The Relationship between Posture and Equilibrium and the Auriculotemporal Nerve in Patients with Disturbed Gait and Balance

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Balance is defined as a state of equilibrium or parity characterized by cancellation of all forces by equal opposing factors. This is the act of maintaining an upright posture (static balance) or in locomotion (dynamic balance or gait). This system depends on vestibular function, vision, and proprioception to maintain posture, to navigate in one's surroundings, to coordinate motion of body parts, to modulate fine motor control, and to initiate the vestibuloculomotor reflexes. These parts of the vestibular system provide our brains with information about changes in head movement with respect to the pull of gravity. Besides the visual, vestibular, and skeletal systems which may contribute to balance disorders, the dental (stomatognathic) system may also contribute to balance disorders. It is when all four of these systems are in coordination with one another that equilibrium and balance will maintain a proper gait and posture. This paper will be show through normal anatomical and neurological processes how the stomatognathic system influences these activities.

Methods and Materials

. It has been recognized that there is a high association between the mandible, the cervical neck and body posture.[1] [2] If the orientation of a patients head and body posture (static balance) changes when the mandible is repositioned with an intraoral appliance[3] the next logical trial is to determine if the dynamic balance or gait is affected.

Gait is a complex motor skill that requires the integration of mechanisms of locomotion with those of balance, motor control and musculoskeletal function. The ability to walk is basic to the quality of life. The classification of gait disorders is difficult because the differing forms of gait often overlap one another. Patient complaints are of varying criteria such as slowness, unsteadiness, weakness, stumbling and falling, pain, numbness and heaviness. Other clinical features include abnormal stance, freezing, loss of balance, short stride, inappropriate postural adjustments when rising from a chair or turning, stiff legs, leg apraxia, easily fatigued, foot drag, and difficulty climbing up or down stairs. In the examination of gait, examiners observed the whole body when the patient walks. This review followed the classification of Jankovic (2001), Nutt and Sudarsky The various gait disorders can be categorized as seen in Table 1.

Table 1. Physical Signs of Gait Disorders

Physical signs	Description	Associated signs
Hemiparetic gait	Extension and circumduction of one leg	Weakness on the affected side; hyperreflexia; extensor plantar response; flexed arm
Paraparetic gait	Stiffness, extension, adduction, and scissoring of both legs	Bilateral leg weakness, hyperreflexia, spasticity, and extensor plantar responses
Sensory gait	Unsteadiness of walking when visual input is withdrawn	Positive Romberg sign; decreased position sense
Steppage gait	Weakness of foot	Atrophy of distal leg

	dorsiflexors; foot-drop; excessive flexion of hips and knees when walking; short strides; unilateral or bilateral	muscles; decreased ankle reflex; possible sensory loss
Cautious gait	Wide-based, careful, slow steps	Associated often with anxiety, fear of open spaces, and fear of falling
Apraxic gait	Difficulty initiating a step; freezing; feet almost stuck to floor; turn hesitation; shuffling gait	Hypokinesia; muscular rigidity; grasp reflexes; possible resting tremor, dementia, or urinary incontinence
Propulsive or retropulsive gait	Body's center of gravity appears to be either in front or behind the patient, who is struggling to keep his or her feet up to center of gravity; festination	Hypokinesia; muscular rigidity; postural instability
Ataxic gait	Wide-based gait; incoordination; staggering; decomposition of movements	Dysmetria; dysdiadochokinesia; tremor; postural instability
Astasia	Primary balance disorder	Postural instability
Waddling gait	Wide-based gait; swaying; toe-walk; lumbar lordosis; symmetrical	Proximal muscle weakness of lower extremities
Dystonic gait	Sustained abnormal posture of the foot or leg; distorted gait; hyperflexion of hips	Action-related gait disturbance; atypical presentations
Choreic gait	Irregular, dance-like gait; slow and wide-based; spontaneous knee flexion and leg raising	Athetotic and choreic movements of the upper extremities
Antalgic gait	Limping; avoidance of bearing full weight on the affected leg; limitation of range of movement	Pain in lower extremity aggravated by leg, hip, and thigh movement as well as weight bearing
Vertiginous gait	Unsteady gait; falling to one side; postural imbalance	Vertigo;nausea; nystagmus
Psychogenic (hysteric) gait	Bizarre and nonphysiologic gait; different varieties; rare fall or injury	Give-way weakness; Hoover sign; other signs of conversion

Ten patients with various forms of balance and gait disorder (Table 2) diagnosed by their primary physician's were seeking treatment in the dental office for various dental problems (crowns, bridges, dentures, etc.). After completing a self evaluation questionnaire the patient was given a dental examination, a treatment plan was set and a consent form was signed by each patient. Each dentist then evaluated what type of gait problem the patient exhibited and how long it has existed. In addition, a comprehensive TMD examination was also performed.

Criteria for inclusion of patients in this evaluation for gait movement disorders included the presence of certain symptoms and signs attributed to the characteristics of TMD. The data consisted of the following: (1) signs obtained from an oral evaluation (2) symptoms obtained from the patient questionnaire (3) symptoms determined immediately after treatment, one, three and six months after treatment by questionnaire and evaluation.

After normal temporomandibular joint disorder (TMJD) evaluation, the patients were found to have a malpositioned maxillomandibular jaw relationship and/or missing posterior teeth which deepened the bite. Each patient's maxillomandibular vertical was opened at 2 mm increments to find their physiological normal maxillomandibular relationship. Upon finding the specific increase in vertical dimension at which the symptoms and dysfunctions were eliminated or greatly improved impressions of each patient were made and a removable oral orthotic device called the Neurocranio Vertical Distractor (NCVD) was made by a commercial laboratory. The patented NCVD covers the posterior mandibular molar teeth, and contains a lingual bar along the gingival margin of the anterior teeth. The NCVD appliance is constructed for each individual patient. The

posterior clear orthodontic acrylic which covers the posterior teeth has a platform which allows vertical adjustments. The posterior base is designed with stabilizing pins to help adjust the maxillomandibular relationship. The acrylic portion can also be added to and adjusted if necessary. The NCVD is to be worn 24 hours a day, seven days a week, for a period of 6 months. The appliance was only to be removed for daily oral hygiene. Patients were instructed to eat, sleep, and go about daily activities with the appliance in place.

TABLE 2

PATIENT	GAIT DISORDER TYPE
Patient # 1	Propulsive/Astasia gait
Patient # 2	Apraxie/Propulsive gait
Patient # 3	Dystonic gait
Patient # 4	Antalgic/Dystonic gait
Patient # 5	Choreic /Ataxic gait
Patient # 6	Waddling/Ataxic gait
Patient # 7	Vertiginous/Sensory/Cautious/Ataxic gait
Patient # 8	Paraparetic gait
Patient # 9	Cautious gait
Patient #10	Antalgic gait

Each patient was video taped as they walked about the office and up a flight of stairs both prior and after receiving the NCVD appliance. When the patients received their appliances, there was an **immediate** change in their gait and/or balance. The patients where able to walk normally with a steady gait and also ascend a flight of stairs without

much difficulty. These improvements were monitored and evaluated upon return visits to the dental office at 1 week, 1, 3 and 6 month intervals and the improvement in their balance and gait disorder was consistent. These were activities they were not able to do prior to appliance insertion. At the six month visit, the posterior acrylic had worn down and was refreshed with additional (Table 3). At six months gait disorder movements of the patients were partially returning which was evident by their moving slower and more deliberately. Upon refreshing the posterior acrylic to the physiological normal height previously determined, the patient's normal/improved gait returned immediately.

