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Understanding Chronic Subjective Dizziness

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In this important edition of "Striking the Right Balance," Dr. Rick Balys discusses how to look for signs that your patient may have chronic subjective dizziness (CSD). He discusses the cause, best practice treatment strategies, and helpful tips to improve rehabilitation outcomes. As audiologists, we can help our patients who may have chronic subjective dizziness by screening for co-existing medical-psychiatric diagnoses by using tools such as the Dizziness Handicap Inventory (DHI) or the Hospital Anxiety and Depression Scale (HADS).

If you would like to be more involved in all things vestibular, please sign up for the Vestibular Special Interest Group. Sign up by simply emailing janine.verge@cdha.nshealth.ca. Also, check out the CAA Vestibular Special Interest Group's Facebook page for information on interesting links, research, and to communicate with other audiologists who are interested in vestibular testing.

In no other diagnosis do the fields of neurology and psychology become so intimately intertwined. Successful outcomes are not only dependant on your knowledge, but also on your approach, reassurance, validation of symptoms, and selective explanation of the neurophysiology. Chronic subjective dizziness (CSD), still referred to as phobic positional vertigo (PPV) in some countries, is one of the most common forms of dizziness.

CSD can be primary, which may be triggered by a stressful event or an exacerbation of a chronic state of stress and anxiety, or it can be secondary to another vestibular disorder. Any vestibular disorder or even a side effect of a medication can trigger CSD.

Symptoms

Patients present with vague descriptions of chronic dizziness, disorientation, disconnected, unsteady, floating, or rocking sensation when standing or walking which mostly resolve when sitting or lying. Symptoms are chronic and can be exacerbated by motion, visual stimulus or social stimulus (fluorescent lighting, computer screens, crowds, busy visual environments).[1] Anxiety and vegetative symptoms are common with up to 75% of patients with CSD having a past history of anxiety or depression.[2] There is also a great deal of overlap between the symptoms associated with CSD, post-concussive syndrome, migraine associated dizziness and chronic pain syndromes which suggests there may be a final common neurophysiological pathway.

Some features of chronic subjective dizziness are[3]:

1. Persistent nonvertiginous dizziness lasting 3 months or more

2. Hypersensitivity to motion stimuli, including a patient's own movement and motion of objects in the visual surround
3. Difficulty with precision visual tasks, such as reading or using a computer

Signs and Objective Testing

The vestibular examination is usually normal, although it may reveal an underlying vestibular trigger. Tests of gait and balance may look abnormal initially but they improve with distraction techniques involving difficult mental tasks such as counting backward from 100 by 3s. The visual and vestibular stimulus during the vestibular exam may trigger the "dizzy" sensation but there will be no objective findings.

Investigations

Videonystagmography testing is normal; however, it often results in an exacerbation of symptoms which can persist for several days after testing. Although patients may report difficulty hearing in crowds or with background noise, the audiogram is normal.

Posturography may reveal some clues to the diagnosis. Querner demonstrated increased sway (0.5 – 19Hz) during easy tasks which improves during difficult tasks.[4] Krafczyk also demonstrated increased sway (3.5 – 8Hz) and suggested this may be due to co-contraction of anti-gravity muscles.[5] Sway may be a natural response to any stressors – we inherently rock our children (and sometimes ourselves) when they are upset. Ohno demonstrated increased sway in healthy, asymptomatic college students who suffer from anxiety compared to control.[6]

Theories on Pathophysiology

One of the proposed mechanisms is triggering of a "threat state." There may be a separate balance control system used when there is a perceived threat. This system is normally activated when standing at a height or on unsteady ground such as ice. Holmberg was able to induce a postural sway pattern similar to that seen in CSD by applying vibration to the legs of healthy volunteers.[7] The vibration reduced the somatosensory input and, he suggests, controls shifted to high risk posture. Even the anticipation of re-applying the vibration induced the same postural sway pattern indicating higher cortical centers are involve in this reflex mechanism. He suggested "threat systems can alter balance performance depending on context and anxiety state."[7] Stahl suggests that there is a failure to readapt the "high risk postural control strategies" which were triggered by a perceived threat or abnormal stimulus for the vestibular apparatus.[2]

