Rehabilitation & Performance INSTITUTE

The Vestibular System

The Vestibular System

ANATOMY & PHYSIOLOGY OF THE VESTIBULAR SYSTEM

Vestibular System Includes: Vestibule (sensory organ), CN VIII, brainstem vestibular nuclei, cerebellar pathways, vestibule-ocular reflexes (VOR), vestibulo-collic reflexes (VCR), vestibulospinal reflexes (VSR).

Purpose: provides information about head motion and orientation in respect to gravity *AND* generates eye movements to promote gaze stabilization and postural righting responses involving the head and trunk.

Vestibule:

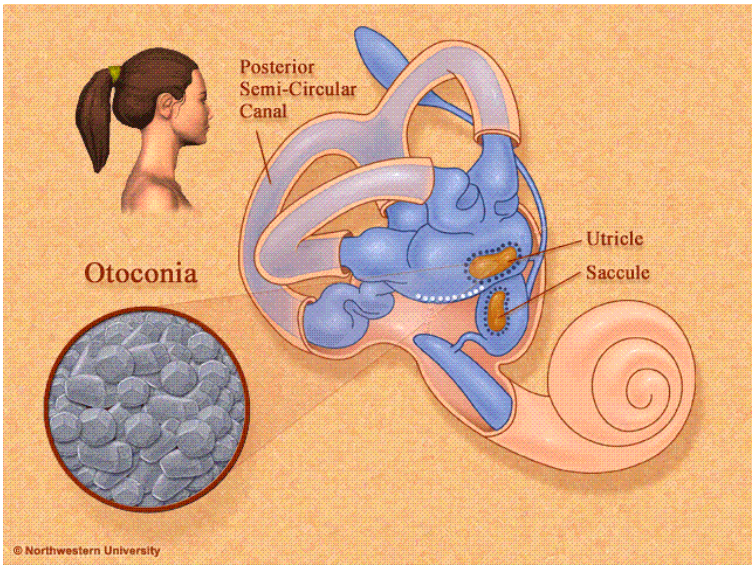
1. **Semicircular Canals:**
    - 3 bony canals in each ear- superior/anterior, posterior, and horizontal
    - Canals positioned at 90 deg angle from one another with horizontal canal tipped back 20-30 deg
    - They detect angular accelerations of the head through displacement of cupula
        - The **cupula** is embedded with sensory hair cells that sits within the **ampullated** ending of the each canal
        - It is displaced by the fluid that fills the canal (**endolymph**)
    - Function in a "push-pull" relationship- the excitation of one canal inhibits other
        - For example:
            - If you tip your chin forward and turn your head right, you stimulate your right horizontal canal and inhibit your left horizontal canal
            - If you touch your nose to your left knee, you stimulate your left anterior canal and inhibit your right posterior canal

Let's get oriented: http://www.vestibularseminars.com/vestibularmodelvideos.html


Posterior canal is located 45 degrees to the sagittal plane.

2. Otolith Organs (Utricle and Saccule)

- Medial portion of vestibule
- Semicircular canals originate from the utricle
- Sensory hair cells are embedded within the membrane (macula) of each
- Calcium carbonate crystals called **otoconia** are attached to both the medial wall of saccule and floor of utricle
    - o They enable the otoliths to detect tilts and translations of the head, because they respond primarily to linear acceleration forces (gravity).



Vascular Supply:

1. **Anterior Vestibular Artery:** supplies anterior & horizontal semicircular canals, utricle
2. **Posterior Vestibular Artery:** supplies posterior semicircular canal, saccule

Nerve Supply:

- **CN VIII** divided into 2 parts:
    - **Superior** portion innervates anterior & horizontal canals, utricle
    - **Inferior** portion innervates posterior canal, saccule

Vestibular-Ocular Reflex (VOR):

- Generates compensatory eye movements in order to stabilize gaze during head movements **(YouTube)**

Balance and Postural Control:

- Brain uses vestibular input to help it stabilize the head and body in space through neck, trunk, hip mm. activation
- Activation of distal mm. is primarily responsibility of the somatosensory system
- **Vestibulo-Spinal Reflex (VSR):** maintains vertical alignment of trunk
- **Vestibulo-Collic Reflex (VCR):** activates neck mm. to stabilize the head in space

PATHOLOGY AND CONDITIONS OF THE INNER EAR

Benign Paroxysmal Positional Vertigo (BPPV)

- BPPV occurs as a result of otoconia detaching from the otolithic membrane in the utricle and collecting in one of the semicircular canals (posterior or horizontal).
    - When the head moves, the otoconia shift, which stimulates the cupula to send false signals to the brain producing vertigo and triggering nystagmus.
    - It is typically unilateral, but can be bilateral
    - Most typical subtype: canalithiasis (free floating otoconia within the affected canal) in the posterior semicircular canal.
- Symptoms:
    - Dizziness (lightheadedness) that lasts < 2 minutes
    - Imbalance
    - Difficulty concentrating
    - Nausea
- Common problematic movements: Looking up, Rolling over, Getting out of bed
- Onset: Following trauma, Prolonged inactivity, Following surgery, Idiopathic, viruses

Vestibular Neuritis and Labyrinthitis

- Results from inflammation of the nn. of the inner ear, which disrupts transmission of sensory information from the ear to the brain.
- Inner ear infections are usually viral of nature
    - I.E.- Herpes (cold sores, chicken pox, shingles), influenza, measles, rubella, mumps, polio, hepatitis, Epstein-Barr
- The vestibulo-cochlear n. is usually involved (CN VIII).
    - One branch affects balance, the other hearing
    - When the brain receives mismatched signals when info is transmitted, vertigo onsets
    - "Neuritis" affects the branch associated with balance (no change in hearing)
    - "Labyrinthitis" affects both branches (hearing and dizziness)
- Symptoms:
    - Dizziness to violent spinning
    - Nausea
    - Vomiting
    - Unsteadiness and imbalance
    - Difficulty with vision
    - Impaired concentration
    - Tinnitus/ hearing loss

Cervicogenic Dizziness

- Afferent input from the cervical spine to the vestibular nuclei; minor input from the paravertebral mm.; disruption of cervical proprioceptive input.

Differential Diagnosis

- **Orthostatic Hypotension:** dizziness with supine to sit
- **Meniere's Disease:** Commonly over diagnosed or misdiagnosed peripheral disorder
    - Fullness in ear
    - Hearing loss
    - Tinnitus
    - Nausea
    - Postural ataxia
- **Vertebral Artery Insufficiency:** This is a medical Emergency!
    - Vertigo/ dizziness
    - HA
    - loss of consciousness
    - Gait disturbance
    - Visual disturbance
    - Nausea
    - Upper limb paraesthesias
- **Central Nervous System Disorders:** tumor, cerebellar involvement, CVA, TIA, MS, Head injury
    - Abnormal oculomotor exam
    - Resting exam
    - Pure vertical nystagmus
    - Abnormal smooth pursuit, saccades
    - Signs of CNS disorder (UMN signs)
    - Constant vertigo
- **Peripheral Vestibular loss:**
    - BPPV
    - Vestibular neuritis
    - Labyrinthitis
    - Acoustic Neuroma
- **Cervicogenic Dizziness:**
    - Neck pain
    - Trouble concentrating
    - Vision problems
    - Nystagmus- spontaneous central position
- **Others**: Allergies, Side effects of drugs, Neurological disorders, other psychological disorders

ASSESS

Medial History:

- Neurological: stroke, MS, migraine, seizure, CHI, cervical cord compression
  - Dylantum- given for seizures can cause down-beating nystagmus
- Cardiovascular: arrhythmias, orthostatic hypotension
- Endocrine: DM
- Orthopaedic: arthritis, RA, AS, DS
- Psychological: anxiety, panic

History Algorithm for the "Dizzy" Patient: Refer to flow chart to help with thought process of where to go with examination.

Examination:

- **Auditory Screen:** "Weber Test" for unilateral hearing loss
  - Vibrating tuning fork on top of head; if sound lateralizes= + for ipsilateral conductive loss or CL nerve-type deafness (512 Hz fork)

- **Gaze Stability Assessment:** Take patient's glasses off
  - **Spontaneous nystagmus**
    - Peripheral: mixed horizontal/torsional, acute lesions, prominent with fixation removed
    - Central: vertical/torsional, acute or chronic, prominent with fixation present
  - **Gaze evoked nystagmus**
    - Gaze held L, R, up, down 20-30 degrees from centered resting position; observe for nystagmus
    - Peripheral: unilateral direction, prominent with fixation removed
    - Central: direction changing, prominent with fixation present
      - Meds, Substance abuse, brainstem/Cerebellar disorder, congenital
  - **Pursuit:** Abnormal pursuit sign of CNS dysfunction, not localizing
  - **Saccades:**
    - Assess volitional horizontal saccades with special attention to: amplitude, duration, synchrony
    - Dysfunction indicative of CNS dysfunction (pons or Cerebellar)- possible MS
  - **Static v. Dynamic Visual Acuity**
    - Oscillate at 2Hz (in 15 seconds, move 30 times) and read eye chart
    - Compare static v. dynamic- >1 line degradation considered significant
  - **Head Impulse/ Thrust Test**
    - Tests VOR with rapid rotation of the head (<20 degrees)
    - Move in plane of canal of interest
    - Helps to test function of vestibular nn.

        - Normal response: gaze fixed on examiner's nose
        - Abnormal:
            A.) Ipsilateral Vestibular-Ocular Reflex (VOR): eyes travel with skull during thrust and corrective saccade in direction opposite required to go back
            B.) Central Vestibular Dysfunction: eyes over-correct for head impulse and need corrective saccade in direction of head thrust
    - **Post Head-Shaking Induced Nystagmus**
        - 2 Hz for horizontal or vertical head shaking for 15 seconds
        - Fixation removed
        - Peripheral: >3 beats considered clinically significant with unilateral vestibular dysfunction
    - **Hyperventilation induced dizziness/nystagmus**
        - 40 seconds- 1 breath/second
        - Anxiety: Symptoms within 20 seconds without nystagmus
        - Nystagmus induced= unilateral vestibular hypofunction or cerebellar pontine angle tumor
    - **Block Fixation with Frenzel Lenses**
        - Use with spontaneous nystagmus
        - Gaze holding
        - Head Shake Nystagmus
        - Hyperventilation

- **Balance/ Vestibulospinal Testing:**
    - Past pointing
    - Static balance- rhomberg, SLS
    - Reactive balance
    - CTSIB (Clinical Test for Sensory Interaction for Balance)
        - Firm surface, EO
        - Firm surface, EC
        - Compliant surface, EO
        - Compliant surface, EC← vestibular bias
        - Fuduka step test: Rotation >45 degrees with 50 steps with EC= +
- **Gait:**
    - EO
    - EC
    - Pivot
    - Head motion
    - Tandem walk

BPPV Special Tests:

- Posterior Canal:
    - **Dix-Hallpike Maneuver**: Examiner stands to side of patient, who sits upright and turns head towards examiner. Hold head and move patient rapidly to head-hanging position. First to one side and then the other. Can show burst of nystagmus after 5-10 seconds.
    - **Sidelying Test:** 45 degrees rotation, 10 degrees extension
- Horizontal Canal:
    - **Supine Roll Test:** Elevate head 30 degrees to get // to gravity. Turn head side to side to keep eye contact- at least 60-90 degrees.
      += "geotropic" nystagmus (beating towards the ground that lasts seconds) (Effected side has *stronger* nystagmus)
- Positional Changes for Patients with Pre-existing Ortho issues:
    - Severe LBP- hip/knees flexed at 90/90
    - <20 deg C/S rotation- get remainder or rotation from torso
    - Dec. C/S extension- tilt table
    - Tight hamstrings- perform SL test

BRANDT-DAROFF EXERCISES: FAIR DIAGNOSTIC TOOL FOR BPPV TO LOCALIZE CANAL IN LESION

STEP 1: Sit on the edge of the bed. You can also do this exercise on the floor or any flat surface.

