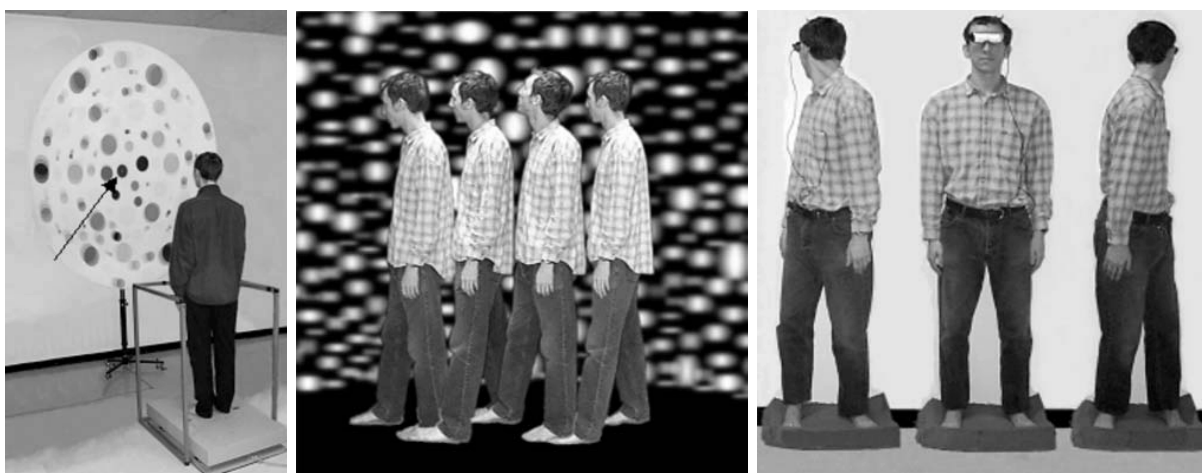


Fig. 1. Examples of the type of visual stimulation employed in the desensitisation of patients with chronic dizziness and visual vertigo. Left: a rotating disk. Centre: gait exercises during planetarium projection. Right: optokinetic stimuli delivered by the EyetrekTM or virtual reality systems; in this case the patient stands on foam to decrease the accuracy of the lower limb proprioceptive input. Modified from Pavlou et al. 2004.

ing crowds, traffic, curtains being drawn, or films with car chase scenes. Repetitive visual patterns like the stacks of cans in supermarket shelves, ironing striped shirts, walking past a repetitive patterned fence seem to be relevant. Some patients also mention that moving the eyes, reading and flickering or fluorescent light can make them feel dizzy.

On the one hand we know that, in many of the situations described, the visual environment contains too much information (e.g. the optokinetic stimulus provided by shelved goods plus movement of other shoppers plus flickering lights plus the patient's own head movements). In other scenarios, the self-motion information conveyed by the visual and the vestibulo-proprioceptive systems are not in agreement, for instance when watching disco lights or motion rich cinema films (e.g. movement of large visual scenes is normally interpreted as self-motion but in such cases this is not corroborated by the vestibular system creating inter-sensory conflict). In addition, patients with vestibular lesions are more prone to be influenced by visual stimuli, as part of the sensory substitution process which takes place during vestibular compensation. Furthermore, research has shown that patients with the visual vertigo syndrome are 'visually dependent', in that, for them, vision is the predominant force driving balance and spatial orientation. For these reasons, it should come as no surprise that intense visual motion stimuli and visuo-vestibular conflict can produce disorientation and imbalance. Convergence between visual and vestibular inputs occurs at multiple levels, from the medulla up to the cerebellum and cortex where such

symptoms might originate. Indeed secondary vestibular neurons cannot distinguish between sources of afferent activity (Barmack, 2003) thus explaining why visual motion input can recreate normal and abnormal vestibular sensations.

The reasons why some specific vestibular patients develop visual dependence and visual vertigo are not known, but migraine appears to be a predisposing factor (Drummond, 2005). We do know, however, that visual vertigo can be improved by specific rehabilitation (Pavlou et al., 2004). This consists of progressive exposure to visual motion stimuli and visuo-vestibular conflict during the rehabilitation program (see Figures; slightly modified from Pavlou et al., 2004).

