American Physical Therapy Association/Neurology Section

Vestibular Rehabilitation SIG

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Vestibular Rehabilitation SIG Officers:

Message from the Chair

Susan L. Whitney,
PhD, PT, NCS, ATC, FAPTA
VR SIG Chair

Dear Vestibular SIG members,

I would like to thank all of the people who helped to make CSM a success in California. Your vestibular elected and appointed members did a great job with the programming at the meeting. Fun was had by all and the learning opportunities were plentiful for our specialty area. The new Vestibular SIG shirt was launched and the SIG fact sheets were seen for the first time by the members. We hope to have them online and available to members soon after CSM. Please keep checking at www.neuropt.org. There were over 500 people Thursday night at the SIG meeting.

Thanks go to Micro Medical Technologies who donated the video goggles for our SIG meeting in San Diego. Also thanks go to all those who donated books including Linda Luxon (Textbook of Audiological Medicine), Joseph Furman (Vestibular Disorders- A case study approach), Alan Desmond (Vestibular Function Evaluation and Treatment), Gary Jacobson and Neil Shepard (Balance function assessment and management), Neurotology (Furman and Whitney), VHI balance and vestibular kits, BPPV teaching CDs from Bernard Cohen, and Susan Herdman (Vestibular Rehabilitation, 3rd edition).

I would like to take this opportunity to thank Sharan Zirges for all of her help over the last 5 years as newsletter editor and then as newsletter co-editor her last year. She made a wonderful contribution to the SIG and treated the content in the newsletter with great respect. Sharan needed to move on because of family commitments. She is already missed and we hope that as opportunities arise, that she can rejoin us later in her career. Her laughter and positive attitude were a great addition to the Vestibular SIG leadership.

APTA has just launched the first fact sheet about what is vertigo, which was written by Melissa S. Bloom, PT, DPT. Congratulations to Melissa for writing the first one to be published by the APTA. It is located at www.moveforwardpt.com. APTA has also published a new document related to FAQs for insurance companies about vestibular physical therapy. The SIG also provided a document that reviews the literature related to the need for acute intervention for persons with vestibular disorders. The URL is: **http://www.apta.org/AM/Template.cfm?Section=FAQs5&Template=/TaggedPage/TaggedPageDisplay.cfm&TPLID=355&ContentID=49058**

The advanced Neurology Section Vestibular course will be offered in San Diego September 11 7 12 and also in Pittsburgh October 9 & 10, 2010. We hope to see some of you there. There are 12 of us who are working on the course and content.

"Balance Assessment in Different Practice Settings" Balance and Falls SIG Meeting

Becky Olson-Kellogg, PT, DPT, GCS
VR SIG Nominating Committee &
Abstract of the Week Committee

Following the annual business meeting for the Balance & Falls SIG, Linda Csiza, PT, DSc, NCS, Melissa Fong, PT, DPT, NCS, Tammie Keller Johnson, MS, DPT, and Jennifer Ellis, MS, PT presented programming on assessing patients balance across a variety of practice settings. Linda Csiza served as the moderator (in Leslie Allison's absence), and the other 3 panel members represented various practice settings.

Tammie Keller Johnson opened the presentation representing inpatient rehab facilities, which is her area of clinical practice. One of the greatest challenges for physical therapists in this area of rehab is the declining length of stays for patients in these settings. Certainly this impacts the outcomes obtained prior to moving onto the next stage in the rehab process. Tammie presented a case report of a 45yo male attorney who suffered a MCA stroke. Balance assessments that Tammie utilized with this gentleman included the FIM, TUG, FGA, and Gait Velocity. Overall, in the inpatient acute rehab setting, Tammie highlighted that a common problem with balance assessments is experiencing floor effects. So, it is important for therapists practicing in this area to select an assessment tool that is not too advanced for patients at this point in their rehab.

Melissa Fong continued the presentations addressing the outpatient rehab settings. She highlighted that following a literature search, it is evident that there is currently no standardized core set of balance outcome

Melissa went on to highlight 6 considerations for selecting a balance assessment for all practice settings (regardless of what the setting is), emphasizing that all of these criteria must be considered when selecting an outcome measure:

1. Appropriateness: Does the outcome measure match the specific purpose of the assessment?
2. Reliability: It is always our responsibility to select a reliable tool, regardless of the practice setting
3. Validity: Again, the practice setting should not influence this.
4. Interpretability: How meaningful is the score? Are there norms for comparison? Again, the setting should not matter.
5. Responsiveness: How sensitive is the measure to change over time? This area is likely most influenced by practice setting.
6. Acceptability & Feasibility: What is the burden on the patient / staff / facility when implementing this outcome measure?