STEP 2: Turn your head left, at a 45-degree angle, so that your chin is halfway to your left shoulder. After you have turned your head to the left, lie down on your right side. While lying down, your head should still remain at a 45-degree angle, which is not against the flat surface, yet not pointing toward the ceiling. **Stay in this position for 30 seconds. If you are experiencing vertigo, continue this position for one minute or until vertigo subsides.**

STEP 3: Sit up and into the normal sitting position as when you started. Remain sitting for **30 seconds**.

STEP 4: Turn your head right, at a 45-degree angle. Again, this angle would be turning your head so that your chin halfway meets with your right shoulder. After you have turned your head to the right, lie down on your left side. Remember that your head should remain at a 45-degree angle during this exercise. You should be facing halfway between the flat surface you're lying on and the ceiling. **Stay in this position for 30 seconds. If you are experiencing vertigo, continue this position for one minute or until vertigo subsides.**

STEP 5: Return again to the sitting position on the edge of your bed. Stay sitting for **30 seconds.** This exercise completes one set.

Complete five repetitions in the morning, five repetitions at noon and five repetitions in the evening.

Cervicogenic Dizziness Special Tests:

- **Modified Neck Torsion Nystagmus Test**
  - Head still, neck moves: rotate body underneath head; stabilize head position
    - If (+) for symptoms suspect neck involvement
  - Neck still, head and neck move together (en bloc)
    - If (+) for symptoms suspect vestibular system

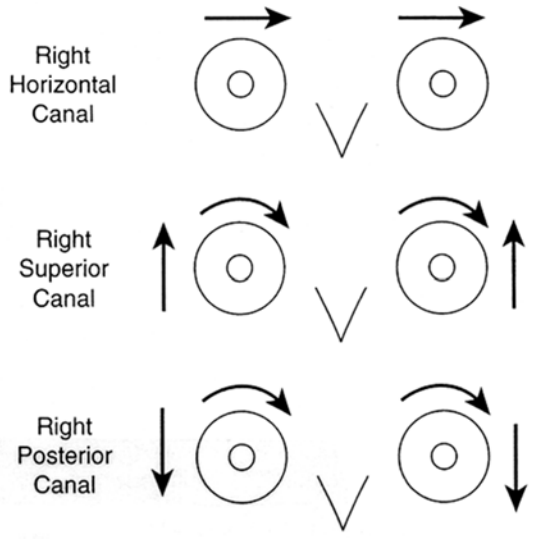
<u>How to Evaluate Nystagmus</u> (YouTube)

There are certain eye movements that are married to dysfunctions of particular canals. If the eye movements are not clear, then it may be difficult to come up with a definitive diagnosis. Clear interpretation of the eye movements can rule in or out a disorder.

- There are two phases of nystagmus:
  - Slow phase ("Drift") – vestibular activity is responsible for this phase
  - Fast phase ("Beat") – brainstem centers generate this "reset" phase
- Canal specific movements: ***slow phase component of VOR***

  Right posterior canal (RPC) = Left torsion & Down
  Right horizontal canal (RHC) = Left
  Right anterior canal (RAC) = Left torsion & Up

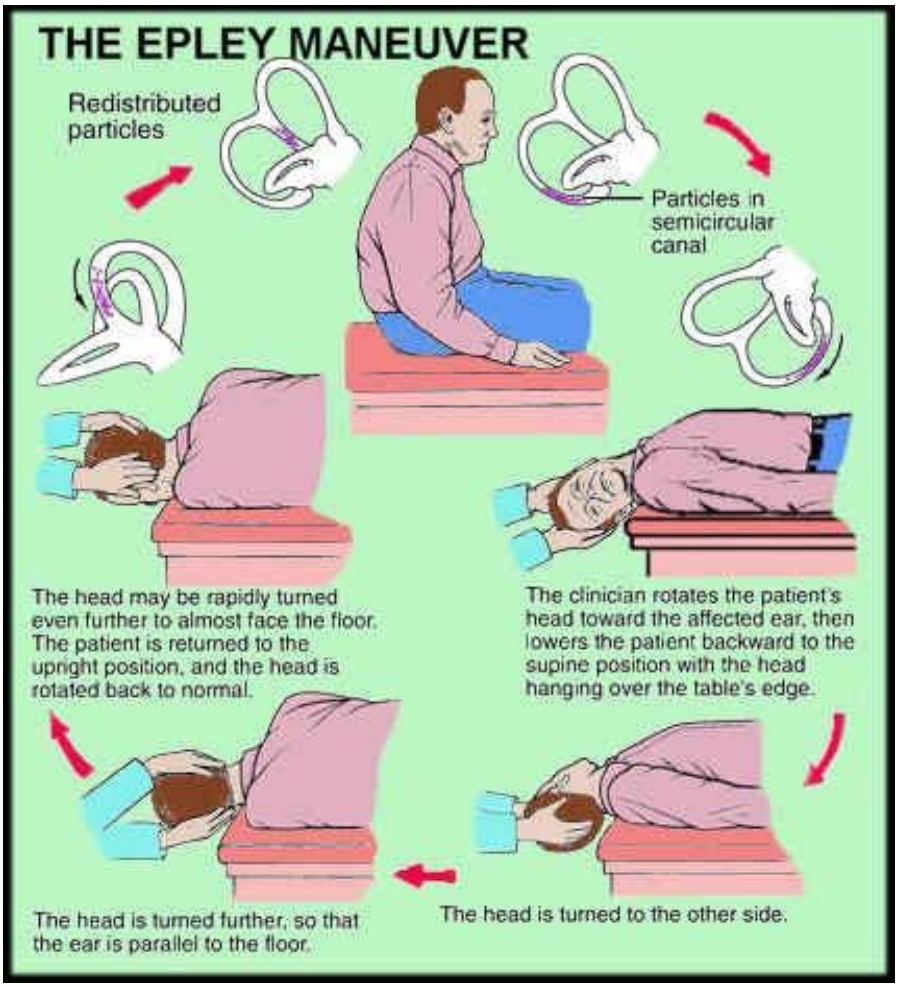
  Left posterior canal (LPC) = Right torsion & Down
  Left horizontal canal (LHC) = Right
  Left anterior canal (LAC) = Right torsion & Up



RESET THE SYSTEM

Canalith Repositioning Procedures:

- **BPPV: Posterior Canal:**
  - **Epley Flip (YouTube)**



Patient's head rotated so that the loose particles slide out of posterior semicircular canal and back into the utricle.

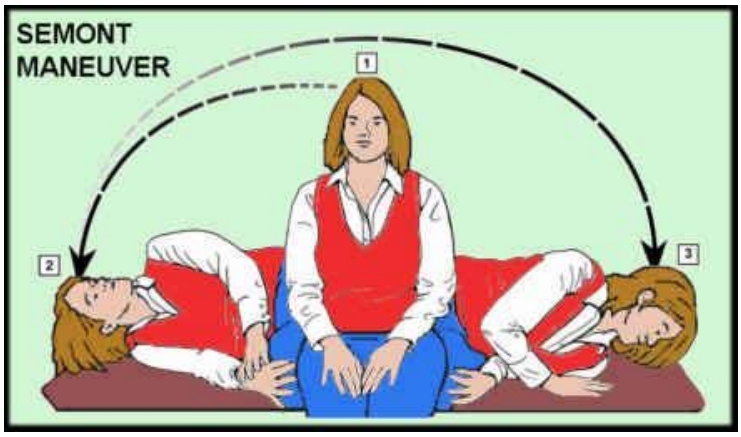
A. Dix-Hallpike Test Position- if patient is affected on the right, turn head so right ear is down to the ground.

B. Rotate patient's head so right ear is up towards ceiling. Hold for 30 seconds

C. Patient rolls onto left side and head is rotated until nose points towards the floor. Hold for 30 seconds.

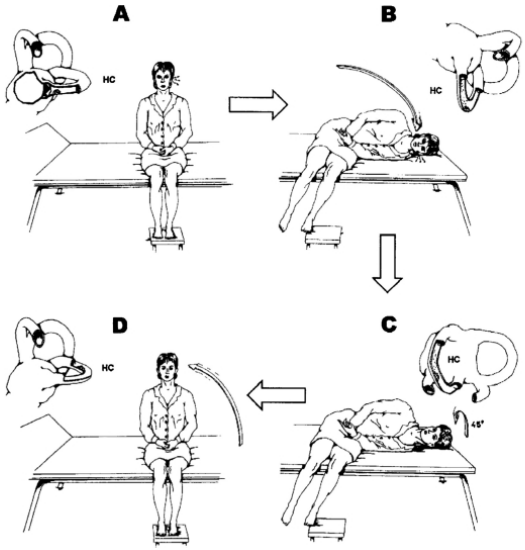
D. Patient is lifted into sitting position with head facing left.

- **Semont Maneuver**



Speed from position #2 to #3 is critical.
Nystagmus in position #3 should be consistent with position #2 (upbeating and R torsion in this example).

- **BPPV: Horizontal Canal: Gufoni's Maneuver**



Patient sits on the side of table with head straight.
Steadily moved into sidelying position on the unaffected side and remains in this position one minute after the end of the geotropic nystagmus.
Head turned steadily 45 degrees downward and is held for 2 minutes.
Patient returns to sitting position.

