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Loma Linda University
School of Allied Health Professions
in conjunction with the
Faculty of Graduate Studies

The Effects of Neck Muscle Fatigue on the Vestibulo-Ocular Reflex

By

Amer Al Saif

A Dissertation submitted in partial satisfaction of
the requirements for the degree
Doctor of Philosophy in Rehabilitation Science

December, 2011

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Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

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I dedicate this work to all my future Cervicogenic Dizziness patients...

LIST OF PUBLICATIONS

This present work is based on the following papers, which are referred to in the text by their roman numerals;

- I. **AlSaif A**, Johnson E. Cervicogenic Dizziness: Implications for Physical Therapy. Indian Journal of Physiotherapy & Occupational Therapy. 2011;5(4):6-11.
- II. **AlSaif A**, Johnson E, Trato J, Daher N, Alyahya D, Dobariya E, Doshi N, Gianchandani S, Kode N, Mehrotra S, Nair S, Parikh P, Ramadasse A, Ramani S. The Effects of Neck Muscle Fatigue on the Vestibulo-Ocular Reflex in Humans. Spine. In Review.
- III. **AlSaif A**, Johnson E, Daher N, Trato J, Alyahya D, Dobariya E, Ramadass A, Ramani S. Journal of Neurologic Physical Therapy. The Influence of Neck Muscle Fatigue on Dynamic Visual Acuity. In Review.

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ABBREVIATIONS

VOR	Vestibulo-Ocular reflex
VSR	Vestibulo-Spinal reflex
DVA	Dynamic Visual Acuity
EMG	Electromyography
CNS	Central Nervous System
JPE	Joint Position Error
SPNT	Smooth-pursuit neck torsion test
CGD	Cervicogenic Dizziness
VBI	Vertebrobasilar insufficiency
SCG	Superior cervical sympathetic ganglion
CCN	Central cervical nucleus
WAD	Whiplash-associated disorder
NTNT	Neck torsion nystagmus test
NTSPT	Neck torsion smooth pursuit
MTT	Manual traction test
cDVA	Computerized dynamic visual acuity
AE	Head repositioning absolute value
mVAS	Modified Visual Analog Scale
MVC	Maximum voluntary contraction
CCR	Cervical Collic Reflex
COR	Cervical Ocular Reflex
CSR	Cervical Spinal Reflex
VCR	Vestibular Collic Reflex

ABSTRACT OF THE DISSERTATION

The Effects of Neck Muscle Fatigue on the Vestibulo-Ocular Reflex

by

Amer Al Saif

Doctor of Philosophy, Graduate Program in Rehabilitation Science
Loma Linda University, December 2011
Dr. Eric Johnson, Chairperson

In order to maintain balance, the brain receives and processes input from multiple sensory systems including the visual, vestibular, and somatosensory systems. The information from these systems is sent to the brain where the information is then combined, organized and interpreted. This results in the output of reflexes (vestibulo-ocular reflex [VOR] and vestibulospinal reflex [VSR]). The VOR and the VSR work together with other motor systems to control gaze stability and control whole body equilibrium. Impairment in any of these sensory systems can result in dizziness, loss of balance, and visual instability. While the contributions of the visual, vestibular, and somatosensory systems on visual stability have been well described in the literature, contribution of cervical spine somatosensation on visual stability is not fully understood.

The overall aim of the present work was to investigate whether impaired somatosensory information from the cervical spine, caused by neck muscle fatigue, directly influences dynamic visual stability in healthy young and healthy older participants. Results indicated that healthy young and healthy older participants who were fatigued had significantly poorer Dynamic Visual Acuity (DVA) than those who were not fatigued ($P < .05$).

In Conclusion, our research suggests that when assessing vestibular function in patients complaining of dizziness and/or visual disturbance with a history of neck trauma,

one may speculate that VOR dysfunction could have a cervical origin due to somatosensory disturbance, which may lead to visual disturbances and dizziness

CHAPTER ONE

INTRODUCTION AND REVIEW OF THE LITERATURE

Understanding Balance

To maintain balance, the brain receives and processes input from multiple sensory systems including the visual, vestibular, and somatosensory systems.^{1,2} Information goes to multi-modal sensory integration areas of the brain and interact there to maintain postural equilibrium, body orientation and oculomotor controls.¹ Imbalance and dizziness may occur if there is a sensory mismatch between the vestibular system, the somatosensory system and the visual system.¹⁻³ A sensory mismatch can result from several causes, including impaired cervical spine somatosensory input.^{1,2}

Irregular Cervical Somatosensory Input

The literature suggests that somatosensory information from the cervical spine can be altered by several mechanisms.¹ For example, somatosensory input can be altered by direct trauma to the neck such as with a cervical flexion–extension injury (whiplash). This can lead to impaired cervical spine muscle function causing a sensory mismatch between somatosensory information from the cervical spine and input from the visual and vestibular systems.^{1,4,5} Muscle fatigue has been shown to modify the discharge of sensory receptors affecting proprioception and postural stability.⁴⁻⁶

Muscle Fatigue Affects Somatosensory

Stapley et al⁵ examined whether patients with cervical spine whiplash injuries had an increase in postural body sway after contractions of the dorsal neck muscles. Sway was measured during stance in 13 patients before and after they performed 5 minutes of isometric dorsal neck muscle contractions and after recovery. The authors found that after performing the contractions, seven patients had signs of fatigue via electromyography (EMG) and the other six patients had no fatigue and no increased sway.⁵ This study demonstrated the link between neck muscle fatigue and impaired postural control.⁵ It also suggested that balance and postural control could be altered in healthy participants by stimulating the neck, for example fatiguing it, because excessive stimulation produces abnormal kinesthetic input to the central nervous system (CNS) causing a sensory mismatch between the somatosensory information from the cervical spine and input from the visual and vestibular systems.^{6,7}

Neck Muscle Fatigue and Postural Control

Nicolas et al⁸ investigated the effects of cervical muscular fatigue on postural control under three sensory conditions (no vision, no vision-foam support, and vision) in two different conditions (no fatigue and fatigue of the neck extensor muscles) in 14 healthy adults.⁸ The authors reported that cervical muscle fatigue yielded increased center of foot pressure displacement in the absence of vision.⁸ This study demonstrated a link between neck muscle fatigue and impaired postural control.⁸

Neck Somatosensory Assessment

Revel et al⁹ assessed cervicocephalic kinesthetic sensibility in patients with cervical pain. Their results showed that patients with cervical pain had significantly less accurate

head repositioning performance than the control group.⁹ Heikkila and Astrom¹⁰ investigated the effects of neck somatosensory disturbance in patients with cervical whiplash trauma. Their results showed that patients with chronic dysfunction after whiplash trauma were significantly less accurate than the control group in their ability to relocate their heads in space after an active displacement of moved the head away from the reference position.^{10,11} Therefore, deficits in relocation accuracy are considered to reflect abnormal afferent input from the neck joint and muscle receptors.^{1,9-12}

Joint Position Error (JPE) and Muscle Fatigue

Pinsault and Vuillerme¹³ investigated the relationship between neck muscle fatigue and performance on the JPE test. A total of 9 young healthy adults were asked to perform the cervicocephalic relocation test to the neutral head position, that is, to relocate the head on the trunk, as accurately as possible, after full active cervical rotation to the left and right sides.¹³ This experimental task was done in two different conditions: no fatigue and fatigue of the scapula elevator muscles.¹³ Results showed that less accurate and less consistent repositioning performances were observed in those with fatigue than those without fatigue.¹³ Their results demonstrated abnormal afferent input from the neck joint and muscle receptors in fatigued subjects.¹³

Altered Cervical Afferent Input and Oculomotor Control

Deficits in oculomotor control have been demonstrated in patients with cervical whiplash injuries.¹⁴ Tjell and Rosenhall¹⁴ developed the smooth-pursuit neck torsion test (SPNT), which is considered to be specific for detecting eye movement disturbances due to altered cervical afferent input. Tjell and Rosenhall¹⁴ demonstrated that the altered smooth-pursuit eye movement control in torsion of the neck was evident in persons with

whiplash but not in persons with vestibular disorders, which also suggests that cervical spine structures may cause loss of eye movement control in whiplash patients. This would explain some of their reported symptoms, such as dizziness.¹⁴ In our review of the literature, we saw that altered cervical afferent input can cause many different symptoms and dysfunctions.^{1,2,5,6} One of the dysfunctions could be loss of eye movement control as previously reported.¹⁴ However, this may suggest that the neck may directly influence the vestibular complex, and as a result may have an impact on VOR.¹⁵ Herdman et al¹⁵ demonstrated that there is a significant relationship between age and dynamic visual acuity scores in healthy participants.^{15,16} Specifically, older participants had poorer visual acuity during head movement than younger participants due to age related loss of vestibular dysfunction.¹⁵

Hypothesis: Neck May Directly Influence VOR

There is evidence to suggest that the neck may directly influence vestibular function.¹⁷ For example, stimulation of the deep neck mechanoreceptors has a measurable impact on the VOR in cats.^{1,17} Irregular cervical proprioceptive input may give rise to a mismatch of sensory input and as a result cause an asymmetry to the VOR.¹⁷ Pardoan et al¹⁴ investigated the interaction between neck proprioception and the VOR.¹⁴ They used a rotary chair to passively rotate 16 healthy subjects facing forward and with their heads passively turned 70 degrees to either side.¹⁴ The VOR gain tended to be lower when the subjects were rotated with their heads turned opposite to the direction of rotation compared to when they were rotated in the same direction but with their head facing forward.¹⁴ The results of this study suggest that there is a measurable interaction between neck proprioception and the VOR in subjects with normal vestibular function.¹⁴ Also, abnormal neck muscle proprioceptive signals may give rise to asymmetric functioning of

the VOR and contribute to postural and visual instability.¹⁴ The interaction between cervical spine proprioception and the VOR has been observed in a very limited number of studies and its impact on the human VOR is not fully understood.^{1,14,15} The present study was designed to address this issue. The purpose of the present work was to investigate whether impaired somatosensory information from the cervical spine, caused by neck muscle fatigue, directly influences dynamic visual stability in healthy young and healthy older participants.

CHAPTER TWO

CERVICOGENIC DIZZINESS: IMPLICATIONS FOR PHYSICAL THERAPY

Amer A. AlSaif, MPT, DPT¹ and Eric G. Johnson, DSc, PT, NCS²

Abstract

Cervicogenic Dizziness (CGD) is a relatively new, emerging area in the medical literature and physical therapy practice. Approximately 60% of patients with whiplash-associated disorder experience dizziness due to impaired neck proprioceptive input. Patients with CGD typically describe their dizziness as vertigo, lightheadedness, blurry vision, disequilibrium, and/or nausea. Physical therapy interventions for CGD include orthopedic and vestibular rehabilitation strategies. The purpose of this paper is to discuss the etiology of cervicogenic dizziness, describe the proposed pathophysiology, and introduce the physical therapy examination and intervention process for patients with CGD.

Key words: Cervicogenic Dizziness, Cervical Vertigo, Whiplash Associated Disorder, Orthopedic Manual Physical Therapy, Physical Therapy.

Introduction

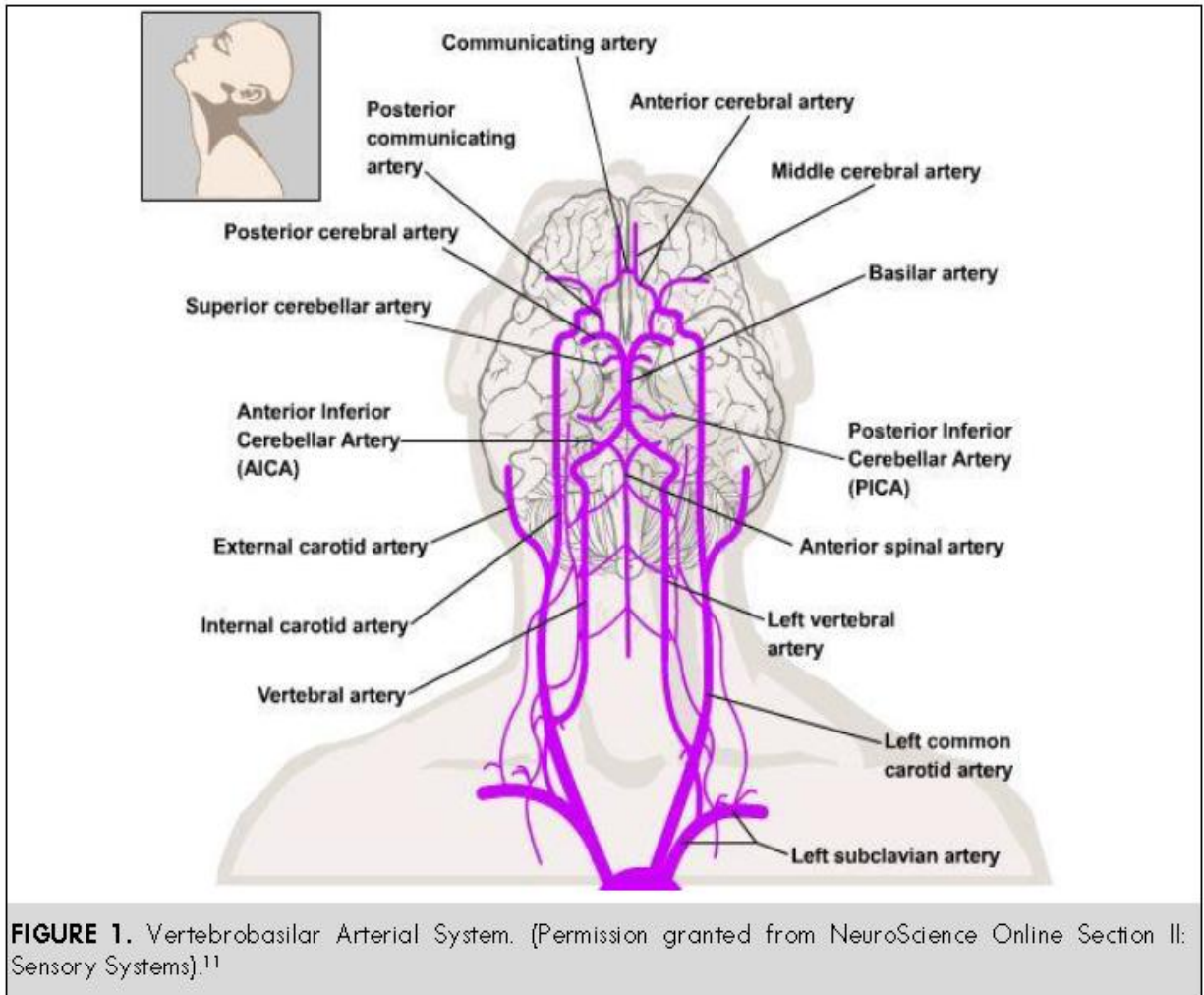
Balance is maintained through a complex interaction between the visual, vestibular, and somatosensory systems.^{1,2} Multimodal sensory integration occurs in the central nervous system where motor responses are generated to coordinate head and body orientation, postural stability, and gaze stability during head movements.¹ When normal sensory integration is impaired, imbalance and dizziness often occur.¹⁻⁴ Dizziness is one of the most common medical problems in many countries, including Asia and the United States, especially in the elderly population.^{5,6} Dizziness is a nonspecific symptomatic description resulting from numerous pathologies including viral or bacterial infection, head trauma, neurological disease, psychological conditions, orthostatic hypotension, migraine and/or

headache disorders, pharmacology, vertebrobasilar insufficiency, and vestibular disorders.^{1,2,7} Dizziness can also be caused by orthopedic impairments affecting the cervical spine and, in such cases, is referred to as “cervicogenic dizziness.”^{1,7} Cervicogenic dizziness has been defined as “a non-specific sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck.”⁷ It is different from vestibular-driven dizziness in that symptoms are usually not as intense and it rarely includes vertigo.⁷ It is an emerging area of clinical practice with a growing body of evidence that supports the pathophysiology and physical therapy management of patients with cervicogenic dizziness. Therefore, the purpose of this paper is to provide a brief overview of the theory and clinical application of managing patients with cervicogenic dizziness.