Table 3

Results

	Normal Gait Achieved	Abandoned Walking Aid	1 Month Eval	3 Month Eval	6 Month Eval
Pt. 1	Yes	X	O	O	O
Pt. 2	Yes		O	DNR	-/+
Pt. 3	Yes		O	O	-/+
Pt. 4	Yes	X	O	-/+	-/+
Pt. 5	Yes		O	DNR	DNR
Pt. 6	Yes	X	O	O	O
Pt. 7	Yes	X	O	O	O
Pt. 8	Yes		O	O	-/+
Pt. 9	Yes		O	O	DNR
Pt. 10	Yes		O	DNR	-/+

O = No Change
\- = Abnormal Gait Returned
\+ = Refreshment of Appliance / Normal Gait Returned
DNR = Did Not Return

Discussion

Balance is one the leading health concerns for people over the age of 65 and more than 1/3 of this population suffers a fall in the United States each year.[4] Balance is also dependent on good muscle strength and joint mobility. Disorders especially in the elderly that result from visual problems (including cataracts, glaucoma, diabetic retinopathy, and macular degeneration) can cause peripheral neuropathy which affects the positional sense of the legs and feet. Among older adults, falls are the leading cause of death. They are also the most common cause of nonfatal injuries and hospital admissions for traumatic brain injuries (TBI) in the elderly. Twenty to thirty percent of people who fall suffer moderate to severe injuries such as bruises, hip fractures, or head traumas.[5] Many people who fall, even those who are not injured, develop a fear of falling. This fear may cause them to limit their activities, leading to reduced mobility and physical fitness, and increasing their actual risk of falling.[6] These injuries can limit normal activities of daily living and consequently limit independent living. They also can increase the risk of early death. Men are more likely to die from a fall. After adjusting for age, the fall fatality rate in 2004 was 49% higher for men than for women and women are 67% more likely than men to have a nonfatal fall injury. The risk of being seriously injured in a fall increases with age. In 2001, the rates of fall injuries for adults 85 and older were four to five times that of adults 65 to 74.[7] The multiplicity of medications ingested by the elderly can also contribute to vestibular imbalance effects. The total direct cost of all fall injuries for people 65 and older in 2000 was slightly more than $19 billion: $0.2 billion ($179 million) for fatal falls, and $19 billion for nonfatal falls[8].In addition there are psychological causes, as well as other unknown causes that may cause balance disorders.

One of the unknown causes that routinely has not been considered concerns the neurological aspects of the stomatognathic system.

The ability to move about without hindrance is an important factor in the quality of life for the young and the old. Dizziness is reported to be the most common complaint for patients over 75 years of age and the elderly seek treatment for dizziness more frequently than for hearing loss, and, that 3,200 per 100,000 new cases of dizziness per year result in a visit to a primary care physician.[9] In this group 68% were found to have benign paroxysmal positional vertigo, 9% neurologic problems, 5% Meniere's disease, 4% vestibular areflexia, 8% a rare cause such as a psychiatric or vascular disorder, and no cause was found in 6%.[10].

Balance control is accomplished by an autoregulating reflex reaction with inputs from the sensory systems of the eyes, ears, muscles and joints relative to your environment. Additionally it relies on the brain and the brainstem's ability to process this information. If our balance control is abnormal, we then must exert conscious effort to control our balance and this leads to compensatory symptoms that may cause fatigue.

Some of the most common balance disorders are:

(A) Labyrinthitis – which is an inflammation usually caused by a virus within the labyrinth; this is the part of the inner ear that contains the organs of balance which may cause dizziness or imbalance.

(B) Benign Paroxysmal Positional Vertigo (BPPV) - a brief, intense sensation of vertigo that occurs because of a specific positional change of the head caused possibly by a problem in the vestibular system of the inner ear. The cause of BPPV is unknown although is it may be caused by an inner ear infection, head

injury, or aging. This type of vertigo comes and goes and if left untreated, may occur for years.

(C) Post-traumatic vertigo is dizziness resulting from a head injury, concussion, or whiplash.

(D) Tinnitus is buzzing or ringing in the ears and can occur with dizziness, but it may be a symptom in itself.

(E) Meniere's disease is an inner ear fluid balance condition, which causes repetitive attacks of vertigo and is accompanied by pressure in the ears, vomiting, buzzing and/or ringing, and a partial hearing loss which can fluctuate during an episode. The cause of Meniere's disease is also unknown.

Determining the cause of a balance disorder and what treatment options are appropriate may be diagnostically complex and difficult because of the existence of underlying medical conditions. Ear infections, blood pressure changes, vision problems and medications contribute to balance disorders.[11] Medical conditions such as multiple sclerosis, stroke and Parkinson's disease can contribute to balance disorders. The otolaryngologist who specializes in diseases and disorders of the ear, nose, throat, and neck with expertise in balance disorders usually performs a combination of observational tests during physical examination and takes a detailed medical history. This physician makes an assessment of the patient walking and determines whether the patient can walk in a straight line without veering side to side. He makes another observation for stumbling and/or hesitation or whether the patient can walk heel to toe.

Another assessment by the physician evaluates the function of the cerebellum. This part of the brain, if damaged, can produce movement, but the movements become

jerky, making it difficult to get hands and legs to stop moving at the position the patient desires.[12] The third assessment is eye and head movement functions, indicating the patient's ability to coordinate movements of the eyes and head while walking or running.

The tests the physician would order are the audiometric (hearing) test and the nystagmography test. The audiometric test evaluates the patient's ability to follow visual objects with their eyes and how the eyes respond to different types of information that is inputted into the vestibular system. The eyes are monitored by electrodes in the Electro-nystagmography test (ENG) or by an infrared video camera (IR-Video Nystagnography) which helps determine the patient's balance system problems.

The physician will probably also order imaging tests such as a CAT scan or MRI of the brain and head to see the inner ear structures as well as the nerve connections of these organs to the brain.

Anatomy of the Motor and Vestibular Systems

Motor activity can be either voluntary or involuntary. Voluntary movements are planned and started by a 'forward feeding' control mechanism and are maintained and controlled by a 'feedback loop'. These muscles and movements act as a lever system on bones and joints (contraction and relaxation). Posture is a body pose from one position to another. Involuntary movements comprise reflexes, such as the stretch reflexes and autonomic functions such as cardiac and respiratory reflexes. We have motor systems in the central nervous system (CNS) which are organized in the cerebral cortex, brainstem, spinal cord, cerebellum, and basal ganglia. The cerebral cortex contains the motor areas

from which commands originate that go to the brainstem and spinal cord and activate voluntary muscle movement. The brainstem contains descending motor pathways that modulate the motor activity within the spinal cord. It processes and coordinates the neural signals received from the cortex, the cranial nerves, (vestibular, trigeminal, etc.) and sensory neurons. The spinal cord is involved in automatic and stereotypic motor reactions to peripheral stimuli known as reflexes. The myotatic stretch reflex is the crucial monosynaptic reflex for the maintenance of the body posture in a human being. The main function of the basal ganglia is to initiate and stop movements. They receive input from all cortical levels, process the information and then send output to the premotor and motor areas of the cerebral cortex. The cerebellum helps regulate movements and posture including eye movements and balance. The cerebellum is particularly concerned with conjugate movements between agonist and antagonist muscle groups. Motor learning is programmed into the cerebellum and it controls the sequence of movements and makes corrective adjustments to complex movements.

The purpose of the vestibular system is to sense changes in motion or rotation of the head in space. The information is processed through three different sources: the eyes (visual system), general proprioceptive receptors throughout the body (senses), and vestibular receptors in the inner ear (balance system). The vestibular labyrinth within the inner ear is contained within the temporal bone. This structure contains three semicircular canals (3SC) (anterior, posterior, and lateral) within the left and right side of the temporal bone which detect head acceleration and angulations and act as counter balances for each other (fig.1). The semicircular canals contain endolymph and have a swelling at each end of it called the ampulla. Within this ampulla is the cupula, a

gelatinous barrier that surrounds sensory cilia. Flow of the endolymph inside the semicircular canals displaces the cupula, thereby bending the cilia. This activates or inhibits the sensory cells depending on the direction of the bending. Rotational movements of the head produce flow of the endolymph in the semicircular canals. The orientation of the semicircular canals ensures that rotation of the head is perceived in all conceivable planes by producing changes in the activity of the receptor cells in one or more of the semicircular canals. The brain monitors the rotational movements of the head by reading the pattern of activity produced by all receptors.