Videos for each of the above procedures found at:
http://www.neurology.org/content/suppl/2008/06/09/70.22.2067.DC2 and
www.vestibularseminars.com

REINFORCE THE CORRECTION

Patient Education: Post Treatment Considerations following Repositioning:

- Sleep upright with 2 or more pillows
- Avoid quick head turns
- Avoid exercises that would involve looking up/down or rotating (i.e. swimming)
- Postural education
- Transitioning from different positions
- ADLs/ANLs- activity modification
- Driving

RELOAD THE SOFTWARE

Visual Stabilization Exercises: Improve VOR

- VOR x 1: head moving, target stationary
- VOR x 2: head and target moving in opposite directions
- 2 Target VOR: gaze held on target followed by head movement
- Imaginary Target: gaze held on target, close eyes, move head, open eyes to assess accuracy
- *Things to Remember:*
    - Have to move at least 1Hz
    - Change to specific canals: horizontal, vertical, diagonal
      Otolithic: utricular translations; saccular mini tramp/BOSU
    - Print should be legible with head still
    - Alter visual target to busy environment
    - Vary target distance

Saccadic Training:

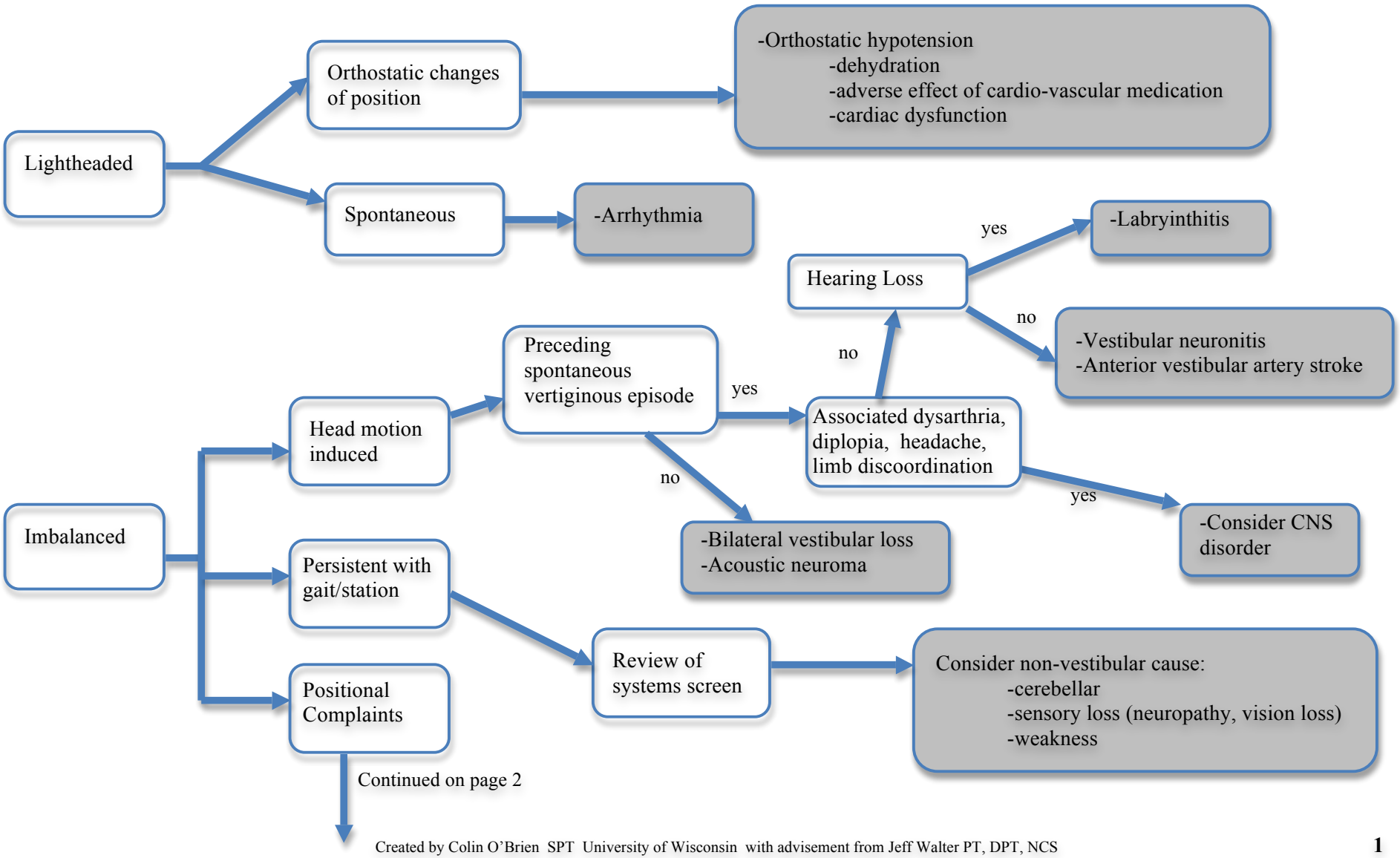
- Large words for reading
- Visual scanning
- Progressing to smaller words
- Teaching how to use peripheral vision to manage complex environments
- Busy background visual scanning (shopping in the grocery, etc.)

Balance Training:

- Gait with head and body turns
- Static standing balance with EO/EC
- Optokinetic stimulation
- Habituation: turns with and without ball toss

History Algorithm for the "Dizzy" Patient

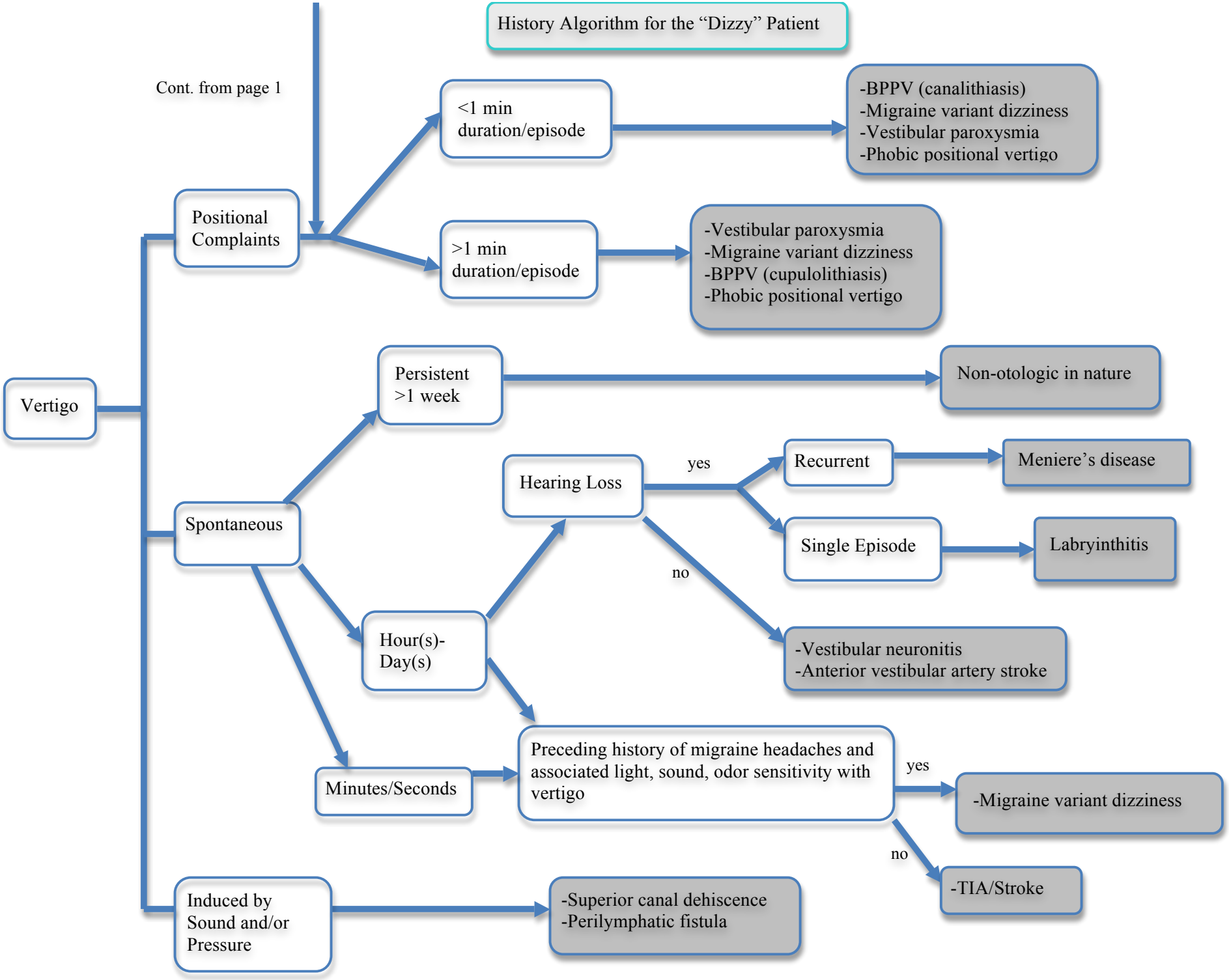
The following algorithm is designed to be a basic guide in the taking of a history from a "dizzy" patient. First the "dizzy" patient must have their chief complaint specified into: vertigo (true spinning), light headed (sense of feeling faint, or passing out), or imbalanced (unsteady or tipsy). A few of the most common diagnostic criteria are then provided to differentiate conditions. Finally the most common conditions are found at the end of each branch. This is not a substitution for a thorough history and exam.



Continued on page 2

History Algorithm for the "Dizzy" Patient

Cont. from page 1



BMC Geriatrics

RESEARCH ARTICLE Open Access

Gluteal muscle composition differentiates fallers from non-fallers in community dwelling older adults

Mario Inacio[1], Alice S Ryan[2], Woei-Nan Bair[1], Michelle Prettyman[1], Brock A Beamer[2] and Mark W Rogers[1*]

Abstract

Background: Impaired balance, loss of mobility and falls are major problems associated with changes in muscle in older adults. However, the extent to which muscle composition and related performance measures for different lower limb muscles are associated with falls in older individuals is unclear. This study evaluated lower limb muscle attenuation, intramuscular adipose tissue (IMAT) infiltration and muscle performance in older fallers and non-fallers.

Methods: For this cross-sectional study, fifty-eight community dwelling older individuals (>65 years) were classified into fallers (n = 15) or non-fallers (n = 43). Computed tomography (CT) was used to determine muscle attenuation and intramuscular adipose tissue (IMAT) of multiple thigh and hip muscles. Muscle performance was assessed with isokinetic dynamometry.

Results: For both groups, Rectus Femoris showed the highest muscle attenuation and lowest IMAT infiltration, and Gluteus Maximus and Gluteus Medius/Minimus muscles had the lowest muscle attenuation and highest IMAT infiltration. Fallers exhibited lower muscle attenuation and higher IMAT infiltration than non-faller participants in most muscles, where the gluteal muscles were the most affected ($p < 0.05$). Fallers also showed a lower peak hip abduction torque ($p < 0.05$). There were significant associations ($r = 0.31$ to 0.53) between joint torques and muscle composition, with the strongest associations between Gluteus Medius/Minimus and hip abduction strength.

Conclusions: While fallers were generally differentiated from non-fallers by muscle composition, the most affected muscles were the proximal gluteal muscles of the hip joint accompanied by lower hip abduction strength, which may contribute to impaired balance function and increased risk for falls.

