GAIT DISORDERS

Rodger J. Elble, MD, PhD
Department of Neurology
Southern Illinois University School of Medicine
P.O. Box 19643
Springfield, IL 62794-9643
relble@siumed.edu

Anatomy and physiology of locomotion

Successful locomotion requires the integrated control of posture and movement. Postural control is necessary for static stability during stance and dynamic stability during locomotion. Starting, stopping and turning are particularly difficult because postural shifts and limb movements must transition the body from one static or dynamic steady state (e.g., standing) to another (e.g., walking).

Cats with high cervical cord transections exhibit rudimentary locomotor activity when they are suspended in a sling with paws touching a moving treadmill.(1) Such studies in laboratory animals and humans(2) revealed that the basic locomotor rhythm emerges from spinal neuronal networks.(3) This spinal pattern generator of locomotion is integrated with complex motor pathways that include virtually all supraspinal motor centers (Figure 1).

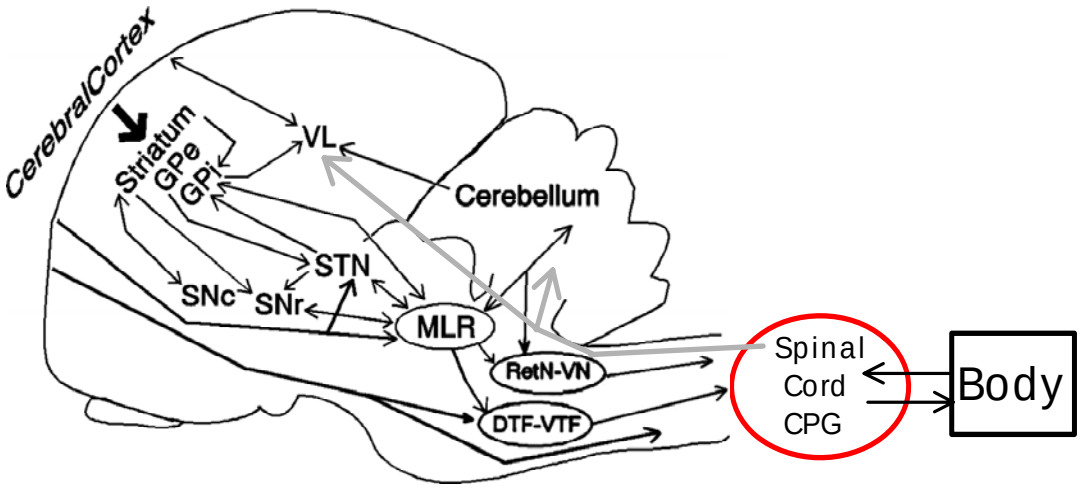

Figure 1: Schematic diagram of the principal neural pathways of locomotor control. GPe and GPi = globus pallidus externa and interna; VL = ventrolateral thalamus, SNc and SNr = substantia nigra pars compacta and reticulata; STN = subthalamus; MLR = midbrain locomotor region; VN = lateral vestibular nucleus; RetN = pontomedullary reticular nuclei; CPG = spinal locomotor network (central pattern generator); DTF-VTF = dorsal and ventral tegmental fields.

The reticulospinal and vestibulospinal pathways in the ventrolateral spinal quadrant are important for the activation of spinal locomotor networks and for the recovery of locomotion after spinal cord injury.(4-6) These bulbospinal pathways are modulated by the cerebellar vermis and fastigial nuclei so that postural tonus is maintained in harmony with the support and swing phases of the gait cycle.(7) The lateral vermal and paravermal cerebellar cortex and the interposed (globose and emboliform) nuclei influence locomotor rhythmicity and phasic coordination of body segments through connections with rubrospinal, lateral pontomedullary reticulospinal, and vestibulospinal pathways.(7-9) The dorsal spinocerebellar pathway carries sensory feedback from the periphery to the cerebellum, and the ventral spinocerebellar pathway carries peripheral sensory feedback and output from the spinal pattern generator (a.k.a. spinal efferent copy).(7) The cerebellum also receives a cerebral efferent copy via corticobulbocerebellar pathways, involving the pontine, reticular and olivary nuclei. With these sensory and cortical inputs, the cerebellum plays a critical role in the feedback and feedforward (anticipatory) control of locomotion.(7)

The midbrain locomotor region (MLR; pedunculopontine nucleus) of the dorsolateral mesopontine tegmentum contains cholinergic and glutaminergic neurons that have complex reciprocal and bilateral connections with the basal ganglia, cerebellum and other brainstem nuclei.(10, 11) The MLR and its connections play a critical role in the initiation of gait and control of posture. Brief electrical stimulation of the MLR in cats induces rapid walking, followed by running, and excitatory glutaminergic inputs to MLR from the subthalamus and motor cortex promote locomotion.(12-14) GABAergic inputs to the MLR from the substantia nigra pars reticulata (SNr) and the globus pallidus interna (GPi) inhibit locomotion.(14) Lesions in the MLR produce akinesia(15), and the increased SNr and GPi activity in Parkinson disease probably causes akinesia and freezing through abnormal GABAergic inhibition of the MLR.(16)