In order for the brain to perceive the activity of the vestibular system, it must receive afferent impulses from various parts of the vestibular system. When there is bending or deflection of the cilia within the semicircular canals, activation of the vestibular nerves occur. Nerve afferents are located in the superior and inferior vestibular ganglia, which are within the internal auditory canal near the cerebello-pontine angle. The superior vestibular nerve innervates the lateral and anterior semicircular canals. The inferior vestibular nerve innervates the posterior semicircular canal. Sensory neurons of the vestibular nerves terminate in four (4) vestibular nuclei. Primary vestibular afferents in the superior vestibular nerve bifurcate and end on two different vestibular nuclei. The superior and the medial nuclei then synapse and go to the cerebellum. The inferior vestibular nerve bifurcates and terminates on the lateral, inferior, and the medial vestibular nuclei which then synapse within the brainstem. The superior vestibular nuclei along with the inferior, medial and lateral nuclei form the vestibular nuclear complex.

The vestibular complex receives afferents from other parts of the central nervous system, especially from the spinal cord, the reticular formation, parts of the

mesencephalic nuclei and the cerebellum. Afferent neurons contribute to the adaptation of vestibular reflexes in response to changing conditions.

The Vestibular Nuclei act on four main regions:

1.) Motoneurons of the Spinal Cord

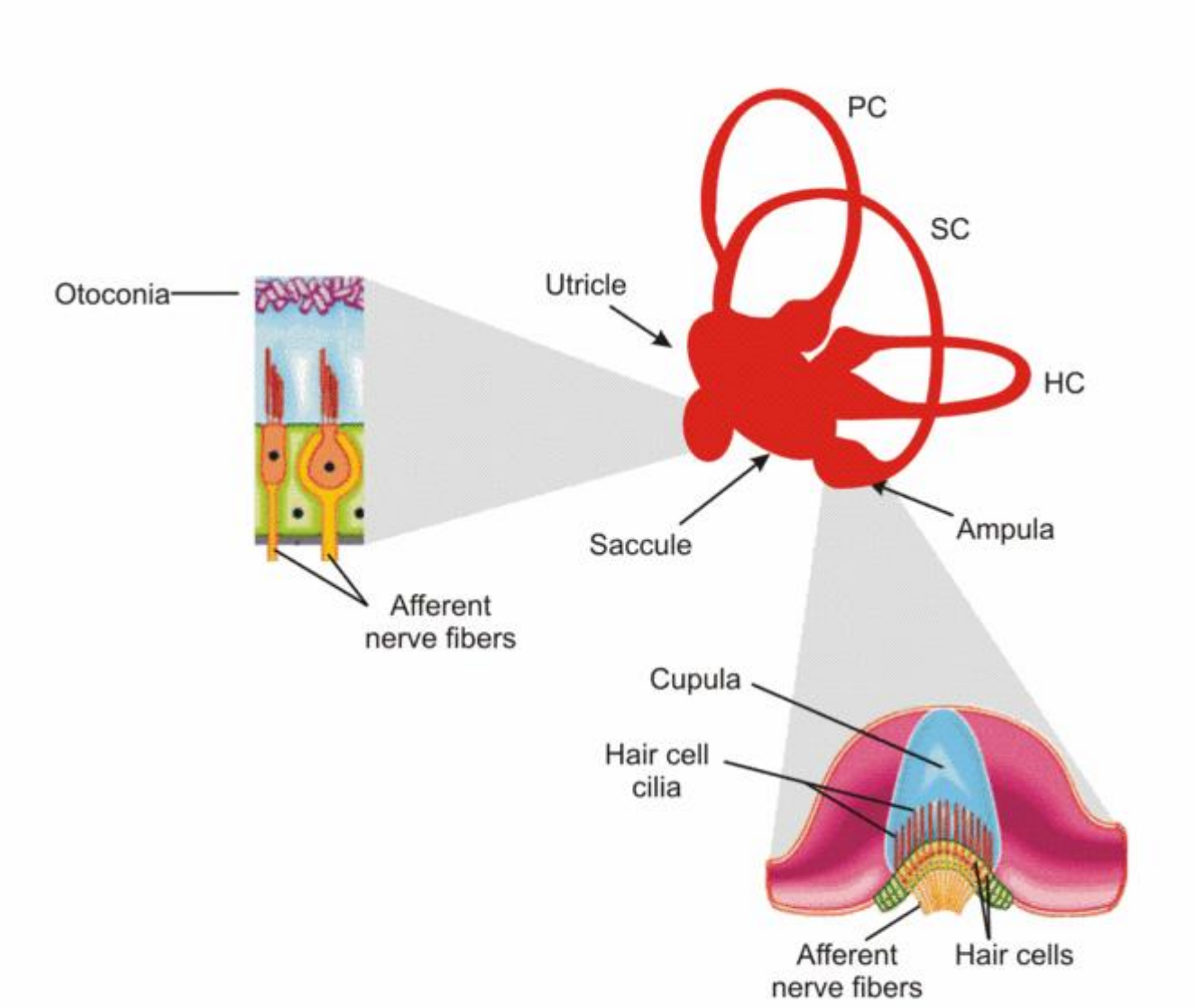
2.) Motoneurons of the nuclei of the extraocular muscle's

3.) The cerebellum.

4.) The reticular formation

The processes that exist primarily within the brain stem control many of the vestibular reflexes. Connections within the vestibular cortex, thalamus, and the reticular formation enable the vestibular system to contribute to the integration of arousal and conscious awareness of the body and to discriminate between movements of one's self and one's environment.

Most of the efferent fibers to the spinal cord come from the lateral vestibular nuclei from the lateral vestibulospinal tract (fig. 2). This tract produces strong effects on the muscles that result in posture during static and locomotive states and influences the coordination of limb movements for equilibrium and posture. The smaller medial vestibulospinal tract (fig3) arises from the medial vestibular nucleus which is the terminus for motoneurons. It primarily functions to stabilize the head in space. These descending vestibulospinal tracts act on both gamma and alpha motor neurons that go to axial and proximal limb muscles to maintain the body against gravitational forces keeping an individual in an upright position.


PC
SC
Otoconia
Utricle
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Saccule
Ampula
Afferent
nerve fibers
Cupula
Hair cell
cilia
Afferent
nerve fibers
Hair cells