Keywords: Older age, Falls, Muscle composition, Muscle performance

Background

Among older adults, falls and related injuries are major health care problems that diminish quality of living and contribute to increased morbidity and mortality [1-4]. Thus, identifying the characteristics of individuals at higher risk for falls in order to establish effective falls prevention interventions is of major clinical significance [5-8].

Impairments of balance and mobility are important risk factors contributing to age-associated falls [8,9]. During everyday activities, protective stepping is normally a commonly used strategy for stabilizing balance in response to postural challenges. However, balance control through stepping is problematic for many older individuals, especially in the mediolateral (M-L) direction where vulnerability to imbalance and fall-related injuries may be heightened [8,10]. For example, when steps were randomly evoked in 12 different directions by waist-pull postural perturbations, an age-associated reduction in balance recovery effectiveness through stepping was identified particularly for the lateral direction among prospectively identified fallers [11]. A possible reason for diminished lateral balance function may be that older individuals less often engage in activities, such as single limb weight bearing tasks, sideways whole-body movements and other tasks that require vigorous use of the hip abductor-adductor muscles important for lateral

* Correspondence: mrogers@som.umaryland.edu
[1]Department of Physical Therapy and Rehabilitation Science, University of Maryland School of Medicine, Baltimore, MD, USA
Full list of author information is available at the end of the article

balance control [10,12-14]. In this regard, older individuals have lower maximum isokinetic hip abduction torque implicated in lateral balance control than younger adults, and these age-related decrements in strength are greater for older individuals at higher risk for falls than for those at lower fall risk [7,15]. Although arthritic degeneration with older age can affect muscle composition [16], it is conceivable that the age-related reductions in physical activity patterns may also impact changes in muscle composition that affect balance and mobility particularly involving the mediolateral direction.

Aging brings about changes in skeletal muscle frequently manifested as primary sarcopenia [17]. Primary sarcopenia is characterized by an age-related reduction in muscle mass, strength, quality [18-20] and altered metabolism [21]. These changes often include increased intramuscular adipose tissue (adipose tissue within the epimysium (IMAT)) [22-24] and are associated with physical frailty, loss of mobility and increased risk for fractures [25,26]. Furthermore, IMAT infiltration and the density of the skeletal muscle fibers (muscle attenuation) may differentially affect the lower limb musculature [16,21,27].

Although sarcopenia and its associated muscle composition characteristics have received much attention, further clarification is needed concerning their relationship to clinically relevant functional impairments such as diminished muscular performance, balance function and the propensity to fall. Hence, the purpose of this study was to evaluate muscle attenuation and infiltration of IMAT by computed tomography (CT), and muscular performance in older fallers and non-fallers. We hypothesized that the proximal muscles of the hip would demonstrate greatest age-related changes in muscle composition. Furthermore, we anticipated that worse muscle composition would be associated with poorer muscular performance and that elderly fallers would show greater impairments in muscle composition and muscular performance than non-fallers.

Methods

Subjects

Participants were recruited mainly by community older adult newspaper advertisements in the Baltimore/Washington area. In order to recruit a sample of otherwise healthy community dwelling older individuals that would be able to undergo muscle composition and strength testing procedures without major confounding factors, participants aged 65 years or older were screened over the telephone by the recruitment staff, followed by a medical examination performed by a physician geriatrician. Exclusion criteria consisted of the following: 1) cognitive impairment (Folstein Mini Mental Score Exam < 24); 2) sedative use, such as Valium, Xanax or Lunesta; 3) unable to walk independently without the use of an assistive device; 4) any clinically significant functional impairment related to musculoskeletal, neurological, cardiopulmonary, metabolic, or other general medical problem; 5) engaged in a dedicated and structured physical exercise regimen for 3 or more days per week beyond more routine casual or recreational physical activity; and 6) Centers for Epidemiological Studies Depression Survey score greater than 16. The falls assessment was performed during the telephone screen and consisted of the fall history in the 12 months prior to enrollment. A fall was identified based on the criteria adopted by the World Health Organization [28]. This assessment identified 43 individuals (n = 21 males, n = 22 females) to be non-fallers and 15 (n = 5 males, n = 10 females) to be fallers. Descriptive information about the study participants is presented in Table 1. All subjects provided written informed consent that was approved by the research ethics committee from the Institutional Review Board of University of Maryland, Baltimore and the Baltimore Veteran's Administration

Table 1 Comparison between non-faller and faller groups on the sample demographics

	Non-fallers (*n=43*)	Fallers (*n=15*)	*p*
Age (yr)	74.0 ± 1.1	73.0 ± 1.1	*0.56*
Height (*cm*)	166.9 ± 1.3	165.6 ± 2.5	*0.63*
Weight (*kg*)	75.1 ± 2.2	79.0 ± 5.0	*0.42*
BMI ($kg.m^{-2}$)	26.7 ± 0.5	28.2 ± 1.3	*0.21*
Fat Mass (*kg*)	26.7 ± 1.1	31.0 ± 2.5	*0.09*
Fat-Free Mass (*kg*)	45.8 ± 1.4	47.2 ± 3.4	*0.65*
% Fat Mass	35.4 ± 0.9	38.3 ± 1.7	*0.12*
Co-morbidities (%)			
Hypertension	22.4	13.8	*0.27*
Hyperlipidemia	31.0	19.0	*0.19*
Diabetes	3.4	1.7	*0.56*
Coronary disease	1.7	3.4	*0.56*
Edema (lower limbs)	3.4	3.4	*1.00*
Painful joints	19.0	15.5	*0.65*
Osteoarthritis	24.1	13.8	*0.20*
Osteoporosis	10.3	6.9	*0.53*
Depression	1.7	6.9	*0.18*
Incontinence	1.7	1.7	*1.00*
Asthma	5.2	1.7	*0.32*
Number of reported falls in previous 12 months (%)			
1	0	66.7	
2	0	26.7	
3	0	6.7	

Data is expressed as Mean ± SEM. Comparisons significant for $p < 0.05$.

Research and Development prior to participation (HP-00040282).

Procedures

This study adopted a single session cross-sectional design where participants underwent a continuous computed tomography (CT) scan (Siemens Somatom Sensation 64 Scanner), from the 2nd lumbar vertebra (L2) to the patella while lying in supine position. From the whole scan, three regions of interest were selected for analysis, abdominal, hip and mid-thigh regions, specifically at the level of the 3rd lumbar vertebrae, 3rd sacral vertebrae and 50% of femur's length respectively (Figure 1). The following muscles were selected: Psoas (PS), Gluteus Maximus (GMax), Gluteus Medius and Minimus (GMm), Vastus Lateralis (VL), Rectus Femoris (RF), Hamstrings (Ham) compartment and Adductor Magnus and Longus (Add). Muscle cross section area (CSA), Intramuscular adipose tissue (IMAT) content and muscle attenuation were determined with MIPAV (Medical Image Processing, Analysis and Visualization, v 7.0, NIH) analysis software by tracing the fascia around the selected muscles. Scanning and analysis procedures have been previously reported in detail [24], with CT data expressed as cross-sectional area of tissue (cm2), and using Hounsfield units (HU) for muscle area between 30 to 80 HU, fat as −190 to −30 HU, and low density lean tissue as 0–29 HU [24]. In addition, the IMAT content was normalized to the respective muscle's size $\left(\text{normalized IMAT} = 100 * \frac{\text{IMAT}}{CSA}\right)$ and expressed as a percentage of the muscle's CSA (cm^2) [29].

Height (cm) and weight (kg) were measured to calculate body mass index (BMI, kg/m^2). Fat mass and lean tissue mass (fat-free mass = lean + bone) were determined by Dual-energy X-ray Absorptiometry (Prodigy, LUNAR Radiation Corp., Madison, WI). A trained radiology technician performed all DXA scans. Our past referenced CV's for total body percent fat, fat tissue mass, lean tissue mass, and BMC are 1.4%, 1.4%, 0.7%, and 0.4%, respectively [30].

Isokinetic strength testing (Biodex System 4, Biodex Medical Systems, Shirley, NY) performed by blinded trained research staff involved performing bilateral concentric contractions at $60^{o/s}$ for knee flexion-extension (seated), hip flexion-extension (supported standing), and hip abduction-adduction (supported standing). An external stabilization frame was used to minimize extraneous body movements while standing similar to previous study [7]. Knee flexion-extension was performed from 90° to 5° of knee flexion, hip flexion-extension was performed from 0° to 45° of hip flexion and hip abduction-adduction was performed from 0° to 30° of hip abduction. Due to a change in isokinetic measurement system used during the study and to procedural errors on some of the tests, the number of participants used in the analysis of the isokinetic performance variables was 12 non-fallers and 10 fallers. All peak torque data were normalized to

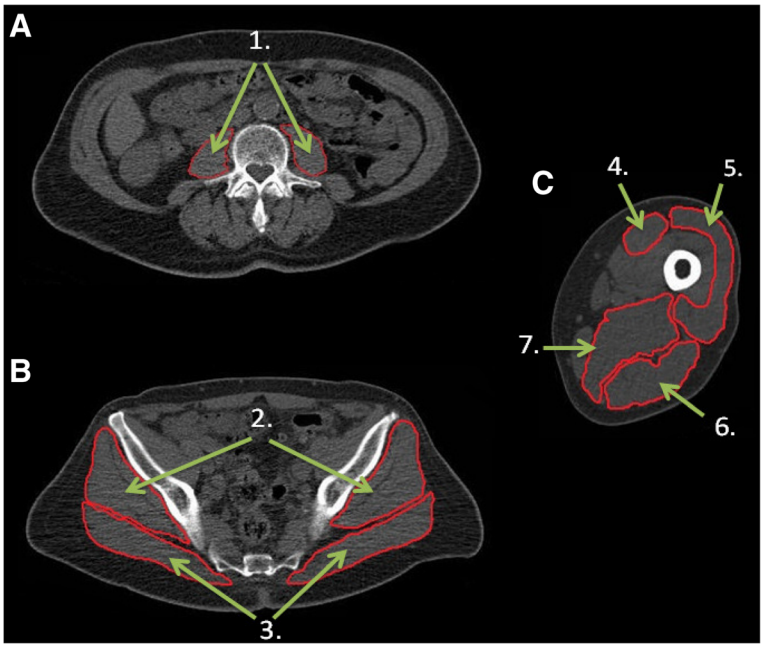


Figure 1 Representative example of computed tomography (CT) scans from an older adult showing A) abdominal scan; B) hip scan; C) thigh scan. 1. Psoas; 2. Gluteus Medius and Minimus; 3. Gluteus Maximus; 4. Rectus Femoris; 5. Vastus Lateralis; 6. Hamstrings compartment; 7. Adductor Magnus and Longus.

height and weight of each subject using the following equation:

$$\text{Normalized Torque} = \frac{\text{Peak Torque}}{Height * Weight}$$

Statistical analyses

Statistical analyses were performed using SPSS (v.17) statistical software. Considering the muscle composition measures did not have a normal distribution (Shapiro-Wilk test, $p < 0.05$), comparisons between non-fallers and fallers groups on muscle attenuation, IMAT and CSA were performed by a Mann–Whitney U test. Between-muscle comparisons on each individual group were performed by a Kruskal-Wallis one-way analysis of variance, followed by a series of Mann–Whitney U tests to test for pairwise comparisons where Bonferroni correction for multiple comparisons was applied. Muscle performance measures were normally distributed (Shapiro-Wilk test, $p > 0.05$), thus between-group comparisons of peak joint torque were performed by independent samples t-test. Associations between joint torque and muscle composition data were performed using Pearson's correlation. Significance was set at $p < 0.05$.

