TRAUMATIC BRAIN INJURIES AND OLDER ADULTS: THE IMPLICATIONS OF NECK STRENGTH, MUSCLE ACTIVATION, AND RANGE OF MOTION

BY

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DISSERTATION

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ABSTRACT

Fall-related traumatic brain injuries (TBIs) are a major cause of morbidity and mortality in older adults. Previous research has focused on non-modifiable risk factors, such as age and gender. However, some potential modifiable risk factors to fall-related TBIs in older adults may be related to neck musculature and function. Yet, these risk factors have received less scientific scrutiny in older adults. Thus, the current project quantified isometric neck strength, active and passive range of motion, and neck muscle activation latency in response to postural perturbation in 57 participants. Participants were divided based on age with 20 Young (18 – 30 years old), 23 Young-Old (60 – 74 years old), and 14 Old-Old (75 – 89 years old) adults being included. Participants underwent isometric neck strength testing in four directions using a custom device, active and passive neck ROM were quantified using a standard goniometer, and neck muscle activation latency was quantified using electromyography in response to anterior and posterior translations. The results of the project revealed older adults have reduced isometric neck strength, when compared to young adults, with the Old-Old adults displaying the greatest declines. Furthermore, active and passive neck ROM significantly decreased with advanced age and Old-Old adults displayed the greatest reductions in ROM. Finally, neck muscle activation latency time significantly increased with age in response postural perturbation. The significant age-related differences to neck muscle strength, ROM, and muscle activation latency may be responsible for the elevated prevalence of fall-related TBI in older adults. The findings of this project may be used with future research to identify possible rehabilitation techniques to improve neck muscle strength, ROM, and muscle activation latency in older adults, establish the link between neck musculature and function and head kinematics during a fall, and establish screening and prevention protocols for this significant health problem.

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CHAPTER 1: BACKGROUND

A traumatic brain injury (TBI) is a disruption of normal function of the brain as a result of damaging forces.[1] Fall-related TBIs are a major cause for morbidity and mortality in older adults.[1,2] It is estimated that about 80% of TBIs in older adults are a result of the head hitting the ground or other surface during a fall.[3] In 2013, the CDC reported that there were approximately 2.8 million TBIs that resulted in emergency department visits, hospitalization, and deaths.[1] The highest rates of TBIs are in adults over the age of 75. Falls accounted for almost four times as many hospitalizations and more than twice as many TBI-related deaths than motor vehicle accidents.[1] In addition to a higher incidence rate, older adults have worse health outcomes following a TBI, including extended hospitalizations and greater than 10% fatality rate.[3]

Despite fall-related TBIs being a major health issue for older adults, there is limited knowledge on how to prevent them. The significant negative health impacts from fall-related TBIs makes it imperative that cost effective evaluation and prevention programs be developed. To maximize success, preventative strategies in older adults should target modifiable risk factors. Previous research examining fall-related TBIs has focused on non-modifiable risk factors such as age or gender.[4] Potential modifiable risk factors that have received minimal scientific inquiry in older adults are neck strength, muscle activation, and range of motion (ROM).

In the sports medicine literature, reduced neck strength, slower muscle activation, and decreased ROM have been found to be significantly associated with an increased risk of concussions (mild TBIs). The neck is responsible for controlling and stabilizing the head. With sufficient neck strength and muscle activation, the neck musculature may decrease head acceleration at impact and reduce the magnitude of impact forces on the brain.[5] In a large epidemiological study, it was found that less neck strength was a significant predictor of sports-

related concussion in a sample of young adults.[6] This finding has been supported by several other studies, which found that greater neck strength resulted in significantly less head acceleration at impact in young adults.[7-13] Additionally, activation of the sternocleidomastoid (SCM) and the upper trapezius muscles is important for head stabilization and reducing head impact severity.[13-19] For instance, during simulated backwards falls in healthy young adults, it was shown that SCM activation contributes to the prevention and modulation of head impact.[15] Conversely, active ROM indicates the extent the muscles can move the head and passive ROM reveals the extent passive structures inhibit motion. Limited neck ROM may contribute to fall-related TBIs because the neck cannot counteract the movement of the head during a fall to prevent impact. For instance, if an individual experiences a right sideways fall, sufficient left lateral flexion ROM would be necessary to counteract the right lateral neck motion of the fall.[20]

While neck strength and activation have been indicated as potential modifiable risk factors of TBIs in young adults, there remains a dearth of knowledge of risk factors in older adults. The aims of this innovative proposal are to better understand age-related differences in neck muscle strength, muscle activation latency, and ROM, which may shed light on the implications of the neck musculature in fall-related TBIs. By providing background information for screening and rehabilitation programs to prevent fall-related TBIs, this study could ultimately create a paradigm shift in the aging literature that will lead to a reduction in fall-related TBIs. To accomplish these aims, background information on the neck, age-related differences in muscle strength, age-related differences in muscle activation, and age-related differences in ROM will be examined. By discussing each of these areas, we will highlight the limitations of the current literature. First, an overview of the structure of the neck will be presented to provide context for the project.

1. Overview of the Structure of the Neck

The neck extends from the base of the skull to the level of the first rib.[21] The neck provides a stable and dynamic platform for the head; mobility of the neck provides the ability for the head to scan the environment.[21,22] The neck is a highly complex structure, with seven vertebrae that form the bony support, multiple muscles, and numerous cervical nerves.[21] The primary movers of the neck are the sternocleidomastoids (SCM), splenius capitis, and upper trapezius muscle.[21]

The SCM originates on the manubrium and the medial end of the clavicle; it then inserts on the mastoid process.[21,23] If the SCM contracts unilaterally, then it can rotate the head to the opposite side or cause lateral flexion; if SCMs contract bilaterally, then neck flexion occurs.[21,23] The trapezius is the most superficial muscle in the back. It originates from the superior nuchal line, external occipital protuberance, ligamentum nuchae, and the spinous processes of C7 to T12.[21,23] The trapezius is a large muscle and divided into upper, middle, and lower portions, which have different actions. The upper trapezius can extend the neck, along with contributing to lateral flexion and rotation.[21,23] There are many other deep muscles located on the posterior and lateral sides of the neck and attach to individual vertebrae.[21] The muscles which extend along the vertebral column and insert on the individual vertebra can create lateral flexion or rotation when activated unilaterally. If the muscles are contracted bilaterally, then extension occurs.[21] The deep muscles also serve a protective function to hold the head and neck upright.[21] Anterior to the upper cervical vertebra are several thin flat muscles that attach to the vertebra from the base of the skull; these muscle are involved in neck flexion and lateral flexion.[21]

The neck is responsible for the control of the head and the amount of movement is determined by the active range of motion (ROM). Active rotation ROM of the neck is ~50° in

each direciton.[22,24,25] Rotation primarily occurs at the atlantoaxial joint, with ~40° of rotation possible.[22,24] Still, with the coupled movement as a result of the various interconnections within the cervical spine, an additional 4° to 7° of rotation can occur at the other joints (C2-C3 through C6-7).[22,26] Active flexion ROM is ~40° and extension is ~50°.[25] Through cadaver and radiograph studies, it is estimated that ~25° of flexion and extension can occur at the atlanto-occipital joint, ~15° can occur at the atlantoaxial joint, and 10° to 20° can occur at each joint, C2-C3 to C6-C7.[22] It is further estimated that lateral flexion can result in ~22° of active ROM in each direction, with similar accessory motions as flexion and extension.[25]

The neck is a complex structure of the body. To best understand how aging may affect neck muscle strength, muscle activation latency, and ROM, it is critical to understand the existing research around age-related differences. In the next section, age-related differences to muscle strength will be discussed. In addition, the current literature surrounding age-related differences to neck muscle strength will be examined with the current limitations of the investigations explained. By highlighting the limitations of the current literature, we will set up the background for Aim 1 and provide context for our hypothesis that neck strength decreases with age, with the oldest participants having the least amount of neck strength.

2. Age-Related Differences to Neck Isometric Strength

During the aging process, global neuromuscular changes occur which result in decreased strength. Overall, muscle strength increases up to the third decade of life.[27] Muscle strength remains constant until the fifth decade of life, but is followed by a decline with increased age.[27,28] While research has shown that overall strength decreases with age, different muscle groups are suggested to have varying rates of decline. Grip strength has been shown to decline at about 1% per year after the age of 50 years, and increasing to a 3% per year decline after 70 years.[28] Knee

extensor and flexor isometric muscle strength also decreases with age, but showed significantly greater reductions than grip strength.[29,30] In a 10 year period, adults 46 to 78 years old experienced a 12% to 17% reductions in isometric knee strength.[29,30] The evidence of age-related strength changes to trunk muscles is less conclusive.[31] The data shows that the rectus abdominis, internal oblique, and external oblique muscles decrease as much as 48% with age and older adults have poorer muscle quality, but the impact aging has on trunk strength and function is unclear.[31] Conversely, other investigations have determined that trunk instability may be contributing to decreased postural control.[32,33]

The neck provides a stable and dynamic platform for head control.[34,35] Therefore, it is important to understand specific age-related declines. Neck strength has been studied in young and middle-aged adults, but no significant strength differences were found between the ages of 20 and 59 in isometric cervical flexion and extension.[36] Conversely, a different investigation found that neck strength decreased 24% to 30% in flexion and 10% to 16% in extension, between the ages of 20 and 74 years. Another study discovered a 30% to 45% decrease of isometric neck strength from ages of 20 to ≥60 years.[37] However, these studies are not without limitations. These investigations did not examine age-related differences in right and left lateral flexion. As sideways falls are common,[38] understanding the age-related differences of right and left lateral flexion strength is critical to understanding the implications of neck strength in supporting the head during a fall. Additionally, only one study examined participants up to the age of 74 years.[39] While this information does give a vague overview of the potential age-related differences in neck strength, there is a lack of information surrounding specific age-related strength differences (60-74 years old vs 75+). As individuals over the age of 75 years' experience the greatest incidence of fall-related TBIs, it is critical to understand the declines in

neck strength in this age group. As it is known that strength declines accelerate with age, it would be expected that participants in the old-old group (75+ years old) would have the greatest strength differences. By understanding the difference in the young-old group (60-74 years old) and the old-old group, it will allow for a better comprehension of the implications of neck strength on fall-related TBIs. Furthermore, understanding this difference will allow for the development of screening and rehabilitation programs to prevent fall-related TBIs. It is expected that neck strength will be greater with age. These age-related differences may also be accompanied by ROM differences. Decreased ROM may be contributing to an inability to counteract the downward motion of the head during a fall. Understanding age-related differences of active and passive neck ROM is necessary to understand the implication of the neck in fall-related TBIs.

3. Age-Related Differences in Range of Motion

While ROM has not been studied as a risk factor in concussions in young adults, older adults may have less neck ROM than young adults.[40,41] The decreased ROM may be a risk factor for fall-related TBIs because older adults may have insufficient ROM to counteract the direction of the head during a fall. It is important to examine both active and passive ROM. Active ROM will indicate the ability of the muscles to move the head.[42] Conversely, passive ROM will examine the passive structures of the neck, such as joint capsules, tendons, and boney structures, as they may limit ROM.[42] There is some cross-sectional data that suggests that active neck ROM may decreases as much as 33% between the ages of 20 years and 90 years.[40,41] Additionally, it has been suggested that passive ROM may decrease 0.5º per year in females between the ages of 20 years and 59 years.[40,41] However, the literature on active and passive ROM does not distinguish ROM declines through each decade of life. As adults over the age of 75 years are at

the greatest risk for fall-related TBI,[1] understanding the age-related differences to active and passive ROM are necessary in order to understand the potential implications of the neck musculature on fall-related TBIs. Examining active ROM will provide insight into how the neck muscles may be able to counteract the direction of the head during a fall. Examining passive ROM may provide information to the extent that passive structures are limiting ROM with age. By obtaining both active and passive neck ROM, this information may provide the foundation for understanding how neck musculature and passive structure impact neck ROM and how these potential age-related differences can be examined in a screening program and improved with a rehabilitation program.