The MLR connects with the raphe nuclei of the caudal midline pons, which correspond to the so-called ventral and dorsal tegmental fields (VTF and DTF). Stimulation of the VTF increases antigravity muscle tone, and stimulation of the DTF decreases antigravity muscle tone. The DTF and VTF control postural tonus during locomotion in conjunction with neighboring reticulospinal and vestibulospinal pathways.(17)

The corticospinal and corticobulbar pathways are needed for flexible, adaptive locomotor control.(8) This “highest level control” is accomplished through rich cortical connections with the basal ganglia, thalamus and cerebellum. The frontal cortex participates in the purposeful modification and initiation of locomotion through connections with the MLR, basal ganglia and spinal networks.(8, 18) Locomotion requiring precise foot placement (e.g., walking on a grid or balance beam) is impossible without the frontal lobes. The lateral cerebellar hemispheres evolved in concert with the frontal lobes, so it is not surprising that this part of the cerebellum also participates in precision limb placement during novel situations requiring visual guidance of locomotion.(7)

Features of normal walking

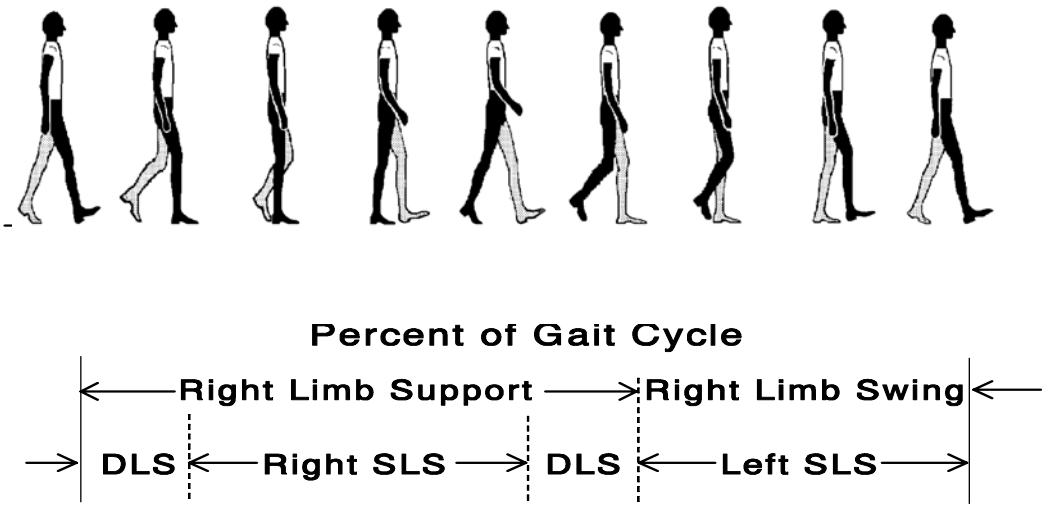

Figure 2: Phases of the normal gait cycle, expressed as percentage of total stride. DLS = double limb support and SLS = single limb support.

The gait cycle is defined as the time between successive heel-floor contacts with the same foot (Figure 2). One gait cycle consists of two steps (one stride). From right heel-floor contact to left toe-off is a period of double-limb support, which lasts approximately 10% of the total gait cycle. This phase of the cycle is followed by the left swing phase, which is simultaneous with and equal to the right single-limb support phase. The time from left heel-floor contact to right toe-off comprises the second of two double-limb support phases in a gait cycle and is followed by the right swing phase and left single-limb support phase. Stride length (length of two successive steps) and cadence (steps per minute) determine the velocity of walking (= stride length x cadence ÷ 2). The magnitudes of arm swing, toe-floor clearance, and hip and knee rotations are proportional to stride length and gait velocity, whereas the percentage time in double-limb support increases with reductions in gait velocity.(19, 20)

The total-body center of mass oscillates vertically at a frequency equal to the cadence and horizontally at one-half the cadence. During a gait cycle, the two maxima in vertical oscillation occur in the middle of right and left single-limb support, and the two minima occur in the middle of the two phases of double-limb support. The left- and right-most horizontal excursions of the center of gravity occur at the times of mid left and right single-limb support. These vertical and horizontal excursions of the center of mass are optimized in such a way that the center of mass (body) moves forward with the least amount of expended energy. Consequently, most gait disturbances increase expended energy.(21)

An assessment of ongoing (steady-state) walking is not an adequate gait exam. A complete gait exam (Table 1) includes an assessment of static posture, postural control (e.g., pull test), balance (tandem gait), steady-state walking, and transitional movements (starting, stopping, sitting, turning and rising from a chair).