Jennifer Ellis represented the homecare setting. She highlighted the one criteria that is most influenced by the homecare setting is whether the outcome measure is practical and purposeful. Issues of practicality are encountered by homecare therapists on a daily basis, and this is certainly true in the selection of outcome measures also. Jennifer reinforced that one balance assessment doesn't tell us everything we need to know; instead each test just gives us a slice of the pie. The BESTest has been a beneficial tool for homecare therapists as it takes a systems approach, which in the end, provides the therapist with more comprehensive information about that patient's balance.

Mark your calendars!!!

CSM 2011 is returning to NEW ORLEANS!!!!!

February 9-13, 2011

CERVICOGENIC DIZZINESS:
PERSPECTIVES ON EVALUATION AND TREATMENT

Kenda Fuller, PT, NCS
Nominating Committee, Chair

CSM 2010 Vestibular SIG meeting Program
Speaker Rob Landel, PT, DPT, OCS Associate Professor of Clinical Physical Therapy Division of Biokinesiology and Physical Therapy University of Southern California

This was clearly a topic of interest as evidenced by the large number of attendees, from both the Neurology Section and the Section on Orthopedics. Dr. Landel's basic premise is: Patients with cervical dysfunction can present with complaints of dizziness and disequilibrium associated with impairments of the cervical spine. Traditionally considered a diagnosis of exclusion, cervicogenic dizziness rarely presents in isolation, making recognition and treatment of this condition challenging. Current practice suggests that a combination of vestibular rehabilitation, balance retraining and manual therapy techniques may be effective in treating persons with this condition.

The main idea is that cervicogenic dizziness results from a sensory mismatch between somatosensory information from the cervical spine and input from the visual and vestibular systems. In the history the dizziness is often vaguely described. Usually it is not vertigo: there is no reported spinning, but rather a feeling of unsteadiness or imbalance. The report may be floating, spacey, feeling off. Important in the report of lightheadedness is that the complaint is not pre-syncopal. Often there is a complaint of difficulty concentrating and focusing on a single task, or multitasking. Another common complaint that can be examined is oscillopsia resulting in poor smooth pursuit tracking. Concurrent complaint of neck pain occurs with an onset that is temporally related. It is also common that pain and dizziness severity co-vary.

Cervicogenic dizziness is typically described as a diagnosis of exclusion; central causes should be ruled out including abnormal oculomotor tests and there should be no resting nystagmus or abnormal smooth pursuits, or saccades. In addition, there should be no other signs of CNS disorder or UMN signs. Unilateral or bilateral peripheral vestibular causes should be ruled out, including passive head shake, head thrust, Hallpike.

The major implicating findings on examination are ROM impairments, cervical muscle weakness and fatigue and decreased postural control, easily noted as increased sway on CTSIB, SLS, tandem stance. Often there are oculomotor abnormalities when the head is placed in different positions resulting in a positive smooth pursuit neck torsion test and an abnormal Dynamic Visual Acuity test (DVA).

Impaired cervical muscle function (extensors as well as flexors), including strength, control, and endurance, are often seen. Impaired cervical kinesthetic ability is manifested by increased joint position error. We must realize that the upper cervical spine is high in muscle spindles and there is direct access to vestibular nucleus and central cervical nucleus. Deep cervical flexors are poorly recruited compared to superficial flexors. Cervical extensors are more susceptible to fatigue in some patients. Muscle fatigue modifies discharge of sensory receptors and affects proprioception. It has been found that those who demonstrate increased fatigue also demonstrate increased postural sway. Therefore, neck weakness, among other impairments (ROM, segmental mobility, soft tissue mobility and flexibility) should be examined in patients with a suspected cervical component to their dizziness. The therapist should look for increased joint position errors. Examination should include the determining the status of head stabilized while body rotates under stable head compared to symptoms when the head and neck rotate together. Other findings that point to possible involvement of the somatosensory systems are hypersensitivity to temperature, or post-traumatic stress.

In conclusion, Dr. Landel concluded that in patients with complaints of dizziness and imbalance, in addition to standard tests for central and peripheral vestibular dysfunction, and the various tests for postural control, should be evaluated for cervical impairments, neck joint repositioning and neck muscle endurance. The overlap of oculomotor function should also be considered.

We thank Dr. Landel for his contribution to the success of the Vestibular Rehabilitation Special Interest Group presentations at APTA/Combined Sections Meeting!

Special Thank You!!