Results

Age, height, weight and BMI were not significantly different between groups. Approximately two thirds of the faller group experienced a single fall in the previous 12 months while the remainder of the group had two or more falls (Table 1). In accordance with the exclusion criteria, the existing co-morbidities are presented in Table 1. Due to the different gender ratio in the groups, gender was used as a covariate to assess any potential gender differences on the different groups. The results from this initial analysis of covariance showed that both genders were similar in every muscle and fall classification group for percent IMAT and muscle attenuation, with only the exceptions of Psoas and Vastus Lateralis muscles in the non-faller group for normalized IMAT (see Additional file 1, Table A1 for further details). Considering that, in general, gender appeared not to impact the present results, the entire data set was used in the analyses and described below.

Between-muscle comparisons

Multiple significant differences were found between muscle groups (see Additional file 2, Table A2 for the complete analysis). Overall, Gluteus Maximus muscle attenuation was lower than all of the other muscles ($p < 0.0024$), whereas Rectus Femoris had the highest muscle attenuation ($p < 0.0024$). In the non-faller group, Gluteus Medius/Minimus and Adductors had significantly higher muscle attenuation than Gluteus Maximus but lower values than all other muscles ($p < 0.0024$). In the faller group, Gluteus Medius/Minimus, Vastus Lateralis and Adductors had similar muscle attenuation ($p > 0.0024$), which was greater than Gluteus Maximus but less than all of the other muscles ($p < 0.0024$).The percentage of IMAT infiltration was highest for Gluteus Maximus ($p < 0.0024$) and lowest for Rectus Femoris ($p < 0.0024$) in both groups. In the non-faller group, the relative adipose infiltration was similar between Gluteus Medius/Minimus and Gluteus Maximus ($p > 0.0024$). In the faller group, Gluteus Medius/Minimus and Psoas muscles had similar relative adipose tissue infiltration ($p > 0.0024$) that was significantly less than that for Gluteus Maximus, but more than for the other muscles ($p < 0.0024$).

Between-group comparisons

Muscle CSA was similar between groups for all of the muscles ($p > 0.05$). However, the non-faller group had higher muscle attenuation values for most muscles compared with the faller group ($p < 0.05$, Figure 2). Likewise, percent IMAT in the Psoas, Rectus Femoris, Hamstrings, Adductors, Gluteus Maximus and Gluteus Medius/Minimus was higher in the faller group ($p < 0.05$, Figure 3). Normalized peak joint torque differed between the groups only for hip abduction, which was greater for the non-fallers ($p < 0.05$, Table 2).

Associations between joint torque and muscle composition

Hip abduction torque was inversely associated with Gluteus Medius/Minimus percent IMAT infiltration ($r = -0.49$, $p < 0.01$) and positively associated with muscle attenuation ($r = 0.53$, $p < 0.01$). Hip extension torque was inversely associated with Gluteus Maximus percent IMAT infiltration ($r = -0.39$, $p < 0.05$). There was a trend for an association between hip extension torque and Gluteus Maximus muscle attenuation ($r = 0.31$, $p = 0.056$). Similarly, hip flexion torque was inversely associated with Psoas percent IMAT infiltration ($r = -0.46$, $p < 0.01$) and positively associated with muscle attenuation ($r = 0.34$, $p < 0.05$). Knee flexion torque was associated with Hamstrings muscle attenuation ($r = 0.41$, $p < 0.01$), but not with IMAT infiltration. Additionally, the normalized knee extension torque was not associated with any of the muscle composition measures for the Vastus Lateralis and Rectus Femoris. A similar lack of associations was observed between hip flexion torque and Rectus Femoris, as well as between hip adduction torque and Adductors.

Discussion

This was among the first studies to investigate whether age-related changes in lower limb muscle composition

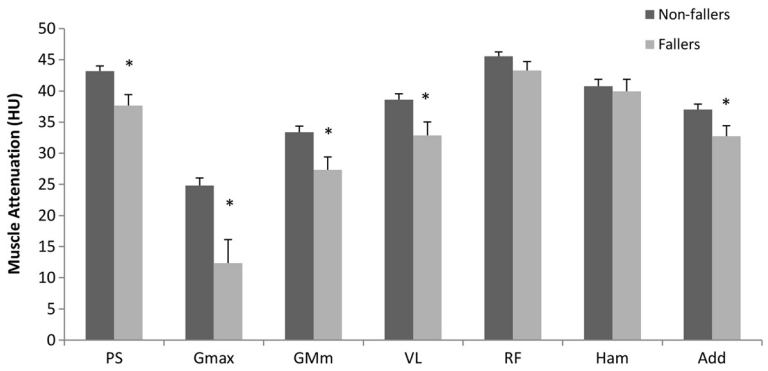


Figure 2 Comparison between non-faller and faller groups for muscle attenuation (*HU*). Data expressed as Mean ± SEM. * indicates significant difference ($p < 0.05$).

and performance differentiated between faller and non-faller cohorts of older adults who were reportedly not residing in community care or other health care settings, and have not scored zero in any item of the Instrumental Activities of the Daily Living (IADL) impairment screening tool. Twenty-six percent of the participants reported falling one or more times during the yearlong monitoring period prior to testing. This incidence of falls generally resembled past studies [3,7,8,31].

Between-muscle comparison

As hypothesized, muscle composition changes differed between lower limb muscles. Muscle attenuation analysis revealed several differences between the selected muscles across the two groups, where Rectus Femoris had the highest values and Gluteus Maximus and Gluteus Medius/Minimus showed the lowest values. These data suggested that the gluteal muscles may be particularly susceptible to composition changes with advancing age as evidenced by lower muscle density and reduced force generating capacity compared with other muscles of the lower limb [20].

Considering that muscle attenuation is inherently related to muscular adipose content [32], our findings for the adipose tissue infiltration were not surprising. In both groups, when adiposity was expressed relative to the specific muscle's cross-sectional area, we observed that Gluteus Maximus and Gluteus Medius/Minimus showed the highest relative infiltration of adipose tissue. Although previous reports have demonstrated age-related differences in composition between different muscles [16,21,27], we found the most prominent composition changes for the proximal muscles of the hip. This novel finding has clinical relevance as it indicates that disproportionate changes may occur involving the proximal hip musculature. These changes may not only affect overall muscle function, but may especially impact the ability to stabilize standing balance in the frontal plane. In this connection, osteoarthritis is associated with hip muscle dysfunction and impaired balance [33,34]. Although only a

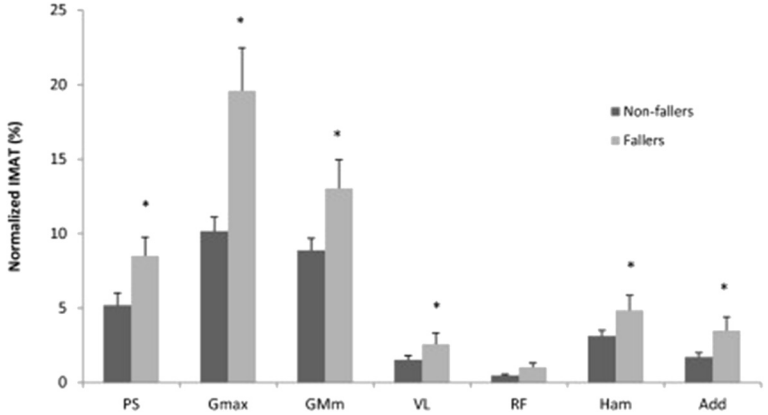


Figure 3 Comparison between non-faller and faller groups for normalized intramuscular adipose tissue (IMAT) (%). Data expressed as Mean ± SEM. *indicates significant difference ($p < 0.05$).

Table 2 Comparison between non-faller and faller groups on normalized peak isokinetic torque (N.Kg $^{-1}$)

	Non-fallers (*n=12*)	Fallers (*n=10*)	*p*
Knee Extension	0.71 ± 0.03	0.61 ± 0.03	*0.06*
Knee Flexion	0.31 ± 0.01	0.32 ± 0.02	*0.64*
Hip Extension	0.37 ± 0.03	0.36 ± 0.03	*0.73*
Hip Flexion	0.59 ± 0.02	0.54 ± 0.03	*0.12*
Hip Abduction	0.53 ± 0.02	0.41 ± 0.03	* *0.01*
Hip Adduction	0.22 ± 0.02	0.22 ± 0.02	*0.90*

Data is expressed as Mean ± SEM. *indicates significant difference ($p < 0.05$).

small percentage of our study participants reported some type of mild osteoarthritis that did not affect daily living activities, it is possible that even asymptomatic osteoarthritic changes in the hip joints may be contributing to the observed muscle composition changes.

Between-group comparison

Muscle CSA was not different between the groups for any of the muscles, and therefore does not likely confound the interpretation of the results. Nonetheless, even though the classical measurement of sarcopenia (muscle size) was not different between the groups, other muscle composition changes, such as skeletal muscle density and adipose infiltration, may be different between individuals and potentially impact performance and functional activities.

To our knowledge, this study has identified previously unreported differences in muscle composition between older fallers and non-fallers. Fallers had lower muscle attenuation in most muscle groups, with Gluteus Maximus and Gluteus Medius/Minimus showing the greatest reductions in muscle density. In addition, normalized IMAT was higher in the faller group for most of the muscle groups but especially for Gluteus Maximus and Gluteus Medius/Minimus. In particular, IMAT infiltration in Gluteus Maximus of the fallers was double that of the non-fallers. These findings differentiating between fallers and non-fallers are especially noteworthy because they were not just attributable to increased BMI as these were non-obese older adults. While several muscle composition measures identified differences between the groups, these differences were only reflected in impaired performance for the hip abductor muscles, where fallers were weaker than non-fallers.

Associations between joint torque and muscle composition

Because of the inherent inverse relationship between IMAT infiltration and muscle attenuation, it was expected that joint torque and muscle attenuation would be positively associated, while torque and adipose infiltration would be negatively associated. These results indicated that the muscle composition changes were not only greater among fallers but were also associated with their poorer muscle performance.