Etiology of Cervicogenic Dizziness

There are several proposed mechanisms leading to cervicogenic dizziness including mechanical compression of the vertebral artery system, irritation of the cervical sympathetic nervous system, and abnormal proprioceptive input from the upper cervical spine.^{1,2,7-9} Mechanical compression of the vertebral artery system can produce vertebrobasilar insufficiency (VBI).^{8,9} The vertebral arteries are branches of the subclavian arteries arising from the aortic arch.⁹ The vertebral arteries travel superiorly through C6-C1 transverse foramen, migrate horizontally around the posterior arch of the atlas, enter the foramen magnum, and merge with one another to form the basilar artery.^{8,9} The vertebral arteries encounter several soft tissue and bony structures capable of producing mechanical compromise.⁹ Muscle tightness in the upper cervical spine can potentially occlude the vertebral arteries reducing brainstem perfusion and causing VBI.^{7,9,10} In particular, the vertebral arteries travel between the anterior scalene and longus colli

muscles as well as under the inferior capitis oblique and intertransversarius muscles.⁷ Cervical spine osteophytes and forward head posture are also potential contributors to vertebral artery compression and VBI.^{9,10} Irritation of the cervical sympathetic ganglia has also been theorized to contribute to cervicogenic dizziness.^{1,13} The cervical ganglia are paravertebral ganglia of the sympathetic nervous system and travel adjacent to the arterial network and cervical musculature antero-lateral along the vertebral bodies.^{12,13} The cervical sympathetic ganglia consist of the superior, middle, and inferior cervical ganglion. The superior cervical sympathetic ganglion (SCG) is the largest and is located at the level of the second and third cervical vertebrae.^{1,12,13} The SCG is posterior to the internal carotid artery and internal jugular vein, and anterior to the longus capitis muscle.^{12,13} Upper cervical spine muscle tightness, bony anomalies, and/or poor cervical spine posture can potentially compromise this ganglion, leading to hypoperfusion of the vertebral and carotid arterial network causing dizziness consistent with cervicogenic dizziness.^{1,12,13} (Figure 1)



Abnormal proprioceptive input from the upper cervical spine also contributes to dizziness. The somatosensory system detects peripheral stimuli from sensory receptors, including mechanoreceptors located in human skin.^{1,2} Mechanoreceptors (Pacinian corpuscles, Meissner's corpuscles, Merkel's discs, and Ruffini corpuscles) mediate peripheral stimuli including pressure, touch, pain, temperature, and proprioception.^{1,2,14-16} There are an abundance of mechanoreceptors in the upper cervical spine that primarily transmit impulses through nerve cells originating from C2 dorsal root ganglion.^{2,16,17} The mechanoreceptor input from the upper cervical segments (Occiput-Atlas, Atlas-Axis, Axis-

C3), particularly from the upper cervical spine muscles, report directly to the vestibular nuclear complex and the superior colliculus.^{1,2,17} The upper cervical spine mechanoreceptors also converge in the central cervical nucleus (CCN), which serves as a pathway to the cerebellum for integrating and organizing vestibular, ocular, and proprioceptive sensory input.^{2,13,16,17} The CCN also sends coordinated information to the cortex for maintenance of postural equilibrium and body orientation (Figure 2).^{1,2,17,18} Cervical spine reflexes contribute to head orientation, eye movement control, and postural stability; cervical collic reflex, the cervical ocular reflex, and cervical spinal reflex, respectively (Figure 3).^{1,2} These cervical reflexes work in conjunction with vestibular collic reflex, vestibular ocular reflex, and the vestibular spinal reflex.^{1,2,17,19} Abnormal sensory input from the vestibular and upper cervical spine can lead to dizziness, unsteadiness, and visual disturbance.^{1,2,13,17,19}

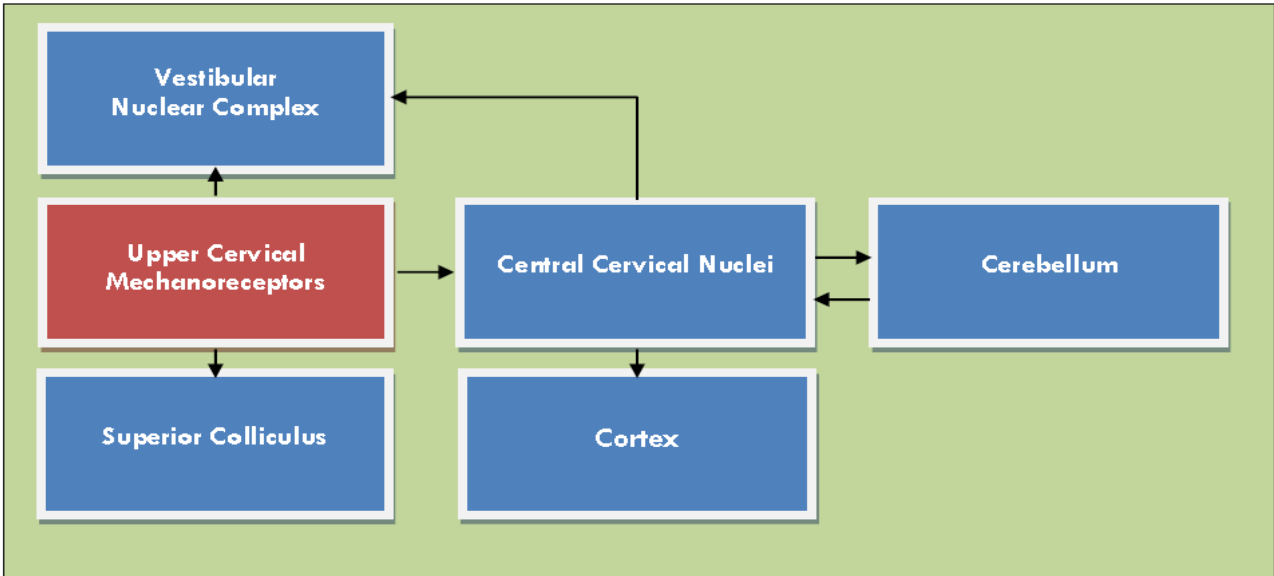


FIGURE 2. Important neurological pathways between the upper cervical spine and the central nervous system concerning head and body orientation as well as head-eye coordination. Postural balance and visual stability is dependent upon healthy neural communication.

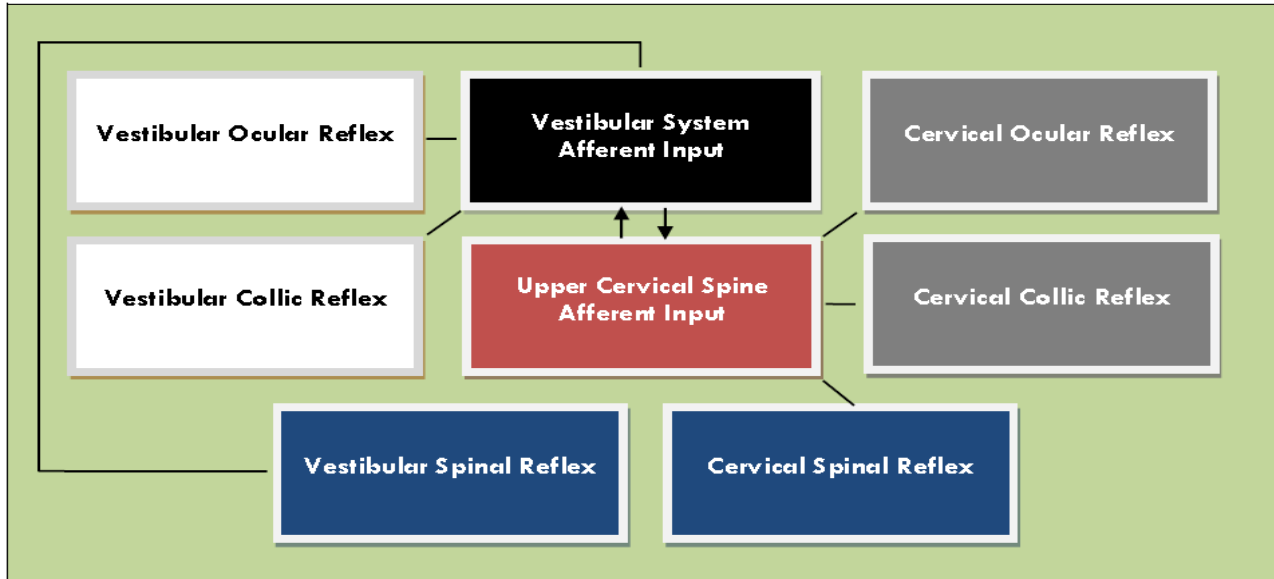


FIGURE 3. Cooperative reflexes between the vestibular system and the upper cervical spine. These reflexes are dependent upon healthy integrative central nervous system communication, particularly with the vestibular nuclear complex, in order to maintain normal postural balance and visual stability.

Cervical Spine Trauma

Abnormal cervical somatosensory input can alter somatosensory control and negatively impact postural stability and vision.¹ Cervical spine trauma, such as whiplash-associated disorder (WAD), can impair cervical somatosensory function by causing ischemia, inflammation, and stress.¹ Moreover, evidence suggests that direct trauma to the neck can lead to cervical spine muscle fatigue that ultimately modifies the discharge firing rate of sensory receptors, thus affecting joint position of the head and neck as well as postural stability.^{3,20-22,24-27} Deficits in oculomotor function have also been described in the literature in patients with WAD.^{1,28} Tjell and Rosenhall²⁸ reported abnormal smooth-pursuit eye movements when the neck was rotated under a stable head in WAD patients. Additionally, greater loss of eye motor control was identified among WAD patients complaining of dizziness.^{1,28} Problems of convergence and diplopia have also been associated with WAD patients.¹ It is estimated that approximately 60% of all WAD patients develop dizziness.¹

Physical Therapy Evaluation

The physical therapy evaluation includes subjective and objective components. Because CGD is one of numerous types of dizziness, the evaluation strategy can be challenging.^{1,7} According to Wrisley et al, CGD is a diagnosis of exclusion, meaning that competing causes of dizziness must be ruled out.⁷ Physical therapists need to carefully review the past medical history and ask specific questions about the patient's dizziness in order to determine that the dizziness is cervicogenic.⁷ Episodic dizziness lasting minutes to hours is a common complaint in patients with CGD.⁷ They may also report a general sense of disequilibrium or lightheadedness as well as visual disturbances. Vertigo is rarely a chief complaint in CGD patients.^{1,7} Circumstances that frequently produce their dizziness

include neck pain.^{1,7} An example of a CGD Physical Therapy Subjective Examination Form is provided in Table 1. The physical examination for CGD patients includes a medical screening component that is performed first (Table 1).^{1,7} The medical screening component includes three different phases: screening of the (1) cervical spine stability, (2) cervical vascular system, and (3) central nervous system. If any of these medical screening tests produce positive or abnormal results, the physical therapist must refer the patient to a physician for further medical consultation. Otherwise, the physical therapist continues with the physical examination. The vestibular system is examined to determine whether the dizziness is being caused by the peripheral or central vestibular system.⁷ If the peripheral vestibular system examination is positive, appropriate vestibular rehabilitation interventions are implemented. If the central vestibular examination is positive, the physical therapist should refer the patient to a physician for further medical consultation. Otherwise, the physical therapist continues the physical examination.⁷ The following tests may help determine whether the dizziness is being caused by the neck. The neck torsion nystagmus test (NTNT) is performed by stabilizing the patient's head and rotating his or her body underneath (Figure 4).²⁸ The NTNT is positive if nystagmus is elicited. The neck torsion smooth pursuit (NTSPT) is performed by observing ocular smooth pursuit with the patient's head in neutral followed by neck rotation under a stable head. A positive NTSPT results when smooth pursuit is normal in the neutral position and abnormal when the neck is in the rotated position.^{1,28} The joint position error (JPE) test examines cervical spine proprioception. The JPE is performed by asking the patient to sit 90 cm away from a fixed target while wearing a head strap with a laser pointer, as shown in (Figure 5).^{22,23} The physical therapist asks the patient to look straight ahead at the center of the fixed target. This is the starting position.²³ The physical therapist then asks the patient to close their eyes, rotate their neck as far as they can, and return their head to the starting

position with as much precision as possible.²³ A normal JPE requires the patient to be within 4.5 degrees of the starting position. ²³ The manual traction test (MTT) is performed while the patient is seated (Figure 6).¹ The test is considered positive if the compression increases or produces dizziness and traction relieves it.¹



FIGURE 4. Neck Torsion Nystagmus Test.



FIGURE 5. Joint Position Error Test.

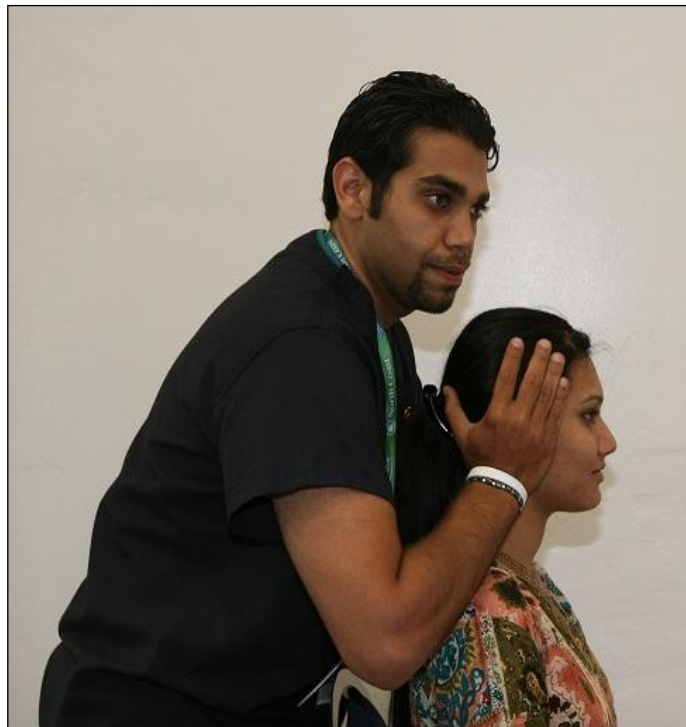


FIGURE 6. Manual Traction Test.

TABLE 1. Physical Therapy Cervicogenic Dizziness Examination Form.

Physical Therapy Dizziness Examination Form		
Patient:	Date:	Date of Birth:
Referral Source:	Diagnosis:	Occupation:
SUBJECTIVE EXAMINATION		
1. Chief complaint and date of onset:		
2. Mechanism of injury:		
3. Tempo of symptoms: constant/episodic seconds/episodic minutes/episodic hours		
4. Do you experience spells of vertigo?	YES NO	mVAS range ____/10; currently ____/10
5. Do you experience disequilibrium?	YES NO	mVAS range ____/10; currently ____/10
6. Do you experience lightheadedness?	YES NO	mVAS range ____/10; currently ____/10
7. Do you experience oscillopsia?	YES NO	mVAS range ____/10; currently ____/10
8. Circumstances that exacerbate/produce symptoms:		
9. Fall History:		
10. Past Medical History:		
11. Medications:		
12. Do you have steps in your home?	YES NO	
13. Do you smoke/drink, and if so, how much?	YES NO	
14. Do you have trouble sleeping?	YES NO	
15. Was previous functional level normal?	YES NO	
ORTHOPEDIC ASSESSMENT		
1. Cervical Spine Stability: Alar Ligament/Sharp-Purser/Lateral Shear + OR -		
2. Positional Tolerance Testing: + OR -		
3. Cervicogenic Dizziness Testing: NTNT/NTSPT/NPPT/MTT/JPE ____ cm difference + OR -		
4. Cervical Spine AROM/PROM:		
NEUROLOGICAL ASSESSMENT		
5. Proprioception: + OR -		
6. CNS Testing: Babinski/Clonus/DTR/RAM/Finger to Nose EO and EC + OR -		
VESTIBULAR ASSESSMENT		
7. Central Vestibular Testing:	Ocular Alignment / Spontaneous Nystagmus / Gaze Evoked Nystagmus Smooth Pursuit / Saccadic Eye Movement / VOR Cancellation Test	
8. Peripheral Vestibular Testing:	Hallpike Dix Test / Roll Test / Head Thrust Test/ Dynamic Visual Acuity Head – Shaking Nystagmus Test	
BALANCE TESTS:		
9. Balance Testing: Romberg/Sharpened Romberg/Timed Up and Go/Functional Reach Test + OR -		
10. Berg Balance Scale: ____/56; Dynamic Gait Index: ____/24		
DIZZINESS INVENTORY:		
16. DHI Score: ____/100; ABC Score: ____/100		
TREATMENT PLAN:		
RECOMMENDATION TREATMENT PLAN: _____ DAYS/WK FOR _____ WKS.		