We would like to send a special thank you to Sharan Zirges for all her hard work and dedication to the Vestibular SIG over the past 5 years as newsletter editor. We greatly appreciated all your efforts and look forward to working with you again in the future in new ways!!

Mild head injury and dizziness

A Summary from the Multisection CSM 2010 Concussion programming

By Susan L. Whitney,
PhD, PT, NCS, ATC, FAPTA
VR SIG Chair

Persons with mild head injury often experience dizziness. Twenty-six percent of 100 patients with post-concussive syndrome report dizziness on the Rivermead symptom scale after 3 months.[1] Another group of authors reported that 32% of 141 patients with mild TBI report dizziness after 5 years.[2] It is very common for post concussed persons to find their way to a physical therapist that specializes in vestibular disorders because of their dizziness complaints.

Often as physical therapists we see a cascade of symptoms after mild TBI. Various investigators classified the symptoms into somatic, emotional, cognitive and also sleep disturbance.[3] The somatic symptoms include visual concerns, dizziness, headaches, light sensitivity, balance disturbance, and nausea. Any vestibular therapist would state that this is what we often see in a person with vestibular disease. Emotional symptoms include increased irritability, sadness and being nervous. Cognitive symptoms typically experienced include difficulties with memory, the perception of "fogginess", fatigue, slowed cognition, and attentional deficits. Sleep disturbance has been described as experiencing too much or too little sleep. Patients also often report having difficulty reading and that if they read too long, they will develop a headache.

Mild head injury results in neuropathological changes that largely reflect a functional disturbance rather than a structural one. Concussion may or may not involve loss of consciousness and the resolution of symptoms follows a sequential course in almost all people. Mild TBI is typically associated with grossly normal structural neuroimaging studies, yet patients have significant functional sequale from the concussion. Post concussion disorders are not just for athletes- the problems noted above are also in evidence after auto accidents, falls, and even after abuse.

Recovery from concussion appears to be somewhat dependent on age. Younger people in their teens often have a longer recovery period than people in their twenties.[4 5] Generally, 90% of persons post mild head injury have recovered within 4 weeks.[6] Lovell et al recently studied 200 high school athletes who were tested with ImPACT (a neuropsychological test) within 7 days of concussion and also when they were clinically recovered while undergoing fMRI.[7] Hyperactivation within the brain predicted clinical recovery time and the resolution of the hyperactivation correlated with recovery on the computerized ImPACT testing.[7]

The team approach to management of persons post concussion seems to be advised. Professions typically involved on the team include the physician (general practice, physical medicine and rehabilitation, neuro-ophthalmologist, neuro-otologist) , neuropsychologist, athletic trainer, and the physical therapist. Generally, physical therapy is only consulted if the person is not getting better and has complaints of dizziness and imbalance.

Post concussion migraine is a common complaint, even in people who have no history of prior migraine. To be diagnosed with migraine, one must meet the International Headache Society criteria for migraine (headache classification committee of the IHS) [Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Cephalalgia 1988 8: 1-96.] Neuhasuer et al[8] developed criteria for migraine dizziness which were slightly modified by Marcus et al[9, 10] that provide a structured interview process that can guide the physical therapist in indentifying if the person post concussion has met criteria for either migraine or migraine dizziness. Often pharmacologic intervention is the only way to get the headaches manageable. Use of anti-migraine drugs is often difficult for families to understand, especially in younger children, but can reduce the person's dizziness and headache symptoms. Headache may or may not be associated with dizziness in persons with migraine.[8]

Mild head injury and dizziness (cont.)

As vestibular physical therapists, it is not uncommon to see convergence spasm or convergence insufficiency during the oculomotor examination of the patient. Convergence spasm results in the eyes moving medially and papillary constriction. Weber et al[11] have reported convergence spasm as a result of midbrain compression. In the past, convergence spasm has been related to inorganic causes.[12] Little is known about convergence spasm or insufficiency, yet it is often demonstrated in children and adults post mild TBI.

One of the key aspects to recovery of the person post concussion is "brain rest". It is imperative that persons not increase their headache, dizziness, sleep problems, or other co-morbid symptoms. Our experience suggests that if headache, dizziness, or sleep problems persist, the person does not get better. Since mild TBI sets off a metabolic cascade, it is important that symptoms be minimized. This almost always means not working or going to school until symptoms of headache, dizziness, confusion , lack of sleep, restlessness and others have almost completely resolved. If return to activity occurs too quickly, patients typically regress and get worse. Gradual return to work and play is the norm rather than the exception in persons who are struggling post concussion to resume activities. Playing computer games and texting can also contribute to symptoms, so these activities are reduced also as part of their physical therapy education program. Most families to not understand what activities cause brain overload, so it is important to be very clear with what is and what is not acceptable.

