In a Spin

Vestibular rehabilitation therapy can train the CNS and the brain to acclimate to conflicting input.

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Patients with vestibular disorders will do anything to avoid movements that provoke episodes of dizziness. So they may look askance at clinicians who use vestibular rehabilitation therapy. This treatment actually provokes the symptoms of dizziness so the central nervous system and the brain can acclimate to asymmetrical / conflicting input. In so doing, the central vestibular pathways in the brainstem and cerebellum can correctly integrate the vestibular, visual and somatosensory signals and produce a correct response.

VRT has been in the literature for 50 years and is gaining ground as an important effective non-medical treatment for chronic vestibular disorders. Before detailing the particulars of VRT, however, I'd like to explain vestibular disorders and their effect on the body.

Patients with vestibular disorders may have had insult or damage to either the peripheral (labyrinth part of the inner ear) or central (brainstem or cerebellum) portions of the vestibular mechanism. Common inner ear disorders include labyrinthitis, vestibular neuritis, herpes zoster oticus (shingles), vestibular migraine, labyrinthine ischemia and Meniere's disease. Many of these patients may have had a short phase of acute vertigo or surgery to treat intractable inner ear disease. Once out of the acute phase, they may be left with chronic symptoms that affect spatial orientation, gaze stabilization or balance.

Vestibular disorders affect the vestibulo-ocular reflex (VOR), which controls eye movement and gaze stabilization during active head movement. These disorders also affect the vestibulospinal reflex (VSR) which influences postural stability, translated through the musculoskeletal system and antigravity muscles. Patients with vestibular disorders may present with defects in one or both symptoms of gaze stabilization problems or unsteadiness. This usually occurs when they're challenged by uneven surfaces, quick turns or with reduced vision.

For those with slow, insidious vestibular changes that culminate over years, patients will not experience vertigo, but rather a loss of equilibrium and increased unsteadiness with ambulation. This often occurs in older adults or those with various non vestibular or nonotologic-related disease processes. People who've had bilateral vestibular losses, secondary to aminoglyside toxicity, usually present with an associated complaint of oscillopsia during head movement. Oscillopsia is the term for blurred vision that occurs with head movement when a person has an uncompensated vestibular weakness.

VRT consists of systematic repetitive exercises and protocols that ameliorate motion-provoked symptoms, as well as enhance postural stability and equilibrium.

Cawthorne[1] and Cooksey[2] discussed the benefit of active eye and head movement exercise for patients who experienced labyrinthine problems. Since then, research and clinical experience have greatly advanced the scientific application of this treatment methodology. [3-7]

The underlying physiological basis for VRT focuses on the plasticity of the central nervous system. VRT doesn't't regenerate or treat the damaged vestibular end-organ itself. Instead, it works by allowing the central nervous system and the brain to acclimate or adapt to asymmetrical / conflicting input from the VOR and VSR.

Possible mechanisms include the spontaneous rebalancing of tonic activity in the vestibular nuclei, recovery of the VOR through adaptation, and the habituation effect, which lessens the response to the same stimuli over time. Theoretically, central compensation should occur within 90 days following dysfunction or loss of one of the vestibular systems. But many lesions, particularly those that occur with rapid onset, don't benefit from this compensation phenomenon. Moreover, patients are reluctant to perform active head motions that produce symptoms of dizziness. This may be a primary reason central compensation doesn't't occur in many people. Another complicating factor is that patients take drugs that suppress the peripheral vestibular or CNS function. Therefore, these medications will delay or prevent the central nervous system from relearning or adapting to asymmetrical sensory input. Dizzy patients, in their heightened state of anxiety about becoming dizzy (especially while at work or driving), rely on those pharmaceuticals to suppress their symptoms. This suppression, while allowing the person to be somewhat functional, inhibits the brain's ability to naturally compensate and fix the problem.

Patients who are in the midst of a labyrinthine storm secondary to labyrinthitis, vestibular neuronitis, or active Meniere's disease will not benefit from VRT. In cases where the disorder is still active and not yet stabilized, the brain is not allowed to recognize what it's being asked to fix. Once the condition has stabilized and the attack(s) have subsided, the brain will have a much clearer picture of the asymmetry it must acclimate to.

Gans[8], Shepard[9], Black[10], and Cohen[11], as well as others have supported and promoted vestibular rehabilitation strategies correlating with specific underlying categorization of the functional disability. Many well-meaning practitioners continue to use the 50-year old Cawthorne and 30-year-old Brandt exercises for dizzy patients, regardless of the patient's diagnosis or condition. These self-administered treatments have not been shown to have the same degree of efficacy as VRT. Clinical experience and the literature strongly indicate that vestibular rehabilitation success is related to applying the correct treatment methodology to the appropriate corresponding dysfunction, rather than leaving it to chance with a global-type protocol.