Physical Therapy Intervention

Physical therapy intervention has been shown to be effective in reducing CGD symptoms. According to Wrisley et al⁷, CGD symptoms typically increase with neck pain. Therefore, treating neck pain among this group of patients is one of the main objectives for physical therapists. Intervention strategies may include: (1) orthopedic manual techniques specific to the cervical spine region, (2) head and neck proprioceptive rehabilitation program, and (3) cervical-ocular motor exercises. Cervical spine pain and inflammation can be treated with a variety of physical therapy modalities, including cryotherapy, thermotherapy, ultrasound, and cervical spine traction.^{1,7} Cervical spine hypomobility is common among CGD patients and may increase symptoms of dizziness.^{1,7} However, cervical hypomobility can be treated with joint specific mobilization techniques and tissue/age-specific stretching programs.^{1,7} Cervical spine proprioception impairments can be treated with a specific proprioceptive rehabilitation program. The proprioception program includes slow, passive head movements with fixed-target gaze exercises (the clinician passively moves the patient's head while the patient maintains a fixed gaze on a stationary target).^{22,23} The program can be progressed by doing active head movements rather than passive head movements. Also, the clinician may progress the program by instructing the patient to perform active head movements while maintaining their gaze on a fixed target with their trunk passively or actively moved.²³ Another way of performing the program is to instruct the patient to close their eyes and actively rotate their head, return to the starting position, and open their eyes.²³ If the patient cannot see the target, they can keep their eyes open and continue rotating their head until they can see the target.^{22,23} This training provides the patient with information about cervical spine joint position sense and can be performed with restricted peripheral vision using foveal

glasses.^{1,7,22,23} Input from cervical spine afferent nerves can alter the function of the oculomotor system.^{1,7,23}

Thus, oculomotor training is important to reduce potential extraocular muscle weakness.²⁸ Extraocular motor function can be managed using smooth-pursuit (patient keeps head still while eyes follow a moving target), saccades (patient keeps head still and quickly moves eyes between targets), X1 adaptation exercises (patient moves head from side to side while maintaining the gaze on a stationary target), and X2 adaptation exercises (patient moves head and a hand-held target in opposite directions while maintaining gaze on moving target at all times).^{1,22,28} All extraocular exercises can be progressed by increasing the speed of movement, range, duration, and frequency. Also, the exercises could be progressed by gradually decreasing the stability of support and changing from static to dynamic positions.^{1,22,28}

Conclusion

Cervicogenic dizziness is often the result of a sensory mismatch between the vestibular, somatosensory, and visual afferent inputs. Physical trauma involving the cervical spine, such as whiplash injury, is a common mechanism of injury in CGD patients. Physical trauma contributes to impairment in the upper cervical spine proprioceptive input leading to symptoms including disequilibrium and dizziness. In order to determine the origin of the patient's dizziness, the physical therapist must exclude all competing causes of dizziness. Once CGD has been confirmed, appropriate interventions are implemented to reduce cervical spine pain and inflammation, improve cervical spine proprioception, improve cervico-ocular function, and restore joint and soft tissue range of motion and mobility.

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CHAPTER THREE
THE EFFECTS OF NECK MUSCLE FATIGUE ON THE VESTIBULO-OCULAR
REFLEX IN HUMANS

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Abstract

Study Design: Double-blinded, prospective, randomized, controlled trial.

Objectives: To determine whether neck muscle fatigue affects dynamic visual acuity in healthy young participants.

Background: Postural and visual stability are dependent upon efficient and accurate central processing of visual, vestibular, and somatosensory afferent input. Inaccurate sensory information from dysfunctional sensory end organs leads to a sensory mismatch, causing postural and/or visual instability. The interaction between cervical spine proprioception and the VOR has been observed in a very limited number of studies and its impact on the human VOR is not fully understood.

Main Outcome Measures: Dynamic visual acuity (DVA) and cervical joint position error (JPE).

Methods: Thirty healthy young subjects (ages 21 to 30 years) participated in the study. Participants were randomly divided into an experimental group (n=15) and a control group (n=15). The experimental group received an exercise designed to induce neck muscle fatigue and the control group received non-fatiguing sham exercises.

Results: There were significant differences in mean DVA between the two groups ($.26 \pm .11$ LogMar versus $.003 \pm .02$ LogMar, $p = .001$). Subjects in the experimental group had a significant decline in their DVA compared to the control group. Dynamic visual acuity strongly correlated with neck muscle fatigue ($r = .79$, $p < .001$). No significant differences in JPE were observed between the two groups ($p = .20$) and no significant correlations between JPE and neck muscle fatigue were observed ($r = .23$, $p = .21$).

Conclusion: The results of this study suggest that neck muscle fatigue negatively impacts DVA. Although not statistically significant, cervical spine proprioception as measured by the JPE in the experimental group was diminished after fatigue.

Keywords: Cervicogenic dizziness, cervical vertigo, whiplash associated disorder, physical therapy, dynamic visual acuity, joint position error.

Introduction

Postural and visual stability are dependent upon efficient and accurate central processing of visual, vestibular, and somatosensory afferent input.^{1,2} This afferent input undergoes multi-modal sensory integration in several areas of the brain and brainstem in order to provide efferent output to maintain postural equilibrium and oculomotor control.² Inaccurate sensory information from dysfunctional sensory end organs leads to a sensory mismatch, causing postural and/or visual instability.¹⁻³ Sensory mismatch can result from several causes including, but not limited to, vestibular disorders, neurological disease, pharmacology, and cervical spine trauma.^{1,2} Somatosensory information from the cervical spine can be altered as a result of direct trauma.¹ Cervical spine trauma can lead to cervical spine muscle fatigue and modify the discharge firing rate of upper cervical sensory receptors.^{1,4,5} The upper cervical spine is transmitted through nerve cells originating mainly from C2 dorsal root ganglion.^{1,2} Mechanoreceptors in the upper cervical spine converge in the central cervical nucleus (CCN).¹⁻³ The CCN serves as a pathway to the cerebellum, which integrates and organizes vestibular, ocular, and proprioceptive information.^{1,2,4} Disturbances in the neural connections between the three sensory systems can lead to mismatched sensory input, causing conflicts among all inputs from the different sensory systems which cause dizziness, unsteadiness, and visual disturbance.¹⁻⁴ For example, altered somatosensory input, particularly from the upper cervical spine structures, can disturb the vestibular system.¹⁻³ Clinical research shows that when experiencing such disturbances, patients become less able to utilize vestibular information to resolve inaccurate and irregular information from the somatosensory and

visual systems.^{1,2} Moreover, evidence suggests that upper cervical muscle fatigue may be an important contributing factor to altered postural stability in people with neck pain because neck muscle fatigue has been shown to modify the discharge of sensory receptors in neck muscles and affect proprioception.^{2,4-7}

Pinsault and Vuillerme⁷ investigated the relationship between neck muscle fatigue and cervical spine proprioception using the joint position error test (JPE). Subjects were asked to relocate their head on trunk as accurately as possible after full active cervical rotation to the left and right sides.⁷ Subjects were randomized into cervical spine muscle fatigue and control groups.⁷ Less accurate and less consistent cervical joint repositioning was observed in the fatigue group which they correlated with abnormal afferent input from the neck joint and muscle receptors.⁷

Revel et al⁸ assessed cervicocephalic kinesthetic sensibility in patients with cervical pain. Their results demonstrated that patients with cervical pain had significantly less accurate head repositioning performance compared to the control group.⁸ Heikkila and Astrom⁹ investigated the effects of neck somatosensory disturbance in patients with cervical whiplash trauma. They found that patients with chronic dysfunction after whiplash trauma were significantly less accurate than a control group in their ability to relocate their heads in space after actively rotating their head away from the reference position.^{9,10}

Nicolas et al¹¹ investigated the effects of cervical muscular fatigue on postural control under multiple sensory conditions and determined that cervical muscle fatigue increased the center of foot pressure displacement in the absence of vision.¹¹ Their results indicated that there is a correlation between neck muscle fatigue and impaired postural stability.¹¹

Stapley et al¹² examined whether patients with cervical spine whiplash injuries had an increase in postural body sway after contractions of their dorsal neck muscles.¹² Sway was measured during stance in 13 patients, before and after performing 5 minutes of isometric dorsal neck muscle contractions.¹² They found that after performing the contractions, seven subjects had signs of fatigue via electromyography and increased sway.¹² This study demonstrated the link between neck muscle fatigue and impaired postural stability.¹² It also suggested that balance and postural control could be altered in healthy subjects by inducing fatigue in the neck muscles.¹² Deficits in oculomotor control have also been reported in patients with cervical trauma.¹

Tjell and Rosenhall¹³ observed that altered smooth-pursuit eye movement occurred in subjects with neck trauma when their neck was rotated. These findings suggest that normal eye movement is partially dependent upon accurate sensory input from the cervical spine.¹³ There is also evidence to suggest that the cervical spine influences eye movements via the vestibular system.¹⁴ Stimulation of the deep cervical spine mechanoreceptors has a measurable impact on the vestibulo-ocular reflex (VOR).^{1,14}

Pardoan et al¹⁴ investigated the interaction between neck proprioception and the VOR.¹⁴ They used a rotary chair to passively rotate 16 healthy subjects facing forward and with their heads passively turned 70 degrees to either side.¹⁴ The VOR gain tended to be lower when the subjects were rotated with their heads turned opposite to the direction of rotation compared to when they were rotated in the same direction but with their head facing forward.¹⁴ The results of this study suggest that there is a measurable interaction between neck proprioception and the VOR in subjects with normal vestibular function.¹⁴ Also, abnormal neck muscle proprioceptive signals may give rise to asymmetric functioning of the VOR and contribute to postural and visual instability.¹⁴

The interaction between cervical spine proprioception and the VOR has been observed in a very limited number of studies and its impact on the human VOR is not fully understood.^{1,14,15} The purpose of this investigation was to determine the effects of cervical muscle fatigue on dynamic visual acuity (DVA) and Joint Position Error (JPE).

Methods

Participants

The study was conducted in the Department of Physical Therapy, Loma Linda University, Loma Linda, California. Thirty adults participated in the study. Fifteen participants (8 males and 7 females) were randomized into the experimental group and 15 participants (7 males and 8 females) into the control group. Informed consent was acquired prior to the beginning of the study. The study protocol was approved by the Institutional Review Board at Loma Linda University. Potential participants were recruited based on the following criteria; healthy young adults between the ages of 21 to 30 years without current cervical spine pain or history of cervical spine whiplash injury. Participants were required to pass all comprehensive screening tests to ensure normal function of the somatosensory, visual, and vestibular systems. Screening tests included all of the following: cervical stability (alar ligament test, Sharp-Purser test, and lateral shear test), cervical vascular screening using the modified vertebral artery test, vestibular system screening (Hallpike-Dix Test, Roll Test, Head Thrust Test, Head Shaking-Induced Nystagmus Test, and condition 5 of the NeuroComSMART Balance Master®¹⁶, static and dynamic visual acuity and cervical spine somatosensory integrity (JPE). To be included in the study, all screening test results had to be negative.

Sample Size

We recruited a cohort of 30 participants. A post power analysis with an effect size of 1.1 according to the change in DVA between cervical spine muscle fatigue and control groups, an alpha level of .05, and a sample size of 30 indicated that the power was .89.

Randomization

Randomization of group assignment was accomplished using a computer generated random sequence.

Study Design

The two main outcome variables were the VOR as measured by the computerized dynamic visual acuity (cDVA) and cervical joint position sense as measured by the JPE test. The VOR and JPE measurements were taken pre and post intervention. Data were collected for all participants between 8 am and 12 pm to minimize the effects of normal fatigue.¹⁷ (Figure 7).

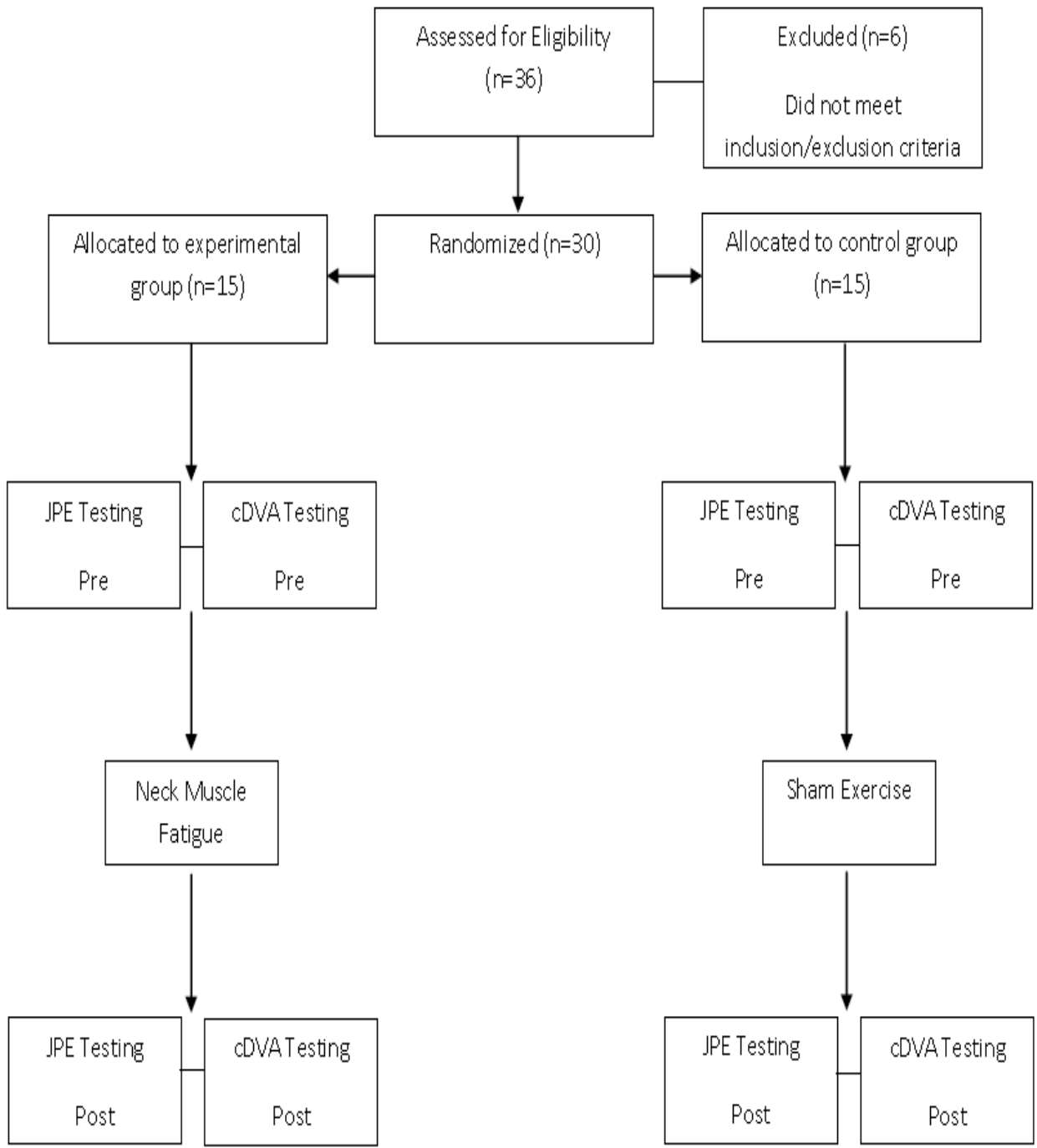


FIGURE 7. CONSORT flow diagram of study.