As vestibular physical therapists, generally we are referred patients who complain of dizziness and balance complaints that have not resolved. When treating these patients, intensive exercise is not advised. Careful progression of the exercise program over time appears to be effective in relieving symptoms and improving postural control.[13]

References

1. Ingebrigtsen T, Waterloo K, Marup-Jensen S, Attner E, Romner B. Quantification of post-concussion symptoms 3 months after minor head injury in 100 consecutive patients. J Neurol 1998;245(9):609-12.

References (cont.)

2. Masson F, Salmi LR, Maurette P, Dartigues JF, Vecsey J, Garros B et al. [Characteristics of head trauma in children: epidemiology and a 5-year follow-up]. Arch Pediatr 1996;3(7):651-60.
3. Iverson GL, Gaetz M, Lovell MR, Collins MW. Relation between subjective fogginess and neuropsychological testing following concussion. J Int Neuropsychol Soc 2004;10(6):904-6.
4. Iverson GL, Brooks BL, Collins MW, Lovell MR. Tracking neuropsychological recovery following concussion in sport. Brain Inj 2006;20(3):245-52.
5. Echemendia RJ, Putukian M, Mackin RS, Julian L, Shoss N. Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. Clin J Sport Med 2001;11(1):23-31.
6. Collins M, Lovell MR, Iverson GL, Ide T, Maroon J. Examining concussion rates and return to play in high school football players wearing newer helmet technology: a three-year prospective cohort study. Neurosurgery 2006;58(2):275-86; discussion -86.
7. Lovell MR, Pardini JE, Welling J, Collins MW, Bakal J, Lazar N et al. Functional brain abnormalities are related to clinical recovery and time to return-to-play in athletes. Neurosurgery 2007;61(2):352-9; discussion 9-60.
8. Neuhauser HK, Radtke A, von Brevern M, Feldmann M, Lezius F, Ziese T et al. Migrainous vertigo: prevalence and impact on quality of life. Neurology 2006;67(6):1028-33.
9. Marcus DA, Kapelewski C, Jacob RG, Rudy TE, Furman JM. Validation of a brief nurse-administered migraine assessment tool. Headache 2004;44(4):328-32.
10. Marcus DA, Kapelewski C, Rudy TE, Jacob RG, Furman JM. Diagnosis of migrainous vertigo: validity of a structured interview. Med Sci Monit 2004;10(5):CR197-201.
11. Weber KP, Thurtell MJ, Halmagyi GM. Teaching NeuroImage: Convergence spasm associated with midbrain compression by cerebral aneurysm. Neurology 2008;70(15):49-50.
12. Sarkies NJ, Sanders MD. Convergence spasm. Trans Ophthalmol Soc U K 1985;104 (Pt 7):782-6.
13. Alsalaheen B, Mucha A, Morris LM, Whitney SL, Furman JM, Camilo-Reddy CE et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. Journal of Neurologic Physical Therapy 2010;34:87-93.

Vestibular SIG Roundtable Discussion CSM 2010
"Building an Ideal Dizziness and Balance Program"

Julie Tilson, PT, DPT, MS, NCS
Vestibular SIG Vice Chair

Over 50 therapists attended the Vestibular SIG Roundtable discussion "Building an Ideal Dizziness and Balance Program" to learn and share ideas for vestibular rehabilitation program development. The discussion was lead by moderator Colin Grove (University of Wisconsin Hospital & Clinics) and panelists: Janene Holmberg (Intermountain Hearing and Balance Center), Janet Callagan (Massachusetts General Hospital), Sapan Palkhiwala (Complete Balance Solutions Institute for Rehabilitation), and Kenda Fuller (South Valley PT). The group discussed 3 main questions from the moderator. Answers from panelists and participations are summarized below.

Question 1:
What are the most important elements of starting a successful dizziness and balance program?

Build strong relationships with physicians in your community.