When considered collectively, the findings indicated that changes in lower limb muscle composition in general, and of the gluteal muscles in particular, distinguished between older fallers and non-fallers. It is widely recognized that sarcopenic changes are related to loss of muscle mass, reduced muscle strength, physical frailty, and increased risk of fracture [19,26]. Such changes have also been associated with loss of mobility [25,27,35,36] and limitations in balance function [37]. For example, increased muscle fat infiltration has been shown to be related to reduced knee extensor muscle strength, as well as future mobility limitations among well-functioning older individuals [25,36]. Similarly, IMAT accumulation in the thigh has been identified as a predictor of 6 minute walk distance, stair ascent/descent, and Timed Up and Go time [35]. Among older adults with a history of back pain, increased trunk muscle fat infiltration predicted diminished functional capacity especially for balance outcomes [38]. Hence, the present findings have implications for mobility disability related to falls among community dwelling older individuals.

With respect to balance function, protective stepping is normally a primary strategy for recovering balance in the everyday environment that becomes more prevalent and problematic for those at greater risk for falls, especially in the mediolateral direction [8,10,11]. An age-associated reduction in the maximum hip abductor-adductor muscle torque generating capacity has been linked with impaired protective stepping in the lateral direction and prospective risk for falls [7,14]. Although the non-fallers in this study demonstrated greater torque production than fallers only for hip abduction, we generally found significant associations between peak joint torques and muscle composition. The strongest associations were found for Gluteus Medius/Minimus muscles that contribute to hip abduction torque and are important for lateral balance stability. We propose that the present results may be indicative of an underlying mechanism contributing to functional deficits in frontal plane balance stability experienced by older individuals at greater risk for falls [37,39]. The substantial composition changes for Gluteus Maximus among the faller group also has relevance to lateral stability during gait where older adults have demonstrated an increased reliance on Gluteus Maximus (hip and back extensor muscle) contributions to controlling mediolateral balance when compared to younger adults [40]. Additionally, as a primary hip extensor muscle, Gluteus Maximus IMAT infiltration could contribute to impaired recovery from a trip or slip while walking via forward or backward protective stepping [37,39,41].

From a rehabilitation perspective, previous studies have shown that muscle composition can be improved with exercise training and nutrition in older individuals [23,42-46]. Therefore, the present results may help to guide exercise interventions that target proximal hip muscle composition and performance to enhance balance and mobility, two important factors in fall prevention.

Among the limitations of this study is the extent to which the results may be more generally applicable to older individuals beyond community dwelling older adults who are relatively healthy and functionally independent. Thus, frail older people with other morbidities affecting functional capacity may present different muscle composition profiles than those identified here. Additionally, although we found significant associations between muscle composition and muscular performance, the cross-sectional design of the study has a limited potential for inferring causality. A small and similar percentage of individuals in both groups reported experiencing some type of osteoarthritis, which has been associated with reduced muscle composition and quality [16,47]. Although unlikely, it is possible that it may be impacting the outcome measurements. Furthermore, the statistical power for the strength measurements may have also been affected due to the smaller sample size used in this particular test. Another limitation is that we did not account for possible differences in participants' physical activity levels. However, the impact of this limitation may have been minimized as none of the subjects were participating in a regular vigorous exercise program. The smaller sample size in the faller group was a further limitation of the study. However, the incidence of falls involving 26% of our subjects generally resembles prior studies [3,7,8,31]. It is also acknowledged that the retrospective self-report for falls may have underestimated their true incidence compared with a calendar method [48]. Nevertheless, self-report remains as an important source of information about falls occurring in the community and our present data may bolster the notion that individuals self-reporting falls are physiologically distinct from those who do not.

Conclusions

In summary, the findings indicated that age-related changes in muscle composition are not equivalent throughout the lower limb musculature. While fallers were differentiated from non-fallers by greater IMAT infiltration and greater reduction in muscle attenuation of several muscles, the most affected muscles were Gluteus Maximus, Medius and Minimus. These regionally disparate changes in muscle composition may influence directional changes in lateral balance function, and possibly, successfully recovering from a trip while walking or backward falls.

Additional files

Additional file 1: Table A1. Between-gender comparisons in the non-faller and faller groups.

Additional file 2: Table A2. Between-muscle comparisons in the non-faller and faller groups.

Competing interests

The authors have no conflicts of interest related to the content of this manuscript.

Acknowledgments

The authors acknowledge the Claude D. Pepper Older Americans Independence Center, University of Maryland School of Medicine, Baltimore, MD, USA, and the assistance of D. Yungher, J. Morgia and K. Riddle. The helpful comments of Dr. Odessa Addison on a draft manuscript are gratefully acknowledged. This work was supported by the National Institute on Aging at the National Institutes of Health (R01AG029510, P30AG028747), Claude D Pepper - Older Americans Independence Center Grant (OAIC) NIH/NIA P30 AG028747, University of Maryland Advanced Neuromotor Rehabilitation Research Training (UMANRRT) Program, supported by the National Institute of Disability and Rehabilitation Research post-doctoral training grant (H133P100014) and a VA Research Career Scientist Award to Alice S. Ryan.

Authors' contributions

MI carried out data collection and analysis, and drafted the manuscript. AR helped to draft the manuscript and with CT analysis or data analysis. WB and BB participated in the design of the study. MP participated in the design and coordination. MR conceived the study and helped to draft the manuscript. All authors read and approved the final manuscript.

Author details

[1]Department of Physical Therapy and Rehabilitation Science, University of Maryland School of Medicine, Baltimore, MD, USA. [2]Division of Gerontology and Geriatric Medicine, Baltimore VAMC GRECC and Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, USA.

Received: 10 September 2013 Accepted: 13 March 2014
Published: 25 March 2014

References

1. Alexander BH, Rivara FP, Wolf ME: **The cost and frequency of hospitalization for fall-related injuries in older adults.** *Am J Public Health* 1992, **82**(7):1020–1023.
2. Englander F, Hodson TJ, Terregrossa RA: **Economic dimensions of slip and fall injuries.** *J Forensic Sci* 1996, **41**(5):733–746.
3. Hornbrook MC, Stevens VJ, Wingfield DJ, Hollis JF, Greenlick MR, Ory MG: **Preventing falls among community-dwelling older persons: results from a randomized trial.** *Gerontologist* 1994, **34**(1):16–23.
4. Stevens J: **Fatalities and injuries from falls among older adults–United States, 1993–2003 and 2001–2005.** *MMWR Morb Mortal Wkly Rep* 2006, **55**(45):1221–1224.
5. Brauer SG, Burns YR, Galley P: **A prospective study of laboratory and clinical measures of postural stability to predict community-dwelling fallers.** *J Gerontol A Biol Sci Med Sci* 2000, **55**(8):M469–M476.
6. De Rekeneire N, Visser M, Peila R, Nevitt MC, Cauley JA, Tylavsky FA, Simonsick EM, Harris TB: **Is a fall just a fall: correlates of falling in healthy older persons. The Health, Aging and Body Composition Study.** *J Am Geriatr Soc* 2003, **51**(6):841–846.
7. Hilliard MJ, Martinez KM, Janssen I, Edwards B, Mille ML, Zhang Y, Rogers MW: **Lateral balance factors predict future falls in community-living older adults.** *Arch Phys Med Rehabil* 2008, **89**(9):1708–1713.
8. Maki BE, Holliday PJ, Topper AK: **A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population.** *J Gerontol* 1994, **49**(2):M72–M84.
9. Tinetti ME, Speechley M, Ginter SF: **Risk factors for falls among elderly persons living in the community.** *N Engl J Med* 1988, **319**(26):1701–1707.
10. Mille ML, Johnson ME, Martinez KM, Rogers MW: **Age-dependent differences in lateral balance recovery through protective stepping.** *Clin Biomech (Bristol, Avon)* 2005, **20**(6):607–616.