Computerized Dynamic Visual Acuity Test (cDVA)

We used the computerized DVA (cDVA) InVision™ system to determine the participant's ability to accurately perceive objects while actively moving their head.¹⁸ The cDVA is commonly used when evaluating patients with possible VOR dysfunction and other vestibular deficits.^{1,19,20} Static visual acuity was determined followed by dynamic visual acuity (Figure 8).¹⁹ The difference between the two test scores was calculated and the result was converted into LogMar, which is a measure of visual acuity loss. According to Herdman et al¹⁹, changes of one line (0.1 LogMAR) or less (≤ 0.1) are considered normal and changes of two or more lines (> 0.2) are abnormal.¹⁹ The cDVA is a reliable and sensitive tool for identifying patients with vestibular dysfunction resulting from an impaired VOR.^{19,20} The sensitivity of the cDVA is reported to be 94.5% and specificity 95.2%.¹⁹ The positive predictive value is 96.3% and the negative predictive value is 93%.¹⁹

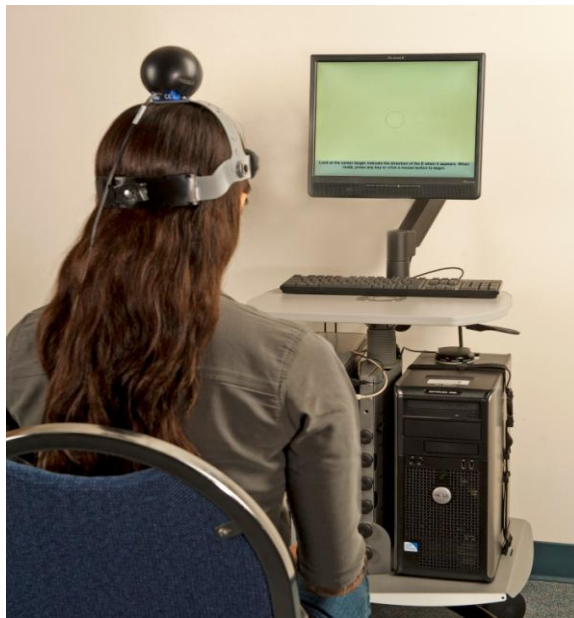


FIGURE 8. Computerized dynamic visual acuity test.

Joint Position Error Test (JPE Test)

We used the JPE test to measure the cervical spine somatosensory system.⁸ Participants sat on a chair 90cm away from a fixed target on a wall while wearing a head strap with a top-mounted laser pointer (Figure 9).^{8,21-23} Participants were instructed to focus on the target then close their eyes and remember the starting position of the head, specifically the “zero target”. Keeping their eyes closed, participants maximally rotated their head to one side, and then returned to the starting position as accurately as possible.²¹⁻²³ No speed instructions were given.^{8,22,23} The new position of the laser light was noted and the distance from the starting point was measured (reposition error distance).^{9,21-23} All participants repeated the same test ten times to each side in a random order.^{8,21} The head repositioning absolute value (AE) in centimeters was calculated.^{8,21} A normal JPE is less than or equal to 4.5 degrees.^{1,8,9,12,21}



FIGURE 9. Head-mounted laser pointer for joint position error testing.

Modified Visual Analog Scale (mVAS)

We used the mVAS to subjectively measure the participant's upper posterior neck muscle fatigue level.⁷ Participants in both groups rated their level of fatigue before and after the intervention. The mVAS was a number scale from 0 to 10.^{7,24} A score of 0 was defined as no fatigue at all and 10 was defined as the most fatigue imaginable.²⁴

The Cervical Spine Isometric Contraction Protocol

Participants in the experimental group sat on a customized neck exercise machine. With their head and neck in a neutral position, participants isometrically resisted a weighted stack load for 5 minutes (Figure 10).⁶ The load used in the weight stack was equal to 30% of the participant's maximum voluntary contraction (MVC)^{6,7} which was determined for each subject using a dynamometer. Participants performed 3 MVC's and the average reading obtained from the dynamometer (in pounds) represented the MVC for each participant.^{6,7} In order to limit trunk muscles from contributing during the contraction, a normal gait belt was used during the MVC and the 5 minute isometric exercise to stabilize their trunk to the back support of the neck exercise machine.⁶ Participant's neck fatigue level was taken immediately following the 5 minute isometric exercise.⁷



FIGURE 10. Cervical neck muscle fatigue machine.

Sham Exercise Protocol

Participants sat on a chair facing a wall while wearing a head strap with a top-mounted laser pointer attached. Four targets of the same shape and width (center, down, left, and right) were fixed on the wall at the subject's eye level. The sham exercise began with the participant's head in a neutral position (eyes straight ahead). Participants pointed the laser inside the large circle of the center target. From this starting position, participants were instructed to point the laser inside the second target by moving their head slightly then returning the laser to the center target. Participants repeated the same activity for all targets as follows: center-down, center-right, and center-left for a total of 5 minutes (including two 1 minute breaks). This sham exercise was designed to not fatigue the cervical muscles. The participant's eyes were open during this exercise and no speed instructions were given.

Masking and Blinding

Data was collected by researchers blinded to group assignment. Participants were instructed not to reveal intervention allocation to the researchers during data collection.

Statistical Methods

Data was analyzed using the statistical package SPSS for Windows version 19.0 (SPSS, Inc., Chicago, IL).^a Frequencies and relative frequencies were computed for gender, and means and standard deviations (SD) for the continuous variables age, DVA, and JPE. The chi-square test of independence was used to assess the relationship between gender and type of group at baseline, and the *Kolmogorov-Smirnov test* was applied to test for the normality of continuous variables. The Mann-Whitney U-test was performed to compare the mVAS scores between the two groups at baseline. The difference between mVAS, DVA, and JPE values at baseline and at the end of the study were calculated and these differences were compared between the two groups using the independent sample t-test. Spearman's correlation and regression analysis were used to examine the relationship among DVA, JPE, and mVAS at the end of the study. The level of statistical significance was set at $p < .05$.

Results

Fifteen participants were randomized into the experimental group and fifteen into the sham group. The demographic and baseline clinical characteristics of the two study groups are presented in Table 2. There were no significant differences between the two groups at baseline for gender, age, DVA, JPE, and mVAS. At the end of the study, there were significant differences in the mean DVA between the two groups ($.26 \pm .11$ Vs $.003 \pm .02$, $p = .001$, Table 3). Participants who were fatigued had significantly poorer DVA than those who were not fatigued (Figure 11). There were, however, no significant differences in mean JPE between the two groups ($p = .20$, Table 2). Results showed that the DVA strongly correlated with the mVAS score ($r = .79$, $p < .001$, Figure 12). No significant correlations were observed between the mVAS score and JPE ($r = .23$, $p = .21$). A stepwise linear regression was conducted to determine the effect of JPE and mVAS on cDVA score. Results indicated that mVAS was a significant predictor of DVA ($R^2 = .59$, $p < .001$). Approximately sixty percent of the variability in the DVA was explained by its relationship to the fatigue score.

TABLE 2. Mean (SD) of baseline characteristics at baseline for the participants in the experimental group and control group (N=30)

Variables	Experimental (n=15)	Control (n=15)	P-Value
Gender ⁺			.72*
Male	8 (53%)	7 (47%)	
Female	7 (47%)	8 (53%)	
Age (years)	25.3 (1.8)	25.8 (2.4)	.50**
DVA	.06 (.04)	.07 (.03)	.34**
JPE	4.54 (1.02)	4.95 (1.25)	.33**
mVAS	.33 (.7)	.20 (.6)	.06***

DVA= Dynamic Visual Acuity, JPE = Joint Position Error, mVAS = modified Visual Analog Scale.

* chi-square test.

** Independent sample t-test.

*** The Mann-Whitney U-test.

+ Results are presented as frequency (%).

TABLE 3. Comparison of mean (SD) of post minus pre DVA and JPE between the two groups

Variables	Experimental (n=15)	Control (n=15)	P-Value *
DVA	.26 (.11)	.003 (.02)	.001
JPE	.97 (1.07)	.44 (1.40)	.201

DVA=Dynamic Visual Acuity, JPE= Joint Position Error

* Independent sample t-test.

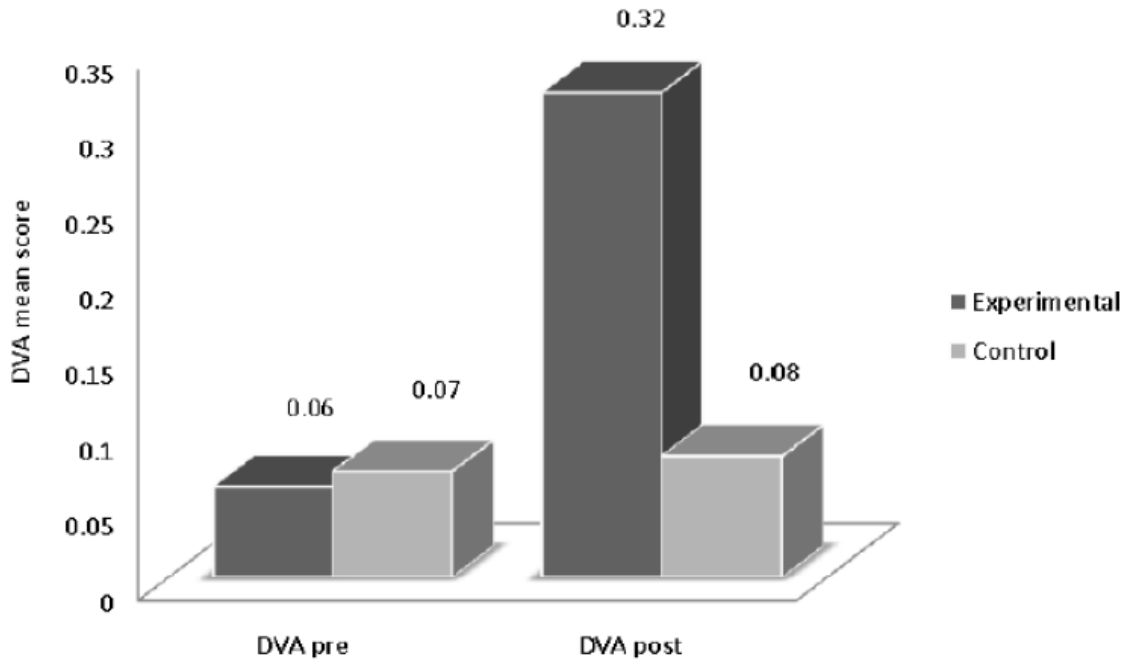


FIGURE 11. Comparison of Dynamic Visual Acuity (DVA) by Group and Time Assessment.

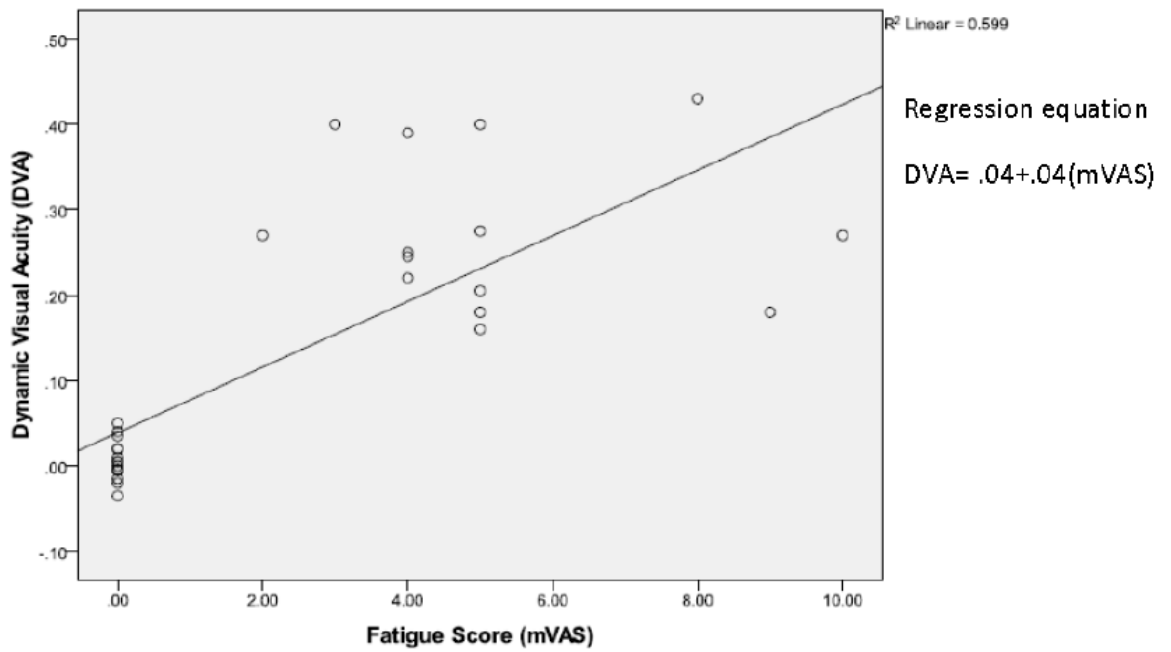


FIGURE 12. Relationship between Fatigue Score and Dynamic Visual Acuity (N=30).

Discussion

The purpose of the present investigation was to determine the effect of neck muscle fatigue on dynamic visual acuity in healthy young adults. Thirty young healthy participants were randomly assigned into either a neck muscle fatigue group or a sham group. Dynamic visual acuity test scores were used to measure the gaze stability, and JPE test scores were used to measure cervical joint repositioning accuracy. Our results determined that neck muscle fatigue negatively impacts DVA. Although not statistically significant, a trend was observed suggesting that neck muscle fatigue negatively impacts JPE, as determined by less accurate and less consistent repositioning performances in the fatigue group versus sham group. Our findings are consistent with existing literature, which reports that reduced proprioceptive acuity contributes to sensory mismatches and possibly an asymmetry of the VOR.^{1,14} This phenomenon is probably due to disturbances in the neural connections between the three sensory systems (somatosensory, vestibular, and vision) that can lead to mismatched sensory input, causing conflicts among all inputs from the different sensory systems.^{1,2,25,27-29} Moreover, our results demonstrated a strong positive linear relationship between DVA and neck muscle fatigue.^{7,30} This is likely because neck muscle fatigue has been shown in previous literature to modify the discharge of sensory receptors in neck muscles and affect proprioception.^{1,2,4,12} Consequently, neck muscle fatigue may affect the neural connections between the three sensory systems, which may be the main cause of the gain increase in VOR.^{1,14} Thus, when assessing vestibular function in patients complaining of dizziness and/or visual disturbance with a history of neck trauma, one may speculate that VOR dysfunction could have a cervical origin due to somatosensory disturbance, which may lead to visual disturbances and dizziness. We identified several limitations in the present study. Our study was conducted on healthy normal young adults. Future research should include healthy adults of various ages. Also,

future studies should include patients with whiplash associated disorder (WAD) in order to measure the effects of neck trauma and pain on dynamic visual acuity. Finally, the effects of neck muscle fatigue on DVA should be conducted in standing versus sitting.