3.2. *Motorist disorientation syndrome*

Driving, particularly in motorways (highways), can be uncomfortable for patients with chronic dizziness. Occasionally, patients report a sensation that the car is tilting or veering to one side. This sensation can be compelling because, often, these patients see their mechanic or change their car before seeing their doctor. This 'motorist disorientation syndrome' also seems to be, at least partly, visually determined because patients describe problems in visually deprived areas (top of a hill), visually challenging conditions (e.g. simultaneously overtaking and being overtaken by a car). Indeed the coexistence of visual vertigo and motorist disorientation syndrome in the same patient is not rare.

Going round a bend, as when driving in a roundabout, can also disorient patients but here there may be

a predominantly vestibularly mediated component. It must be kept in mind that driving through a curve is different to just turning round while walking. The radius of curvature is large while driving so the conditions are equivalent to being centrifuged and that is why you are being pushed sideways against the door. In this case the unusual stimulation (sideways linear acceleration) acting on a damaged otolith system maybe responsible for the symptoms. Page and Gresty, who first described this syndrome in vestibular patients, favoured an otolith based explanation (Page and Gresty, 1985). The additional, non congruent, visual stimulation and the non physiological posture, on vibrating cushions which numb somatosensory inputs, make driving a kind of sensory deprivation experiment in which vestibular asymmetries may re-emerge. Psychological components usually in the form of panic and avoidance behaviour can be contributory. In fact, in patients with no vestibular history and findings, a psychological disorder maybe the only mechanism responsible (Taylor et al., 2002). In these patients, however, there usually is more 'panic' and less 'veering and tilting' of the car.

In patients with motorist disorientation syndrome comprising tilting car illusions, previous history/findings of vestibular disease and no panic component, treatment will be based on vestibular rehabilitation with the addition of visuo-vestibular conflict and optic flow stimuli. In patients with predominant panic symptoms but neither car tilting illusions nor vestibular disease, the treatment is predominantly psychiatric, often combining medication (e.g. SSRI, anxiolitics) with cognitive behavioural therapy (Townend and Grant, 2006).

3.3. *Psychological presentations, phobic postural vertigo, panic attacks*

It is universally agreed that the frequency of psychological symptoms is high amongst vestibular patients. During the acute stages of vertigo, anxiety levels are high, patients often thinking that they have suffered a stroke or a heart attack (Godemann, Linden et al., 2004; Pollak et al., 2003). Direct vestibulo-autonomic projections and vestibular connections to the limbic system may play a part as well (Fuller et al., 2004).

Outside the acute vertigo situation the role played by psychological mechanisms is far from clear. In chronic stages, some 30–40% of vestibular patients suffer from anxiety and depression and these may be the presenting or dominant symptom (Eckhardt-Henn et al., 2008). Also, a diagnosis of 'phobic postural vertigo' is used to describe patients with transient sensations of

unsteadiness in whom postural balance is entirely normal on extensive clinical examination (Brandt, 1994). Patients may deny psychological disturbance but excessive anxiety or an obsessive-compulsive personality may be apparent. Panic attacks, in which patients describe autonomic symptoms, catastrophic thoughts and avoidance behaviour can also turn up in dizzy patient clinics mimicking vestibularly initiated chronic dizziness. A complicating factor is that in approximately 20–30% of patients with these 'psychogenic' syndromes a clear vestibular onset such as BPPV, migraine or vestibular neuritis can be elicited (Brandt, 1994). Another, that some symptoms of the visual vertigo and motorist disorientation syndromes overlap with phobic postural vertigo and panic attacks and, unfortunately, diagnostic and classification criteria differ considerably between workers in the field. It may well be that some of the presentations described in this section, e.g. the motorist, visual or phobic postural syndromes, are just common modes of reaction that humans have to different disorders. A vestibular disorder or a psychological problem may lead to a similar final clinical result. In summary, the interface between vestibular and psychological mechanisms is far from clear at this point in time (Staab, 2006). Although future research may improve this situation, patients cannot wait for their treatment so, how do we proceed?