A diagnosis-based strategy approach to VRT can produce successful outcomes. These strategies link the underlying physiological changes with the patient's functional symptoms. This group of patients typically presents symptoms that are provoked with active head movement, often at a particular frequency of motion, and in a particular direction. They may not be able to ride down a street with numerous telephone poles while they look out the side window. It's common for patients to express a sensation of motion sickness while they look at certain patterns of floor tiles or wall coverings. One of the most common complaints is difficulty walking down

an aisle of a grocery store while turning the head from side-to-side, and up-and-down, while shopping.

The following three VRT approaches can be used independently or with one another, depending on the patient's needs:

Adaptation. This approach will reset or retune the VOR by repetitive activities. These activities will include those situations or movements that provoke the symptoms the patient has been trying to avoid. Activities will incorporate coordinated head and eye movement, along with head and eye movement with full ambulation. Presenting the patient with multiple, simultaneous acceleration stimuli in various planes will provide an excellent activity with a minimum of space, cost and equipment.

To promote adaptation, have the patient sit on a balance ball while he turns his head from side-to-side and reads two separate word lists. Activities that disrupt the predictability of gaze stabilization or somatosensory input will be useful. gaze stabilization exercises may progress from easy to more difficult. For example, the patient can perform side-to-side head turns while seated on a stationary chair, then perform the head turns while seated on a ball.

We can assess baseline, serial, or final performance with any technique that evaluates the VOR function. This may be as simple as testing dynamic visual acuity with a Snellen Eye chart, or as complicated as using vestibular autorotation testing, which provides a computerized analysis of eye and head velocity.

Substitution. This strategy will strengthen the weakened systems by reducing the person's dependence on the remaining system. Or, in cases in which a sensory modality is missing, substitution protocols will work to make the remaining systems more trustworthy. A patient with a weakened vestibular system is forced to make it more dominant by reducing or challenging the somatosensory input.

He can do so by standing on a trampoline. The dynamic and unpredictable surface of the trampoline forces postural stability to depend on the vestibular and visual sensory modalities. The visual sense could then be eliminated or disrupted by having the patient close his eyes or watch a moving visual stimulus while maintaining his balance. To maintain balance, the patient would have to depend on the vestibular system.

To evaluate the patient's performance, we can use tests of postural stability with dynamic surface and absent vision. A simple version of this is Horak and Schumway-Cook's classic Clinical Test of Sensory Integration of Balance. A more complex test would be the computerized dynamic posturography, used by NASA to evaluate balance function of returning shuttle astronauts.

Canalith Repositioning and Liberatory Maneuvers. These strategies treat the No. 1 cause of vertigo in older adults - Benign Paroxysmal Positional Vertigo (BPPV). Diagnosing and treating patients with BPPV depends on the therapist's skill in understanding the patient's eye movement associated with nystagmus. Nystagmus is the involuntary eye movement that is the cardinal clinical indicator of BPPV. Nystagmus and the patient's report of vertigo will usually be matched in time and intensity.

Because of the transitory nature of the nystagmus – it lasts less than 20 seconds – the ability to capture the nystagmus on videotape is invaluable. New technologies using infrared video cameras inserted in goggles allows therapists to review the tape and document treatment efficacy. If the treatment is successful, the patient will not experience nystagmus or vertigo. This technology is simple and requires only a TV/VCR. Clinicians must make a differential diagnosis in BPPV, identifying the involved canal, as well as the canalithiasis vs. cupulolithiasis variant.

Therapy models of VRT also exist, one of which is self-directed exercise at home. Each program is individually designed for patients (based on their test results) to include situations that bring on their symptoms. This approach is most commonly used with people who don't require supervision during exercising. Best results occur when they spend 20-30 minutes per session, two to three times daily. Most patients report a significant reduction in symptoms in two to four weeks.

Clinician-directed exercises are also available for patients whose symptoms are severe and who require supervision during exercises. Therapy sessions usually include using various vestibular therapy equipment: balance balls, trampolines, mats or dense foam. More sophisticated systems use computerized tests of balance function, which provide quantitative data on limits of stability or rhythmic weight shift. Fall prevention is also emphasized, particularly with older patients. Typically, the patient participates in one or two 60-minute session per week, with an average of seven to twelve sessions. As the patient progresses, he's given some self-directed, home exercises to quicken improvement.

Fifty years or literature, clinical experience, and a growing patient acceptance has solidified VRT as an important and efficacious non-medical treatment for chronic vestibular disorders.

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