Fig. 1

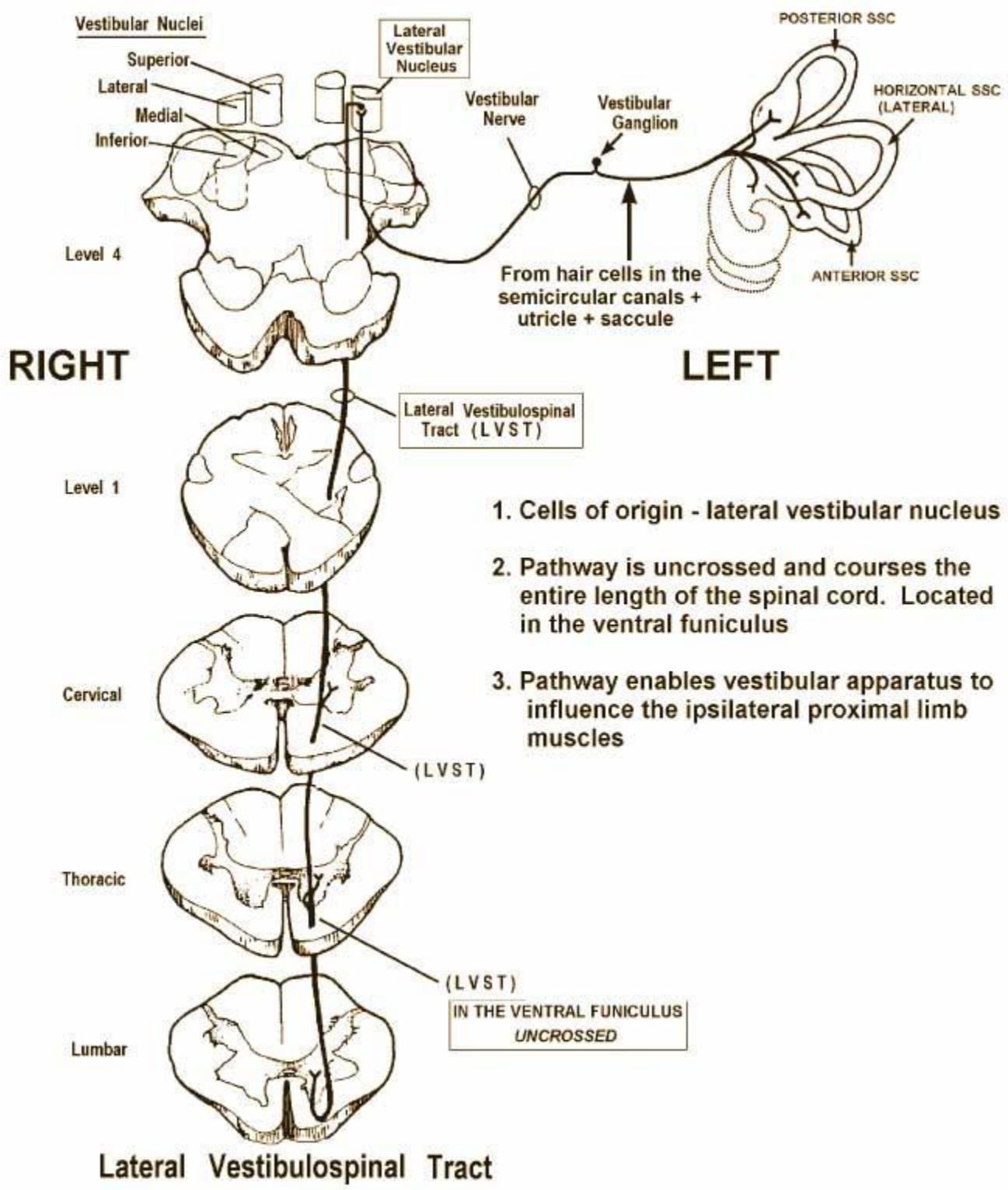


Fig. 2 http://www.neuroanatomy.wisc.edu/virtualbrain/BrainStem

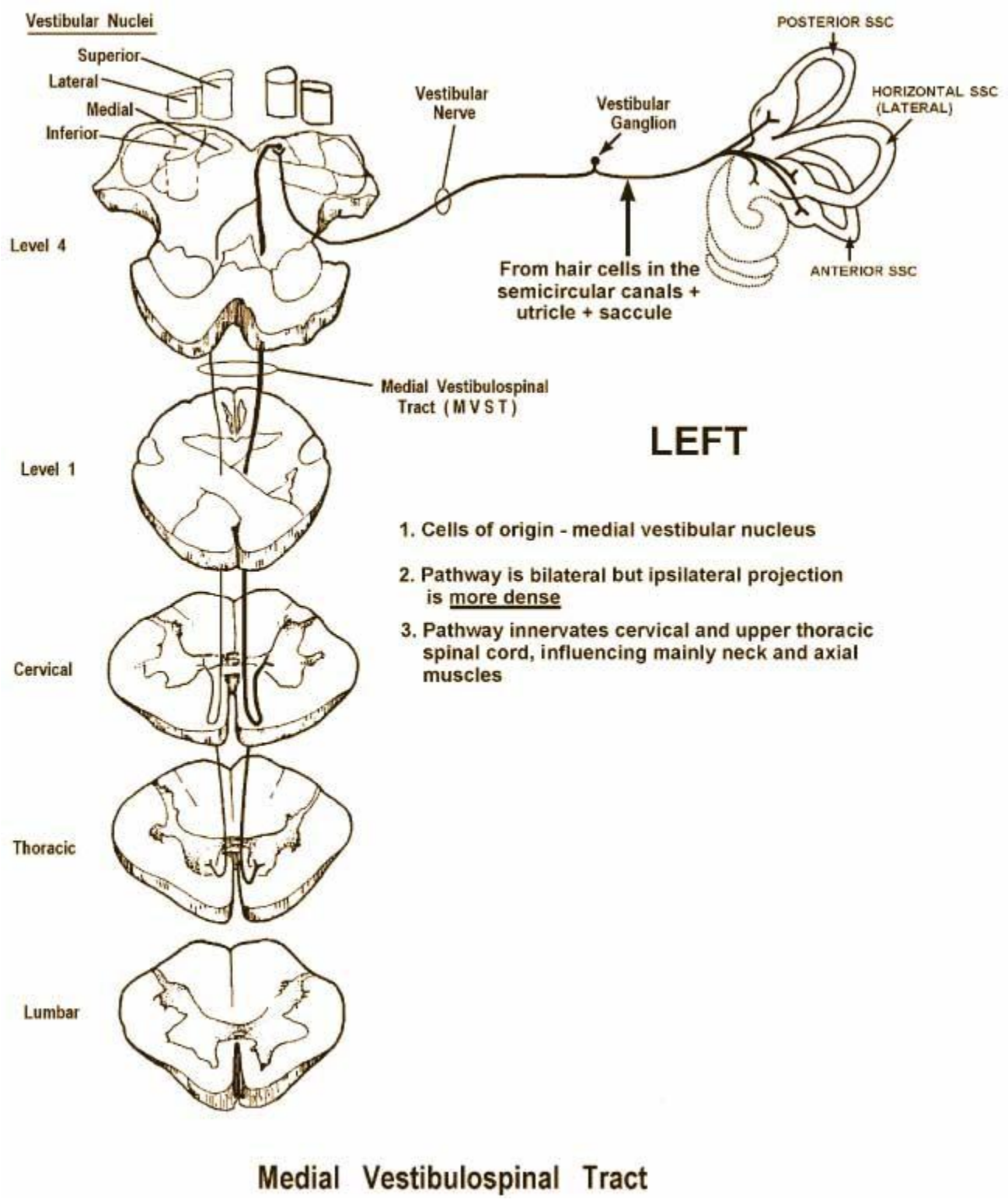


Fig. 3 http://www.neuroanatomy.wisc.edu/virtualbrain/BrainStem

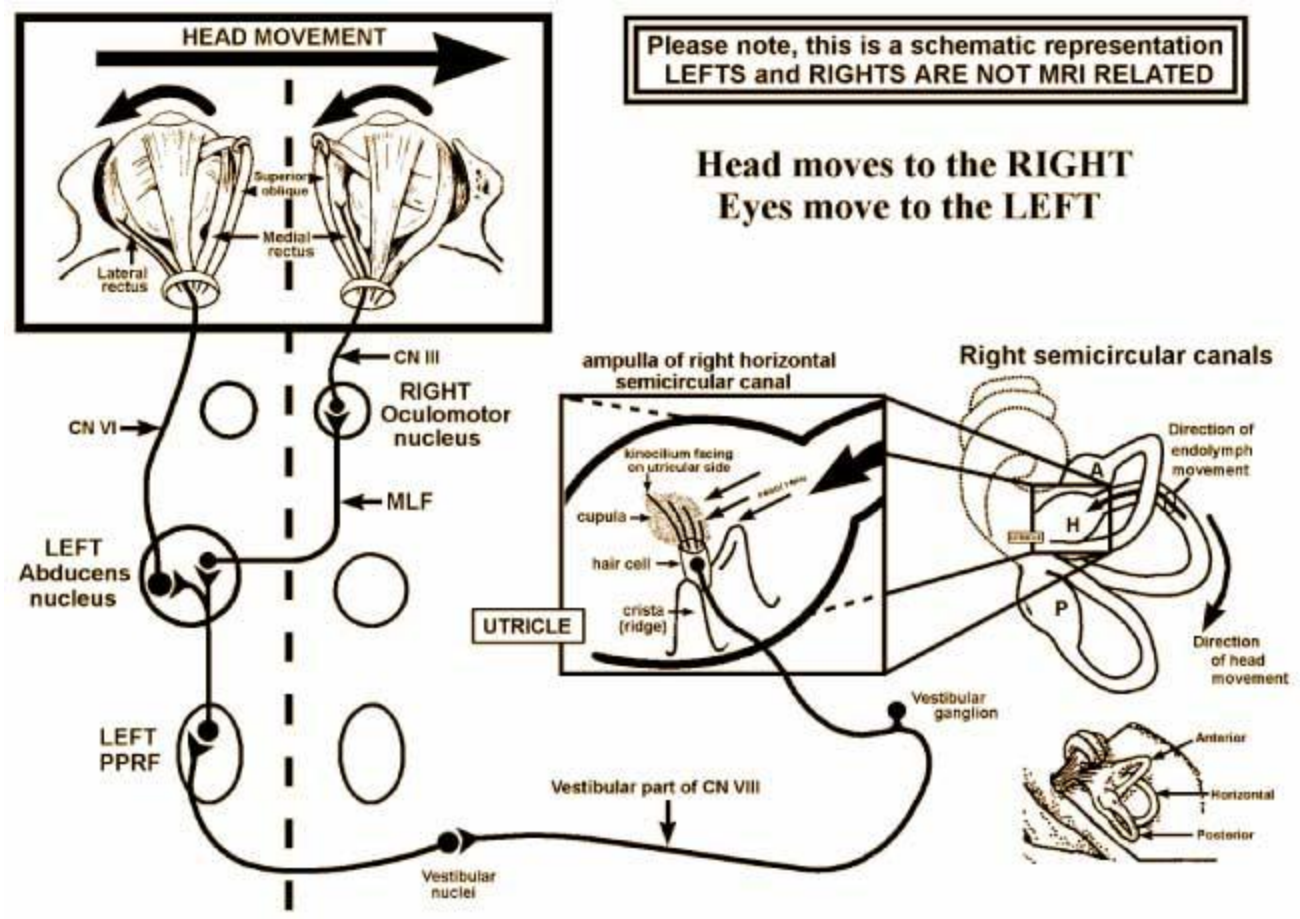
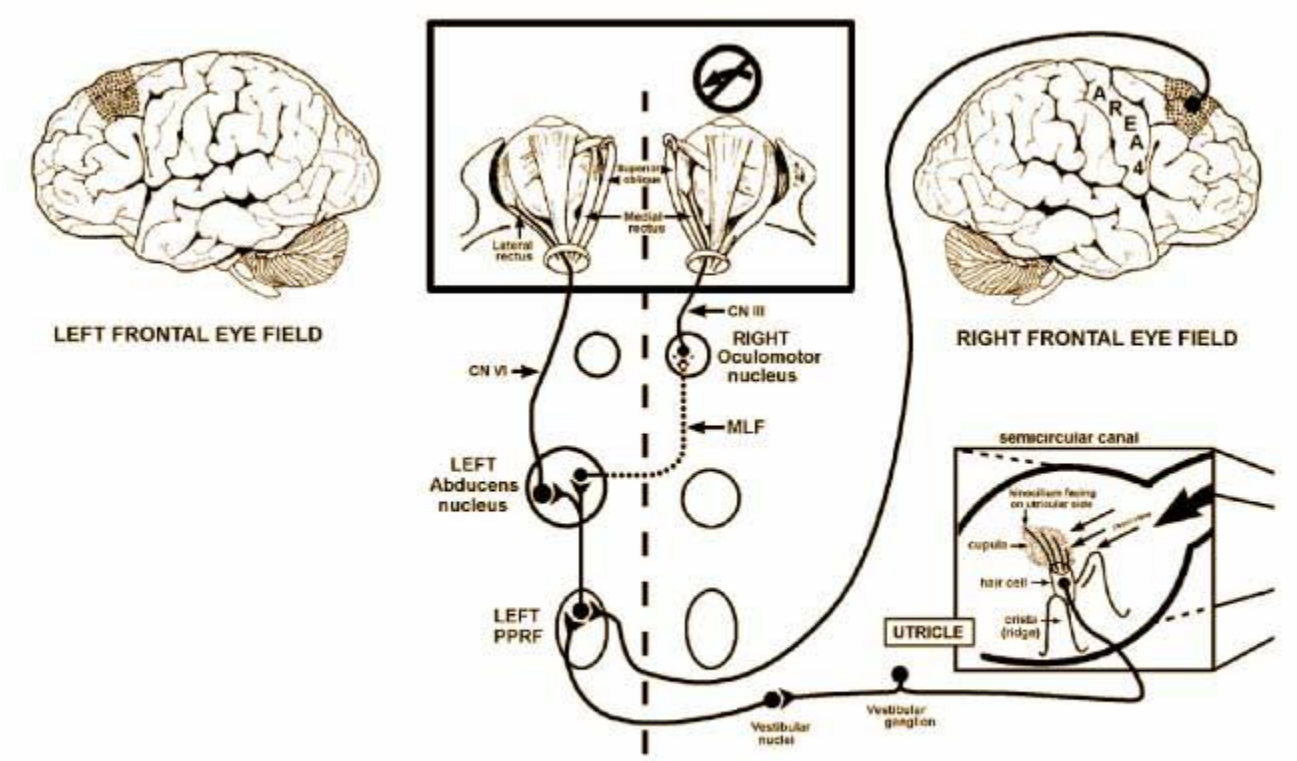


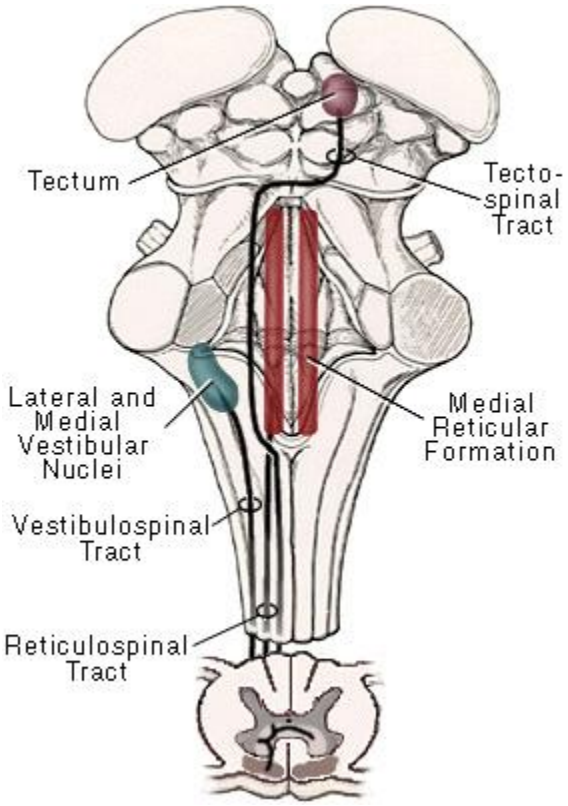
Fig.4 http://www.neuroanatomy.wisc.edu/virtualbrain/BrainStem



Lesion of the RIGHT MLF = inability to turn the RIGHT eye past the midline to the LEFT = INTERNUCLEAR OPTHALMOPLEGIA (INO), No atrophy of the RIGHT medial rectus, Diplopia when attempting to look LEFT of the midline, also there is nystagmus of the LEFT eye

Fig. 5 http://www.neuroanatomy.wisc.edu/virtualbrain/BrainStem

Fig. 6


Tectum
Tecto-
spinal
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Lateral and
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Nuclei
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Formation
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Tract