Conclusion

The results of this study suggest that neck muscle fatigue negatively impacts dynamic visual acuity. Although not statistically significant, cervical spine proprioception in the experimental group was diminished after fatigue as measured by the JPE. The clinical application of these findings suggests that patients with CGD may experience improved DVA and cervical proprioception through rehabilitation efforts directed at reducing cervical muscle fatigue.

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Suppliers

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CHAPTER FOUR
THE INFLUENCE OF NECK MUSCLE FATIGUE ON DYNAMIC
VISUAL ACUITY

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Abstract

Background and purpose: Postural and visual stability are dependent upon efficient and accurate central processing of visual, vestibular, and proprioception afferent input. Inaccurate sensory information from dysfunctional sensory end organs leads to a sensory mismatch, causing postural and/or visual instability and symptoms of dizziness or motion sensitivity. The interaction between cervical spine proprioception and the vestibular ocular reflex (VOR) has been observed in a very limited number of studies and its impact on the human VOR is not fully understood. The purpose of this study was to determine the effect of neck muscle fatigue on dynamic visual acuity in healthy participants.

Methods: Thirteen healthy adults ages 41 to 50 years participated in this study. Seven participants were randomized into an experimental group and six into a control group. The experimental group performed an exercise designed to induce neck muscle fatigue and the control group performed a non-fatiguing sham exercise. Primary outcome measures were the dynamic visual acuity (DVA), cervical joint position error (JPE), and Modified Visual Analog Scale (mVAS).

Results: There were significant differences in the mean DVA between the two groups ($.30 \pm .10$ Vs $.001 \pm .02$, $p = .003$), and in mean mVAS score (5.00 ± 1.53 Vs 0.00 ± 0.00 , $p = .001$). Participants who were fatigued had significantly poorer DVA than those who were not fatigued. There were, however, no significant differences in mean JPE between the two groups ($p = .13$)

Discussion and conclusions: Neck muscle fatigue decreased DVA. Our research suggests that VOR dysfunction can have a cervical origin due to somatosensory disturbance leading to visual disturbances.

Background and Purpose

Gaze stability is dependent upon a healthy interaction between the visual, vestibular, and somatosensory systems.¹⁻³ Cervicogenic dizziness is an area of physical therapy practice that directly considers the cervical spine contribution to postural and oculomotor impairments. Neck muscle fatigue and pain have been shown to modify the discharge of sensory receptors affecting cervical spine joint position sense and postural stability.⁴⁻⁶ Oculomotor deficits including abnormal smooth pursuit and vestibulo-ocular reflex (VOR) impairments have also been observed in patients with cervical whiplash injuries.^{7,8} The interaction between cervical spine proprioception and the VOR has been observed in a very limited number of studies and its impact on the human VOR is not fully understood.^{1,8,9} The central nervous system (CNS) receives and processes sensory input from the somatosensory, vestibular, and visual systems. Resultant motor responses contribute to gaze stability and postural control.^{1,2} The somatosensory system detects peripheral stimuli from sensory receptors, including mechanoreceptors located in human skin.² Mechanoreceptors (Pacinian corpuscles, Meissner's corpuscles, Merkel's discs, and Ruffini corpuscles) mediate peripheral stimuli including pressure, touch, pain, temperature, and proprioception.^{1,2,10} There are an abundance of mechanoreceptors in the upper cervical spine that primarily transmit impulses through nerve cells originating from C2 dorsal root ganglion.^{1,2} The mechanoreceptor input from the upper cervical segments (Occiput-Atlas, Atlas-Axis, Axis-C3), particularly from the upper cervical spine muscles, report directly to the vestibular nuclear complex (VNC) and the superior colliculus.^{1,2} The upper cervical spine mechanoreceptors also converge in the central cervical nucleus (CCN), which serves as a pathway to the cerebellum for integrating and organizing vestibular, ocular, and proprioceptive sensory input.^{1,2,11} The CCN also sends coordinated information to the cerebellar cortex for maintenance of postural equilibrium and body

orientation.^{1,2,11} Cervical spine reflexes contribute to head orientation, eye movement control, and postural stability; cervical collic reflex (CCR), cervical ocular reflex (COR), and cervical spinal reflex (CSR), respectively.^{1,2} These cervical reflexes work in conjunction with vestibular collic reflex (VCR), vestibular ocular reflex (VOR), and the vestibular spinal reflex (VSR).^{1,2} Abnormal sensory input from the vestibular and upper cervical spine can lead to dizziness, unsteadiness, and visual disturbance.^{1,2} The purpose of this investigation was to determine the effects of cervical muscle fatigue on dynamic visual acuity (DVA) and Joint Position Error (JPE).

Methods

The study was conducted in the Department of Physical Therapy, Loma Linda University, Loma Linda, California. Thirteen healthy adult participants ages 41 to 50 years participated in the study. Seven participants (4 males; 3 females) were randomized into the experimental group and six participants (3 males; 3 females) into the control group. Informed consent was acquired prior to the beginning of the study. The study protocol was approved by the Institutional Review Board at Loma Linda University.

Potential participants were recruited based on the following criteria; healthy adults between the ages of 41 to 50 years without current cervical spine pain or history of cervical spine whiplash injury. Participants were required to pass all comprehensive screening tests to ensure normal function of the somatosensory, visual, and vestibular systems. Screening tests included all of the following: cervical stability (alar ligament test, Sharp-Purser test, and lateral shear test), cervical vascular screening using the modified vertebral artery test, vestibular system screening (Hallpike-Dix Test, Roll Test, Head Thrust Test, Head Shaking-Induced Nystagmus Test, and condition 5 of the NeuroComSMART Balance Master^{®12}, static and dynamic visual acuity testing, and cervical spine

somatosensory integrity testing (JPE). To be included in the study, all screening test results had to be negative. The two main outcome variables were the VOR, as measured by the computerized dynamic visual acuity (cDVA), and cervical joint position sense, as measured by the JPE test. The VOR and JPE measurements were taken pre and post intervention. We recruited a cohort of 13 participants. A post power analysis with an effect size of 1.1 according to the change in DVA between cervical spine muscle fatigue and control groups, an alpha level of .05, and a sample size of 13 indicated that the power was .85.

Data were collected for all participants between 8am and 12pm to minimize the effects of fatigue.¹³ Randomization of group assignment was accomplished using a computer generated random sequence. Data was collected by researchers blinded to group assignment. Participants were instructed not to reveal intervention allocation to the researchers during data collection. We used the computerized DVA (cDVA) InVision™ system to determine the participant's ability to accurately perceive objects while actively moving their head.^{14,15} The cDVA is commonly used when evaluating patients with possible VOR dysfunction and other vestibular deficits.^{1,15-17} Static visual acuity was determined followed by dynamic visual acuity (Figure 13).¹⁶

The difference between the two test scores was calculated and the result was converted into LogMar, which is a measure of visual acuity loss.^{15,16} According to Herdman et al¹⁶, changes of one line (0.1 LogMAR) or less (≤ 0.1) are considered normal and changes of two or more lines (≥ 0.2) are abnormal.¹⁶ The cDVA is a reliable and sensitive tool for identifying patients with vestibular dysfunction resulting from an impaired VOR.¹⁵⁻¹⁷ The sensitivity of the cDVA is reported to be 94.5% and specificity 95.2%.¹⁶ The positive predictive value is 96.3% and the negative predictive value is 93%.¹⁶ We used the JPE test to measure the cervical spine somatosensory system.¹⁸ Participants sat on a chair 90cm away from a fixed target on a wall while wearing a head strap with a top-

mounted laser pointer (Figure 14).¹⁸⁻²¹ Participants were instructed to focus on the target then close their eyes and remember the starting position of the head, specifically the “zero target”. Keeping their eyes closed, participants maximally rotated their head to one side, and then returned to the starting position as accurately as possible.¹⁹⁻²¹ No speed instructions were given.^{18,20,21} The new position of the laser light was noted and the distance from the starting point was measured (reposition error distance).^{12,22} All participants repeated the same test ten times to each side in a random order.^{18,19} The head repositioning absolute value (AE) in centimeters was calculated.^{18,19} A normal JPE is less than or equal to 4.5 degrees.^{1,18,19,22,23}

We used the mVAS to subjectively measure the participant’s upper posterior neck muscle fatigue level.²⁴ Participants in both groups rated their level of fatigue before and after the intervention. The mVAS was a number scale from 0 to 10.^{24,25} A score of 0 was defined as no fatigue at all and 10 was defined as the most fatigue imaginable.²⁵ Participants in the experimental group sat on a customized neck exercise machine. With their head and neck in a neutral position, participants isometrically resisted a weighted stack load for 5 minutes (Figure 15).⁶ The load used in the weight stack was equal to 30% of the participant’s maximum voluntary contraction (MVC)^{6,24} which was determined for each subject using a dynamometer.

Participants performed 3 MVC’s and the average reading obtained from the dynamometer (in pounds) represented the MVC for each participant.^{6,24} In order to limit trunk muscles from contributing during the contraction, a normal gait belt was used during the MVC and the 5 minute isometric exercise to stabilize their trunk to the back support of the neck exercise machine.⁶ Participant’s neck fatigue level was taken immediately following the 5 minute isometric exercise.²⁴ Participants in the control group sat on a chair facing a wall while wearing a head strap with a top-mounted laser pointer attached. Four

targets of the same shape and width (center, down, left, and right) were fixed on the wall at the subject's eye level. The sham exercise began with the participant's head in a neutral position (eyes straight ahead). Participants pointed the laser inside the large circle of the center target. From this starting position, participants were instructed to point the laser inside the second target by moving their head slightly then returning the laser to the center target. Participants repeated the same activity for all targets as follows: center-down, center-right, and center-left for a total of 5 minutes (including two 1 minute breaks). This sham exercise was designed to not fatigue the cervical muscles. The participant's eyes were open during this exercise and no speed instructions were given.



FIGURE 13. Computerized dynamic visual acuity test.



FIGURE 14. Head-mounted laser pointer for joint position error testing.



FIGURE 15. Cervical neck muscle fatigue machine.

Data was analyzed using the statistical package SPSS for Windows version 19.0 (SPSS, Inc., Chicago, IL).^a Frequencies and relative frequencies were computed for gender, and means and standard deviations (SD) for the continuous variables age, DVA, and JPE. The chi-square test of independence was used to assess the relationship between gender and type of group at baseline, and the *Kolmogorov-Smirnov test* was applied to test for the normality of continuous variables. The Mann-Whitney U-test was performed to compare the mVAS scores between the two groups at baseline. The difference between mVAS, DVA, and JPE values at baseline and at the end of the study were calculated and these differences were compared between the two groups using Mann-Whitney U-test. Spearman's correlation was used to examine the relationship among DVA, JPE, and mVAS at the end of the study. The level of statistical significance was set at $p < .05$.

Results

Seven participants were randomized into the experimental group and six into the sham (control) group. The demographic and baseline clinical characteristics of the two study groups are presented in Table 4. There were no significant differences between the two groups at baseline for gender, age, height (cm) and weight (kg), DVA, JPE, and mVAS ($p > .05$). At the end of the study, there were significant differences in the mean DVA between the two groups ($.30 \pm .10$ Vs $.001 \pm .02$, $p = .003$, Table 5), and in mean mVAS score (5.00 ± 1.53 Vs 0.00 ± 0.00 , $p = .001$, Table 5). Participants who were fatigued had significantly poorer DVA than those who were not fatigued (Figure 16). There were, however, no significant differences in mean JPE between the two groups ($p = .13$, Table 2). In the experimental group, there were no significant correlations between DVA and mVAS score ($\rho = .63$, $p = .13$). Also, no significant correlations were observed between the mVAS score and JPE ($\rho = .43$, $p = .33$).

TABLE 4. Mean (SD) of baseline characteristics at baseline for the participants in the experimental group and control group (N=13)

Variables	Experimental (n=7)	Control (n=6)	P-Value
Gender ⁺			
Male	4 (57%)	3 (50%)	.62*
Female	3 (43%)	3 (50%)	
Age (years)	44.0 (2.4)	44.5 (2.4)	.67**
DVA	.05 (.03)	.08 (.06)	.35**
JPE	5.50 (1.39)	5.29 (.94)	.89**
mVAS	0.0 (.0)	0.0 (.0)	

DVA= Dynamic Visual Acuity, JPE = Joint Position Error, mVAS = modified Visual Analog Scale.

* Fisher's exact test.

** Mann-Whitney U-test.

⁺ Results are presented as frequency (%).

TABLE 5. Comparison of mean (SD) of post minus pre DVA , JPE, and mVAS between the two groups

Variables	Experimental (n=7)	Control (n=6)	P-Value*
DVA	.30 (.10)	.001 (.02)	.003
JPE	.34 (.48)	.02 (.21)	.13
mVAS	5.00(1.52)	.00 (.00)	.001

DVA=Dynamic Visual Acuity, JPE= Joint Position Error, mVAS = Modified Visual Analog Scale

* Mann Whitney U test.

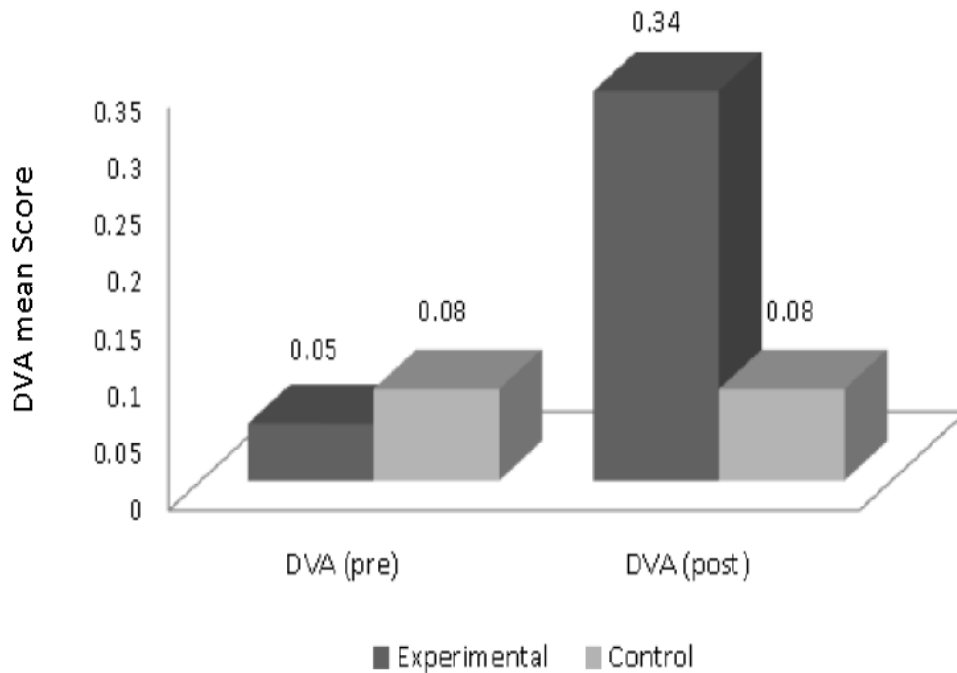


FIGURE 16. Comparison of Dynamic Visual Acuity (DVA) by Group and Time Assessment.

Discussion

The purpose of this study was to determine the effect of neck muscle fatigue on dynamic visual acuity in healthy participants. Thirteen healthy adults were randomly assigned into either a neck muscle fatigue group or a sham (control or sham exercise) group. Dynamic visual acuity test scores were used to measure the gaze stability and VOR function and JPE test scores were used to measure cervical joint repositioning accuracy. Our results suggest that neck muscle fatigue negatively impacts DVA. Although not statistically significant, a trend was observed suggesting that neck muscle fatigue negatively impacts JPE, as determined by less accurate and less consistent repositioning performances in the fatigue group versus sham (control or sham exercise) group. Our findings are consistent with existing literature, which reports that reduced proprioceptive

acuity contributes to sensory mismatches and possibly an asymmetry of the VOR.^{1,8} This phenomenon is likely due to disturbances in the neural connections between the three sensory systems (somatosensory, vestibular, and vision) causing conflicts among inputs from the different sensory systems.^{1,2,11,26-28}

Thus, when assessing vestibular function in patients complaining of dizziness and/or visual disturbance with a history of neck trauma, one may speculate that VOR dysfunction could have a cervical origin due to somatosensory disturbance, which may lead to visual disturbances and dizziness. Future research should include patients with whiplash associated disorder (WAD) in order to measure the effects of neck trauma and pain on dynamic visual acuity. Also, the effects of neck muscle fatigue on DVA should be conducted in standing versus sitting.