4. Age-Related Differences in Neck Muscle Activation

Age-related strength declines result from various processes.[43] The total muscle mass determines the force-generating capacity of the muscle, yet muscle atrophy occurs with aging and physical inactivity.[44] However, other research has shown that aging does not always present with significant external atrophy, which may indicate that muscle atrophy alone does not explain the declines in strength output.[27,43] Neuromuscular changes may also contribute to strength declines and lead to increases in muscle activation latency.

Muscle activation latency is the duration between the onset of a perturbation to the onset of muscle activity. In response to postural perturbation, older adults had increased muscle activation latency in the distal leg muscles.[45] Another investigation determined that older adults had delayed anticipatory muscle activity and larger compensatory muscle responses in lower extremity and trunk muscles as a result of a postural perturbation.[46] In a study examining startle responses, it was discovered that older adults (age range 70 – 80 years) had similar muscle activation patterns to younger adults in the anterior lower extremity, trunk muscles, and SCM,

but older adults were delayed by approximately 20 ms.[47] While this study found significantly increased muscle activation latency in SCM muscle activation in the older participants, the authors did not distinguish specific age-related differences (65-74 years old and 75-85 years old).[47]

Increased muscle activation latency of the neck muscles may result in reduced ability to mitigate head acceleration at impact. Furthermore, it would be expected that muscle activation latency increases with age, yet there is limited knowledge on specific age-related differences to muscle activation latency. Understanding specific age-related neck muscle activation latency is one of the first steps to identifying the implications of the neck musculature in fall-related TBIs. There are several processes that may be involved in increased muscle activation latency. Two probable explanations for this increase may be changes in muscle fiber and slowed neural processing.

4.1. Age-related differences to muscle fibers

It is estimated that there is a ~1% loss of motor units per year beginning in the sixth decade of life.[48] Various studies have examined age-related differences in muscle fiber types. It has been shown that with aging there is an overall loss of Type I and Type II fibers and a reduction in the size of Type II fibers.[43] The number of Type II fibers have greater declines than the number Type I fibers. With more Type I fibers persisting into advanced age, this may explain age-related declines to muscle strength and increases in muscle activation latency as Type I fibers generate lower amounts of force and have slower contractibility.[43,49]

In young adults, the SCM has similar amounts of Type I and Type IIa fibers.[50] With aging, the SCM takes on a slower muscle phenotype. In muscle samples of 60 to 83-year-old males the area of Type II fibers decreases, the number of Type I fibers increase, and the muscle

fiber size remains unchanged.[50] The changes to the SCM are similar to previously reported age-related muscle fiber changes in the extremities.[43,49] With the slower muscle phenotype, it would be expected that neck strength would decrease and neck muscle activation latency would increase with aging, yet there is a lack of data to support this claim. While changes to muscle fiber type may contribute to declines in strength and increases in muscle activation latency, another potential reason may be changes to neural processing.

4.2. Age-related differences to neural processing

With aging, there are various changes to the neurological system, which results in slowed adaptation to perturbations.[44-47,50-55] Thresholds of excitability increase with age for cutaneous sensation and proprioception.[51-53] Visual sensitivity decreases with age.[54] Vestibular function also decreases, which diminishes the nervous system's ability to resolve multimodal sensory conflicts.[55] These changes may inhibit an individual's ability to respond to a perturbation.

In labyrinthine-defective (vestibular deficit) middle-aged subjects, greater muscle activation latency has been shown in the SCM during unanticipated head righting experiments; this may suggest that activation of SCM and other neck muscles is partially mediated by the vestibulocollic reflex (VCR) pathway.[16,17] While the VCR pathway may contribute to muscle activation in the neck, little is known about the exact pathway.[56] Another pathway implicated in neck muscle activation is the medial vestibulospinal reflex (VSR) pathway.[56,57]

The VSR pathway connects the macula, crista ampullaris, visual system, and axial and limb muscles to the brainstem and cerebellum to maintain postural and balance.[56,57] The VSR pathway consists of the lateral and medial tract. The lateral VSR originates in the ipsilateral lateral vestibular nucleus and receives input from the macula of the otolitic and cerebellum. Efferent vestibular signals are carried ipsilateral in the spinal cord to neurons in all spinal levels.

The lateral VSP pathway produces monosynaptic activation of ipsilateral trunk and proximal limb extensors to generate antigravity postural motor activity or protective extension.[56,57] The medial VSR pathway originates from the contralateral medial, superior, and descending vestibular nuclei.[56,57] When angular rotation of the head is sensed by the semicircular ducts, information is transmitted to the medial VSR pathway. When the medial VSR pathway is activated, it bilaterally activates motor neurons in the cervical spinal cord to stimulate the neck muscles to coordinate head and neck motion.[56,57] The two different pathways allow the trunk and neck to act independently when responding to perturbation.

It has been suggested that a reflexive mechanism may be necessary to mitigate forces on the head at impact in young adults.[18,19] Older adults have a slowed transmission speed in the lateral VSR pathway, which results in greater amounts of postural sway.[58] If the medial VSR pathway is also impacted by age, then the neck muscles would have a slowed ability to respond to perturbations and be unable to support the head. Thus Aim 2 sets out to understand the age-related differences of muscle activation latency during perturbation. We hypothesize that participants in the old-old group will have the greatest amounts of muscle activation latency. By identifying the specific age-related muscle activation latency response to perturbation, we will have a better understanding the implications of muscle activation latency in fall-related TBIs. Furthermore, screening and rehabilitation programs can be developed to improve how the neck supports the head during a fall.

Thus, the proposed project aims to quantify age-related differences to neck strength, active and passive ROM, and muscle activation latency. Neck strength, ROM, and activation are crucial for supporting the head, yet has received far too few investigations into age-related differences. Because the neck maintains stability of the head, declines in strength, ROM, and

activation may place older adults at a greater risk of TBI during a fall. Investigating age-related neck muscle strength, ROM, and activation may initiate a paradigm shift in the aging research and lead to great discoveries to decrease fall-related TBIs, improve overall strength in aging, and enhance quality of life in older adults.

CHAPTER 2: AGE-RELATED DIFFERENCES TO NECK MUSCLE STRENGTH AND RANGE OF MOTION

1. Introduction

Fall-related traumatic brain injuries (TBI) are a major cause in morbidity and mortality in older adults.[1] It is estimated that about up to 80% of TBIs in older adults result the head hitting the ground or other surface during a fall.[3] In 2013, the CDC reported that there were approximately 2.8 million TBIs that resulted in emergency department visits, hospitalization, and deaths.[1] The highest rates of TBIs are in older adults over the age of 75. Falls accounted for almost four times as many hospitalizations and more than twice as many TBI-related deaths than motor vehicle accidents.[1] In addition to a higher incidence rate, older adults have worse health outcomes, extended hospitalizations, and a greater than 10% fatality rate following a TBI.[3] Despite fall-related TBIs being a major health issue for older adults, there is limited knowledge on how to prevent them.[59]

In contrast, the sports medicine literature has focused on risk factors to mild TBIs. One potential modifiable risk factor has received considerable attention in sports medicine literature for mild TBIs and may translate to fall-related TBIs is neck muscle strength.[60] The neck is responsible for the controlling and stabilizing the head. With sufficient neck strength, the neck musculature may decrease head acceleration at impact and reduce the magnitude of impact forces on the brain.[5] In a large epidemiological study, it was found that lower neck strength was a significant predictor of sports-related concussion in a sample of young adults.[6] This finding has been supported by several other studies, which found that greater neck strength resulted in significantly less head acceleration at impact in young adults.[7-9,11-13]

While neck strength has been indicated as potential modifiable risk factors of mild TBIs in young adults in sports, there is limited knowledge of the implications of the aging process on the neck muscles. During the aging process, there are well defined global neuromuscular changes that result in decreased strength.[27,28] Grip strength declines at about 1% per year after the age of 50 years and 3% per year decline after 70 years.[28] Knee extensor and flexor isometric muscle strength also decrease with age; in a 10 year period, adults 46 to 78 years old experienced a 12% to 17% reductions in isometric knee strength.[29,30] Conversely, neck strength has been studied in young and middle-aged adults, yet no significant strength differences were found between the ages of 20 and 59 in isometric cervical flexion and extension.[36] However, it has been suggested between the ages of 20 and 74 years, neck strength decreased 24% to 30% in flexion and 10% to 16% in extension and a another study discovered a 30% to 45% decrease of isometric neck strength in flexion and extension from ages of 20 to ≥60 years.[37,39]

However, these studies are not without limitations. These investigations did not examine age-related differences in right and left lateral flexion. As sideways falls are common and a leading cause of fall-related TBIs,[38,61] understanding the age-related differences of right and left lateral flexion strength is critical to understanding the implications of neck strength in supporting the head during a fall. Additionally, only one study examined participants up to the age of 74 years.[39] While this information does give a vague overview of the potential age-related differences in neck strength, there is a lack of information surrounding specific age-related strength differences (60-74 years old vs 75-89 years old). As individuals over the age of 75 years' experience the greatest incidence of fall-related TBIs,[1] it is critical to understand the declines in neck strength in this age group.

In addition to age-related differences to neck strength, changes to neck range of motion (ROM) may also contribute to fall-related TBIs. Neck ROM can be assessed actively and passively. Active ROM indicates the ability of the muscles to move the head, while passive ROM examines the passive structures of the neck, such as joint capsules, tendons, and boney structures, as they may limit ROM.[42] There is some cross-sectional data that suggests that active neck ROM may decreases as much as 33% between the ages of 20 years and 90 years.[40,41] Additionally, it has been suggested that passive ROM may decrease 0.5º per year in females between the ages of 20 years and 59 years. However, the literature on active and passive ROM does not distinguish specific changes to active ROM with different aged groups, nor is there information on age-related differences to passive ROM over the age of 60. As adults over the age of 75 years are the greatest risk for fall-related TBI,[1] understanding the age-related differences to active and passive ROM are necessary in order to understand the potential implications of the neck musculature on fall-related TBIs. If active ROM is decreased, this may indicate the neck muscles are insufficient to counteract the downward force on the head during a fall. Furthermore, if passive ROM is decreased, it may indicate passive structures of the neck are interfering with potential to counter the downward movement of the head.

As it is known that strength and ROM declines accelerate with age, it was hypothesized that participants over the age 75 of years would have the lowest levels of neck strength and ROM. To understand the age-related differences in neck muscle strength and ROM, we quantified neck isometric strength and active and passive ROM in four directions in a Young (18 – 30 years), a Young-Old (60 – 74 years) and an Old-Old (75 – 89 years) group. It was hypothesized that both Old groups would have significantly lower neck strength and ROM than

the Young group and the Old-Old group would have lowest levels of neck muscle strength and ROM.

2. Methods

2.1. Participants

A total of 57 participants were recruited from the local community through online newsletters and a database of past participants. Participants were divided into groups based on age. 20 participants (10 male, 22.3±3.8 years) age 18 – 30 years old comprised the Young group, 23 participants (13 male, 67.2±3.8 years) age 60 – 74 years old comprised the Young-Old group, and 14 participants (4 male, 81.1±5.3 years) age 75 – 89 years old comprised the Old-Old group. To be included in the study, potential participants had to be 18 to 30 or 60 – 89 years old, able to read and speak English, able to ambulate independently, self-report normal or corrected to normal vision, and self-report normal or corrected to normal hearing. Participants were excluded from the study if they had allergies to adhesives on their skin, report a history of chronic neck or back pain, a history of neck dysfunction including but not limited to referred pain, numbness, or paraesthesia, a history of spinal fractures or deformities, current neurological disorders, history of vertigo or trauma to the head or neck, currently experiencing uncontrolled cardiorespiratory problems, or a current or a history of vestibular disease.