1. Invest the time to build professional relationships.
2. Be and look professional in every way: your dress, your marketing/educational materials, and your written and oral communication.
3. Approach potential referral sources with the confidence that you can help them manage their patients with dizziness and disequilibrium.
4. Don't limit yourself to just a few physicians. Build relationships with as many as possible and look beyond Otolaryngologists and Neurologists (e.g. Internists, Primary Care, Opthamologists, Gerontologists, care providers at skilled nursing facilities, board and care, and wellness facilities)
5. Educate physicians about the value of vestibular rehabilitation.
    a) Share summaries of the literature supporting VR (e.g. Hillier 2007 Cochrane Systematic Review: http://mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD005397/frame.html)
    b) Emphasize the quality of care that you provide and back it up with data.
    c) Present at Grand Rounds
    d) Meet with frequent referrers monthly to review patient progress

Question 2:
Working on a 'shoe string'. If you could have only the equipment that you could fit in a single duffle bag, what would it be? (listed in random order)

Panelists and attendees emphasized the benefit of being able to provide quality Vestibular Rehabilitation on a tight budget. They emphasized the therapist training (not equipment) was the most important element of good practice. Here are some of low budget items that panelists and attendees reported they would pack in their VR on a shoe string duffle bag:

1. Foam (for balance training)
2. 2x4 plank (for balance training)
3. Tuning fork (for testing peripheral sensation)
4. Eye chart (for testing dynamic visual acuity)
5. Metronome (for teaching exercises)
6. Frenzel lenses (for eye movement examination)
7. Vestibular model (for teaching about BPPV)
8. Brock's string (for testing convergence and divergence)

Question 3:
If you had a blank check and could buy any piece of equipment possible, what would you buy?

Panelists and attendees admitted that although VR can be practiced on a shoe string, fancy toys are fun and can enhance the options available to patients. If the discussion group had an endless supply of cash, here are some of the wish list big ticket items mentioned during the discussion:

1. Proprio 5000
2. Infrared/video Frenzel Goggles
3. Fall prevention harness systems
4. Epley Omniax System

(Note: The equipment lists are in no way endorsed by the Vestibular SIG, Neurology Section, or APTA)

Overall, the session provided an excellent opportunity to share ideas, challenges, and solutions for building an ideal dizziness and balance program. For those you were not able to join us – we hope that this provides you with a few fresh ideas for success!

Clinical assessment of the Oculomotor Systems

Part two of a three-part series on Oculomotor control

Michael Shubert, PhD, PT
Assistant Professor
Johns Hopkins Medicine

Knowing the integrity of the oculomotor system in patients with reports of dizziness and imbalance is useful to assist in identifying the site of lesion and formulating a home exercise program with realistic goals. In this article, clinical assessment of the oculomotor systems will be covered, to include how the tests should be performed and which abnormal signs indicate pathology. This article does not include all aspects of the vestibular oculomotor exam (i.e. integrity of the peripheral labyrinth by tragal pressure) but instead focuses on oculomotor control systems.

Five major oculomotor control systems should be considered as part of the clinical examination:

1. Gaze stability – the ability to maintain stable gaze without the generation of other eye movements (such as jerk nystagmus) while looking straight ahead, left, right, up and down;
2. Smooth pursuit – the ability to move the eyes with smooth, continuous motions in order to follow the movement of a target of interest and maintain the moving image on the fovea (the most sensitive part of the retina);
3. Saccade – the ability to move the eyes in a single, rapid movement to refixate a target of interest onto the fovea;
4. Optokinetic nystagmus – reflexive eye movements composed of jerk nystagmus (slow and quick eye rotations) while viewing moving objects that occupy 80% or more of the visual field of view.
5. Vestibulo-ocular reflex (VOR) – the ability to keep the eyes stable in space during a head motion by generating an eye rotation of similar magnitude but opposite direction from the head motion.

Gaze stability (i.e. fixation in center, left, right, up & down) evaluates the ability to hold the eyes in a fixed direction without drifting off the target. Gaze fixation is a function of the central nervous system and enables us the ability to maintain foveal fixation of a still target of interest, without the production of eye movement. However, pathology within the peripheral vestibular system as well as the oculomotor control centers may cause gaze instability. Common signs of abnormal gaze stability include nystagmus or the presence of other repeating eye movements (i.e. saccades). When gaze nystagmus is direction fixed, enhanced in the dark (target removed), and increased when looking in the direction of the fast component - the peripheral vestibular labyrinth is considered the source of pathology. In this case, the nystagmus will always beat towards the more active neural side, which typically is away from the affected side in vestibular neuronitis or towards the affected side in presentations of irritative Ménières disease.

When gaze nystagmus is of central origin, the dominant abnormalities often are pure vertical or pure torsional nystagmus, and direction changing nystagmus. Sites of lesion in central evoked gaze nystagmus depend on whether the eye movements are horizontal or vertical. The nucleus propositus hypoglossi (medulla) and medial vestibular nuclei, (medulla/pons) are the primary neural substrate mediating horizontal gaze, while the interstitial nucleus of Cajal (midbrain) is the prime neural substrate for maintaining vertical (up or down) gaze. While these are the primary sources for maintaining gaze, the cerebellum and brainstem also participate in gaze stability.