Conclusion

The results of this study suggest that neck muscle fatigue impairs DVA and cervical spine proprioception. Our research suggests that VOR dysfunction can have a cervical origin due to somatosensory disturbance leading to visual disturbances.

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CHAPTER FIVE

DISCUSSION

Cervicogenic dizziness (CGD) is often the result of a sensory mismatch between the vestibular, somatosensory, and visual afferent inputs. Physical trauma involving the cervical spine, such as whiplash injury, is a common mechanism of injury in CGD patients. Physical trauma contributes to impairment in the upper cervical spine proprioceptive input leading to symptoms including disequilibrium and dizziness. Gaze stability is dependent upon a healthy interaction between the visual, vestibular, and somatosensory systems.¹⁻³ Neck muscle fatigue and pain have been shown to modify the discharge of sensory receptors affecting cervical spine joint position sense and postural stability.⁴⁻⁶ Oculomotor deficits including abnormal smooth pursuit and vestibulo-ocular reflex (VOR) impairments have also been observed in patients with cervical whiplash injuries.^{7,8} The interaction between cervical spine proprioception and the VOR has been observed in a very limited number of studies and its impact on the human VOR is not fully understood.^{1,8,9} The purpose of the present work was to determine the effect of neck muscle fatigue on dynamic visual acuity in humans. Thirty young healthy participants between the ages of 21 to 30 were randomly assigned into either a neck muscle fatigue group or a sham group. Thirteen healthy adults between the ages of 41 to 50 were also randomly assigned into either a neck muscle fatigue group or a sham. Dynamic visual acuity test scores were used to measure the gaze stability, and JPE test scores were used to measure cervical joint repositioning accuracy. Our results determined that neck muscle fatigue negatively impacts DVA. Although not statistically significant, a trend was observed suggesting that neck

muscle fatigue negatively impacts JPE, as determined by less accurate and less consistent repositioning performances in the fatigue group versus sham group in both aging groups. Our findings were consistent with existing literature, which reports that reduced proprioceptive acuity contributes to sensory mismatches and possibly an asymmetry of the VOR.^{1,14} This phenomenon is probably due to disturbances in the neural connections between the three sensory systems (somatosensory, vestibular, and vision) that can lead to mismatched sensory input, causing conflicts among all inputs from the different sensory systems.^{1,2,25,27-29} Moreover, the results of this study demonstrated a strong positive linear relationship between DVA and neck muscle fatigue. This is likely because neck muscle fatigue has been shown in previous literature to modify the discharge of sensory receptors in neck muscles and affect proprioception.^{1,2,4,12} Consequently, neck muscle fatigue may affect the neural connections between the three sensory systems, which may be the main cause of the gain increase in VOR.^{1,14} Thus, when assessing vestibular function in patients with a history of neck trauma complaining of dizziness and/or visual disturbance, one may speculate that VOR dysfunction could have a cervical origin due to somatosensory disturbance, which may lead to visual disturbances and dizziness. We identified several limitations in the present study. Future research should include healthy participants of various ages. Also, future studies should include patients with whiplash associated disorder (WAD) in order to measure the effects of neck trauma and pain on dynamic visual acuity. Finally, the effects of neck muscle fatigue on DVA should be conducted in standing versus sitting. The results of this present work suggest that neck muscle fatigue negatively impacts dynamic visual acuity. Therefore, VOR dysfunction can have a cervical origin due to somatosensory disturbance leading to visual disturbances. Although not statistically significant, a trend was observed suggesting that neck muscle fatigue negatively impacts

JPE, as determined by less accurate and less consistent repositioning performances in the fatigue group versus sham (control or sham exercise) group.

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APPENDIX A
DATA COLLECTION SHEET

SECTION 1

*Subjects ID: CGD _____ *Date: _____ *Gender: _____

*Age: _____ *Height: _____ *Weight: _____

*BMI: _____ *Occupation: _____ *Dominant Hand: _____

SECTION 2

*Modified Visual Analog Scale (mVAS): Before Exercise (/ 10); After exercise (/ 10)

* Alar Ligament Test: (+/-) * Sharp-Purser Test: (+/-)

* Lateral Shear Test: (+/-) * Head Thrust Test: (+/-)

* Hallpike Dix Test: (+/-) * Roll Test: (+/-)

* Condition Five: (+/-)

*Computerized Dynamic Visual Acuity (cDVA) – *Before Exercise:*

Test Results	
Left Direction	Right Direction
_____ LogMAR (+/-)	_____ LogMAR (+/-)

*Joint Position Error (JPE) – Before Exercise:

Trial #	Test Results (centimeter)		Test Results (degrees)	
	Left Direction	Right Direction	Left Direction	Right Direction
(1)				
(2)				
(3)				
(4)				
(5)				
(6)				
(7)				
(8)				
(9)				
(10)				
	Average:____ (+/-)	Average:____ (+/-)	Average:____ (+/-)	Average:____ (+/-)

NOTE: If any section 2 tests are positive, sections 3 is skipped and sections 4 and 5 signed.

SECTION 3

*Computerized Dynamic Visual Acuity (cDVA) – After Exercise:

Test Results	
Left Direction	Right Direction
_____ LogMAR (+/-)	_____ LogMAR (+/-)

*Joint Position Error (JPE) – After Exercise:

Trial #	Test Results (centimeter)		Test Results (degrees)	
	Left Direction	Right Direction	Left Direction	Right Direction
(1)				
(2)				
(3)				
(4)				
(5)				
(6)				
(7)				
(8)				
(9)				
(10)				
	Average:____ (+/-)	Average:____ (+/-)	Average:____ (+/-)	Average:____ (+/-)

SECTION 4

The subject didn't pass the following test(s) in section (2) _____. The subject was given the results of the evaluation and advised to follow up with his/her physician for further medical consultation.

Subject Signature: _____ Date: _____

Investigator Signature: _____ Date: _____

SECTION 5

All information on this form has been reviewed and is complete and correct.

Reported by: _____ Signature: _____

Supervised by: Dr. Eric Johnson OR Dr. Amer Al Saif

APPENDIX B
INFORMED CONSENT

The Effects of Neck Muscle Fatigue on the Vestibulo-Ocular Reflex as Measured by
Computerized Dynamic Visual Acuity

Purpose and Procedures

You are invited to participate in this research project because you are a normal, healthy individual between the ages of 21–30 or 41–50 years old. Screening will be performed on all potential research participants, all to determine normal balance function of the head, neck, and body.

The purpose of this project is to investigate whether altered balance information from the cervical spine caused by neck muscle fatigue directly influences visual stability in healthy individuals.

If you decide to participate, your date of birth, height and weight will be recorded. You will be required to fill out several questionnaires before and after testing. We will use a random number table to place you into either an “experimental group” or a “control group.” Each group will perform a different neck exercise protocol for five minutes. We will measure your visual stability using a device called the Dynamic Visual Acuity. The Dynamic Visual Acuity measures the clarity of vision while actively moving your head. We will also measure your neck position sense using a test called the Joint Position Error test. The Joint Position Error test measures your neck position sense with your eyes closed. These two measurements will be taken in both groups before and after testing. The entire process will take approximately 60 minutes.

_____Initial

_____Date

Risks

Participating in this study involves minimal risk. A mild to moderate discomfort (soreness) may be felt in the upper neck extensor muscles similar to working out at the gym. In order for us to minimize the risk as much as possible, we will perform a series of evaluations to assess cervical stability. The evaluations will not be uncomfortable and if you don't pass any of the evaluations you will not be included in the study. Also, in the event that you don't pass any of the evaluations, we will give you the results of the evaluations and advise you to follow-up with your physician for further medical consultation. We will also ensure that the neck exercises are performed correctly and smoothly.

Benefits

You are not likely to personally benefit from participation in the study; however, the expected benefit to science is to determine if neck muscle fatigue affects visual stability when a person's head is moving.

Participant's Rights

Participation in this study is voluntary. Your decision whether or not to participate or terminate at any time will not affect your present or future relationship with the Loma Linda University Department of Physical Therapy.

Confidentiality

Any published document resulting from this study will not disclose your identity and your name will not appear on the data collection form. An ID number will be used instead of your name.

Additional Costs/ Reimbursement

There is no cost to you for your participation in this study beyond the time involved to participate. You will receive a \$5 Starbucks gift card after completing the study.

Impartial Third Party Contact

If you wish to contact an impartial third party not associated with this study regarding any question or complaint you may have about the study, you may contact the Office of Patient Relations, Loma Linda University Medical Center, Loma Linda, CA 92354, phone (909) 558-4674, e-mail patientrelations@llu.edu for information and assistance.

_____ Initial

_____ Data

Informed Consent Statement

I have read the contents of the consent form and have listened to the verbal explanation given by the investigators. My questions concerning this study have been answered to my satisfaction. I hereby give voluntary consent to participate in this study. I have been given a copy of this consent form. Signing this consent document does not waive my rights nor does it release the investigators, institution, or sponsors from their responsibilities. I may call and leave a voice message for Eric Johnson, DSc during routine office hours at this number (909) 558-4632 ext. 47471 or e-mail him at ejohnson@llu.edu, if I have additional questions and concerns. I have been given a copy of this consent form.

Signatures

Signature of Subject

Date

I have reviewed the contents and this consent form with the person signing above. I have explained potential risks and benefits of this study.

Investigator

Date

APPENDIX C

CERVICOGENIC DIZZINESS: AN EMERGING AREA OF CLINICAL PRACTICE

Eric G. Johnson, DSc, PT, NCS¹ and Amer A. AlSaif, MPT, DPT²

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²PhD Candidate, Loma Linda University, Department of Physical Therapy, Loma Linda, CA.

Abstract

Cervicogenic Dizziness (CGD) is an emerging area of physical therapy clinical practice. The past medical history of patients with CGD often includes cervical spine trauma and the physical examination usually reveals cervical spine impairments contributing to mechanical compression of the vertebral artery network, irritation of the cervical sympathetic nervous system, and/or impaired neck proprioception. Patients with CGD typically describe their dizziness as a vague sense of impaired orientation or disequilibrium that has a close temporal relationship with neck pain. Physical therapy interventions for CGD include orthopedic and vestibular rehabilitation strategies. The purpose of this paper is to introduce the etiology of cervicogenic dizziness and provide a brief overview of the physical therapy examination and intervention process.

Introduction

Cervicogenic Dizziness (CGD) is an emerging area of physical therapy clinical practice. Although CGD has been a topic of academic discussion for more than a decade, it has not been widely adopted into clinical practice. Progress is being made to bridge this gap and the American Physical Therapy Association (APTA) now offers a certificate of competency in CGD through successful completion of an evidenced-based post-graduate course.¹ Certification in CGD serves as an excellent complement to the APTA certificate of competency in vestibular rehabilitation.² Effective clinical management of dizziness is an integral component of improving balance. Balance is dependent upon normal interaction between the visual, vestibular, and somatosensory systems.^{3,4} Within the central nervous system, multimodal sensory integration facilitates motor responses to coordinate head and body orientation, postural stability, and gaze stability during head movements.³ In the absence of normal sensory integration, imbalance and dizziness often occur.³⁻⁶ Dizziness is

one of the most common medical problems in the United States and other countries, especially in the elderly population.^{7,8} Dizziness is caused by numerous pathologies including viral or bacterial infection, head trauma, neurological disease, psychological conditions, orthostatic hypotension, migraine and/or headache disorders, pharmacology, vertebrobasilar insufficiency, and vestibular disorders.^{3,4,9} Dizziness can also be caused by impairments in the cervical spine and, in such cases, is referred to as “cervicogenic dizziness.”^{3,9} Cervicogenic dizziness has been defined as “a non-specific sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck.”⁹ Symptoms of CGD are usually not as intense as vestibular-driven dizziness and it rarely includes vertigo.⁹ There is a growing body of evidence supporting the pathophysiology and physical therapy management of patients with CGD; however, it is still not widely adopted in clinical practice. Therefore, the purpose of this paper is to provide an overview of the theory and clinical application of managing patients with CGD.

Etiology of Cervicogenic Dizziness

There are several proposed mechanisms responsible for causing CGD. These include mechanical compression of the vertebral artery network, irritation of the cervical sympathetic nervous system, and abnormal proprioceptive input from the upper cervical spine.^{3,4,9-11} Muscular tightness in the cervical spine region can lead to mechanical compression of the vertebral artery network and lead to symptoms consistent with vertebrobasilar insufficiency (VBI).^{10,11} The vertebrobasilar arterial network begins with the aortic arch giving rise to the subclavian arteries; which, in turn, give rise to the right and left vertebral arteries.¹¹ The vertebral arteries travel superiorly through the transverse foramen of C6-C1 then travel horizontally around the posterior arch of the

atlas, where they ultimately enter the foramen magnum and merge with one another to form the basilar artery.^{10,11} During the anatomical pathway from the subcalvian arteries, the vertebral arteries navigate several muscular and bony relationships that are capable of producing mechanical compromise.¹¹ In the presence of upper cervical spine muscle tightness or muscle imbalance, neck rotation can potentially compromise the vertebrobasilar and collateral arteries, leading to brainstem ischemia and symptoms consistent with VBI.¹¹⁻¹² The vertebral arteries travel between the anterior scalene and longus colli muscles as well as under the inferior capitis oblique and intertransversarius muscles.⁹ Bony abnormalities and poor cervical spine posture can also contribute to vertebral artery compression and symptoms consistent with VBI.^{11,12} (Figure 1)