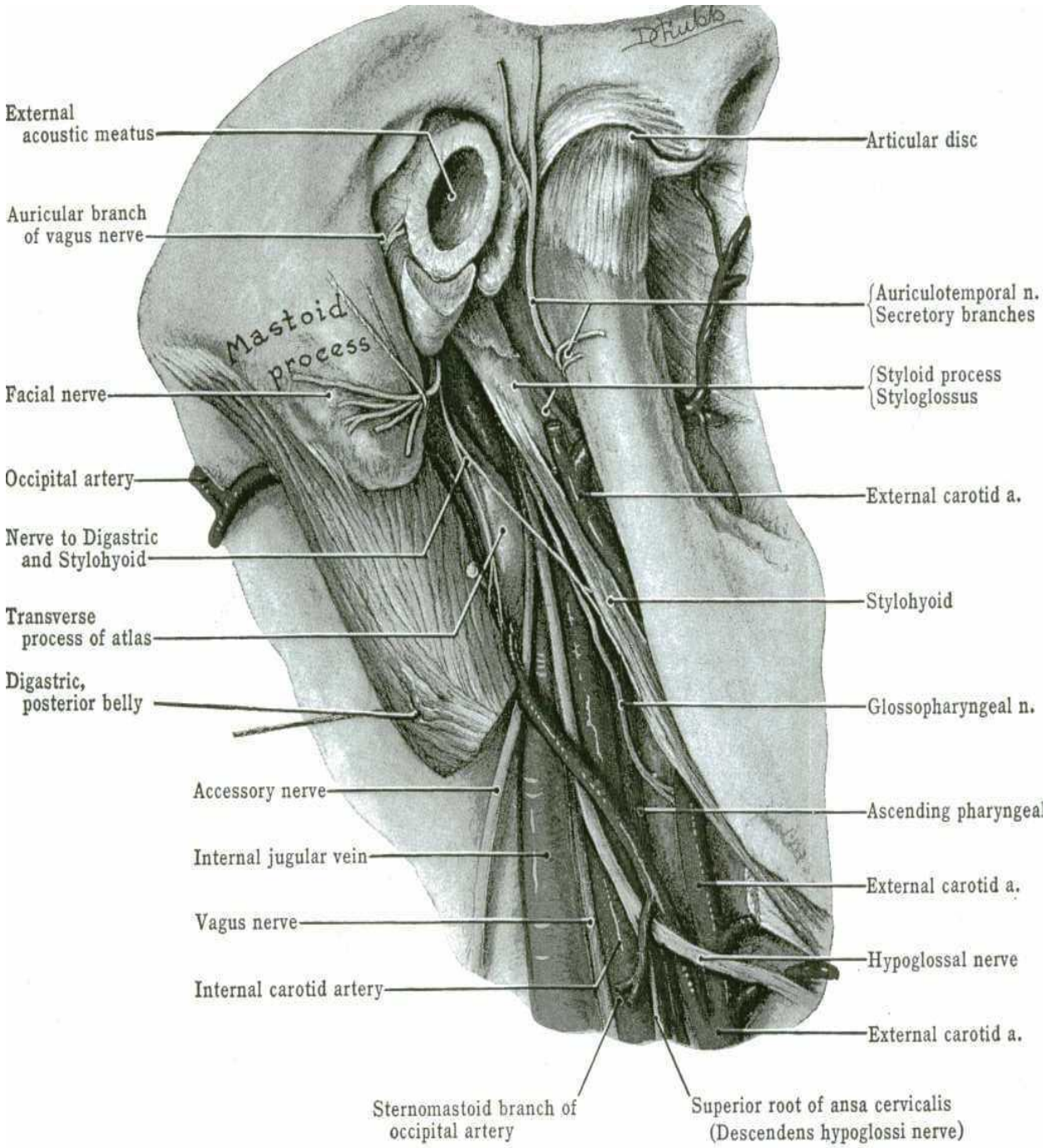

Fig. 7 Auriculotemporal Nerve: Structures deep to the Parotid bed; Courtesy of Grant's Atlas of Anatomy. James E. Anderson, MD: 7th edition. Williams &Wilkins Co. 1978.

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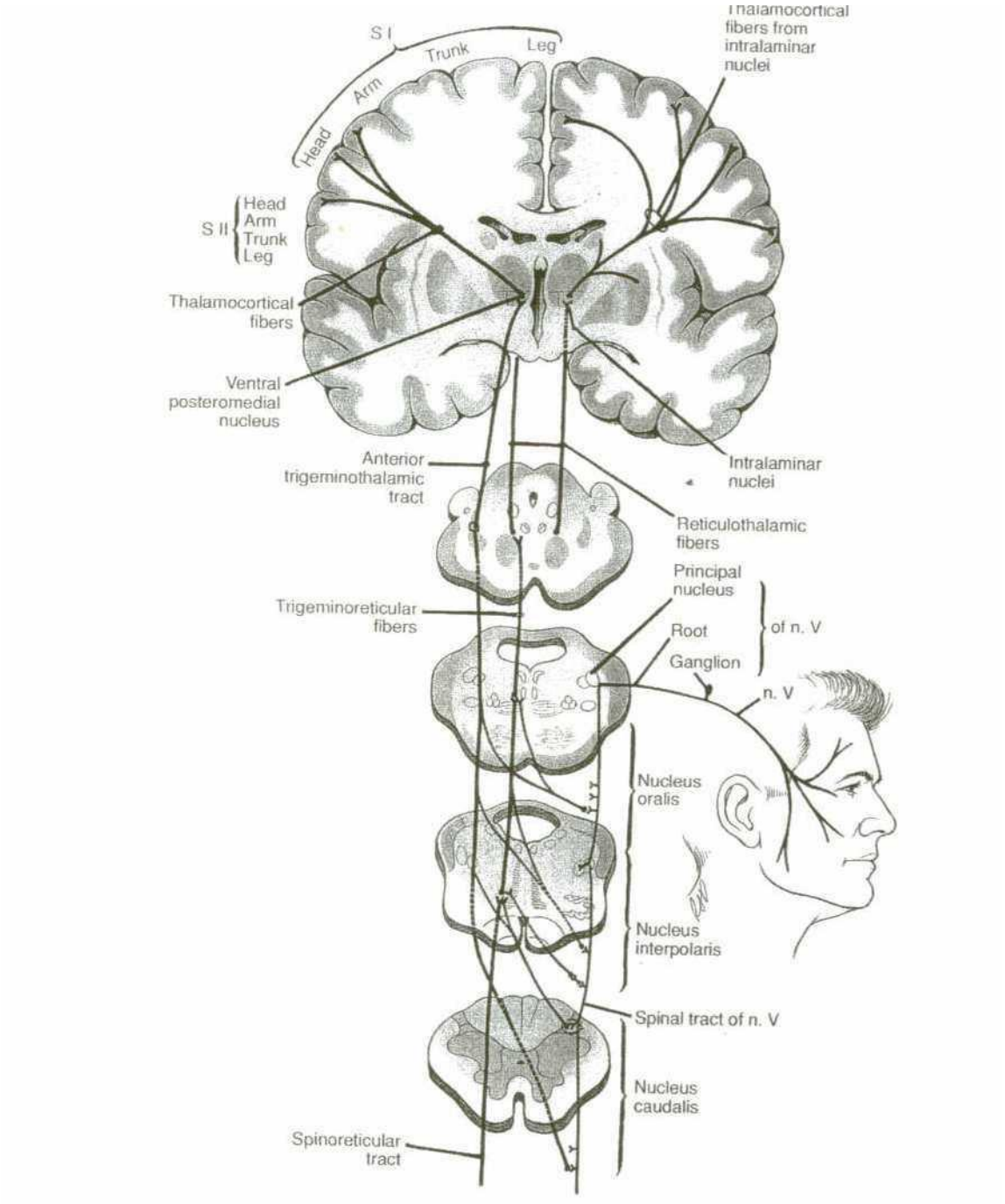

Fig. 8 Stimulation of reticular cells by the trigeminal nerve (trigeminoreticular fibers) run rostally, caudally and reflexively. Figure used with permission from The Human Nervous

System by Charles Noback and Robert Demarest, 2005 ed: Totowa, New Jersey: Humana Press

The superior and medial vestibular nuclei receive input from the superior vestibular nerve and give output to the nuclei of the extraocular muscles. The superior and medial vestibular nuclei along with the tectospinal tracts (originating from the visual center) and medial reticular formation form what is now called the medial longitudinal fasciculus (MLF) which terminates on the Abucent, the Trochlear, and the Occulomotor nuclei (fig 6). These nerve fibers are located in the brainstem and extend through the spinal cord and allow adjustments of the extraocular eye muscles to compensate for different head positions (fig.4).