Tests of gaze stability should be done in the light and dark. The subject is simply asked to look straight, then 30 deg left/right/up/down. In the clinic, the target (finger tip/pen tip – make sure the target is a discrete point, not your whole finger) should be presented at a distance of 20 – 24 inches from the subjects head. To elicit a 30 deg eye rotation at a distance of 20 inches, the target must be moved ± 11.5 inches. At a distance of 24 inches, the target must be moved ± ~14 inches. The examiner should have the patient keep their eyes fixed approximately 10 seconds, or long enough to be confident of the exam's finding. After each movement of the eyes, the patient should be guided back to center to check for rebound nystagmus (central finding where the eyes will beat in the direction of the last eye movement).

Clinical assessment of the Oculomotor Systems

(continued)

Smooth pursuit is very sensitive to identify pathology within the multiple central nervous system pathways used for its generation; however it is not good for localizing where that pathology may be. Central pathways involved in pursuit include multiple cortical areas, basal ganglion regions, brainstem (pons), cerebellum (paraflocculus, dorsal vermis), and oculomotor nuclei. Smooth pursuit is tested in clinic by having your patient follow a target that is moving continuously from side to side and up and down. Many variations of this test are possible (i.e. diagonal, 'H' pattern etc…). The patient should have no difficulty keeping their eyes on the moving target. Abnormal findings include asymmetric / dysconjugate eye movement or saccadic interruptions. Interruptions of pursuit may be unidirectional or bidirectional. Smooth pursuit is very sensitive to age. If the target is moved too quickly, the brain may intentionally recruit saccades in attempt to fixate, which reflects normal function but poor technique. It is therefore important to provide a target velocity that is not too quick. Typically, testing can be done using a frequency range (.2 to .8 Hz) or a velocity range (20 - 40d/s) with the higher units being more difficult. For example, to elicit a 30d/s eye rotation, follow the guidelines outlined in the above gaze stability section, and move the target along the specified distance (11.5 or 14") in 1 second.

Saccade testing is not as sensitive as smooth pursuit for identifying CNS pathology. Horizontal saccades are generated from burst neurons in a region of the pons known as the paramedian pontine reticular formation (PPRF). Vertical and torsional saccades occur from burst neurons in a region of the midbrain known as the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF). As with the pursuit system, many other cortical areas are involved in saccade generation; superior colliculus, cerebellum, frontal lobe, basal ganglia, and thalamus. When testing the saccade system, the clinician should consider these saccade metrics - velocity, onset, and accuracy. Fatigue and certain medications can affect each metric, as do pathologies affecting either the PPRF or riMLF. However, common pathologies that affect saccade velocity include: slow saccades in early stages of mysasthenia gravis which can change to fast saccades in later stages of the disease, cerebrum and cerebellar lesions, and internuclear ophthalmoplegia (INO) due to multiple sclerosis. Common pathologies affecting saccade onset include visual acuity deficits, Parkinson's disease, and other basal ganglia diseases. Common pathologies affecting saccade accuracy (overshoot or undershoot of the target) include; overshoot – cerebellar lesion, ipsilateral saccades in dorsalateral medullary infarct, and undershoot – ipsilateral cerebellar or brainstem lesions, contralateral saccades in dorsolateral medullary infarct, mysasthenia gravis, and cerebral lesions.

Clinical testing of saccades is done by presenting a still target that is ± 30d to the left, right, up, down from the center of the patient. The subject should be able to move both eyes together to the target within 3 saccades. Overshooting the target is considered abnormal.

Optokinetic nystagmus (OKN), originally called train nystagmus for the created jerk nystagmus noticed in train passengers, normally occurs when 80% or more of the subject's visual field is occupied by moving patterns of repeated objects. This reflex involves both smooth pursuit and saccade oculomotor systems generated by a combination of foveal and peripheral retinal stimulation. Many authors suggest OKN testing is most useful to do when abnormalities are seen during pursuit or saccade testing. OKN is best quantified with laboratory testing, but can be qualitatively assessed with a handheld OKN drum (alternating black and white stripes) or using fabric with similar striped patterns. The patterns should be moved horizontally and then vertically across the subject's field of view. The velocity of the moving target should be within the smooth pursuit system, between 20 - 40d/s is typical. Abnormalities in OKN include its absence, dysconjugacy of eye rotation, and asymmetry between vertical and horizontal OKN.