We suggest considering each patient on his/her own merit. Two examples at opposite ends of a spectrum would be, the patient who has no psychiatric history but a picture of vestibular neuritis leading to chronic dizziness with visual vertigo and, another, the patient with general anxiety and panic attacks in the supermarket in whom, despite direct questioning and specialised balance examination, no vestibular features are detected. There is little doubt that the former patient should receive vestibular treatment, usually rehabilitation including visual motion desensitisation, and that the latter should be in the hands of a psychiatrist or cognitive behavioural therapist. The many patients in between these two opposite examples often require combined, individualised treatment. Ideally, vestibular therapists, whatever their background (audio-vestibular or physiotherapy), should have counselling skills so that they can deal with the very common anxiety, depressive or phobic components that patients usually have.

4. **The patient with progressive disequilibrium**

We will briefly review here the topic of the patient with progressive disequilibrium because it is not always

Table 1
Relevant questions and investigations in the patient with chronic disequilibrium (Slightly modified from Bronstein and Lempert 2007)

Oscillopsia? →	Downbeat nystagmus syndrome ->	MRI
	Bilateral loss of vestibular function	Vestibular tests
Cervico-brachial pain? ->	Cord compression; canal stenosis	MRI spine
Sphincter dysfunction?		
Long tract signs?		
Memory loss? ->	Hydrocephalus	MRI head
Incontinence?		
Slowness, tremor? ->	Parkinsonism	Clinical neurolog DAT scan
Motor incoordination? ->	Cerebellar ataxia	Clinical+MRI
Speech disorder?		Paraneoplastic & Genetic tests
Vascular factors? ->	White matter disease	MRI
Strokes?		
Distal numbness? ->	Polyneuropathy	Clinical +EMG

easy to separate the patient with chronic dizziness following episodes of vestibular vertigo, from those patients with true gait unsteadiness, on the basis of the history alone.

Dizziness is usually acknowledged by the patient as a sensation 'in the head'. They may say that they *feel* as if they were drunk but that, in contrast to true drunkenness or gait unsteadiness, friends or colleagues will not notice anything wrong with their balance. In contrast, the patient who describes disequilibrium due to gait unsteadiness will usually volunteer that loss of balance is noticed by themselves and observers alike. They may in fact have fallen or nearly fallen over as a result of the unsteadiness, again in sharp contrast to the patient with chronic dizziness secondary to a poorly compensated vertiginous condition who rarely fall. In most cases, unfortunately, clinical conditions producing gait unsteadiness are progressive.

The diagnosis of the patient with disequilibrium is also heavily guided by the history, in particular the symptoms associated to the disease responsible for the unsteadiness of gait. Table 1 provides a few questions useful in guiding the examination and laboratory investigations required to reach a diagnosis. Please note that an MRI of the brain is not enough – and definitely no substitute for a neurological examination, in many of these conditions. The treatment of these conditions is part of general neurology and will not be discussed here (for review see Snijders et al., 2007).

5. The patient with no history of vertigo nor disequilibrium

Sometimes patients refer a sense of vague and chronic dizziness with no history of 'true' vertigo nor dis-

equilibrium. Hopefully direct questions will guide to specific organs or systems, but this line of enquiry may be negative. A full clinical examination is warranted, including checking for orthostatic hypotension if the history is suggestive; the latter is particularly important in the elderly under drug treatment for high blood pressure when, due to age-related cognitive decline, the history provided by the patient may be unhelpful.

This is a difficult patient group to diagnose and, often, numerous investigations are required, including blood tests to rule out general medical conditions such as anaemia, hypothyroidism or other endocrinological conditions, diabetes or hypoglycemia, brain scans and vestibular function tests. With a non disease-specific history, a neurotic personality and negative investigations, many patients are diagnosed as psychogenic dizziness – rightly or wrongly. Observe, during history taking, if the patient appears to hyperventilate and enquire about frank hyperventilation and anxiety episodes, as well as features which may indicate hyperventilation such as perioral paresthesia. In our opinion, the active hyperventilation test, however, is not so reliable because hyperventilation induces dizziness and unsteadiness in everybody (Sakellari et al., 1997), not only in patients with the hyperventilation syndrome. Only when the patient recognises his/her own typical symptoms during voluntary hyperventilation is the test useful. Blood gases and the opinion of a chest physician may be justified in some cases.