The predictors of CSD are high levels of anxiety at the onset of the trigger, vigilance about vestibular symptoms, catastrophic thinking about possible outcomes, and anxiety which inhibits the emergence of more flexible postural control strategies. Identifying predictors in patients presenting with other vestibular insults allows an opportunity for counselling and reassurance in hopes of avoiding secondary CSD.

Treatment

Treatment involves early diagnosis, reassurance, counselling and validation of their symptoms. When explaining the diagnosis many patients show immediate resistance if stress or anxiety is mentioned as a trigger. I prefer to normalize the symptoms using analogies they and their support person can understand such as their brains having "triggered a threat state – similar to being at the edge of a cliff." Anxiety I refer to as "an irritated or hyperactive brain" which has become very sensitive to sensory stimulus.

Since the early 1990s vestibular rehabilitation therapy (VRT) has been an established treatment option for people with vestibular disorders.[8?10] The cornerstone of an effective rehabilitation program is proper patient education. Understanding the mechanism behind the illness helps alleviate some of the mystery surrounding these frightening and often disabling symptoms. In the CSD population patient education is particularly important, as there is often an element of health anxiety and catastrophization.11 Identifying elements and patterns of CSD or the underlying otological disorder helps reduce some of the anxiety associated with symptoms and refocus attention on the positive benefits of rehab.[11]

A program of VRT would also typically include balance-retraining exercises, exercises to improve gaze-stability and repeated movement exposure to reduce motion sensitivity.[8?10] The exercise elements of a vestibular rehabilitation program are theorized to help promote central nervous system compensation for peripheral or central vestibulopathy.[9,10] As some of the early validation studies included subjects that were symptomatic for periods of time much longer than would be required for central nervous system compensation, Staab theorizes that some of the therapeutic benefits of VRT, particularly with CSD, arise from desensitization to provocative stimuli and reversal of conditioned gait responses.[2] VRT training in the CSD population must be less intense in the initial phases than patients with acute vestibulopathy and must be progressed more gradually.[2] Exercises must also include visual complexity to address the visual symptoms and take place in real-world settings to help with reintegration of activities of daily living.[2]

Many patients will require the temporary use of medications such as SSRIs (selective serotonin reuptake inhibitors), although I explain clearly we are not treating an anxiety disorder, we are using medications to get the brain out of the threat state. Staab reports on 5 open label studies on SSRIs involving over 190 patients.[2] These showed a 50% symptom reduction in 60?70%. There was a 20% dropout rate due to the side effects of the medications. Interestingly, there was a similar response in patients with or without a history of anxiety and depression suggesting they are working through a different mechanism than simply reducing anxiety.

When using any medications in this patient population it is imperative to start at very low doses and increase the dose very slowly as necessary. We have found this population to be very sensitive to any side effects of medications and very quick to give up on them. Allowing quick follow-up for assessment of side effects and reassurance is imperative to ensure patient compliance with medications.

Many patients feel they are unable to work or drive. Limiting activity, however, can further exacerbate symptoms. Work can provide distraction and play an important role in their vestibular rehabilitation. We advocate for an early but gradual return to work program once patients start to show improvement in symptoms while reassuring that they will still have some ‘bad’ days and it is not a sign of more serious disease.

Summary

CSD needs to be identified early and treated with a multidisciplinary approach to avoid long term disability and exacerbation of anxiety and depression. Our lack of a complete understanding should not inhibit our diagnosis, treatment, and compassion. I leave with a quote from Staab “the interface between vestibular and psychological mechanisms is far from clear at this point in time”…but patients are having trouble waiting for us to figure it out.

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