2.2. Experimental Procedures

After completion of the informed consent procedures, participants completed questionnaires related to their health status, demographics, and fall and traumatic brain injury history. To quantify health status, participants were asked 6 questions and responses were ranked on a Likert scale (See Appendix A). Responses to the health status questions were summated with the highest score of 25 indicating worse health status and the lowest score of 6 indicating

best health status. Participant then had their height, weight, and head and neck anthropometrics quantified utilizing standardized procedures. Head circumference was measured at the level of glabella by wrapping a cloth measuring tape around the head. Neck circumference was measured by wrapping the measuring tape around the neck just below the laryngeal protuberance. Neck length was measured with the measuring tape from the occipital protuberance at the base of the skull to the most prominent spinous process (C7).[6]

Next, participants had their fall risk assessed using the short form of the physiological profile assessment (PPA).[62] The PPA consists of tests of visual edge contrast sensitivity, simple hand reaction time, lower extremity proprioception, dominant leg knee strength, and balance on a foam surface. Participants then completed a series of warm up exercises and stretches before the neck isometric strength testing. Participants began with 5 minutes of peddling on a stationary bike with no resistance. Next, participants completed a series of head turns, shoulder rolls, and 30 second neck stretches.

2.2.1. Range of Motion Testing

Active and passive neck ROM was measured in flexion, extension, and right and left lateral flexion using a standard goniometer. An experienced clinician measured the participants with valid and reliable techniques.[25]

Neck Flexion: Participants were in a seated position with their back against the chair. The head was positioned in a neutral position. The fulcrum of the goniometer was aligned with the external auditory meatus, the proximal arm was aligned perpendicular to the ground, and the distal arm was aligned with the base of the nares. For active ROM, the participant moved their head through cervical flexion. The measurement at end range was recorded. For passive ROM,

the examiner moved the participant's neck through neck flexion until a firm end feel was noted. The measurement at end range was recorded.

Neck Extension: Participants were in a seated position with their back against the chair. The head was positioned in a neutral position. The fulcrum of goniometer was aligned with external auditory meatus, the proximal arm was aligned perpendicular to the ground, and the distal arm was aligned with the base of the nares. For active ROM, the participant was asked to move their head through cervical extension. The measurement at end range was recorded. For passive ROM, the examiner moved the participant's neck through neck extension until a firm end feel was noted. The measurement at end range was recorded.

Neck Right and Left Lateral Flexion: Participants were in a seated position with their back against the chair. The head was positioned in a neutral position. The fulcrum of goniometer was over the spinous process of the C7 vertebra, the proximal arm was aligned with the spinous processes of the thoracic vertebrae so that the arm was perpendicular to the ground, and the distal arm was aligned with the midline of the head (using the occipital protuberance for reference). For active ROM, the participant was asked to move their head through right and left lateral flexion. The measurement at end range was recorded. For passive ROM, the examiner moved the participant's neck through neck lateral flexion until a firm end feel was noted. The measurement at end range was recorded.

2.2.2. Neck Strength Testing

Figure 1 depicts the testing set up. Isometric neck strength recordings were collected on a custom-made isometric neck strength measurement device. A TAS501 load cell (Sparkfun Electronics, Boulder, CO, USA) was used to quantify neck strength.

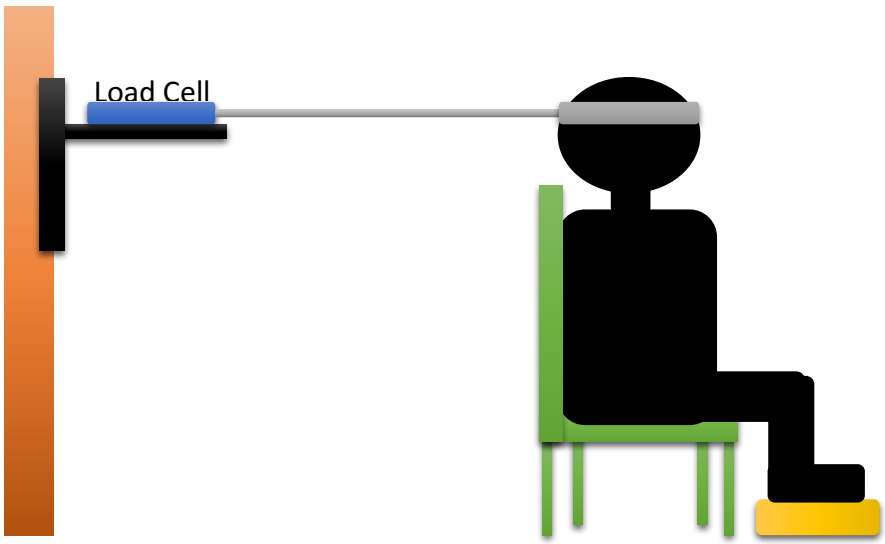


Figure 1: Depiction of participant set up for neck strength measurement

Participants sat in rigid chair in a self-selected neutral position. Velcro straps were affixed around their thorax and abdomen to prevent trunk movement. Furthermore, participants will rest their legs on a box (as displayed in Figure 1) to prevent further lower extremity and trunk movement.[63] In the event that the box collapsed or deformed, the trial was repeated. The load cell was attached to a velcro strap that wrapped around the center of the participant's forehead. The load cell was secured perpendicularly with the forehead. The strap was tightly secured to the head to prevent movement. Participants completed isometric contractions in flexion, extension, right lateral flexion, and left lateral flexion. Each direction was tested three times, for a total of 12 isometric contractions. Before testing each direction, participants performed one submaximal four second contraction to ensure proper technique. After each contraction, participants had a one minute break.

During each contraction, participants had their neck muscle activity quantified with Trigno wireless electromyography (EMG) (Delsys Inc, Natick MA, USA). EMG sensors were affixed bilaterally to the sternocleidomastoid (SCM), upper trapezius, and splenius capitis. Isopropyl alcohol cloths were used to remove dirt and loose skin participles. The EMG sensors

were placed on the skin with adhesive pads. For the SCM, the sensor was placed along the sternal portion of the muscle, with the electrode center 1/3 of the distance between the mastoid process and the sternal notch.[63] For the upper trapezius muscle, the sensor was placed 2 cm lateral to the midpoint of the C4-C5 interspinous distance and orientated along the palpated anterior boarder of the trapezius and in line with the direction of the muscle fibers.[63] For the splenius capitis, a Trigno Mini sensor was used. The enclosure of the sensor was placed on the mastoid process and the mini head was placed at the intersection of the C7-ear line and the line of action of the muscle, which was palpated by the examiner.[63]

2.3. Data Processing

The load cell recorded the output of the isometric contractions at 100 Hz. This output was analyzed using a custom MatLab code (MathWorks, Natick, MA, USA), which output peak isometric strength for each trial. The raw EMG data was processed using the Teager-Kaiser Energy Operator (TKEO).[63-65] The TKEO is a local energy measure for oscillating signals which is proportional to the signal's instantaneous amplitude and frequency.[63-65] The TKEO suppresses baseline activity, where the signals energy is low, relative to the time duration of muscle contraction, where the energy of the signal is high.[63-65] After TKEO calculations, the EMG signal was full wave rectified and low pass filtered using a 5^{th} order, zero phase shift, Butterworth filter with a cutoff frequency of 50 Hz.[66] Next, an EMG envelope was computing using the root mean square (RMS) value of the signal within a sliding window with the attenuation valuate set at 0.997. EMG onset thresholds were set as the instant the signal exceeded 3 standard deviations above baseline levels for a period of 500 ms.[65] The peak EMG amplitude of the signal was calculated as the instant the signal exceeded 15 standard deviations and the Matlab code sorted the peak amplitudes from highest to lowest. The highest peak amplitude was used to calculate

time to peak EMG.[67] Time to peak EMG activity was calculated as the duration in milliseconds between muscle onset and peak EMG amplitude.

2.4. Data Analysis

SPSS Version 25 (IBM Inc., Chicago IL) was used for the data analysis. Descriptive statistics of the groups were calculated using one-way analysis of covariance (ANCOVA), while controlling for gender. Gender was controlled for due to known gender differences throughout the lifespan.[43] One-way ANCOVAs were used to examine if there was a group difference in active and passive ROM in the directions tested (flexion, extension, right and left lateral flexion), while controlling for gender. The three trials of each direction of peak isometric strength and time to peak EMG were averaged for the analysis. A one-way analysis of covariance (ANCOVA) was used to examine if there was a group difference in each of the peak isometric neck strength (flexion, extension, right and left lateral flexion), while controlling for gender. Furthermore, Cohen's d was calculated to determine effect sizes of isometric strength between the groups. Finally, a one-way multivariate analysis of covariance (MANCOVA) was used to examine group differences on time to peak EMG activity, while controlling for gender.

3. Results

Participant demographics are presented in Table 1. One female participant from the Young group was unable to finish the study procedures due to lightheadedness and was excluded from the analysis. Overall, participants were not significantly different on their reported health status. Participants did have significantly different BMI ($F(2,52) = 4.117$).

Table 1: Participant Demographics

	Young	Young-Old	Old-Old	*p*-value
n	19	23	14	-
Gender (% Males)	52.6	56.5	28.6	-
Age (years)	22.3±3.8	67.2±3.8	81.1±5.3	-
BMI	23.2±5.6	27.7±5.3	26.7±4.6	0.02*
Health Status	8.6±1.8	7.6±2.3	8.4±2.6	0.3

notes: Values presented in mean±SD; * denotes significance of $p<0.05$

Table 2 displays the results from the PPA. Based on the one-way ANCOVA, a significant group effect was displayed in reaction time ($F(2,52) = 4.452$), knee strength ($F(2,51) = 7.201$), medial-lateral sway ($F(2,52) = 3.536$), and fall risk score ($F(2,52) = 5.735$).

Table 2: Physiological Profile Assessment as a Function of Age

	Young	Young-Old	Old-Old	*p*-value
PPA Collected Values				
Edge Contrast (dB)	20.7±1.3	20.0±1.8	19.4±2.0	0.11
Reaction time (ms)	218.3±25.4	226.2±36.5	258.5±42.5	0.02*
Proprioception (degrees)	2.5±1.4	2.5±1.4	3.3±1.2	0.20
Knee Strength (Kg)	41.43±8.4	33.3±10.8	29.3±7.6	0.002*
AP Sway (mm)	17.1±9.5	17.4±7.5	21.8±8.0	0.36
ML Sway (mm)	17.0±9.0	20.9±13.2	29.4±16.2	0.04*
Fall Risk Score	-0.03±0.5	0.03±1.1	0.7±0.7	0.006*

notes: Values presented in mean±SD; * denotes significance of $p<0.05$

Table 3 presents the average head and neck anthropometrics. Head and neck circumference were not significantly different across the groups. Based on the one-way ANCOVA, a significant group effect was displayed in neck circumference ($F(2,52) = 4.259$), and neck length ($F(2,52) = 3.958$),

Table 3: Head and Neck Anthropometrics

	Young	Young-Old	Old-Old	*p*-value
Head Circumference (cm)	56.5±2.4	57.2±2.3	56.4±2.6	0.553
Neck Circumference (cm)	34.1±4.5	37.4±4.6	35.9±4.4	0.008*
Neck Length (cm)	12.0±2.4	13.4±1.7	11.4±2.3	0.025*

notes: Values presented in mean±SD; * denotes significance of $p<0.05$

Figure 2 displays active neck ROM by direction tested. One participant in the Old-Old group declined to have extension ROM and strength measured. \ One-way ANCOVAs revealed a significant group difference in active flexion ROM ($F(2,52) = 12.782$, $p<0.001$), active extension ROM ($F(2,51) = 24.469$, $p<0.001$), active right lateral flexion ROM ($F(2,52) = 9.909$, $p<0.001$), and active left lateral flexion ROM ($F(2,52) = 11.139$, $p<0.001$). In all directions tested, the Young group displayed the greatest amount neck active ROM, with the Old-Old group displaying the least.