The vestibular system complex in addition to the receiving input from the vestibular nuclei, receives input from the visual system. Images which are received through the optic nerve enter the visual association area (areas 18 & 19) and terminate in the superior colliculus. The tectospinal neurons (fig. 6) originate from the superior colliculus and descend in the spinal cord and the paramedian pontine reticular formation (PPRF). These neurons influence the extraocular muscles through interneurons and help coordinate neck and back musculature.

The paramedian pontine reticular formation (PPRF) constitutes a portion of what is called the vestibuloocular pathway. These neurons couple and coordinate the movement of the eyes. The PPRF receives input from the vestibular nuclei, cerebral cortex, cerebellum, superior colliculus, and the spinal cord. The output is to the medial longitudinal fasciculus (MLF) and the reticular formation and terminates in the motor nuclei of cranial nerves 3, 4, and 6, which innervate the extraocular muscles. Neurons

from the left vestibular nuclei send axons ipsilaterally through the MLF to excite the neurons of the left occulomotor nucleus and at the same time send axons contralaterally through the MLF to inhibit the neurons of the right Abducens nucleus. In coordination of this pathway may give the patient the perception of dizziness and/or vertigo.

Disturbances of postural reflexes are therefore due in part to the effects on the motoneurons distributed by the vestibulospinal and the reticulospinal tracts. The inhibitory region of the reticular formation is of significant importance. When this region of the reticular formation loses its excitatory input from the cerebral cortex, the result is reduced inhibition of the descending excitatory connections to the spinal cord, thus producing increased motoneuron firing. This increased firing then results as a change of muscle tension in postural muscles usually manifested as a disturbance of the equilibrium.

The Neurological Aspects of Dentistry

The reticular formation receives many direct and indirect projections for the afferent systems of the cutaneous, vestibular, visual, proprioceptive, trigeminal, auditory and autonomic neurons as well as spinal ascending projections. It receives inputs from most subdivisions of the central nervous system (CNS) and then projects to other regions of the body via major interneuronal nerves. Together with the limbic system, it controls the neural mechanism of homeostasis over endocrine and autonomic functions. Muscle reflex responses are also coordinated through the reticular formations craniofacial stimuli from the cranial nerve nuclei.

The midline raphe nuclei are regarded as a major portion of the brainstem's reticular formation. The reticular formation has been divided into multiple subsets and portions of these subsets (medial reticular nuclei, reticularus gigantocellularis, reticularus pontis, reticularus caudalis and others) have always been regarded as playing a role in pain–producing stimuli. Efferent functions of the reticular formation produce postural reflexes and righting reactions and play a critical role in phasic movement and in maintenance of muscle tone. The stimulation of the medial reticular formation brings about postural responses within the neck and labyrinthine reflexes.[13]

Trigeminal afferent neurons, particularly from the spinal tract of V (fig. 8), project directly to the nucleus raphe of the reticular formation and exhibit selective control of the responses (reflex arcs) to their input.[14] Stimulation of the nucleus raphe of the reticular formation can cause a rhythmic tremor of the face, neck and fingers and these movement can include rhythmic movement of the eyelids, alternating protrusion and retraction of the tongue, rhythmic wiggling of the ears and brow, and rhythmic contractions of the platysma muscle.[15] At more caudal levels, <u>all</u> parts of the body may result in tremors and alternation of the flexor and extensor muscles surrounding any joint. Stimulation of the trigeminal afferent projections into the reticular formation would also cause these rhythmic movements to occur. Akinesis or the inability to initiate a voluntary act which is attributed to patients with Parkinsonism may probably be due in part to some disorder or dysfunction within the reticular formation.[16] Reticulospinal neurons are excited by the stimulation of the brain motor cortex and are also stimulated by the nucleus raphe spinal neurons through its reflexive arcs. Without this reticulospinal activity, voluntary activity occurring over the corticospinal tract is unable to initiate

motor movement[17]. If there is excessive abnormal physiological neuronal stimulus, arising from the trigeminal nerve specifically, this would cause interference with the normal conduction of impulses from the cerebral cortex, and it would therefore give rise to enough nerve stimuli to initiate the involuntary movements that cause imbalance disorders.[18]

How would the trigeminal nerve project this excessive stimulus? It occurs through the third division of the trigeminal nerve, the mandibular division, more specifically the auriculotemporal nerve (fig. 7). Examining the anatomical structures presented by the elderly population we discover that many have either had their posterior teeth extracted and/or through normal wearing and grinding of tooth structure lost the youthful vertical dimension (maxillomandibular vertical interrelationship) that once existed. This places the mandibular condyle into a more superior position within the glenoid fossa. This displaces the condylar disc superiorly or posterosuperiorly and causes excess stimuli upon the retrodiscal tissues with its complex of blood vessels and nerves, particularly the auriculotemporal nerve fibers. Sensitization of the afferent input into the auriculotemporal nerve occurs. Impulses travel along the mandibular division of the trigeminal nerve and enter the spinal nucleus of V. They stimulate the reticular formation (nucleus raphe and the medial reticular nuclei) which begins to initiate an inhibitory effect on the reticular formation, thus decreasing voluntary control of the cortex. Involuntary rhythmic tremors and imbalance of posture and gait disturbances begin with the same involuntary acts that occur (in certain cases) through the interneuronal connections of the reticulospinal and the nucleus raphe tracts. Stimulation of the temporomandibular joint tissues is known to evoke a reflexive response in the

tongue and other craniofacial muscles.[19] It can also stimulate postural reflexes initiated through projections to the brainstem's reticular formation. There have been multiple studies concerning the maxillary-mandibular relationship and its positive or negative influence on a patient's posture when the proper vertical dimension is altered.[20 21 22 23] The sensory trigeminal nuclei are of such great magnitude that they mediate the impulses of all the other cranial nerves. Afferent nerves from all parts of the spinal cord have been shown by researchers Glees (1946)[24], Rossi and Brodal (1956)[25], to terminate in the spinal and principal trigeminal nuclei and in the nucleus of the solitary tract. Also the corticoreticular spinal fibers terminate in the same nuclei. In addition to the proprioceptive and exteroceptive impulses entering via direct spinoreticular fibers, the trigeminal fibers influx is of particular importance in maintaining the tonic activity or major influence of the reticular formation (RF)[26]. The RF is an area that spans all levels of the brainstem and is responsible for integrating information from the brain and periphery and linking sensory, motor and autonomic nuclei of the brainstem. The trigeminal nerve, with its influence upon the reticular formation therefore mediates the complex reflexes and functions such as eye movements, posture, feeding, breathing, homeostasis, arousal, sleep, control of vasomotor tone, cardiac output and pain.[27]