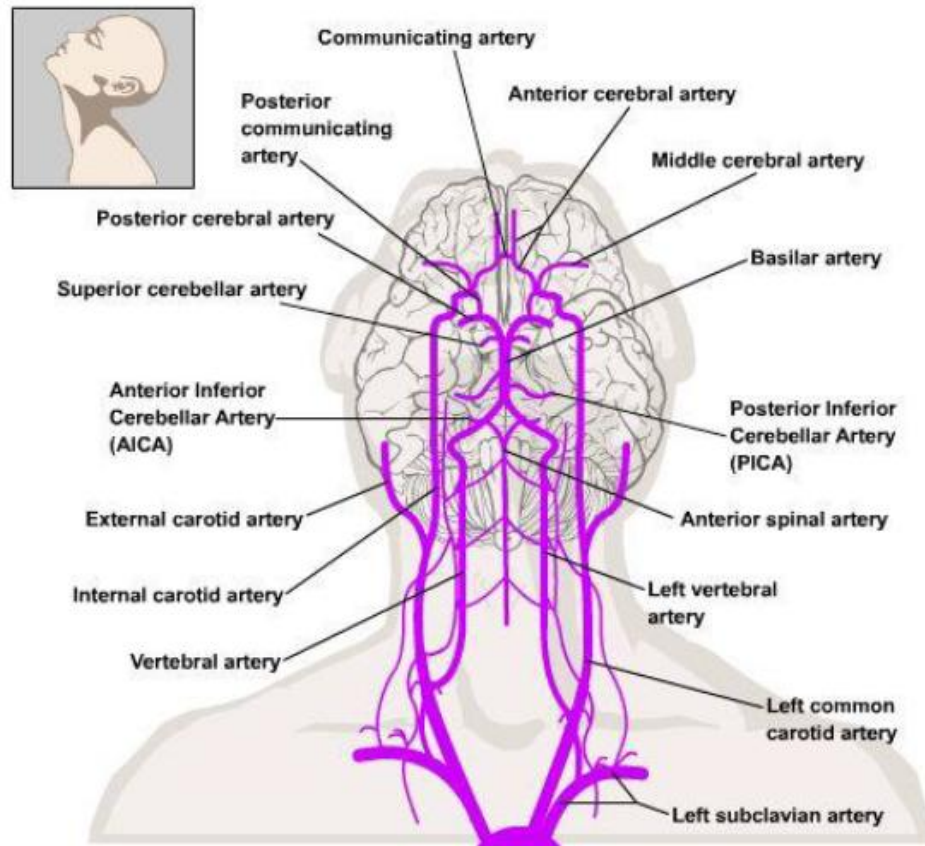


FIGURE 1. Vertebrobasilar Arterial Network.¹³

Another potential source of CGD is Irritation of the cervical sympathetic nervous system.^{3,11} The cervical sympathetic nervous system travels antero-lateral to the cervical vertebral bodies adjacent to the arterial network and cervical musculature.¹⁰⁻¹² The largest of the cervical spine ganglion, the superior cervical sympathetic ganglion, is located at the level of the second and third cervical spine posture can potentially compromise this ganglion, causing vertebral and carotid arterial network hypoperfusion and CGD.¹⁰⁻¹² Abnormal upper cervical spine proprioceptive input, resulting from ischemia, inflammation, or cervical extensor muscle fatigue has also been implicated in patients with imbalance and CGD.^{3,9,14-21} Somatosensory information in the cervical spine

can be impaired by direct neck trauma, such as whiplash associated disorder (WAD).^{3,9,12,15,17,18,20} The resultant injury, or WAD, can lead to cervical spine muscle fatigue and modify the discharge firing rate of sensory receptors affecting joint position sense of the head and neck as well as postural stability.^{5,14-16,18-20} Approximately 60% of all WAD patients develop dizziness.^{15,17} It is proposed that a sensory mismatch occurs between the multimodal sensory inputs from the upper cervical spine and other systems contributing to postural stability and head orientation. This results in symptoms consistent with CGD.^{3-5,14-19} Abnormal extraocular motor function has also been described in the literature after WAD.^{3,22} Tjell and Rosenhall²² reported abnormal smooth-pursuit eye movements when the neck was rotated under a stable head in WAD patients. The authors of the current paper also theorize that abnormal upper cervical spine sensory input impairs the vestibular ocular reflex causing impaired dynamic gaze stability. This theory is based on existing interactive upper cervical spine and vestibular nuclei neural pathways.^{3,4,23,24} Interactive systems of cooperative cervical and vestibular reflexive systems contributing to head and neck orientation (cervico-collic reflex and vestibulo-collic reflex), postural control (cervico-spinal reflex and vestibulo-spinal reflex), and oculomotor control (cervico-ocular reflex and vestibulo-ocular reflex) also exist.^{4,5,23,24} These reflexes are dependent upon healthy integrative central nervous system communication, particularly with the vestibular nuclear complex, in order to maintain normal postural balance and visual stability.^{4,5,23,24} (Figure 2)

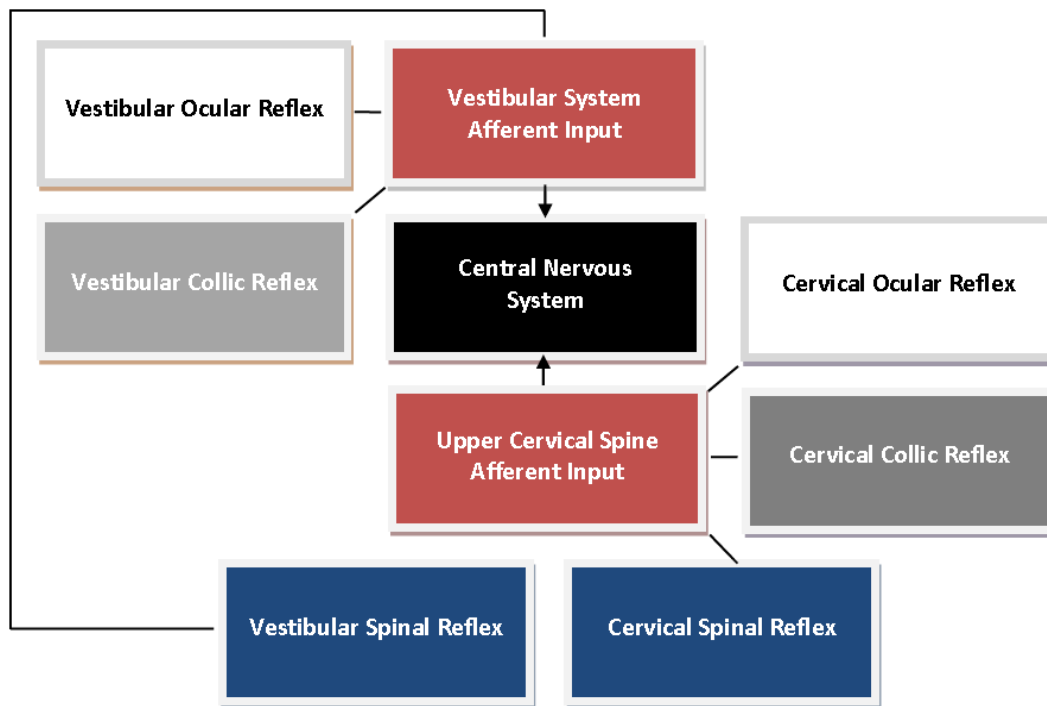


FIGURE 2. Cooperative reflexes between the vestibular system and the cervical spine.

Physical Therapy Examination

The physical therapy examination process requires clinical decision-making that is based on the best available evidence. This process includes subjective and physical examination components. The subjective examination is an important first step in guiding the physical therapist to formulate a clinical hypothesis. During the subjective examination, the physical therapist reviews the patient's past medical history and asks questions aimed at extracting a specific description of the patient's dizziness.⁹ In order for the clinical hypothesis to ultimately arrive at CGD, competing causes of dizziness (described previously) must be eliminated. In particular, the tempo, symptoms, circumstances, and intensity of the patient's dizziness must be determined.⁹ Patients with CGD typically report their dizziness tempo as episodic events lasting several minutes to several hours. Symptoms

are often described as a vague sense of disequilibrium or lightheadedness often accompanied by visual disturbances. Vertigo is usually not reported as a chief complaint. Circumstances that frequently produce or exacerbate their symptoms include neck pain. Additionally, the clinician should determine the patient's current dizziness intensity as well as the range of dizziness over the previous 72 hours using the modified visual analog scale.^{3,9} By collecting this subjective information, the physical therapist can begin to establish a clinical hypothesis.^{7,14,17} The physical examination includes a medical screening component followed by specific tests to assist the physical therapist in determining if CGD is the problem (Table 1). The medical screening component includes screening of cervical spine stability, integrity of the cervical vascular system, and integrity of the central nervous system.^{3,9,11} If the medical screening examination is abnormal, the patient should be referred to a physician for further medical consultation. Otherwise, the physical examination continues with postural analysis, palpation, and range of motion testing.²⁵ Central and peripheral vestibular system testing is performed to determine if the dizziness is being caused by benign paroxysmal position vertigo (BPPV) or vestibulo-ocular reflex (VOR) hypofunction.⁹ If BPPV or VOR hypofunction is found, the appropriate vestibular rehabilitation interventions are implemented.⁹ If central vestibular testing is positive, the patient should be referred to a physician. If vestibular testing is normal, or inconclusive, the physical examination continues with specific tests to determine if the dizziness is coming from the cervical spine (CGD).⁹ The neck torsion nystagmus test (NTNT) is performed by having the patient rotate their body under a stabilized head (Figure 3).²² The NTNT is positive if nystagmus is elicited. The neck torsion smooth pursuit (NTSPT) is performed by first observing ocular smooth pursuit with the patient's head in neutral. The test is then repeated with the patient's body/neck rotated under their stable head.²² A positive NTSPT results when smooth pursuit is normal in the neutral position and abnormal when the

neck is in the rotated position.²² The neck position provocation test (NPPT) is similar in that the patient rotated their body under a stabilized head. The NPPT is considered positive if their symptoms are produced in the latter position. The joint position error test (JPE) is performed by having the patient put on a pair of foveal glasses then look at a stable target on a wall.^{14,17} Foveal vision testing requires modifying glasses with tape to occlude all peripheral vision.^{14,17} The patient then closes their eyes and actively rotates their head and neck to end range. With eyes closed, the patient actively returns to what they think was the initial starting position then opens their eyes. The JPE test is considered positive if the patient is not able to see the stable target.^{14,17,26} The JPE test can also be performed using a head-mounted laser pointer by measuring the distance from the initial position to the returning position in centimeters (Figure 3).^{14,17} The manual traction test (MTT) is performed with the patient seated. The test is considered positive if compression increases or produces dizziness and traction relieves it.



FIGURE 3. Head mounted laser for cervical joint position error testing.

Physical Therapy Intervention

Physical therapy intervention targets specific impairments identified during the physical examination.^{3,9,14,17} Cervical spine pain and inflammation, hypomobility, proprioception, and cervical-ocular motor function are addressed as necessary.⁹ Cervical spine pain and inflammation can be managed with modalities such as cryotherapy, thermotherapy, ultrasound, and cervical spine traction. Cervical spine hypomobility can be managed with mobilization techniques and a tissue-specific and age-specific stretching program.^{9,10,27-30} Cervical spine proprioception impairments can be improved using postural awareness training, passive head movements with fixed target gaze exercises (clinician passively moves patients head while patient maintains fixed gaze on a stationary target), and foveal vision exercises.^{14,17} Foveal vision exercises are performed by having the patient wear foveal glasses and fix their gaze on a stationary wall mounted target.^{14,17} The patient closes their eyes and actively rotates their head, then returns to the starting position and opens their eyes. If the patient cannot see the target, they keep the eyes open and continues rotating their head until they can see the target.^{14,17} This biofeedback training provides the patient information about their cervical spine joint position sense.¹⁴ Cervical-ocular motor function can be managed using free eye-head coupling with pursuits (patient keeps head still while eyes follow a moving target) and saccades (patient keeps head still and quickly moves eyes between targets) as well as X1 (patient moves head from side to side while maintaining gaze on a stationary target) and X2 adaptation exercises (patient moves head and hand-held target in opposite directions while maintaining gaze on moving target at all times).^{14,17} All cervical-ocular exercises can be progressed by increasing the speed, duration, and frequency as tolerated.^{14,17}

Case Study

SUBJECT: The patient was a 24-year-old female college student with a history of chronic dizziness provoked by left neck rotation. The patient described episodic vague dizziness, without vertigo, and general neck stiffness that she attributed to prolonged study sessions over the previous four years. She did not recall any physical trauma to her head or neck. **EXAMINATION:** Postural assessment revealed a forward head position and elevated shoulders. The modified vertebral artery test (mVAT) was positive when performed to patient's left, provoking her symptoms of dizziness, and her verbal responses to questions being asked were markedly delayed. She was referred to her physician for further medical evaluation and a duplex Doppler ultrasound was ordered. The ultrasound findings were normal arterial patency in the bilateral carotid and vertebral arteries. The patient returned to physical therapy for further physical examination. Multiple tender points and stiffness were identified bilaterally in the upper trapezius, sternocleidomastoid, levator scapulae, and anterior scalene muscles through palpation and range of motion testing, respectively. At that time, the clinical hypothesis of cervicogenic dizziness was made. **INTERVENTION:** Manual therapy techniques were performed to improve muscle flexibility and reduce palpable tenderness. The left mVAT was negative after the initial treatment session and the patient was instructed to perform a cervical spine home stretching program and to work on her posture. **OUTCOME:** The patient returned for follow-up testing several weeks later and the mVAT remained negative. The patient reported that she was now able to rotate her head and neck to the left without symptom provocation. **CONCLUSION:** The patient's dizziness was likely the result of mechanical arterial pressure during cervical spine rotation given the muscle tightness and postural abnormalities. The physical therapy hypothesis of cervicogenic

dizziness was effectively managed using orthopedic intervention, home stretching program, and postural awareness training.

Conclusion

There is a growing body of evidence to support effective physical therapy management of CGD. Cervicogenic dizziness can be caused by a variety of problems including mechanical compression of the vertebral artery network, cervical sympathetic nervous system irritation, or impaired upper cervical spine proprioceptive input. The patient history may include physical trauma involving the head and neck and cervical spine postural faults are commonly observed. In order to determine the origin of the patient's dizziness, the physical therapist must conduct thorough subjective and physical examinations to rule out competing causes of dizziness. Once CGD has been confirmed, appropriate interventions are implemented to reduce cervical spine pain and inflammation, restore joint and soft tissue mobility, improve cervical spine proprioception, and improve cervical-ocular eye movement coordination.

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APPENDIX D

CERVICOGENIC DIZZINESS: THEORY AND CLINICAL APPLICATION

Eric G. Johnson, DSc, PT, NCS¹ and Amer A. AlSaif, MPT, DPT²

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Abstract

Dizziness is a common symptom resulting from a variety of medical conditions. An emerging area of physical therapy practice is the management of patients with cervicogenic dizziness. Patients with cervicogenic dizziness typically describe a vague sense of impaired orientation or disequilibrium that is exacerbated by neck pain. There is frequently a past medical history of cervical spine trauma, such as a whiplash associated disorder, and the physical examination often reveals orthopedic problems in the upper cervical spine causing mechanical compression of the vertebral artery network, irritation of the cervical sympathetic nervous system, and/or impaired upper cervical spine proprioception. The purpose of this paper is to introduce the etiology of cervicogenic dizziness and provide a brief overview of the physical therapy examination and intervention process.

Introduction

Balance is maintained through a complex interaction between the visual, vestibular, and somatosensory systems.^{1,2} Multimodal sensory integration occurs in the central nervous system where motor responses are generated to coordinate head and body orientation, postural stability, and gaze stability during head movements.¹ When normal sensory integration is impaired, imbalance and dizziness often occur.¹⁻⁴ Dizziness is one of the most common medical problems in many countries, including Asia and the United States, especially in the elderly population.^{5,6} Dizziness is a nonspecific symptomatic description resulting from numerous pathologies including viral or bacterial infection, head trauma, neurological disease, psychological conditions, orthostatic hypotension, migraine and/or headache disorders, pharmacology, vertebrobasilar insufficiency, and vestibular disorders.^{1,2,7} Dizziness can also be caused by orthopedic impairments affecting the

cervical spine and, in such cases, is referred to as “cervicogenic dizziness.”^{1,7} Cervicogenic dizziness has been defined as “a non-specific sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck.”⁷ It is different from vestibular-driven dizziness in that symptoms are usually not as intense and it rarely includes vertigo.⁷ It is an emerging area of clinical practice with a growing body of evidence that supports the pathophysiology and physical therapy management of patients with cervicogenic dizziness. Therefore, the purpose of this paper is to provide a brief overview of the theory and clinical application of managing patients with cervicogenic dizziness.

Etiology of Cervicogenic Dizziness

Proposed mechanisms causing cervicogenic dizziness include mechanical compression or stenosis of the vertebral artery network, irritation of the cervical sympathetic nervous system, and abnormal proprioceptive input from the upper cervical spine.^{1,2,7-9} Mechanical compression or stenosis of the vertebral artery network can lead to vertebrobasilar insufficiency (VBI).^{8,9} The anatomical pathway of the vertebrobasilar arterial network begins with the aortic arch giving rise to the subclavian arteries; which, in turn, give rise to the right and left vertebral arteries.⁹ The vertebral arteries ascend superiorly through the transverse foramen of C6-C1 before coursing horizontally around the posterior arch of the atlas, where they enter the foramen magnum and merge with one another to form the basilar artery.^{8,9} Over the course of their ascent from the subclavian arteries to the foramen magnum, the vertebral arteries navigate several muscular and bony relationships that could produce mechanical compromise.⁹ In the presence of upper cervical spine muscle tightness, cervical spine rotation can potentially compromise the vertebrobasilar and collateral arteries, leading to brainstem ischemia and symptoms

attributed to VBI.⁹⁻¹⁰ Specifically, the vertebral arteries travel between the anterior scalene and longus colli muscles as well as under the inferior capitis oblique and intertransversarius muscles⁷. Bony anomalies, including osteophytes and spondylosis as well as poor cervical spine posture, have also been suggested as possible contributors to vertebral artery compression and symptoms consistent with VBI.^{9,10} (Figure 1).