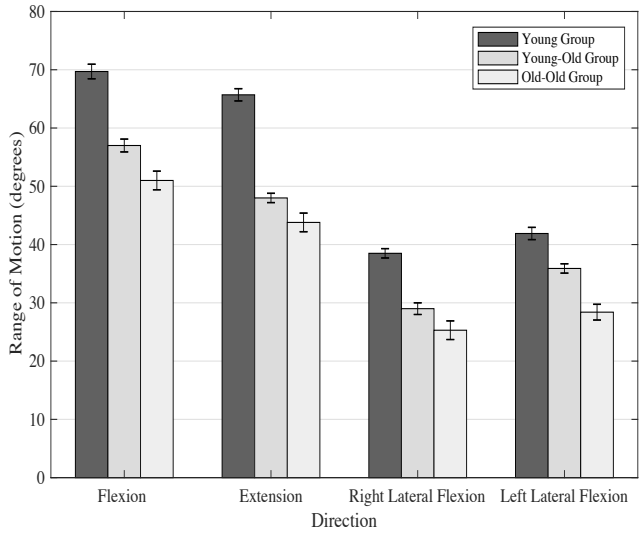


Figure 2: Active Neck Range of Motion

Figure 3 displays the passive neck range of motion by direction tested. One way ANCOVAs revealed a significant group difference in passive flexion ROM ($F(2,52) = 18.503$, $p<0.001$), passive extension ROM ($F(2,51) = 55.686$, $p<0.001$), passive right lateral flexion ROM declined ($F(2,52) = 24.157$ $p<0.001$), passive left lateral flexion ROM ($F(2,52) = 20.806$, $p<0.001$). In all directions tested, the Young group displayed the greatest amount of neck passive ROM, with the Old-Old group having the least amount of ROM.

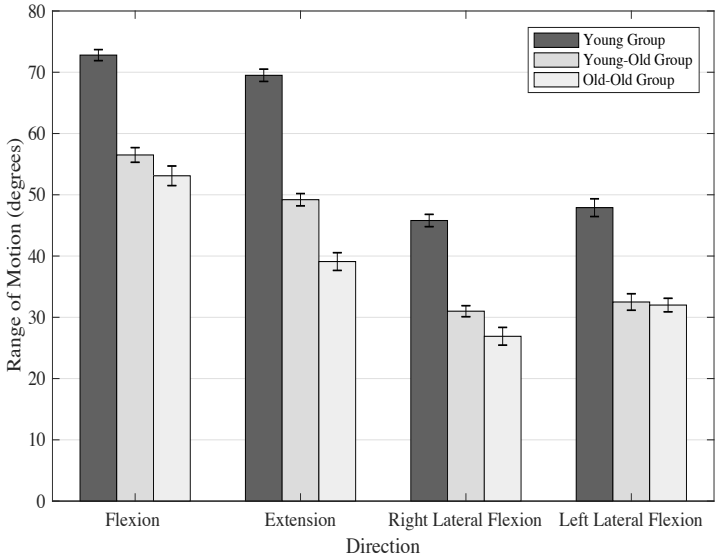


Figure 3: Passive Neck Range of Motion

Figure 4 displays the average neck strength in the directions tested. One-way ANCOVAs did not revealed a significant group effect in flexion ($F(2,52) = 2.345$, $p=0.106$), extension ($F(2,51) = 2.066$, $p = 0.137$), right lateral flexion ($F(2,52) = 1.593$, $p = 0.213$), and left lateral flexion ($F(2,52) = 3.007$, $p = 0.058$).

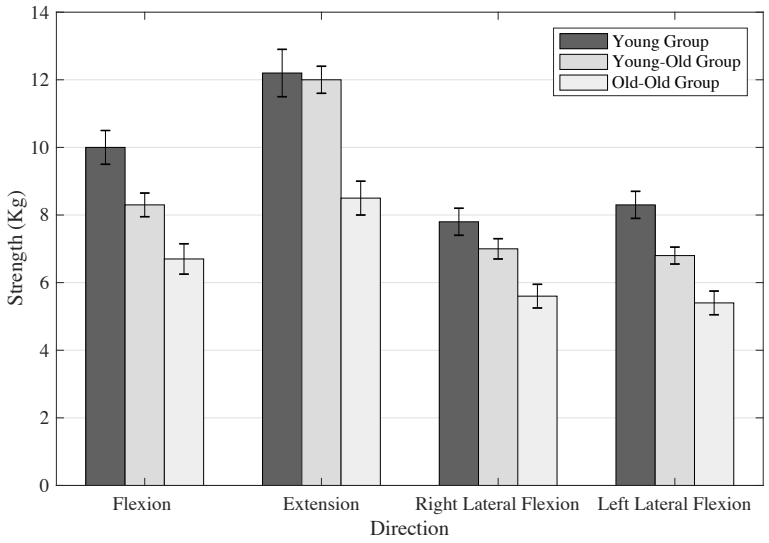


Figure 4: Neck Strength by Direction

Table 4 displays the effects sizes of the tested direction of neck strength by group. Flexion displayed medium to large effect sizes, extension displayed between no effect and large effect size, right lateral flexion displayed a small effect to large effect sizes, and left lateral flexion displayed a small to medium effect sizes.

Table 4: Effect Size of Strength Output by Group

	Young & Young-Old	Young & Old-Old	Young-Old & Old-Old
Flexion	0.39	0.78	0.47
Extension	0.06	0.77	0.94
Right Lateral Flexion	0.25	0.75	0.64
Left Lateral Flexion	0.47	0.16	0.54

Figure 5 displays the average time to peak for neck muscles during isometric flexion; the one-way MANCOVA revealed no statistically significant group differences in time to peak EMG activity ($F(12, 84) = 0.638$, $p = 0.804$, Wilk's $\Lambda = 0.840$).

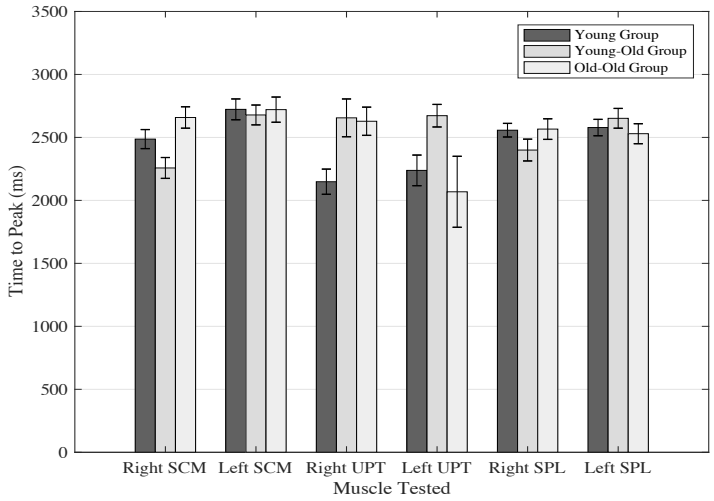


Figure 5: Time to Peak Times of the Neck Muscles in Flexion

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

Figure 6 displays the average time to peak for neck muscles during isometric extension testing; the one-way MANCOVA revealed no statistically significant group difference in time to peak EMG activity ($F(12, 92) = 1.650$, $p = 0.097$, Wilk's $\Lambda = 0.677$).

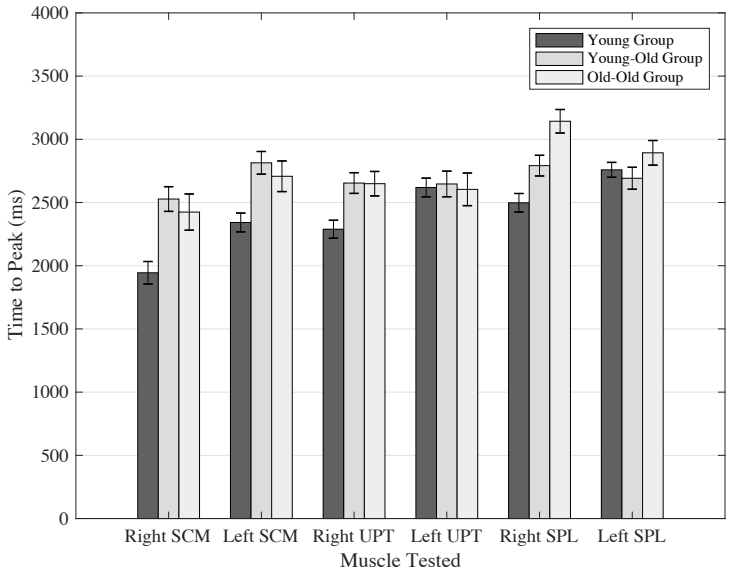


Figure 6: Time to Peak Times of the Neck Muscles in Extension

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

Figure 7 displays the average time to peak for neck muscles during isometric right lateral flexion testing; the one-way MANCOVA revealed a significant group difference in time to peak EMG activity ($F(12, 82) = 3.010$, $p = 0.001$, Wilk's $\Lambda = 0.482$).

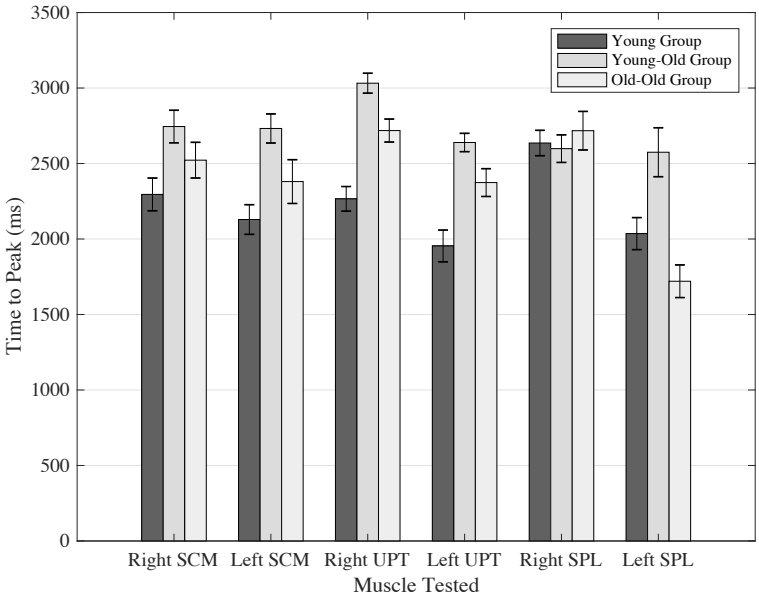


Figure 7: Time to Peak Times of the Neck Muscles in Right Lateral Flexion

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

Figure 8 displays the average time to peak for neck muscles during isometric left lateral flexion testing; the one-way MANCOVA no statistically significant group difference in time to peak EMG activity ($F(12, 76) = 0.750$, $p = 0.475$, Wilk's $\Lambda = 0.750$).