The VOR is tested primarily as a function of the peripheral vestibular labyrinth.

Clinical assessment of the Oculomotor Systems

(continued)

Recent evidence suggests that as many as 7% of cerebellar CVA seen through the emergency department may have an abnormal head impulse test (Kattah et al 2009). However, in these patients other oculomotor anomalies co-exist. The angular VOR (aVOR) is the most sensitive VOR test we can measure in clinic. Because the aVOR is uniquely created to stabilize gaze at head velocities above 1 Hz, rapid head rotations are necessary to assess its integrity. Each of the six semicircular canals (SCC) can be assessed with the head impulse test (HIT). The subjects head should be firmly grasped then rotated with a low amplitude (~ 15d), moderate velocity (~ 200d/s) and high acceleration (~ 3000d/s/s). For each SCC HIT, the examiner needs to ensure the head is rotated in the plane of the SCC (Cremer et al 1998). The vertical SCC are more difficult to test and can be done 1 of 2 ways: A. apply the impulse from neck neutral in the diagonal plane of the SCC or B. rotate the head 45d to one side, maintain this position and then rotate the head in pitch (down for assessing the anterior SCC and up for assessing the contralateral posterior SCC). Repeat for the other side. When testing using method B, recall the anterior SCC are mated with their contralateral posterior SCC (right anterior/left posterior etc…). Therefore separate rotations for up (posterior SCC) and down (anterior SCC) should be made. Abnormality includes a corrective saccade back to the target, which occurs after the head has stopped rotating.

The central VOR pathways can also be assessed using the horizontal head shake nystagmus test. For this test, the patient must be in the dark and the head is oscillated for 20 cycles at 2 Hz. Upon stopping the test, no nystagmus should be present. The presence of horizontal nystagmus suggests asymmetrical peripheral vestibular afference to the brainstem vestibular nuclei. The presence of vertical nystagmus following horizontal head shaking suggests abnormal processing of the vestibular signal and represents a positive central vestibular pathway sign.

The clinical oculomotor exam is crucial to the physical therapist examining patients with reports of dizziness and imbalance.

For those interested to learn about laboratory oculomotor testing or desire a more in depth review of clinical oculomotor testing, readers are encourage to see Jacobsen and Shepard 2008, and Leigh and Zee 2009.

References:

1. Cremer PD, Halmagyi GM, Aw ST, Curthoys IS, McGarvie LA, Todd MJ, Black RA, Hannigan IP. Semicircular canal plane head impulses detect absent function of individual semicircular canals. Brain. 1998 Apr; 121 (Pt 4):699-716.
2. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. 2009. Nov; 40(11):3504-10.
3. Jacobson GP and Shepard N. Balance Function Assessment and Management. Plural Publishing, San Diego, CA. 2008.
4. Leigh RJ and Zee DS. The Neurology of Eye Movements: Book-and-DVD Package (Contemporary Neurology Series, 70). Oxford University Press New York, NY. 2006.

Patient Education Fact Sheet are here

Check out the Vestibular SIG website to find the Patient Education Fact Sheets on a variety of topics relating to vestibular disorders and vestibular rehabilitation. There are 29 fact sheets posted on the website.

Coming soon! Physician Education fact sheets. Stay tuned and visit the Vestibular SIG website frequently to see updates to the resources available to members!
http://www.neuropt.org/go/special-interest-groups/vestibular-rehabilitation

Efficacy of vestibular rehabilitation on chronic vestibular dysfunction: a Journal Article Review

Debbie Struiksma, PT, NCS
VR Nominating Committee

Citation: Topuz O, Topuz B, Ardic FN, Sarhus M, Ogmen G, Ardic F. Efficacy of vestibular rehabilitation on chronic unilateral vestibular dysfunction. *Clinical Rehabilitation* 2004:18; 76-83.

One of the biggest issues facing physical therapists in today's health care environment is the ability to demonstrate the effectiveness of our intervention strategies. Third-party payers, Medicare and patients are all interested in the length of intervention and which interventions get the best outcomes in order to stretch health care dollars. The question of efficacy is a valid question and one that should be addressed. However, it can be challenging to present documented evidence that gives specifics to the intervention strategies used, especially in vestibular rehabilitation. Thankfully over recent years there has been a growing body of evidence to demonstrate the effectiveness of vestibular rehabilitation. Researchers from Pamukkale University, School of Medicine, Turkey have published an article in *Clinical Rehabilitation* to add to the literature. The objective of this study was to assess the efficacy of vestibular rehabilitation exercises on patients with chronic unilateral vestibular dysfunction.