11. Mille M, Hilliard M, Martinez K, Zhang Y, Edwards B, Rogers M: **One step, two steps, three steps more … directional vulnerability to falls in community dwelling older people.** *J Gerontol A Biol Sci Med Sci* 2013, **68**(12):1540-1548.
12. DiPietro L: **Physical activity in aging: changes in patterns and their relationship to health and function.** *J Gerontol A Biol Sci Med Sci* 2001, **56 Spec No 2:**13–22.
13. Gottschall JS, Okita N, Sheehan RC: **Muscle activity patterns of the tensor fascia latae and adductor longus for ramp and stair walking.** *J Electromyogr Kinesiol* 2012, **22**(1):67–73.
14. Rogers MW, Mille ML: **Lateral stability and falls in older people.** *Exerc Sport Sci Rev* 2003, **31**(4):182–7.
15. Johnson ME, Mille ML, Martinez KM, Crombie G, Rogers MW: **Age-related changes in hip abductor and adductor joint torques.** *Arch Phys Med Rehabil* 2004, **85**(4):593–7.
16. Rasch A, Bystrom AH, Dalen N, Berg HE: **Reduced muscle radiological density, cross-sectional area, and strength of major hip and knee muscles in 22 patients with hip osteoarthritis.** *Acta Orthop* 2007, **78**(4):505–10.
17. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, Boirie Y, Cederholm T, Landi F, Martin FC, Michel JP, Rolland Y, Schneider SM, Topinkova E, Vandewoude M, Zamboni M: **Sarcopenia: European consensus on definition and diagnosis: Report of the European Working Group on Sarcopenia in Older People.** *Age Ageing* 2010, **39**(4):412–23.
18. Cawthon PM, Fox KM, Gandra SR, Delmonico MJ, Chiou CF, Anthony MS, Sewall A, Goodpaster B, Satterfield S, Cummings SR, Harris TB: **Do muscle mass, muscle density, strength, and physical function similarly influence risk of hospitalization in older adults?** *J Am Geriatr Soc* 2009, **57**(8):1411–9.
19. Lang T, Cauley JA, Tylavsky F, Bauer D, Cummings S, Harris TB: **Computed tomographic measurements of thigh muscle cross-sectional area and attenuation coefficient predict hip fracture: the health, aging, and body composition study.** *J Bone Miner Res* 2010, **25**(3):513–9.
20. Goodpaster BH, Carlson CL, Visser M, Kelley DE, Scherzinger A, Harris TB, Stamm E, Newman AB: **Attenuation of skeletal muscle and strength in the elderly: The Health ABC Study.** *J Appl Physiol* 2001, **90**(6):2157–65.
21. Nakagawa Y, Hattori M, Harada K, Shirase R, Bando M, Okano G: **Age-related changes in intramyocellular lipid in humans by in vivo H-MR spectroscopy.** *Gerontology* 2007, **53**(4):218–23.
22. Cree MG, Newcomer BR, Katsanos CS, Sheffield-Moore M, Chinkes D, Aarsland A, Urban R, Wolfe RR: **Intramuscular and liver triglycerides are increased in the elderly.** *J Clin Endocrinol Metab* 2004, **89**(8):3864–71.
23. Marcus RL, Addison O, Kidde JP, Dibble LE, Lastayo PC: **Skeletal muscle fat infiltration: impact of age, inactivity, and exercise.** *J Nutr Health Aging* 2010, **14**(5):362–6.
24. Ryan AS, Nicklas BJ: **Age-related changes in fat deposition in mid-thigh muscle in women: relationships with metabolic cardiovascular disease risk factors.** *Int J Obes Relat Metab Disord* 1999, **23**(2):126–32.
25. Visser M, Goodpaster BH, Kritchevsky SB, Newman AB, Nevitt M, Rubin SM, Simonsick EM, Harris TB: **Muscle mass, muscle strength, and muscle fat infiltration as predictors of incident mobility limitations in well-functioning older persons.** *J Gerontol A Biol Sci Med Sci* 2005, **60**(3):324–33.
26. Schafer AL, Vittinghoff E, Lang TF, Sellmeyer DE, Harris TB, Kanaya AM, Strotmeyer ES, Cawthon PM, Cummings SR, Tylavsky FA, Scherzinger AL, Schwartz AV: **Fat infiltration of muscle, diabetes, and clinical fracture risk in older adults.** *J Clin Endocrinol Metab* 2010, **95**(11):E368–72.
27. Tuttle LJ, Sinacore DR, Mueller MJ: **Intermuscular adipose tissue is muscle specific and associated with poor functional performance.** *J Aging Res* 2012, **2012:**1–7.
28. W.H.O: *Falls*; 2012. Available from: http://www.who.int/violence_injury_prevention/other_injury/falls/en/index.html.
29. Bouche KG, Vanovermeire O, Stevens VK, Coorevits PL, Caemaert JJ, Cambier DC, Verstraete K, Vanderstraeten GG, Danneels LA: **Computed tomographic analysis of the quality of trunk muscles in asymptomatic and symptomatic lumbar discectomy patients.** *BMC Musculoskelet Disord* 2011, **12:**65.
30. Ryan AS, Nicklas BJ, Dennis KE: **Aerobic exercise maintains regional bone mineral density during weight loss in postmenopausal women.** *J Appl Physiol (1985)* 1998, **84**(4):1305–10.
31. Lord SR, Rogers MW, Howland A, Fitzpatrick R: **Lateral stability, sensorimotor function and falls in older people.** *J Am Geriatr Soc* 1999, **47**(9):1077–81.
32. Goodpaster BH, Thaete FL, Kelley DE: **Composition of skeletal muscle evaluated with computed tomography.** *Ann N Y Acad Sci* 2000, **904:**18–24.
33. Hatton AL, Sportsphysio JL, Brauer SG, Clark RA, Crossley KM: **Dynamic single-leg balance performance is impaired in individuals with hip chondropathy.** *Arthritis Care Res (Hoboken)* 2013. doi:10.1002/acr.22193.
34. Sims KJ, Richardson CA, Brauer SG: **Investigation of hip abductor activation in subjects with clinical unilateral hip osteoarthritis.** *Ann Rheum Dis* 2002, **61**(8):687–92.
35. Marcus RL, Addison O, Dibble LE, Foreman KB, Morrell G, Lastayo P: **Intramuscular adipose tissue, sarcopenia, and mobility function in older individuals.** *J Aging Res* 2012, **2012:**629–637.
36. Schaap LA, Koster A, Visser M: **Adiposity, muscle mass, and muscle strength in relation to functional decline in older persons.** *Epidemiol Rev* 2012 [*Epub ahead of print*].
37. Grabiner MD, Bareither ML, Gatts S, Marone J, Troy KL: **Task-specific training reduces trip-related fall risk in women.** *Med Sci Sports Exerc* 2012, **44**(12):2410–4.
38. Hicks GE, Simonsick EM, Harris TB, Newman AB, Weiner DK, Nevitt MA, Tylavsky FA: **Trunk muscle composition as a predictor of reduced functional capacity in the health, aging and body composition study: the moderating role of back pain.** *J Gerontol A Biol Sci Med Sci* 2005, **60**(11):1420–4.
39. Owings TM, Pavol MJ, Grabiner MD: **Mechanisms of failed recovery following postural perturbations on a motorized treadmill mimic those associated with an actual forward trip.** *Clin Biomech (Bristol, Avon)* 2001, **16**(9):813–9.
40. Lim YP, Lin YC, Pandy MG: **Muscle function during gait is invariant to age when walking speed is controlled.** *Gait Posture* 2013, **38**(2):253–9.
41. Schulz BW, Ashton-Miller JA, Alexander NB: **Compensatory stepping in response to waist pulls in balance-impaired and unimpaired women.** *Gait Posture* 2005, **22**(3):198–209.
42. Goodpaster BH, Chomentowski P, Ward BK, Rossi A, Glynn NW, Delmonico MJ, Kritchevsky SB, Pahor M, Newman AB: **Effects of physical activity on strength and skeletal muscle fat infiltration in older adults: a randomized controlled trial.** *J Appl Physiol* 2008, **105**(5):1498–503.
43. Prior SJ, Joseph LJ, Brandauer J, Katzel LI, Hagberg JM, Ryan AS: **Reduction in midthigh low-density muscle with aerobic exercise training and weight loss impacts glucose tolerance in older men.** *J Clin Endocrinol Metab* 2007, **92**(3):880–6.
44. Ryan AS, Nicklas BJ, Berman DM, Dennis KE: **Dietary restriction and walking reduce fat deposition in the midthigh in obese older women.** *Am J Clin Nutr* 2000, **72**(3):708–13.
45. Sipila S, Suominen H: **Effects of strength and endurance training on thigh and leg muscle mass and composition in elderly women.** *J Appl Physiol* 1995, **78**(1):334–40.
46. Taaffe DR, Henwood TR, Nalls MA, Walker DG, Lang TF, Harris TB: **Alterations in muscle attenuation following detraining and retraining in resistance-trained older adults.** *Gerontology* 2009, **55**(2):217–23.
47. Conroy MB, Kwoh CK, Krishnan E, Nevitt MC, Boudreau R, Carbone LD, Chen H, Harris TB, Newman AB, Goodpaster BH: **Muscle strength, mass, and quality in older men and women with knee osteoarthritis.** *Arthritis Care Res (Hoboken)* 2012, **64**(1):15–21.
48. Mackenzie L, Byles J, D'Este C: **Validation of self-reported fall events in intervention studies.** *Clin Rehabil* 2006, **20**(4):331–9.

doi:10.1186/1471-2318-14-37
Cite this article as: Inacio *et al.*: Gluteal muscle composition differentiates fallers from non-fallers in community dwelling older adults. *BMC Geriatrics* 2014 **14**:37.

Published in final edited form as:
Restor Neurol Neurosci. 2010 ; 28(1): 57–68. doi:10.3233/RNN-2010-0515.

Postural Compensation for Vestibular Loss

Fay B. Horak, PhD, PT
Department of Neurology Oregon Health and Science University, Portland, Oregon

Abstract

To what extent can remaining sensory information and/or sensory biofeedback compensate for loss of vestibular information in controlling postural equilibrium? The primary role of the vestibulospinal system is as a vertical reference for control of the trunk in space, with increasing importance as the surface becomes increasingly unstable. Our studies with patients with bilateral loss of vestibular function show that vision or light touch from a fingertip can substitute as a reference for earth vertical to decrease variability of trunk sway when standing on an unstable surface. However, some patients with bilateral loss compensate better than others and we find that those with more complete loss of bilateral vestibular function compensate better than those with measurable vestibulo-ocular reflexes. In contrast, patients with unilateral vestibular loss who reweight sensory dependence to rely on their remaining unilateral vestibular function show better functional performance than those who do not increase vestibular weighting on an unstable surface. Light touch of <100 grams or auditory biofeedback can be added as a vestibular vertical reference to stabilize trunk sway during stance. Postural ataxia during tandem gait in patients with unilateral vestibular loss is also significantly improved with vibrotactile biofeedback to the trunk, beyond improvements due to practice. Vestibular rehabilitation should focus on decreasing hypermetria, decreasing an over-dependence on surface somatosensory inputs, increasing use of any remaining vestibular function, substituting or adding alternative sensory feedback related to trunk sway, and practicing challenging balance tasks on unstable surfaces.

Keywords

posture; compensation; adaptation; vestibular loss; rehabilitation

I. Compensation

Immediately after sudden bilateral or unilateral loss of vestibular function, patients are immediately ataxic, with severe postural instability. Over weeks and months, postural stability improves through the process of vestibular compensation that may include a greater reliance on the remaining sensory information, although more challenging tasks on unstable surfaces reveals residual instability. The extent of vestibular compensation, both spontaneous and in response to rehabilitation varies widely among individuals. Some patients, but not others, show great improvements in postural stability when using vision, light touch, remaining vestibular function, or sensory biofeedback. This paper will review several mechanisms for postural compensation after loss of vestibular function, illustrating differences among subjects, and discuss how rehabilitation can facilitate each subject's compensation by augmenting sensory information for postural stability.

Address of Corresponding Author: Department of Neurology, Oregon Health and Science University 505 NW 185th Ave Beaverton, Oregon 97006 Phone: 503-418-2600 Fax: 503-418-2501 horakf@ohsu.edu.

II. Hypermetria

Postural and gait ataxia after vestibular loss is due to increased amplitudes of both reactive and anticipatory postural responses (hypermetria). Hypermetric postural responses, as demonstrated by the large size of muscle and center of pressure responses to surface translations in cats after bilateral labyrinthectory (Fig. 1A), lead to overbalancing and instability as observed in the trajectory of the body center of mass (Figure 1B).[1] Similar postural hypermetria is also seen in human subjects after ototoxic loss of vestibular function as illustrated by the large size of surface reactive torques in response to velocities of surface translations (Fig. 1 C and D).[2] The latency and scaling of postural responses is not changed by loss of vestibular function, although the responses are hypermetric, consistent with a somatosensory trigger of automatic postural responses[3]. Thus, poorly compensated vestibular patients show postural instability partially because their proprioceptively triggered postural responses are too large, not because they lack postural responses. Vestibular hypermetria may result either from reduced cerebellar inhibition of the spinal motor system, for the loss of vestibular inputs reduces the drive to the inhibitory Purkinje cells, or from reactive synaptogenesis of somatosensory inputs to the vestibular nucleus after loss of vestibular drive (Fig. 2). The process of compensation may involve learning how to appropriately calibrate the size postural responses using remaining or augmented sensory information.

III. Sensory Substitution

To what extent can remaining sensory information compensate for the role of vestibular information in controlling postural equilibrium? Our studies have shown that some bilateral vestibular loss subjects are better than others in using their vision[4], light touch on a stable surface[5] or their remaining vestibular function[6] to substitute for missing vestibular information as a vertical reference for trunk orientation in space. For example, although all subjects with severe, bilateral loss of vestibular function are unable to stand on an fast, oscillating surface with eyes closed, half of the subjects we tested were immediately able to maintain their stability when their eyes were open (Fig. 3)[4]. In fact, stability of the head and trunk orientation in space during surface translations was within normal limits in well-compensated subjects but very large in poorly compensated subjects. Surprisingly, the well-compensated vestibular patients had somewhat lower vestibulo-ocular reflex gains than poorly-compensated subjects, suggesting that small amounts of remaining, perhaps distorted, vestibular function is not an advantage for compensation.