The brainstem circuits of the reticular formation also control the reflex changes in blood pressure, heart rate, breathing, and salivation. These autonomic functions along with muscle reflexes and noxious stimuli from sensorimotor control give rise to a patients overall general health as well as gait and balance disorders. In the reticular formation of the pons and the medulla there are two groups of nuclei involved in the control of posture. The nuclei within the pons facilitate spinal reflexes. The nuclei within the

medulla inhibit spinal reflexes. These nuclei project through the medial and lateral reticulospinal tracts of the medial reticular formation to all levels of the spinal cord.[28] [29] The pontine reticular formation projects caudally in the spinal cord through the reticulospinal tract and terminates on and facilitates motor neurons that innervate axial muscles and extensors of the limbs. The medullary reticular formation gives rise to the lateral reticulospinal tract that projects bilaterally and caudally in the front of the lateral columns. This tract produces inhibition of neck and back motor neurons, similar to the medial vestibulospinal fasciculus.[30] These fibers, more importantly, makes polysynaptic inhibitory connections with extensor motor neurons and excitatory connections with flexor motor neurons. This tract can also excite motor neurons innervating extensor muscles and inhibiting flexors. Posture could not be maintained without proper coordination of the neck and leg muscles. (ex: the ilio-psoas muscles of the leg must synchronize its activities with the sternocleidomastoid muscle of the neck to control balance.)[31] Both medial and lateral reticulospinal fibers also modulate reflex action during ongoing movements and produce different effects, depending on the movement in progress at the time.[32] These pathways synchronize ipsilateral functions of the muscles, postural reflexes, activation and relaxation of structures, and coordinate the response to both internal and external stimuli. These RF fibers maintain posture and movement by integrating vestibular, oculomotor and other sensory inputs such as those that come from the trigeminal nerve (spinal nucleus of V) and from the cerebral cortex.[33]

Conclusion

The significance of these findings in this study makes it clear that there is an interrelationship between physical dysfunction of the temporomandibular joint and balance and gait disorders which can be corrected by establishing the proper maxillomandibular relationship with the NCVD appliance. The drastic reduction of signs and symptoms associated with imbalances and gait disorders confirms the conclusion by the authors for the association of the anatomical and physiological pathways of the trigeminal nerve and gait/balance disorders. This data also demonstrates that the trigeminal nerve is complex and that physicians and physical therapists need to coordinate with the dental profession to help maximize treatment for a patient's physical and psychological condition.

Understanding the neuroanatomy of the trigeminal nerve (CN V) shows us how integral the trigeminal nerve is in its effects upon an individual's balance and gait. It is through the reflexes of the motor pathways in conjunction with the trigeminal nerve and its connections with the reticular formation and that of the vestibular system that we are able to observe and prevent elderly patients from falling who have a disturbed gait or imbalance. Providers of this service are easily able to help many of their patients regain their ability walk steadily and improve their balance especially as the patient population increases with age.

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[1] Kendall FP, McCleary EK: Muscles: Testing & Function. #rd ed. Williams and Wilkins. Baltimore, !983

[2] Clinical study of location and reproducibility of three mandibular positions in relation to body posture and muscle function.Tripodakis AP, Smulow JB, Mehta NR, Clark RE.J Prosthet Dent. 1995 Feb;73(2):190-8.

[3] Root GR, Kraus SL, Razook SJ, et. Al: Effect of an intraoral appliance on head and neck posture. J Prosthet Dent 58:421, 1984
[4] Hausdorff JM, Rios DA, Edelber HK. Gait variability and fall risk in community-living older adults: a 1-year prospective study. Archives of Physical Medicine and Rehabilitation 2001;82(8):1050–6.
[5] Sterling DA, O'Connor JA, Bonadies J. Geriatric falls: injury severity is high and disproportionate to mechanism. Journal of Trauma-Injury, Infection and Critical Care 2001;50(1):116–9.
[6] Vellas BJ, Wayne SJ, Romero LJ, Baumgartner RN, Garry PJ. Fear of falling and restriction of mobility in elderly fallers. Age and Ageing 1997;26:189–193.
[7] Stevens JA, Sogolow ED. Gender differences for non-fatal unintentional fall related injuries among older adults. Injury Prevention 2005;11:115–9
[8] Stevens JA, Corso PS, Finkelstein EA, Miller TR. The costs of fatal and nonfatal falls among older adults. Injury Prevention 2006;12:290–5.
[9] Desmond. *Advance for Audiologists*. July/August 2000.
[10] Imbaud Genieys S. Ann Otolaryngol Chir Cervicofac. 2007 Sep;124(4):189-96. Epub 2007 Aug 29. French
[11] Moylan KC, Binder EF. Falls in older adults: risk assessment, management and prevention Am J Med. 2007 Jun;120(6):493.e1-6.
[12] Schmahmann JD. Disorders of the cerebellum: ataxia, dysmetria of thought, and the cerebellar cognitive affective syndrome. J Neuropsychiatry Clin Neurosci. 2004 Summer;16(3):367-78. Review.
[13] Brodal, Per *The Central Nervous System*. 3rd ed. Oxford University Press,2004
[14] Torvik A.: Afferent connections to the sensory trigeminal nuclei, the nucleus of the solitary tract and adjacent structures. An experimental study in the rat. J Comp Neurol. 106:51-142
[15] Ibid
[16] L T Robertson and J P Hammerstad: Jaw movement dysfunction related to Parkinson's disease and partially modified by levodopa**.** J Neurol Neurosurg Psychiatry. 1996 January; 60(1): 41–50.
[17] Torvik A.: Afferent connections to the sensory trigeminal nuclei, the nucleus of the solitary tract and adjacent structures. An experimental study in the rat. J Comp Neurol. 106:51-142
[18] Buisseret-Delmas C, Compoint C, Delfini C, Buisseret P. Organisation of reciprocal connections between trigeminal and vestibular nuclei in the rat. J Comp Neurol. 1999;409:153-68.
[19] Pinganaud G, Bourcier F, Buisseret-Delmas C, Buisseret P. Primary trigeminal afferents to the vestibular nuclei in the rat: existence of a collateral projection to the vestibulo-cerebellum. Neurosci Lett. 1999;264:133-6.
[20] Darling DW, Kraus S, Glasheen-Wray MB: Relationship of Head Posture and the rest position of the mandible. J Prosthet Dent. 1984; 2:111-115
[21] Bracco P. Deregibus A. Piscetta: Effects of different jaw relations on postural stability in human subjects. Neurosci Lett 2004: 356:228-230
[22] Milani RS. De Periere DD. Lapeyre L. Pourreyron L.: Relationship between dental occlusion and posture. J Craniomandib Pract 2000: 18:127-134.

[23] Nicolakis P. Nicolakis M. Piehslinger E. Ebenbichler G. Vachuda M. Kirtley C. et al: Relationship between craniomandibular disorders and poor posture. J Craniomandib Pract 2000: 18:106-112

[24] Glees P, Meyer A, Meyer M. Terminal degeneration in the frontal cortex of the rabbit following the interruption of afferent fibres. J Anat. 1946 Apr;80(Pt 2):101-106.3.

[25] ROSSI GF, BRODAL A. Corticofugal fibres to the brain-stem reticular formation; an experimental study in the cat. J Anat. 1956 Jan;90(1):42-62

[26] Brodal P.; The Reticular Formation of the Brainstem; Anatomical Aspects and Functional Correlations: William Ramsay Henderson Trust/Oliver & Boyd (1957)

[27] Dauvergne C, Ndiaye A, Buisseret-Delmas C, Buisseret P, VanderWerf F, Pinganaud G. Projections from the superior colliculus to the trigeminal system and facial nucleus in the rat. J Comp Neurol. 2004;478:233-47.

[28] Buisseret-Delmas C, Buisseret P. Central projections of extraocular muscle afferents in cat. Neurosci Lett. 1990;109:48-53.

[29] Marfurt CF, Rajchert DM. Trigeminal primary afferent projections to "non-trigeminal" areas of the rat central nervous system. J Comp Neurol. 1991;303:489-511.

[30] Keizer K, Kuypers HG. Distribution of corticospinal neurons with collaterals to lower brain stem reticular formation in cat. Exp Brain Res. 1984;54(1):107-20

[31] A Cunha, T Burke, F França, A Marques: Effect of global posture reeducation and of static stretching on pain, range of motion, and quality of life in women with chronic neck pain: a randomized clinical trial. Clinics vol.63 no.6 São Paulo 2008

[32] I. Engberg, A. Lundberg, R. W. Ryall: Reticulospinal inhibition of transmission in reflex pathways. J Physiol. 1968 January; 194(1): 201–223.

[33] L. Petrosini, D. Troiani, B. Zannoni: Trigeminal stimulation modulates vestibular unitary activity. Cellular and Molecular Life Sciences, Volume 38, Number 3 / March, 1982; 363-365