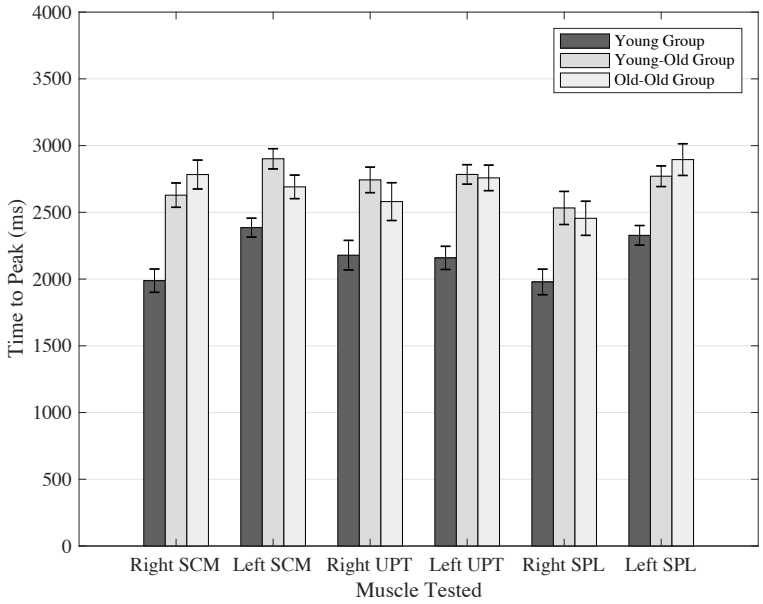


Figure 8: Time to Peak Times of the Neck Muscles in Left Lateral Flexion

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

4. Discussion

Fall-related TBIs are a major cause of hospitalizations and deaths in adults over the age of 75 years.[1] Due to this significant health concern, there have been recent calls for targeted TBI screening, prevention, and rehabilitation protocols in older adults.[59,68] Previous studies have examined non-modifiable risk factors such as age and gender,[61] yet there has been very little information on modifiable risk factors to fall-related TBIs in older adults. A potential modifiable risk factor that has received less scientific scrutiny in this population is age-related differences to neck strength and ROM. The neck is responsible for supporting the head and sufficient strength and function has been shown to decrease head acceleration at impact in young adults.[7-9,11-13] As adults over the age of 75 years are at the greatest risk of fall-related TBIs,[1] it is important to understand age-related differences to neck muscle strength and ROM. The current study examined neck strength and ROM in adults over the age of 75 years and compared the recordings to a younger aged group and a group of young adults. This study revealed that neck

active and passive ROM decreases as much as 44% with advanced age. While neck strength in the current study did not display statistically significant group effects, there was a trend of declining neck strength with age and the *p* values were approaching significances. Furthermore, the effect sizes between the Young and Old-Old group and the Young-Old and Old-Old groups in the strength directions tests were medium to large effects. This may indicate a power problem more than a null finding. Figure 9 depicts a schematic of the potential for head impact as a result of neck strength and ROM.

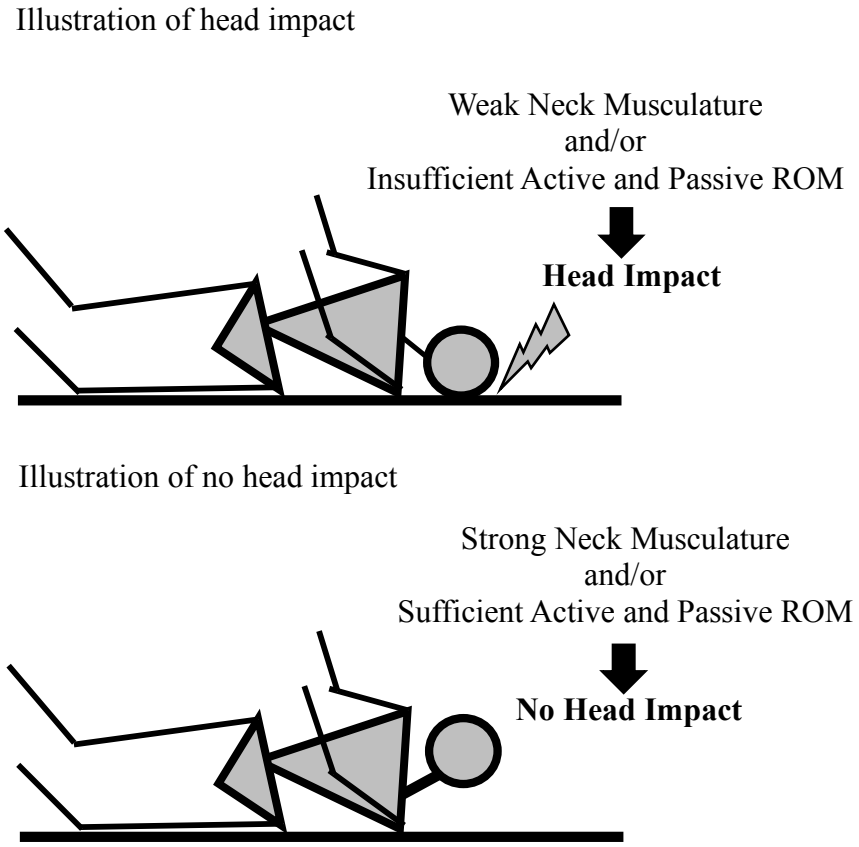


Figure 9: Schematic of Head Impacts as a Function of Neck Strength and Range of Motion

Age-related differences to neck ROM may be placing older adults at a greater risk of fall-related TBIs. As backwards and sideways falls have been implicated as increased risk of TBIs,[61] it is important to understand age-related differences to active and passive neck flexion,

extension, and lateral flexion ROM. It was shown that active ROM was reduced with age, with the Old-Old group displaying the greater reductions in the directions tested. The decreased active ROM may indicate there is a reduced ability of the neck muscles to move the head.[42] Additionally, passive ROM was reduced with age and the Old-Old group displayed the greatest reduction in all directions tested. The decreased passive ROM many indicate the passive structures of the neck, such as joint capsules, tendons, and boney structures, are limiting ROM.[42] Collectively, the reductions of active and passive neck ROM may interfere with the neck's ability to counteract the movement of the head during a fall, which may potentially result in head impact and TBI. The reductions to active and passive ROM may be due to increased muscle stiffness and tone, or forward head posture and thoracic kyphosis. For instance, Kocur et al. found that the SCM and upper trapezius increased in stiffness and tone between the ages of 20 and 70.[69] Conversely, Quek et al found that addressing forward head posture and/or thoracic kyphosis may result in improvements to cervical ROM in older adults.[70]

Previous research has shown that active neck ROM may decrease as much as 33% between the ages of 20 years and 90 years.[40,41] Kuhlman examined adults 20 to 30 years old and 70 to 90 years old and found that the older adults had 12% less flexion active ROM, 32% less extension active ROM, and 22% less lateral flexion active ROM.[40] The current study found greater reductions in flexion and right and left lateral flexion than Kuhlman with age. Furthermore, the current study detailed age-related differences to active neck ROM in two aged groups. It is important to make this distinction, as adults over the age of 75 are at the greatest risk of fall-related TBIs.[1]

In addition to active neck ROM, passive neck ROM was also examined. Age-related differences to passive neck ROM has received very little scientific investigation. One of the few

investigations has shown that passive neck ROM decreased 0.5º per year between the ages of 20 and 59 years in females.[42] However, there is limited information on passive neck ROM after the age of 60 or in male participants.[42] As people over the age of 75 years and males are more likely to sustain fall-related TBIs, understanding passive ROM in this group is critcal.[1,61] Passive ROM is an indicator of how passive structures may interfere with movement. Potentially, knowledge of age-related differences to passive neck ROM may provide insight into the implications of fall-related TBIs. As the passive structures of the neck are restricting the ROM in the examined older groups, this may indicate that older adults may be unable to counteract the movement of the head during a fall, which increases the risk of head impact. With the knowledge gained from this investigation, it may be possible to design screening and prevention protocols to increase active and passive neck ROM. However, more research is needed to identify effective rehabilitation techniques and understand the implications of neck ROM on fall-related TBIs.

In addition to ROM, this study found trending reductions in neck strength with age. In the sports medicine literature, it has been shown that decreased neck strength can result in greater head acceleration at impact and a greater risk of mild TBI.[7-9,11-13] Furthermore, in a recent study, it was shown that healthy older adults experienced a three-fold increase in head impacts, when compared to young adults in experimentally induced sideways falls.[71] The older adults in this experiment may have displayed greater amount of head impacts due to age-related declines to neck strength and/or ROM.[71]

There is limited data pertaining to age-related differences in neck strength after the age of 75 years. Foust et. al.,[39] examined adults 18 to 24 years old and 62 to 74 years old. The results showed that the older adults had as much as 30% less flexion and extension neck strength than the young adults. However, there is no data on neck strength in those over 75 years nor was right

and left lateral flexion tested. Garces et al.,[37] tested the neck strength of young adults (20 – 40 years old) and adults over the age of 60 years. It was shown that the older adult group had as much as 45% less neck strength than the young adults in flexion and extension. These results were consistent with Foust et al study; however, it did not specify the average age of the adults over the age of 60, it did not provide evidence of specific age-related differences, nor did it give data on age-related strength differences of right and left lateral flexion. The current study is the first to document a trend of age-related differences to flexion, extension, and lateral flexion isometric muscle and in older adults over the age of 75 years.

Older adults may experience declines in neck strength due to changes in neck muscle fibers. With age, the SCM takes on a slower muscle phenotype. It has been shown that older adults have a lower number of Type II muscles fibers and a higher number of Type I muscle fibers in the SCM, when compared to a young adult.[50] This slower muscle phenotype may result in decreased force production with age. In addition to decreased force production, the changes to muscle phenotype may also result in a slower rate for force production. The current study examined time to peak EMG activity during the isometric strength testing. While the current study revealed 25% of the muscles tested displayed significant age-related time to peak differences, there was a trend of increased time to peak in several of the muscle. The increasing trend may indicate an additional need to examine dynamic muscle activation in response to perturbation, in addition to neck strength, when assessing for the risk of TBIs.[60]

With the knowledge gained from the current study, the next steps are to identify possible rehabilitation techniques to enhance neck strength and ROM, which may prevent head impact and TBI. Currently, most exercise and fall prevention programs in older adults do not have a focus on neck musculature.[72,73] Several studies have examined the effectiveness of exercise and

strength training on the head/neck complex and the implications in head control with mixed results. Lisman et al.[74] implemented an eight-week isoinertial neck resistance training program on healthy college aged male athletes who had experience with American football tackling to understand the effects of neck strength training on head acceleration during tackling. It was found that the resistance training had minimal increases in strength in extension and left lateral flexion, and resulted in no differences in head acceleration during a tackle.[74] It is important to note that due to the fitness level and age of this group, a ceiling effect may have been experienced which resulted in the null results. Conversely, Eckner et al[10] examined the effects of an eight-week manual resistance neck strengthening program to understand the effects of resistance training on head velocity in response to perturbation in an adolescence sample. It was shown that the resistance training increased neck girth and strength, along with decreasing head linear velocity in response to perturbation.[10] The findings of the Eckner et al[10] study indicate resistance training of the neck muscles may be a feasible approach to improving head kinematics at impact. Thus, future studies should examine the effectiveness of resistance training on the neck muscles in older adults and the implication of increased neck strength on head kinematics during perturbation.

4.1. Limitations

While this study was the first to examine neck ROM and strength in adults over the age of 75 years and display novel findings, several limitations should be discussed. First, we utilized a relatively small convenience sample of healthy adults. This may result in skewed ROM and strength output. It is likely that less healthy, frailer older adults, would have greater age-related declines. Moreover, this study may not be sufficiently powered to display statistically significant age-related differences in neck strength. Furthermore, nutritional intake, current physical activity

level, and past physical activity levels were not collected; this information may beneficial to describe the reasons for the age-related differences. In the Old-Old group, there are more females than males; this may be due to the advanced age of the group being greater than the life expectancy of the United States.[75] Additionally, the link between neck ROM and strength in older adults and fall-related TBIs has not been well established, which may limit our interpretation.

4.2. Future Directions

With the current study filling some foundational knowledge gaps, future studies are needed to better understand age-related differences to neck strength and ROM, and identify methods to prevent fall-related TBIs. First, the link between age-related differences to neck muscle strength and ROM and head impact in older should be established. Additionally, an interdisciplinary approach should be used to further understand age-related differences to neck strength and ROM. It is possible physical activity level and nutritional intake, among other factors, may influence age-related differences to neck strength and ROM, yet more research is needed. Next, effective rehabilitation protocols to increase neck strength and ROM should be examined. Eckner et al[10] showed improvements in head kinematics after manual resistance training to the neck muscles in adolescences; a similar training protocol may be effective in older adults. Finally, with an interdisciplinary approach, screening protocols for fall-related TBIs should be established to identify older adults at the greatest risk with the goal of implementing an intervention to decrease the risk of fall-related TBIs.