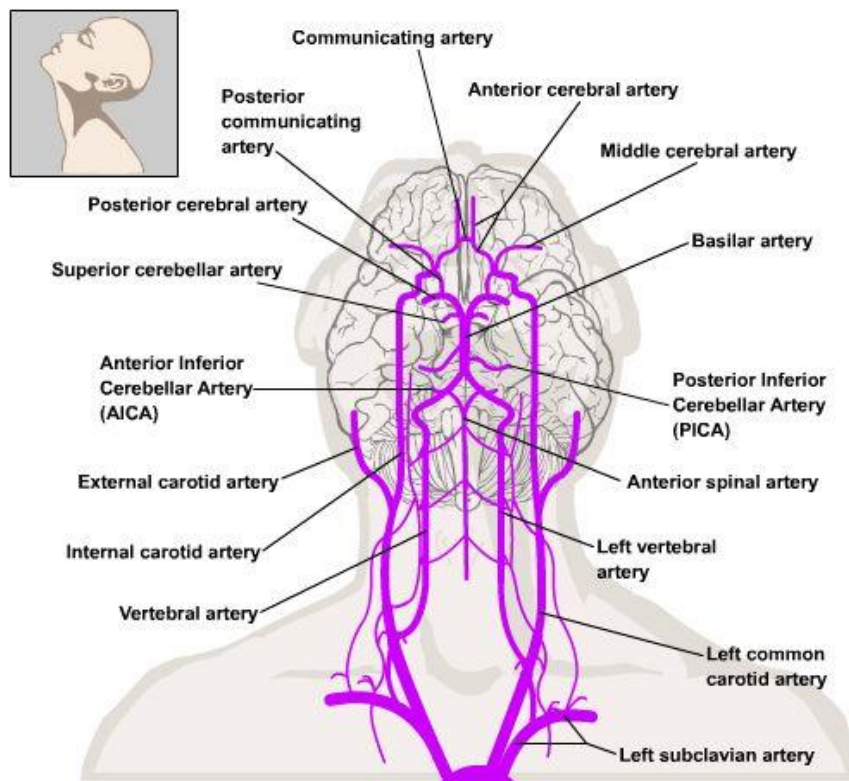


FIGURE 1. Vertebrobasilar Arterial Network. (Permission granted from Neuroscience Online Section II: Sensory Systems).¹¹

Irritation of the cervical sympathetic nervous system has also been theorized to contribute to cervicogenic dizziness.^{1,9} This system runs antero-lateral to the vertebral bodies that are adjacent to the arterial network and cervical musculature.⁸⁻¹⁰ The superior cervical sympathetic ganglion, the largest of the cervical spine ganglia, is located at the level of the second and third cervical vertebrae. Upper cervical spine muscle tightness, bony anomalies, and/or poor cervical spine posture could potentially compromise this ganglion, leading to hypoperfusion of the vertebral and carotid arterial network causing dizziness consistent with cervicogenic dizziness.⁸⁻¹⁰ Abnormal proprioceptive input from the upper cervical spine has also been implicated in patients with cervicogenic dizziness.^{1,7,12-15} Upper cervical spine afferent sensory disturbances resulting from ischemia, inflammation, or cervical extensor muscle fatigue may be responsible for the dizziness and imbalance experienced by these patients.^{1,7,16-19} Cervical spine somatosensory information can be altered by several mechanisms, including direct trauma to the neck, such as whiplash associated disorder.^{1,7,10,13,15,16,18} The resultant injury can lead to muscle fatigue, which has been shown to modify the discharge firing rate of sensory receptors affecting joint position sense of the head and neck as well as postural stability.^{3,12-14,16-18} As many as 60% of all patients with whiplash-associated disorder develop dizziness.^{13,15} It is postulated that a sensory mismatch occurs between the multimodal sensory inputs from the upper cervical spine and other systems that contribute to postural stability and head orientation, producing symptoms consistent with cervicogenic dizziness.^{1-3,12-17} Abnormalities in motor function of the eyes have also been described in the literature after cervical spine whiplash injuries.^{1,20} Tjell and Rosenhall²⁰ reported altered smooth-pursuit eye movements while the neck was rotated in post-whiplash patients. The authors of the current paper also theorize that abnormal sensory input from the upper cervical spine impairs the vestibular ocular reflex, negatively impacting dynamic gaze stability.

This theory is made on the basis of existing neural pathways between the upper cervical spine and the vestibular nuclei.^{1,2,21,22} There is also an interactive system of cooperative cervical and vestibular reflexive systems that contribute to head and neck orientation (cervico-colic reflex and vestibulo-colic reflex), postural control (cervico-spinal reflex and vestibulo-spinal reflex), and oculomotor control (cervico-ocular reflex and vestibulo-ocular reflex).^{2,3,21,22} Research is in progress to support or refute this theory.

Physical Therapy Examination

The physical therapy examination includes subjective and physical examination components. The subjective examination is performed first and is an important step in guiding the physical therapist to formulate a clinical hypothesis. It includes reviewing the patient's past medical history as well as asking questions concerning the characteristics of the patient's dizziness.⁷ This helps the clinician eliminate competing causes of dizziness (described previously). In particular, the clinician should determine the patient's dizziness tempo, symptoms, circumstances, and intensity.⁷ Typical subjective reports of dizziness tempo, symptoms, and circumstance consistent with cervicogenic dizziness are listed in Table 1. Additionally, the clinician should determine the patient's current dizziness intensity as well as the range of dizziness over the previous 72 hours using the modified visual analog scale.^{1,7} This allows the clinician to establish a baseline measurement for purposes of recording subjective progress and patient response to physical therapy examination and intervention during each physical therapy session.^{5,12,15} The physical examination includes a medical screening component followed by specific tests and measures to assist the clinician in determining if cervicogenic dizziness exists (Table 2). The medical screening component is performed first to measure cervical spine stability, integrity of the cervical vascular system, and integrity of the central nervous system.^{1,7,9} If the medical screening

examination is abnormal, the patient is referred back to the physician for further medical consultation. Otherwise, the physical examination continues with vestibular system testing to determine if the dizziness is being caused by benign paroxysmal position vertigo (BPPV) or vestibulo-ocular reflex (VOR) hypofunction.⁷ If BPPV or VOR hypofunction is found, the appropriate vestibular rehabilitation interventions are implemented.⁷ Otherwise, the physical examination continues with specific tests to determine if the dizziness is being caused by the cervical spine (cervicogenic dizziness).⁷ The neck torsion nystagmus test (NTNT) is performed by having the patient rotate his or her body under a stabilized head (Figure 2).²⁰ A positive NTNT is the elicitation of nystagmus. The neck torsion smooth pursuit (NTSPT) is performed by observing ocular smooth pursuit with the patient's head in neutral followed by rotation.²⁰ A positive NTSPT is an abnormal smooth pursuit when the head is in the rotated position but normal when in the neutral position.²⁰ The joint position error test (JPE) is performed by having the patient don a pair of foveal glasses and look at a stable target on a wall.^{12,15} Foveal vision testing require modifying glasses that occlude peripheral vision (Figure 3).^{12,15} The patient then closes his/her eyes and actively rotates his/her head and neck to end range. With eyes closed, the patient actively returns to what he or she thinks was the initial starting position then opens his/her eyes. The test is considered positive if the patient is not able to see the stable target.^{12,15,23} The JPE test can also be performed using a head-mounted laser pointer and the distance from the initial position to the returning position can be measured in centimeters.^{12,15} The manual traction test is performed with the patient seated. The test is considered positive if the traction relieves dizziness (Figure 4). The physical therapist should also examine the cervical spine posture, active and passive range of motion, accessory motion, and palpate cervical spine musculature.²⁴

TABLE 1. Physical Therapy Subjective Examination.

PHYSICAL THERAPY: Subjective Examination	
Past Medical History	Medical record review and patient interview.
Dizziness Tempo	Episodic dizziness lasting from several minutes to hours.
Dizziness Symptoms	Oscillopsia, disequilibrium, lightheadedness, vague dizziness.
Dizziness Circumstances	Causal symptomatic relationship to neck position and neck pain.
Dizziness Intensity	Modified visual analog scale 0–10/10 for current and recent range.

TABLE 2. Physical Therapy Physical Examination.

PHYSICAL THERAPY: Physical Examination	
Medical Screening	<p>Cervical Stability: alar ligament test, Sharp-Purser test, lateral shear test.</p> <p>Arterial Patency: modified vertebral artery test.</p> <p>Central Nervous System: upper motor neuron and cerebellar testing.</p>
Vestibular System	<p>BPPV: Hallpike-Dix test and Roll test.</p> <p>VOR Hypofunction: head thrust test, head shaking-induced nystagmus test, dynamic visual acuity test.</p>
Cervicogenic Dizziness	<p>Neck torsion nystagmus test, neck torsion smooth pursuit test, cervical spine joint position error test, cervical spine manual traction test.</p>
Cervical Spine	<p>Posture, active range of motion, accessory motion, palpation.</p>

Physical therapy intervention

Physical therapy intervention is directed at the specific impairments identified during the physical examination.^{1,7,12,15} It includes reducing cervical spine pain and inflammation, increasing mobility, improving proprioception, and improving cervical-ocular motor function (Table 3).⁷ Cervical spine pain and inflammation can be managed with modalities and manual traction. Cervical spine hypo-mobility can be managed with orthopedic manual therapy using joint mobilization, soft tissue mobilization, and a tissue-specific and age-specific stretching program.^{7,8,25} Stretching parameters have been reported in the literature.²⁶⁻²⁸ Cervical spine proprioception can be managed using postural awareness training, passive head movements with fixed target gaze (clinician passively moves patients head while patient maintains fixed gaze on a stationary target), and foveal vision exercises.^{12,15} The patient fixes his/her gaze on a stationary wall mounted target.^{12,15} The patient closes his/her eyes and actively rotates the head, then returns to the starting position and opens his/her eyes. If the patient cannot see the target, he/she keeps the eyes open and continues rotating the head until he/she can see the target.^{12,15} This provides the patient information about the integrity of the cervical spine proprioception.¹² Cervical-ocular motor function can be managed using free eye-head coupling with pursuits (patient keeps head still while eyes follow a moving target) and saccades (patient keeps head still and quickly moves eyes between targets) as well as X1 (patient moves head from side to side while maintaining gaze on a stationary target) and X2 adaptation exercises (patient moves head and hand-held target in opposite directions while maintaining gaze on moving target at all times).^{12,15} All cervical-ocular exercises can be progressed by increasing the speed, duration, and frequency as tolerated.^{12,15}

TABLE 3. Physical Therapy Physical Intervention.

PHYSICAL THERAPY: Physical Intervention	
Cervical Spine Pain and Inflammation	Modalities and traction.
Cervical Spine Mobility	Joint mobilization, soft tissue mobilization, stretching program.
Cervical Spine Proprioception	Postural awareness training. Passive head movements with fixed target gaze. Foveal vision progression in sitting and standing.
Cervical-Ocular Exercises	Free eye-head coupling with pursuits and saccades. X1 and X2 exercises with progressive functional application.



FIGURE 2. Neck Torsion Nystagmus Test and Neck Torsion Smooth Pursuit Test Positions.



FIGURE 3. Foveal Glasses.



FIGURE 4. Manual Traction Test.

Case Study

SUBJECT: The patient was a 24-year-old female with a history of chronic dizziness provoked by left neck rotation. She reported that she had been avoiding left neck rotation for at least the previous year because of the dizziness. The patient's description of dizziness was episodic vague disequilibrium, without vertigo, and general neck stiffness attributed to prolonged college study sessions over the previous four years. No other known mechanism of injury was reported. **EXAMINATION:** The physical examination began with a screening assessment of the cervical spine vascular integrity. The modified vertebral artery test (mVAT) was negative when performed to her right. The mVAT was positive when performed to patient's left, provoking dizziness, and slowed verbal responses to questions being asked. The patient became very anxious and diaphoretic while in the test position and required approximately 10 minutes to regain her composure afterward. She was referred to her physician for further medical evaluation and a duplex Doppler ultrasound was subsequently ordered. The ultrasound indicated normal arterial patency in the bilateral carotid and vertebral arteries. The patient returned to physical therapy and further physical examination was conducted. Postural abnormalities including a forward

head position and elevated shoulders were noted. Active cervical range of motion was minimally restricted generally with the exception of moderate restriction with left rotation. Palpation revealed multiple tender points and stiffness bilaterally in the upper trapezius, sternocleidomastoid, levator scapulae, and anterior scalene muscles. A resultant clinical hypothesis of cervicogenic dizziness (CGD) was made at this time. It was possible that the cervical postural abnormalities and cervical muscle imbalances could be compromising the vertebral artery during end range of motion, such as the mVAT position. The duplex Doppler ultrasound is typically performed with the cervical spine in neutral which could explain why it was normal. Had it been performed with the patients cervical spine rotated perhaps it would have been positive. A clinical decision was made to intervene with manual therapy directed at restoration of muscle flexibility and improving the cervical posture. INTERVENTION: Strain-counterstrain (SCS) techniques were performed to the bilateral upper trapezius, levator scapulae, anterior scalenes, and SCM muscles based on the palpatory examination. SCS techniques include the following: (1) passively moving the affected joint into its position of greatest comfort, (2) maintaining the position for ninety seconds, and (3) returning the patient slowly back to neutral avoiding any sudden return.²⁹ SCS is defined as the “relief of rheumatic pain by placing a joint in its position of greatest comfort”.³⁰ The dysfunction of proprioceptive reflexes is thought to result in false messages of strain causing a protective muscle spasm. Relief of these false messages of strain can potentially be achieved by applying strain in the opposite direction. This is accomplished by shortening the muscle that contains the false message of strain to the point that it stops reporting strain, for a period of ninety seconds.³¹ When a muscle is shortened, it places the muscle spindle on slack and decreases the afferent discharge of information to the central nervous system, thereby relieving the muscle spasm and improving joint range.^{29,31} The left mVAT was negative after the treatment session. The

patient was instructed to perform a daily cervical spine home stretching program specific to the muscle treated. She was also instructed to improve her cervical posture by performing chin tucks and sitting upright versus slump sitting as much as possible.

OUTCOME: The patient was tested with the mVAT again after several weeks and again one year later; the mVAT remained negative. CONCLUSION: The patient's symptoms of dizziness were likely the result of mechanical arterial pressure during cervical spine rotation due to cervical muscle tightness. The physical therapy diagnosis was cervicogenic dizziness and it was effectively managed using manual orthopedic intervention and a home stretching and postural awareness program.

Conclusion

Cervicogenic dizziness is an emerging area of clinical practice in physical therapy with a growing body of evidence. Cervicogenic dizziness may result from mechanical compression or stenosis of the vertebral artery network, irritation of the cervical sympathetic nervous system, or abnormal proprioceptive input from the upper cervical spine. There is often a history of physical trauma involving the head and neck. In order to determine if a patient's dizziness is coming from the cervical spine, the physical therapist should conduct thorough subjective and physical examinations to rule out competing causes of dizziness. Once cervicogenic dizziness has been diagnosed, appropriate interventions are implemented. In particular, treatment goals may include reducing cervical spine pain and inflammation, reducing cervical spine joint and soft tissue hypomobility, improving cervical spine proprioception, and improving cervical-ocular eye movement coordination.

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