Light touch appears to provide a similar sensory substitution reference to earth vertical as vision for subjects with bilateral vestibular loss.5 Less than 100 grams of light touch of a single fingertip, that provides a sensory reference for earth vertical, but not mechanical support, results in a similar amount of trunk and head stability as vision when standing on a rotating surface. [5] In fact, light touch, like vision, immediately stabilizes posture primarily at the higher frequencies of postural sway in patients with vestibular loss, whereas they stabilize the lower frequencies in control subjects (Fig. 4). We found that the best-compensated patients with unilateral loss of vestibular function were those who used their remaining vestibular function in the intact ear, rather than rely upon their vision or somatosensory function.[6] When UVL subjects stood with eyes closed on a pseudo-randomly rotating surface, on average they depended upon vestibular function for postural orientation about 50% as much as age-matched control subjects. However, the ability to use vestibular information varied widely among UVL subjects, with some subjects able to depend upon their remaining vestibular function as much as controls with bilateral function while others had very little use of their remaining vestibular function for postural orientation. Subjects who were able to depend on their unilateral vestibular function rated their ADL performance7 and balance confidence8 better than those who could not depend on vestibular information when eyes were closed on an unstable surface.

IV. Sensory Addition

Augmenting sensory information for balance control by providing visual, auditory, electrotactile or vibrotactile BF of body sway has been shown to immediately reduce postural sway during stance and gait.[9] However, the extent to which BF successfully improves balance depends on the individual subject. For example, we have shown that the largest reductions of postural sway from audio-BF occurred in subjects with the least vestibular function and that vestibular loss subjects benefit more than healthy controls when standing on a foam surface with eyes closed but not when standing on a firm surface.[9] Also, both healthy and vestibular-loss subjects who tended to be visually-dependent improved the most from BF when their eyes were closed whereas those who tended to be somatosensory-dependent improved the most from BF when they were standing on compliant foam.[9] Thus, sensory BF appears to be used most in the conditions in which it is most needed.

The extent that BF is used may also depend upon how it is presented. Whereas audioBF is most successful when presented as a sigmoidal function of trunk sway, visual biofeedback of trunk sway is most successful when presented as a linear function of trunk sway.[10] Simple alarms at threshold positions of body sway may be just as useful as more complex transformations of postural stability and require further study.

Although in some ways augmented sensory feedback using BF devices appears to substitute for missing vestibular information to improve postural stability, it is not clear whether the nervous system is really increasing sensory weighting or reliance on BF like it does the natural senses or BF is just adding sensory information to reduce sensory noise. We have found that audioBF primarily reduces postural sway at lower frequencies than practice or light touch, perhaps because long loop voluntary control are used for BF. A recent study uses a model of sensory weighting to show that even a very sophisticated vibrotactile BF devise reduces postural sway on an unstable surface primarily by reducing sensory noise and not by increasing sensory reweighting to a vestibular substitute. [11]

We recently showed that BF also can improve dynamic postural stability during gait in patients with unilateral vestibular loss.[12] AudioBF of lateral trunk sway immediately and significantly reduces lateral body CoM and trunk displacements and stance width, although it does not improve the rate of motor learning. In fact, repeated practice of a difficult task, such as walking with narrow base of support and eyes closed results in significant improvement across a single session with retention of performance across two-weeks. However, despite the improved postural stability while using BF, this improvement was not retained without BF. This is what would be expected if BF acted like natural sensory information; improvements made during practice with eyes open would not be expected to transfer to improved balance with eyes closed.

V. Implications for Vestibular Rehabilitation

Studies of postural control following vestibular loss have important implications for rehabilitation of balance disorders. To identify the effects of loss of vestibular function on postural stability, clinicians need to examine patients on unstable surfaces, particularly at medium velocities of surface tilts. Rehabilitation should focus on stabilizing the head and trunk in space since postural responses in the legs may be normal. Rehabilitation should also focus on reducing hypermetric postural responses that destabilize vestibular patients, rather than attempting to facilitate reduced, late or absent postural responses.

The most powerful method for compensating for vestibular loss is sensory substitution or sensory addition. Although patients naturally compensate with remaining vestibular function, vision and touch onto stable surfaces, some patients compensate better than others. Every effort should be made to teach patients to use any remaining, useful vestibular function by practicing

balancing on unstable surfaces without use of vision, since patients who can rely on their remaining vestibular function report the best functional recovery. A cane that provides the nervous system with an external reference to earth via light touch remains the most powerful sensory substitution tool, even more powerful than vision for stabilizing posture.[5]

Sensory biofeedback devices may be useful since the nervous system appears to take advantage of any additional information well correlated with postural sway, whether auditory, visual, or somatosensory, although the characteristics of the feedback signal needs to be optimized for each sense (i.e. linear versus sigmoidal coding) and for each subject (i.e. by normalizing for each subject's limits of stability and sensory thresholds). To date, it appears that biofeedback does not act like as vestibular substitution but rather as a ‘balance prosthesis’. That is, biofeedback adds relevant sensory information about body sway to increase the signal to noise ratio. Studies are needed to determine the extent to which long-term use of BF can result in faster, more automatic use of the additional sensory information. Like a balance prosthesis, biofeedback is useful while it is being applied and not afterwards, so efforts must be made to develop biofeedback systems that are unobtrusive and can be easily suspended when adequate surface or visual references, that provide the first line of sensory substitution, are available.

In conclusion, rehabilitation for loss of vestibular function must be customized for each patient since studies are showing that individuals vary in their sensory dependence and how they compensate for vestibular loss. Control of postural orientation and equilibrium can be significantly improved in patients with bilateral or unilateral vestibular loss as long as it is considered a complex, sensorimotor skill that must be learned with appropriate feedback and active, context-specific training.

Acknowledgments

Supported by NIH grant from NIDCD 004082.

References

1. Inglis JT, Macpherson JM. Bilateral labyrinthectomy in the cat: effects on the postural response to translation. Journal of Neurophysiology Mar;1995 73(3):1181–91. [PubMed: 7608764]
2. Horak, FB. Adaptation of automatic postural responses. In: Bloedel, J.; Ebner, TJ.; Wise, SP., editors. Acquisition of Motor Behavior in Vertebrates. MIT Press; Cambridge: 1996. p. 57-85.
3. Horak FB, Nashner LM, Diener HC. Postural strategies associated with somatosensory and vestibular loss. Exp. Brain Res 1990;82:167–177. [PubMed: 2257901]
4. Buchanan JJ, Horak FB. Vestibular loss disrupts control of head and trunk on a sinusoidally moving platform. J. Vestibular Res 2001-2002;11(6):371–89.
5. Creath R, Kiemel T, Horak F, Jeka JJ. Limited control strategies with the loss of vestibular function. Exp. Brain Res 2002;145(3):323–33. [PubMed: 12136382]
6. Statler, K.; Wrisley, D.; Peterka, R.; Horak, F. Association for Research in Otolaryngology. Daytona Florida: Feb 22-26. 2004 with decreased reliance on vestibular information in people with chronic unilateral vestibular loss. [abstract]
7. Cohen HS, Kimball KT. Development of the vestibular disorders activities of daily living scale. Arch Otolaryngol Head Neck Surg 2000;126:881–887. [PubMed: 10889001]
8. Powell LE, Myers AM. The Activities-specific Balance Confidence (ABC) Scale. J. Gerontology. A. Biol Sci. Med. Sci 1995;50A:M28–34.
9. Dozza M, Horak FB, Chiari L. Auditory biofeedback substitutes for loss of sensory information in maintaining stance. Exp. Brain Res Mar;2007 178(1):37–48. [PubMed: 17021893]
10. Dozza M, Chiari L, Hlavacka F, Cappello A, Horak FB. Effects of Linear versus Sigmoid Coding of Visual or Audio Biofeedback for the Control of Upright Stance. IEEE Transactions 2006;14(4):505–512.

11. Goodworth A, Wall C III, Peterka RJ. Influence of feedback parameters on performance of a vibrotactile balance prosthesis. IEEE Transactions. Submitted.

12. Dozza M, Wall C III, Peterka RJ, Chiari L, Horak FB. Effects of practicing tandem gait with and without vibrotactile biofeedback in subjects with unilateral vestibular loss. J. Vestibular Res 2007;17 (4):195–204.

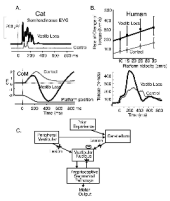


Figure 1.

Hypermetria of postural responses to surface translations in cats and humans with bilateral vestibular loss is illustrated. A. Example of semitendinosis EMG response and CoM and CoP response from 3 trials of forward-right diagonal translations in a cat before and after bilateral labyrinthectomy (adapted from Inglis and Macpherson, 1995). B. Group average of scaling surface reactive responses to increasing backward translation velocities from 7 subjects with chronic, bilateral vestibular loss due to ototoxicity and 7 age-matched control subjects and examples of surface reactive torques from a subject with bilateral labyrinthectomy compared with an age-matched control subject. Schematic of potential explanations for postural hypermetria after bilateral vestibular loss. Hypermetric proprioceptive-triggered postural responses could result either from synaptogenesis or increased efficacy of somatosensory inputs to the vestibular nucleus after loss of vestibular inputs or from loss of vestibular input to the cerebellum, resulting in loss of inhibitory drive to proprioceptive pathways involved in automatic postural responses.

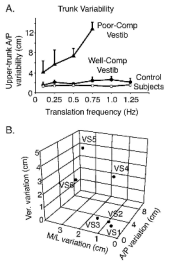


Figure 2.

A. Comparison of upper trunk orientation variability during sinusoidal surface translations with eyes open in 3 well-compensated, 3-poorly compensated vestibular loss subjects, and 10 control subjects. B. Comparison between 3 well-compensated and 3-poorly compensated vestibular loss subjects in head, trunk and leg orientation variation during sinusoidal surface translations with eyes open. Control subjects all showed head, trunk and leg orientation variation below 1 cm. (Adapted from Buchanan and Horak, 2000).

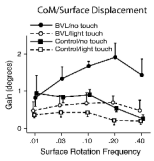


Figure 3.

Effects of light (<100 grams) finger tip touch on postural stability (gain-CoM displacement/ surface displacement) during.01, .03, .10, .20, .40 Hz sinusoidal surface rotation in seven subjects with bilateral vestibular loss and seven age-matched control subjects (adapted with permission from Creath, et al, 2002). Fingertip touch reduces postural sway more in subjects with vestibular loss than controls and subjects with vestibular loss benefit most at high frequencies of surface rotation whereas controls benefit most at low frequencies.