4.3. Conclusion

This was the first study to examine age-related differences in neck ROM and strength in adults over the age of 75 years with comparison to a younger aged group and a young adult

group. Furthermore, this study examined the potential implications of age-related differences to neck ROM and strength on fall-related TBIs. It was shown that neck active and passive ROM displayed age-related differences neck flexion, extension, and right and left lateral flexion. Moreover, isometric neck strength displayed age-related trending reductions with medium to large effects sizes. Understanding these age-related differences are critical for understanding their potential implications in fall-related TBIs and establishing screening and rehabilitation protocols to decrease this significant health problem.

CHAPTER 3: AGE-RELATED DIFFERENCES TO NECK MUSCLE ACTIVATION LATENCY

1. Introduction

Traumatic brain injuries (TBI) are a major cause of morbidity and mortality in older adults, with upwards of 80% of TBIs resulting from head impact during a fall.[1,3] Adults over the age of 75 have the highest rates of TBIs.[1] In 2013, falls accounted for almost four times as many hospitalizations and more than twice as many TBI-related deaths than motor vehicle accidents.[1] In addition to the elevated incidence rate, older adults have worse health outcomes, extended hospitalizations, and a greater than 10% fatality rate following a TBI.[3] Despite fall-related TBIs being a major health issue for older adults, there is limited information concerning their prevention.[59]

The neck is responsible for the controlling and stabilizing the head. In the sports medicine literature, increased neck muscle activation latency has been shown to be a predictor of concussions (mild TBIs) in athletes. With sufficient muscle activation, the neck musculature may decrease head acceleration at impact, reduce the magnitude of impact forces on the brain, and reduce TBI risk.[5] Activation of the sternocleidomastoid (SCM), splenius capitis, and upper trapezius muscles is important for head stabilization and reducing head impact severity.[13-19,76]

While the sports medicine literature indicates neck muscle activation latency as a risk factor for mild TBIs, the connection between neck muscle activation latency and fall-related TBIs is less clear.[60] Muscle activation latency is the duration between a stimulus, such as a perturbation, to the onset of muscle activity. In response to postural perturbations, older adults have increased muscle activation latency in the distal leg muscles, when compared to young adults.[45] It has also been documented that older adults have increased muscle activation latency

and larger compensatory muscle responses in lower extremity and trunk muscles as a result of postural perturbation.[46] In a study examining startle responses as result of a prone fall on a mattress, older adults had similar muscle activation patterns to younger adults in the anterior lower extremity, trunk muscles, and SCM. However, older adults were delayed by approximately 20 ms.[47] While this study found significantly increased muscle activation latency in SCM muscle activation in the older participants, the authors did not distinguish specific age-related differences (65-74 years old and 75-89 years old) to understand the muscle activation latency in older adults who are at the highest risk of fall-related TBI.[47]

There is limited knowledge of the role of the neck musculature during a fall. In an experiment which examined experimentally induced backwards falls, Choi et al.[15] had young adults fall under three different conditions. The first condition was to fall while activating the SCM to support the head and avoid head impact. The second condition was to fall while partially activating the SCM, which resulted in a soft head impact. And the final condition was to not activate the SCM to have minimal efforts to reduce impact severity.[15] The results of this study showed that under the condition with minimal efforts to reduce impact severity, the participants had greater impact velocities than when with full and partial SCM activation. This study highlights that SCM activation can prevent and modulate head impact severity during falls.[15]

With aging, there are several processes which may affect muscle activation. However, neck muscle activation latency due to perturbation in older adults is not well documented. Thus, the purpose of this study is to quantify neck muscle activation latency of a young group (18 – 30 years), a young-old group (60 – 74 years) and an old-old group (75 – 89 years) in response to anterior and posterior translations on the Smart Equitest Research System. It was hypothesized

that both old groups will have significantly longer neck muscle activation latency than the young group.

2. Methods

2.1. Participants

A total of 57 participants were recruited from the local community through online newsletters and a database of past participants. 20 participants (10 male, 22.3±3.8 years) age 18 – 30 years old were categorized into the young group, 23 participants (13 male, 67.2±3.8 years) age 60 – 74 years old were categorized into the young-old group, and 14 participants (4 male, 81.1±5.3 years) age 75 – 89 years old were categorized into the old-old group. To be included in the study, potential participants had to be 18 to 30 or 60 – 89 years old, be able to read and speak English, able to ambulate independently, self-report normal or corrected to normal vision, and self-report normal or corrected to normal hearing. Participants will be excluded from the study if they had allergies to adhesives on their skin, report a history of chronic neck or back pain, a history of neck dysfunction including but not limited to referred pain, numbness, or paraesthesia, a history of spinal fractures or deformities, current neurological disorders, history of vertigo or trauma to the head or neck, currently experiencing uncontrolled cardiorespiratory problems, or a current or a history of vestibular disease.

2.2. Experimental Procedures

After completion of the informed consent procedures completed a health status questionnaire. To quantify health status, participants were asked 6 questions about their health status and responses were ranked on a Likert scale (See Appendix A). Responses to the health status questions were summated with the highest score of 25 indicating worse health status and the lowest score of 6 indicating best health status. Participant then had their height and weight

measured to calculate body mass index (BMI). Participants also completed 10 dominate hand simple reaction time tests. After, participants completed a series of warm up exercises and stretches. Participants began with 5 minutes of peddling on a stationary bike with no resistance. Next, participants completed a series of head turns, shoulder rolls, and 30 second neck stretches.

To examine muscle activation latency, participants underwent dynamic posturography on the Smart Equitest Research System (Natus Medial Inc, Pleasanton, CA, USA) with EMG (Trigno wireless system, Delsys Inc, Natick MA, USA) sensors affixed bilaterally to the sternocleidomastoid (SCM), upper trapezius, and splenius capitis. The EMG sensors were placed on the skin with adhesive pads after isopropyl alcohol cloths were used to remove dirt and loose skin participles. For the SCM, the sensor was placed along the sternal portion of the muscle, with the electrode center 1/3 of the distance between the mastoid process and the sternal notch.[63] For the upper trapezius muscle, the sensor was placed 2 cm lateral to the midpoint of the C4-C5 interspinous distance and orientated along the palpated anterior boarder of the trapezius and in line with the direction of the muscle fibers.[63] For the splenius capitis, a Trigno Mini sensor was used. The enclosure of the sensor was placed on the mastoid process and the mini head was placed at the intersection of the C7-ear line and the line of action of the muscle, which was palpated by the examiner.[63] The EMG sensors were also equipped with a 3-axis accelerometer. Two additional sensors were placed on the center of the forehead, just below the hairline, and on the C7 vertebrae to measure anterior-posterior acceleration of the head and neck.

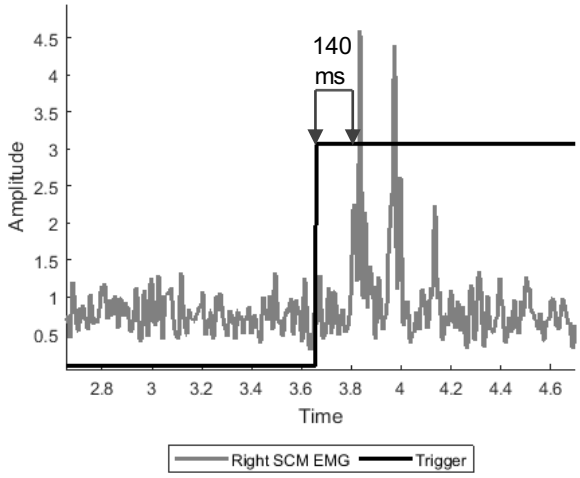
Once the EMG sensor were in place, the participants were fitted into the safety harness for the Smart Equitest Research System. Participants stood with their feet shoulder width apart on the force plates, staring straight ahead with arms at their side. The platform was set to translate anteriorly or posteriorly 6.35 cm in each direction at a velocity of 20 cm/sec.

Participants underwent three trials of the platform translations in the anterior and posterior directions in a randomized order, for a total of six trials. No practice trials will be permitted as this test examines automatic postural responses to unexpected perturbations. The EMG system and the Smart Equitest Research System and EMG system were integrated with an I/O Switch Box and an Delsys Trigger Adapter, which was transmitted to the EMG system at the beginning of each translation.

2.3. Data Processing

The raw EMG data was processed using the Teager-Kaiser Energy Operator (TKEO).[63-65] The TKEO is a local energy measure for oscillating signals which is proportional to the signal's instantaneous amplitude and frequency.[63-65] The TKEO suppresses baseline activity where the signals energy is low relative to the time duration of muscle contraction where the energy of the signal is high.[63-65] After TKEO calculations, the EMG signal will be full wave rectified and low pass filtered using a 2nd order, zero phase shift, Butterworth filter with a cutoff frequency of 50 Hz.[66] Onset of each trial was marked with a signal from the trigger adapter. A custom Matlab (MathWorks, Natick, MA, USA) code detected this onset signal and the onset of the EMG signal. Onset thresholds was set as the instant the signal exceeded 3 standard deviations above baseline levels for a period of 500 ms.[65] Figure 10 displays the EMG time series from the right SCM of a Young participant and an Old-Old Participant. Muscle activation latency was calculated as the onset of EMG signal subtracted from the onset of the trial.

Depiction of EMG series of an Young Participant



Depiction of EMG series of an Old-Old Participant

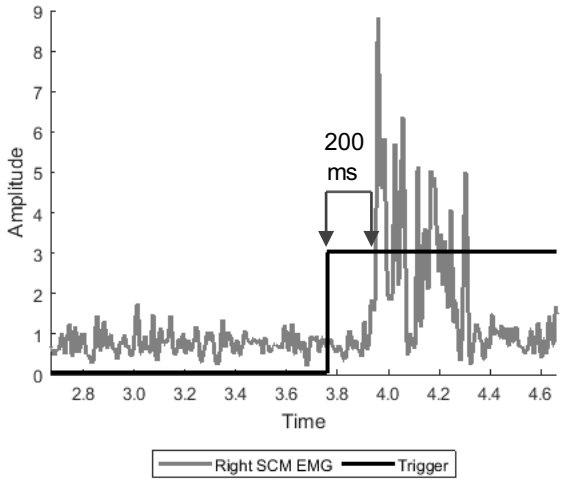


Figure 10: Depiction of EMG series of a Young and an Old-Old Participant

To determine if there was coupling between head and neck movement, raw anterior-posterior acceleration data was processed from the sensors on the head and C7. The acceleration data was band-passed filtered with a 4th order Butterworth filter with a cut-off frequency of 5 Hz.[77] A custom Matlab code detected the onset of anterior-posterior acceleration as the instant the signal exceeded 5 standard deviations above baseline levels for a period of 500 ms. The

absolute difference of onset of each acceleration signal was then used to examine acceleration differences in the head/neck complex. To quantify head movement, a vector sum was used on the three axes of the accelerometer. The vector sum was band-passed filtered with a 4th order Butterworth filter with a cut-off frequency of 5 Hz.[77] A custom Matlab code then normalized the vector sum and detected the peak acceleration.

The force plates calculated center of pressure (CoP) data and a custom Matlab code applied a 4th order low pass Butterworth filter with a cutoff frequency of 10 Hz and calculated average anterior-posterior (AP) and medial-lateral (ML) CoP sway displacement and velocity.

2.4. Data Analysis

SPSS Version 25 (IBM Inc., Chicago IL) was used for the data analysis. Descriptive statistics as a function of group were determined. Muscle activation latencies for each muscle were averaged for the anterior translations and the posterior translations. AP and ML CoP sway displacement and velocity were averaged for the anterior translations and the posterior translations. The coupled head and neck acceleration onset data were averaged for the anterior translations and the posterior translations. The peak head acceleration was averaged for the anterior and posterior translations. A Shapiro-Wilk test of normality was used to assess the normality of the distribution of the muscle activation latency data. A Kruskal-Wallis nonparametric test was used to assess group difference of the muscle activation latencies. A one-way analysis of covariance (ANCOVA) was used to examine group differences of BMI, health status, reaction time, the balance data, coupled head and neck data, and peak head acceleration. When appropriate interactions were examined. Spearman's correlation tests were used to assess the correlation between the anterior and posterior peak head acceleration and the corresponding

direction of the muscle activation latency, along with the correlation between simple reaction time and muscle activation latency.