6. Management of the chronic dizzy patient

There are four components to the treatment of any patient with dizziness or vertigo, whether acute, recurrent or chronic: 1) the treatment of the specific vestibular

lar condition, e.g. BPPV or migraine, 2) non-specific pharmacological treatment of vertigo and associated nausea. 3) the provision of information, counselling and reassurance, 4) physical rehabilitation. These four aspects should be considered for each individual patient and they are all equally important. However, not all patients will require action in all four domains. For instance, patients with BPPV will usually require repositioning treatment and nothing else. Patients with migrainous vertigo, if they do not have interictal or chronic symptoms, may just require antimigraineous drugs but no rehabilitation. In contrast, patients with chronic dizziness typically require counselling and rehabilitation but no drugs. As always in medicine, the treatment has to be tailored to the individual patient. Here, we will summarise some treatment aspects that specifically relate to chronic dizziness.

6.1. Disease-specific treatment

If an active underlying vestibular disorder is identified, such as BPPV, migraine or Meniere's disease, this needs to be treated because, with each vertigo attack, the patient's chronic symptoms tend to increase. By the same token, do not assume that if a patient is in a chronic phase he may not, for instance, have BPPV. After months or even years of experiencing positional vertigo, patients with BPPV develop complex but effective strategies to avoid the offending head positions. Always ask your patient if he/she still has episodic or positional attacks – and always examine your patient even if they deny having them. The treatment of the individual pre-existing disorders which may have led to chronic symptoms of dizziness is, however, beyond the scope of this article (for review see Bronstein and Lempert, 2007).

6.2. Non disease-specific pharmacological treatment

Vertigo or nausea can be improved with medications and these are very useful *for the acute attack or the recurrent episode*. Most experts now believe that long term use of vestibular suppressants and tranquilisers is counterproductive for the process of vestibular compensation. These drugs should only be used for truly acute vertigo and stopped as soon as vertigo begins to recede. Describe briefly the process of vestibular compensation to your patients and explain that, for compensation to occur, the brain must feel some vertigo. This sensation will function as a warning signal and so compensation processes will be set in motion. Essentially,

Table 2

Factors interfering with clinical recovery following vestibular lesions (slightly modified from Bronstein and Lempert 2007)

Age
CNS lesions
Peripheral somatosensory disorders
Visual disorders
Reduced visual acuity
Modified optics (e.g. cataract operation)
Strabismus
Visual Dependence
Cervical or other spine disorders
Psycho-social problems
Medical treatment
Insufficient/inadequate counselling
Surgical procedures
Antivertiginous drugs
Tranquilizers
Lack of mobility
Orthopedic (e.g. hip arthritis)
Excessive bedrest or patient advised not to move
Fear
Avoidance of symptom-provoking situations

no vertigo, no warning signal and, hence, no compensation. Despite this general principle, sometimes it is difficult to wean patients off medication they may have taken for a long time and one has to compromise and let the rehabilitation begin before complete medication withdrawal.

6.3. General support

Reassurance, information and counselling are important. Patients with long term dizziness have often wandered from clinic to clinic, from specialist to specialist, sometimes for months or years. On the basis of normal brain scans some doctor is likely to have said, at some point, 'there's nothing wrong with you', or 'there's nothing that medical science can do for you' or 'it's all in your mind'. We believe that this is not only the wrong approach but also usually not true. Patients may have suffered a genuine vestibular insult in the past, which may not show in conventional vestibular testing, but failures in the vestibular compensation process and/or secondary added psychological problems complicate the situation (see Table 2; also Lacour et al. this Volume). It is a fact that rehabilitation works even in patients with many years of chronic dizzy symptoms and therefore you want your patient to cooperate in this process. For this reason you need to explain the principles of vestibular compensation and rehabilitation. Many useful webpages or leaflets (e.g. the British Brain and Spine Foundation www.bbsf.org.uk or the Vestibular Disorders Association www.vestibular.org) explain