The investigators studied 112 patients (77 women and 35 men) whose mean age was 44 and had a diagnosis of uncompensated unilateral vestibular dysfunction (UVL) confirmed by Caloric testing and without central vestibular lesion, BPPV, Meniere's disease, or polyneuropathy. The patients were treated with an 8 week course of staged vestibular rehabilitation that included components of Cawthorne-Cooksey and Norre exercises. Each exercise was chosen by the investigators to address the vestibulo-ocular system, the somatosensory system, and the vestibulo-spinal system and each exercise was repeated 5 times during each session. The exercises are listed in Table 1.

During the first 2 of the 8 weeks of rehab, the patients attended 30-45 minute exercise sessions, 5 days per week for supervision with the exercises. Subsequently, the patients continued to perform the exercises at home with written home exercise instructions on a daily basis for the remaining 6 weeks.

All patients were examined on the 1st, 15th and 60th days of interventions at which time the Dizziness Handicap Inventory (DHI) and visual analogue scale (VAS) were completed. These were used to quantify the effects of the vestibular exercises on recovery. The patients were divided into groups in order to study the effect of age, gender, and disability level on therapy.

The investigators found no statistical difference between age, gender and disability level groups in response to therapy. There was a 29.21 point reduction on the DHI and 3.85 point reduction on the VAS after the first 2 weeks of treatment. There was no significant difference in VAS and DHI scores between the 2nd week and the end of treatment. The physical subscore of the DHI deteriorated in patients between days 15 and 60. The investigators concluded that there was a fast recovery in the supervised exercise session, whereas there was no significant difference in the home exercise session. They also suggest that either supervised exercise is better than home exercise or that 10 supervised sessions are sufficient to get the end result.

This study did an adequate job of demonstrating the improvements made in patients with unilateral vestibular loss using vestibular rehabilitation as demonstrated by DHI and VAS scores. The study may have been able to demonstrate improved functional gains in this population if an observed functional measurement such as the Dynamic Gait Index (DGI) was used. A confidence score, such as the Activities-specific Balance Confidence (ABC) Scale, may have also been an appropriate subjective assessment for measurement of functional abilities. Using one of these measurements could have possibly explained the deterioration of physical subscores on the DHI between days 15 and 60.

The exercises used for intervention addressed various elements of impairments in the UVL population. However, the exercise program was not progressed to more challenging activities after the initial 10 days of supervised intervention. It is possible that there may have been a different result in physical subscores if VOR exercises were progressed to more challenging positions, such as with gait or in complex visual environments, that would take into account the context specificity of the VOR instead of just the sitting position.

The investigators did a nice job of looking at differences between age, gender and disability level in response to therapy. It should also be noted that the frequency of the intervention strategies used did not follow a plan that is typically used in the United States outpatient rehabilitation system for this population. The conclusion that "10 supervised sessions are sufficient to get to the end result" may be a premature conclusion from this data as it lacks demonstration of an observed functional improvement in the population.

Journal Article Review: Table 1—
Exercises used in vestibular rehabilitation programs

- Movement of the eyes slowly and then fast in all directions in supine and sitting positions.
- Movement of the eyes quickly in horizontal and vertical directions, and then back to the center point slowly.
- Tossing a ball from one hand to the other while following the ball with their eyes in standing.
- Movement of the head horizontally and vertically in supine.
- Movement of the body to left and right recumbent positions and holding each position 20 seconds.
- Movement from supine to sitting and returning to supine.
- Vestibular Ocular Reflex (VOR) exercise in sitting.
- Movement of the head with eyes closed in sitting.
- In sitting, picking up an object from the floor on the right and then on the left.
- Movement from sitting to standing and returning to sitting.
- Standing with feet apart, close together, in tandem, on one leg with their eyes open and closed. All of the positions were repeated on a foam exercise mat.
- Walking forward backward, sideways and along a line, on heel and tiptoes with their eyes open and closed, on both firm and foam surfaces.
- Walking with head turns in yaw and pitch movements on both firm and foam surfaces.
- Sitting in a rotating chair while fixating on a point and moving the trunk while the head position was maintained.

Fun at CSM 2010!!!
Hope you can join us next year in New Orleans

Vertigo-go Dinner at Spaghetti Factory

Vertigo-go Queens—Sue Whitney 2009 and Michelle Gutierrez 2010

Dr. Rob Landel presenting at the Vestibular SIG meeting

Vertigo-go Moo Moo