3. Results

Participant demographics are presented in Table 5. One female participant from the Young group was unable to finish the study procedures due to lightheadedness and was excluded from the analysis. Overall, participants were not significantly different on their reported health status or number of falls in past 12 months. Reaction time ($F(2,52) = 4.452$) and BMI ($F(2,53) = 4.193$) displayed a significant group effect.

Table 5: Participant Demographics

	Young	Young-Old	Old-Old	*p*-value
n	19	23	14	-
Gender (% Males)	52.6	56.5	28.6	-
Age (years)	22.3±3.8	67.2±3.8	81.1±5.3	-
BMI	23.2±5.6	27.7±5.3	26.7±4.6	0.02*
Health Status	8.6±1.8	7.6±2.3	8.4±2.6	0.30
Number of falls	0.3±0.6	0.4±0.7	0.3±0.5	0.81
Reaction time (ms)	218.3±25.4	226.2±36.5	258.5±42.5	0.02*

notes: Values presented in mean±SD; * denotes significance of $p<0.05$

Table 6 presents the balance measures. The majority of the balance measures assessed did not result in significant group differences. The only balance measure that resulted in a significant group differences was anterior-posterior sway velocity ($F(2,53) = 3.544$, $p = 0.036$).

Table 6: Balance Measures from Anterior and Posterior Translations

Anterior Translations

	Young	Young-Old	Old-Old	p-value
AP Displacement (mm)	111.4±28.3	104.2±42.8	114.4±25.2	0.485
ML Displacement (mm)	101.5±33.9	109.2±33.1	110.8±29.2	0.647
AP Velocity (mm/s)	99.1±26.9	92.8±32.3	102.8±26.3	0.686
ML Velocity (mm/s)	68.4±10.3	53.0±26.8	67.9±45.8	0.358

Posterior Translations

	Young	Young-Old	Old-Old	p-value
AP Displacement (mm)	44.6±39.3	46.4±31.3	64.3±52.6	0.278
ML Displacement (mm)	52.4±49.2	41.9±27.3	39.0±30.6	0.544
AP Velocity (mm/s)	72.7±18.1	75.2±33.4	96.0±24.4	0.022*
ML Velocity (mm/s)	64.3±25.8	76.0±30.6	87.0±23.6	0.066

Notes: Values displayed as mean±SD, AP denotes anterior-posterior, ML denotes medial-lateral, values presented in mean±SD; * denotes significance of $p<0.05$

The Shapiro-Wilk test of normality revealed the muscle activation latencies in the anterior and posterior translations were not normally distributed ($p \leq 0.001$). Figure 11 displays the average muscle activation latencies for the anterior translation trials. Of the muscles tested, the Kruskal-Wallis nonparametric test revealed the right and left SCM and the right and left splenius capitis did not display significant group differences ($p>0.05$). The Kruskal-Wallis nonparametric test did reveal a significant group effect for muscle latency of the right upper ($X^2(2) = 7.033$, I = 0.030) and left upper trapezius ($X^2(2) = 12.165$, $p = 0.002$). The Young group displayed shorter muscle activation latency in both muscles when compared to the Young-Old and Old-Old groups. Spearman's correlation tests did not reveal significant correlations between simple reaction time and the muscle activation latency times.

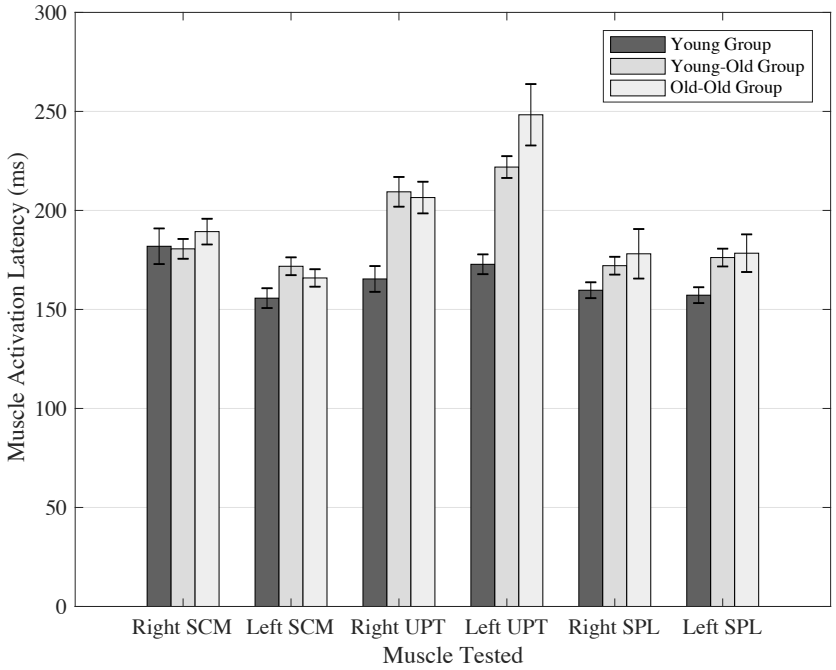


Figure 11: Anterior Translation Muscle Activation Latency as a Function of Group

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

Figure 12 displays average muscle activation latencies for the posterior translation trials. The Kruskal-Wallis nonparametric test revealed the left upper trapezius and the left splenius capitis did not display significant group differences (p>0.05). The Kruskal-Wallis nonparametric test did display a significant group effect in the right ($X^2(2) = 8.969$, $p = 0.011$), and left SCM ($X^2(2) = 11.878$, $p = 0.003$), the right upper trapezius ($X^2(2) = 7.326$, $p = 0.026$), and the right splenius capitis ($X^2(2) = 7.326$, $p = 0.026$). In these muscles, the Young group displayed shorter muscle activation latency when compared to the Young-Old and Old-Old groups. Spearman's correlation tests did not reveal significant correlations between simple reaction time and the muscle activation latency times.

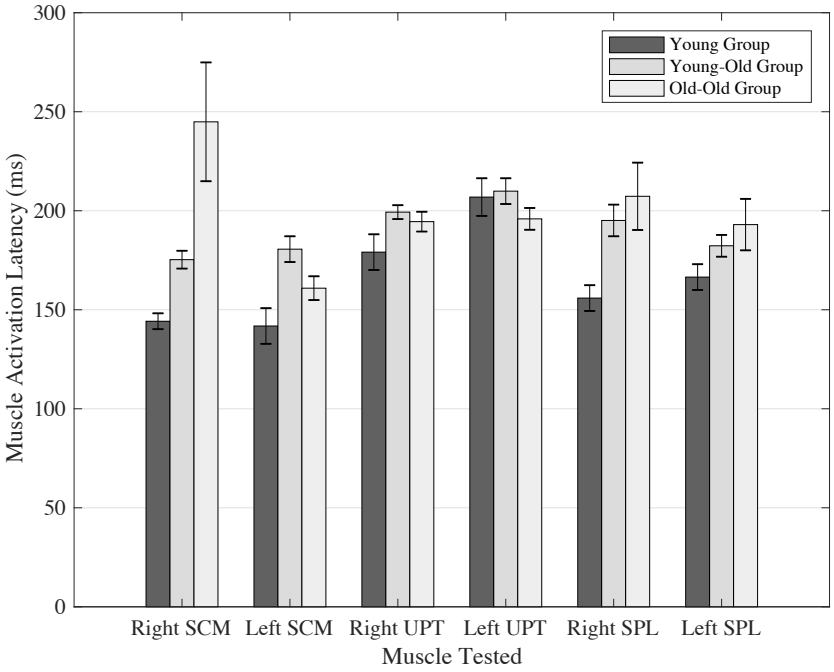


Figure 12: Posterior Translation Muscle Activation Latency as a Function of Group

Notes: SCM denotes sternocleidomastoid, UPT denotes upper trapezius, SPL denotes splenius capitis

Table 7 displays the average difference in coupled head and neck acceleration during anterior and posterior translations. A larger number would indicate a larger difference between when the head and neck began to move after the initiation of the translation. The one-way ANCOVAs revealed there was no significant group difference, yet there was an increasing trend with age.

Table 7: Difference in Head and Neck Acceleration During Translation

	Young	Young-Old	Old-Old	p-value
Anterior Translation (ms)	66.7±35.2	87.1±38.3	101.1±60.2	0.099
Posterior Translation (ms)	53.7±28.6	74.7±56.3	70.3±88.7	0.475

Notes: data displayed as mean±SD.

Table 8 displays the average peak head acceleration during the anterior and posterior translations. The one-way ANCOVAs revealed there was no significant group difference. Spearman's correlation did not reveal significant correlations between the anterior translation

peak head acceleration and anterior muscle activation latencies ($p>0.05$), nor were there significant correlations between posterior translation peak head acceleration and posterior translation muscle activation latencies ($p>0.05$).

Table 8: Peak Head Acceleration During Translation

	Young	Young-Old	Old-Old	p-value
Anterior Translation (g)	2.7±2.0	4.5±7.8	3.5±2.4	0.520
Posterior Translation (g)	3.2±3.4	2.5±1.4	4.0±2.1	0.358

Notes: data displayed as mean±SD.

4. Discussion

Fall-related TBIs are a major cause of morbidity and mortality in older adults.[1] The disproportionate and significant adverse consequences of fall-related TBIs has led to call for novel, targeted TBI screening, prevention, and rehabilitation protocols in older adults.[59,68] The sports medicine literature has identified slowed neck muscle activation in response to perturbation as a risk factor for concussion.[13-19,76] Brain injuries occur when rapid changes in head velocity lead to head displacement.[78] When rapid head displacement and rotation of the head occurs, neck deformation follows, with head displacement being proportional to the force of the neck.[78] Thus, with less muscle activation latency, the muscles may be able to reduce neck deformation which would reduce head displacement and head acceleration and lead to a reduction in brain injuries.[76]

The current study examined neck muscle activation latencies in response to anterior and posterior translations. The results of this study showed that older adults have greater neck muscle activation latency in response to a perturbation. In response to a prone free fall, Bisdorff et al found that a group of older adults (70 to 80 years old) had a reflexive SCM muscle activation latency of 75 ms and young adults (21 to 57 years old) had a reflexive SCM muscle activation latency of 55 ms.[47] There was a similar age-related group difference of 20 ms observed in the

SCM during a sudden drop perturbation.[79] In comparison to previous postural perturbation studies, the participants of the current study displayed similar muscle activation latency to previous reports focusing on lower extremity muscles.[80] For instance, de Freitas et al[80] found during anterior translations, adults age 20 – 25 years old activated the tibialis anterior and rectus femoris activated 130 ms and 165 ms after perturbation, respectively; compared to older adults (aged 60 – 65 years) who activated the tibias anterior 155 ms and the rectus femoris 180 ms after perturbation.[80]

The greater muscle activation latency time, may be placing older adults at a greater risk of fall related TBIs. During a modeling experiment of head impact of an American football athlete, Jin et al.[76] showed when the neck muscles activated 40 ms before impact, the brain injury criteria was reduced by 18% and the cumulative strain damage was reduced by 24%, when compared to the no activation scenario. This suggests that anticipatory neck muscle activation may reduce the risk of TBIs.[76] The majority of the neck muscles tested, which exhibited a significant group effect in the current study, displayed age-related differences of 40 ms or more. Although Jin and colleagues focused on young adults during their simulation, it is logical to speculate that a delay of 40 ms, as seen here, could have catastrophic results.