Table 3
Approach to the patient with chronic vestibular symptoms (slightly modified from Bronstein and Lempert 2007)

Approach	Specific goals – try to establish:
Attempt retrospective diagnosis	1) Did it all start as? BPPV, vestibular neuritis, recurrent vertigo (e.g. Migraine, Meniere's disease), brainstem stroke. 2) Are the original symptoms still present? 3) ... or are we only dealing with chronic dizzy symptoms?
Multifactorial approach	Is vestibular compensation impeded due to additional problems? – Fluctuating vestibular disorder: recurrent vertigo – Visual problems: squints, cataract operation – Proprioceptive deficit: peripheral neuropathy (diabetes/alcohol) – Neurological problems: e.g. ischemic white matter disease – Orthopaedic problems and lack of mobility – Loss of confidence, fear of fall, psychological disorders – Age: all of the above possible but try to identify which
Treatment is multidisciplinary	– Treat any episodic vertigo specifically: * BPPV: repositioning manoeuvres * Vestibular Migraine: migraine prophylaxis * Meniere's disease : low salt diet, diuretics, betahistine – Rehabilitation (and simple counselling): all patients – Treat underlying complicating factors: e.g. orthopaedic, depression, diabetes – Do not prescribe vestibular suppressants or tranquilisers, or stop/reduce them if possible
Make sure the 'chronic dizziness' is not a gait disorder	– "Is your problem a head or a leg problem?" – Observe: Gait (including heel-to-toes gait), postural reactions and Romberg * eye movement and neurological examination: * Bilateral vestibular failure: oscillopsia, unsteady in the dark, abnormal doll's head/head thrust test (Page. . .) * Cerebellum: abnormal eyes, gait/limb ataxia * Parkinsonism: resting tremor, increased tone, akinesia * Spasticity: increased reflexes, Babinski sign * Peripheral neuropathy: distal weakness (cannot walk on heels or toes) and sensory loss * Frontal disorder/Hydrocephalus: gait 'ignition' failure, gait apraxia, shuffling

the need for rehabilitation in the context of promoting vestibular compensation. It is documented that motivated patients, actively engaged in the rehabilitation process, fare better than patients who develop an external locus of control, feeling that they have no power on their clinical outcome (de Ridder et al., 2008).

You may also need to mention that symptoms of anxiety and depression are very common in patients with dizziness – but that this doesn't imply that the symptoms are imaginary or, worse, the result of malingering.

Most patients will have psychological complications. Separating 'organic from psychogenic' or 'primary from secondary' is deeply engrained in our medical training but nowhere is more difficult to separate these than in the patient with chronic 'dizziness'. The effort to distinguish organic from psychogenic may not always be worthwhile and many patients are willing to undertake additional cognitive behavioural, or other psychotherapies if they see this as part of a global 'body and soul' effort. Some patients may need antidepressant medication, particularly on initiating the rehabilitation process. Although there is no evidence one

way or another, the consensus is that antidepressants do not interfere with vestibular compensation or rehabilitation. Indeed, some studies suggest a positive effect of antidepressants on patients with long term symptoms of dizziness (Staab, 2006).

6.4. Rehabilitation

In this chronic group rehabilitation is the most important aspect of the treatment and can be applied even in primary care settings (Yardley et al., 2004). The complexity of the rehabilitation offered to the patient will depend on two factors, 1) How much, if any, rehabilitation and advice your patient has already had, and 2) How much access you have to vestibular rehabilitation services and how good your rehabilitation team is. For the patient who has been ill advised to stay in bed and take tablets if he is dizzy, simple explanations, counselling to begin activity and a progressive reduction in medication may be enough. The other end of the spectrum is the patient who has already done a course of conventional vestibular rehabilitation but has

developed visual vertigo. This patient may need specialised vestibular rehabilitation including optic flow techniques. Although there is considerable variation according to medical practices in different regions and countries, most chronic dizzy patients are likely to be in between these two extremes. For details on the assessment and treatment aspects of rehabilitation see Horak et al., this volume.

A summary of the approach to the patient with chronic vestibular symptoms (adapted from Bronstein and Lempert, 2007) is presented in Table 3.

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