Advanced age is accompanied by various changes that result in slowed muscle activation. The current study revealed simple reaction time did not correlate with muscle activation latency, which may suggest there are numerous factors contributing to age-related differences to muscle activation latency. For example, it has been shown that in young adults, the SCM has similar amounts of Type I and Type IIa fibers.[50] With aging, the SCM takes on a slower muscle phenotype in muscle samples of 60 to 83-year-old males; the area of Type II fibers decreases, the number of Type I fibers increase, and the muscle fiber size remains unchanged.[50] The changes to

the SCM muscle fibers are similar to previously reported age-related muscle fiber changes in the extremities.[43,49] With the slower muscle phenotype, it would be expected that neck muscle activation latency would increase with aging, yet there is a lack of data to support this claim. Additionally, there is limited evidence concerning muscle fiber changes to the splenius capitis and upper trapezius.

While changes to muscle fiber type may contribute to increases in muscle activation latency, another potential reason may be changes to neural processing. With aging, there are various changes to the neurological system, which results in slowed adaptation to perturbations.[44-47,50-55] An important neurological pathway implicated in neck muscle activation is the medial vestibulospinal reflex (VSR) pathway.[56,57] The VSR pathway connects the macula, crista ampullaris, visual system, and axial and limb muscles by the brainstem and cerebellum to maintain postural and balance.[56,57] The VSR pathway consists of the lateral and medial tract. The lateral VSR originates in the ipsilateral lateral vestibular nucleus and receives input from the macula of the otolitic and cerebellum. Efferent vestibular signals are carried ipsilateral in the spinal cord to neurons in all spinal levels. The lateral VSP pathway produces monosynaptic activation of ipsilateral trunk and proximal limb extensors to generate antigravity postural motor activity or protective extension.[56,57] The medial VSR pathway originates from the contralateral medial, superior, and descending vestibular nuclei.[56,57] When angular rotation of the head is sensed by the semicircular ducts, information is transmitted to the medial VSR pathway. When the medial VSR pathway is activated, it bilaterally activates motor neurons in the cervical spinal cord to stimulate the neck muscles to coordinate head and neck motion.[56,57] The two different reflex pathways allow the trunk and neck to act independently when responding to perturbation. Previous research has suggested that a reflexive mechanism may be necessary to mitigate forces

on the head at impact in young adults.[18,19] It has been shown that older adults have a slowed transmission speed in the lateral VSR pathway, which results in greater amounts of postural sway.[58] The current study revealed that older adults displayed significantly greater anterior-posterior sway velocity during posterior translations, which may indicate age-related differences to the lateral VSR pathway. Conversely, while the age-related differences to the medial VSR pathway are less understood, it is probable to speculate that age-relate changes to this pathway may be responsible for the greater muscle activation latency times in the older adults and may indicate an inability to support the head during a fall.

With the evidence of increased neck muscle activation latency with age and the implications of neck muscle activation to reduce TBI,[76] it is important to discuss the potential rehabilitation possibilities. Current fall prevention and exercise programs do not focus on the neck musculature.[72,73] From the sports medicine literature, it has been suggested that neuromuscular training specific to enhancing the neck muscles' dynamic response to perturbation may be beneficial to improving neck muscle activation.[18] Furthermore, it has been shown that volitional and reactive stepping interventions can improve stepping reaction time and fall risk in older adults.[81] Thus, there may be a potential for a dynamic intervention to focus on improving neck muscle activation in older adults, which may prevent fall-related TBIs. Future research should focus on examining rehabilitation techniques to improve neck muscle activation.

4.1. Limitations

While this study provided novel results and insight into the implications of neck muscle activation on fall-related TBIs. A few limitations should be discussed. This study was conducted with a relatively small convenience sample. Furthermore, there are more females than males in the Old-Old group. Collectively, these aspects of the study may skew the results. Additionally,

we examined healthy older adults; less fit or frailer older adults may have greater age-related differences in neck muscle activation latency. Finally, the link between neck muscle activation latency and fall-related TBIs has not been well established, which may limit our interpretations.

4.2. Future Directions

The current study provided information on age-related differences to muscle activation latency. However, more research is needed to understand the implications of the findings on fall-related TBIs. It has been shown that neck muscle activation can reduce the risk of TBI in young adults,[76] yet future research should focus on establishing the link between neck muscle activation latency and head impact in older adults. Furthermore, it has been shown there is a need for a dynamic neck exercise program to improve muscle activation latency.[18] More research is needed to identify an effective rehabilitation technique to improve neck muscle activation latency in older adults. Finally, future research should work to establish screening protocols to identify risk factors and apply an intervention to prevent fall-related TBIs.

4.3. Conclusion

This is the first study to examine age-related differences in neck muscle activation in response to a perturbation in adults over the age of 75 years with comparison to a younger aged group and a young adult group. Furthermore, this study examined the potential implications of age-related differences to neck muscle activation latency on fall-related TBIs. It was shown that neck muscle activation latency increases with age and understanding these age-related differences are critical for understanding the potential implications in fall-related TBIs and working to decrease this significant health problem.

CHAPTER 4: CONCLUSIONS

A traumatic brain injury (TBI) is a disruption of normal function of the brain as a result of damaging forces.[1] Fall-related TBIs are a major cause for morbidity and mortality in older adults.[1,2] It is estimated that upwards of 80% of TBIs in older adults result from the head hitting the ground or other surface during a fall.[3] In 2013, the CDC reported that there were approximately 2.8 million TBIs that resulted in emergency department visits, hospitalization, and deaths.[1] The highest rates of TBIs are in older adults over the age of 75 years.[1] Falls accounted for almost four times as many TBI-related hospitalizations and more than twice as many TBI-related deaths than motor vehicle accidents.[1] In addition to a higher incidence rate, older adults have worse health outcomes, extended hospitalizations, and a greater than 10% fatality rate following a TBI.[3]

The significant negative health impacts from fall-related TBIs make it imperative that cost effective evaluation and prevention programs be developed.[59,68] To maximize success, preventative strategies in older adults should target modifiable risk factors. Previous research examining fall-related TBIs has focused on non-modifiable risk factors such as age or gender.[4] Potential modifiable risk factors that has received minimal scientific inquiry in older adults is neck range of motion (ROM), strength, and muscle activation.

In the sports medicine literature, lower neck strength and slower muscle activation have been indicated as significant predictors for concussions (mild TBIs).[7-9,11-13] The neck is responsible for the controlling and stabilizing the head. With sufficient neck strength and muscle activation, the neck musculature may decrease head acceleration at impact and reduce the magnitude of impact forces on the brain.[5] In a large epidemiological study, it was found that lower neck strength was a significant predictor of sports-related concussion in a sample of young

adults.[6] This finding has been supported by several other studies, which found that greater neck strength resulted in significantly less head acceleration at impact in young adults.[7-9,11-13,82] Additionally, activation of the sternocleidomastoid (SCM) and the upper trapezius muscles is important for head stabilization and reducing head impact severity.[13-19] For instance, during simulated backwards falls in healthy young adults, it was shown that SCM activation contributes to the prevention and modulation of head impact.[15]

Moreover, active ROM indicates the extent to which the muscles can move the head and passive ROM reveals the extent passive structures inhibit motion.[42] Limited neck ROM may contribute to fall-related TBIs because an individual with limited neck ROM may be unable to counteract the movement of the head during a fall to prevent impact. For instance, if an individual experiences a right sideways fall, sufficient left lateral flexion ROM would be necessary to counteract the right lateral neck motion of the fall.[20]

While neck strength and activation have been indicated as potential modifiable risk factors of mild TBIs in young adults, there has been a dearth of knowledge of age-related differences to the neck musculature and function. The current project sought to fill these knowledge gaps by assessing ROM, neck muscle strength, and muscle activation latency in Young (18 – 30 years old), Young-Old (60 – 74 years old), and Old-Old adults (75 – 89 years old). It is imperative to make the distinctions between age group because older adults over the age of 75 years are at the greatest risk of fall-related TBI.[1]

To assess neck ROM, a standard goniometer was used to quantify active and passive ROM in flexion, extension, and right and left lateral flexion. Previous studies have shown that active neck ROM may decrease as much as 33% between age 20 and 90 years, [40,41] and passive neck ROM decreases 0.5º per year between the ages of 20 and 59 years in females.[42] Yet, this

study is the first to study to examine active ROM in two aged groups and the first to quantify passive ROM in a group over the age of 60 years. The results of the current study revealed active and passive neck ROM differences between age groups. As both active and passive ROM were significantly impacted by age, this may suggest that the neck muscles may be insufficient to actively move the head and the passive structure of the neck (ligaments, tendons, etc.) may interfere with the movement.[42] These significant decreases in ROM may be placing older adults at a greater risk of fall-related TBI due to insufficient ROM to counter the downward forces on the head during a fall.

Furthermore, isometric neck strength was quantified in four directions using a load cell affixed to the forehead. Previous studies have shown that the older adults had as much as 30% less flexion and extension neck strength than the young adults.[39] Yet, this is the first study to quantify neck muscle strength after the age of 75 years and in right and left lateral flexion. The result of this study showed a trending decline of neck strength with age in all quantified directions. The age-related decline in neck strength may indicate older adults have insufficient strength to control the head during a fall, which may result in head impact and TBI. As backwards and sideways falls provide the greatest risk of fall-related TBIs,[61] neck muscle strength is important to counteract the downward forces on the head during falls.

Finally, neck muscle activation latency was quantified with EMG in the SCM, upper trapezius, and splenius capitis during anterior and posterior translations. Bisdorff et al showed the SCM had a muscle activation latency 20 ms longer in older adult than young adults during a prone free fall.[47] The current study revealed increased neck muscle activation latency with age in response to postural perturbations. During anterior translations, it was shown that the upper trapezius had longer muscle activation latency times bilaterally with age. During posterior

translations, it was shown the bilateral SCMs, right upper trapezius, and right splenius capitis had longer muscle activation latency times with age. In a modeling experiment of football head collisions, it has been shown that by activating the neck muscles 40 ms before impact, the risk of a brain injury is greatly reduced.[76] Most of the muscles which displayed significant group differences revealed that older adults had muscle activation latency times at least 40 ms greater than young adults. Thus, these observations may indicate that older adults may be at a significantly greater risk of TBI, as they may be incapable of activing their neck muscle quick enough to counter the downward forces of the head during a fall.

This is the first study to examine age-related differences in neck ROM, strength, and muscle activation in adults over the age of 75 years, in comparison to a younger aged group and a young adult group. The novel results gained from this study may be the first to provide data on neck muscles and function as a risk factor for fall-related TBIs in older adults. Collectively, these findings may lead to establishing these modifiable risk factors in fall-related TBIs and work to decrease this significant health problem in older adults. Future research should establish effective rehabilitation protocols to improve neck ROM, strength, and muscle activation latency in older adults, examine head kinematics during falls, establish the link between age-related differences to neck muscle strength, ROM, and muscle activation and fall-related TBIs in older adults, and establish a screening protocol for fall-related TBIs.

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APPENDIX A: HEALTH STATUS QUESTIONNAIRE

Revised Health Status Questionnaire for Traumatic brain injuries and older adults: the implications of neck strength, activation, and range of motion

1. In general, would you say your health is? *(circle one number):*

   Excellent……..……1

   Very Good……...…….2

   Good…….3

   Fair……..…….….4

   Poor……..……...…..5

The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much? *(circle one number on each line)*

		Yes, limited a lot	Yes, limited a little	No, not limited at all
2.	Lifting or carrying groceries …….	3	2	1
3.	Climbing several flights of stairs……	3	2	1
4.	Walking several blocks……..	3	2	1

5. During the past **4 weeks**, how much difficulty did you have doing your work or other regular daily activities as a result of your physical health? (*Circle one number)*

   None at all……..…….…….1

   A little bit…….……...…….2

   Some…….3

   Quite a bit…….4

   Could not do daily work…….……..5

6. How much bodily pain have you had during the past **4 weeks**? (circle one number)

   None……..…….…….1

   Very mild……...…...……..…….2

   Mild……...……..…….3

   Moderate…….……4

   Severe……...……..……..5

   Very Severe…….……6