Table 1: Examination of locomotion

Static Posture: Curvature of spine Head position Pelvic tilt Flexion of knees and hips Stance base	**Postural Control and Balance:** Romberg test Pull test Bending over Reaching while standing Tandem walking
Walking: Coordination and amplitude of upper and lower limb movements Step length, width, rhythmicity, and symmetry; gait velocity Foot-floor clearance and contact Walking path Walking on heels and toes	**Transitional movements:** Sitting and rising from a chair Starting, stopping and turning

A systems classification of gait disorders

Nutt, Marsden, and Thompson introduced a systems approach to the classification of gait disorders in which disturbances of gait are classified as emerging from the highest, middle or lowest levels of motor control.(22) This classification scheme borrows heavily from the classic hierarchical view of motor control. This view of motor control is admittedly overly "vertical", but I find it clinically useful because it encourages me to consider the entire neuraxis and neuromuscular system when deciphering a patient's gait disturbance.

Lowest level gait disorders are caused by diseases of the muscles, peripheral nerves, skeleton, peripheral vestibular system, and anterior visual pathway. When analyzing any gait disturbance, the clinician must remain mindful of the common contribution of secondary muscle deconditioning (type II atrophy), limb contractures, spinal ankylosis and reduced pelvic mobility. These "non-neurologic" conditions are common in older people and in people with many neurologic disorders. Examples of lowest level gait disorders are antalgic gait, arthritic gait, myopathic (waddling) gait, and steppage gait.

Middle level gait disorders are caused by ascending or descending sensorimotor tract lesions, cerebellar pathway damage (ataxia), bradykinesia, hyperkinesias (e.g., chorea, tremor), and dystonia. Examples are hemiparetic gait, spastic (paraplegic) gait, choreic gait, dystonic gait, spinal ataxia and cerebellar ataxia.

Highest-level gait disorders are caused by pathology in the corticobasalganglionic-thalamocortical loop and its connections with the midbrain locomotor region. Highest-level gait disturbances are common in all forms of parkinsonism and dementia. Cortical-basal ganglia-thalamocortical circuits play an important role in selecting desired postures, movements and behaviors, while suppressing undesired or competing postures, movements and behaviors.(23) Damage to these circuits impedes the adaptation of gait to varying environmental and emotional circumstances. Highest-level gait disturbances are particularly severe when both sides of the brain are affected. The characteristics of gait become increasingly bizarre and maladaptive as the underlying disease progresses. The gait disturbance is often most evident in complex, unfamiliar environments and during

transitional movements from one steady-state posture or movement to another (e.g., starting, stopping, standing up, sitting down and turning). Examination of the patient while seated or recumbent usually provides little clue to the characteristics and severity of impaired walking.

Highest-level gait disorders have one or more of the following characteristics:

1. Absent or inappropriate corrective (rescue) reactions to postural perturbation (pull test). Patients "fall like a log" or make surprisingly little attempt to rescue themselves. Rescue reactions often consist of inappropriate limb movement or postural synergies (e.g., leaning or pushing the wrong direction).
2. Inappropriate or bizarre foot placement, postural synergies, and interaction with the environment. Patients may cross their lower limbs inappropriately while walking or turning, lean toward the pivot foot when turning, or lean backward when attempting to rise from a chair or bed.
3. Markedly variable performance, influenced greatly by the environment and emotion. This variability may baffle caregivers (and physicians!) who are unaware of this phenomenon.
4. Start hesitation and freezing, often when seemingly insignificant environmental objects or thresholds are encountered (e.g., a doorway)

Patients with cortical-basal ganglia-thalamocortical pathology can exhibit relatively pure dysequilibrium (subcortical dysequilibrium, frontal dysequilibrium) or freezing (freezing gait), but most patients initially or eventually exhibit signs of both (frontal gait disorder).

Lowest- and middle-level gait disorders differ from highest-level gait disorders in that they exhibit little or no change with alterations in environment or emotion. The clinical characteristics of lowest- and middle-level gait disorders are usually predictable from the neurologic or musculoskeletal deficits revealed by an exam while the patient is seated or recumbent, and these characteristics do not change significantly during transitional movements from one steady state posture or movement to another. Changes in gait are not inappropriate or maladaptive, even though they are predictably limited by the underlying neurologic or musculoskeletal deficit.

Table 2: Clinical terminology of gait disturbances

Gait abnormality or sign	Related terms	Sites of pathology
Cautious gait		Any part of the central or peripheral nervous system involved in locomotion and higher cortical function
Dysequilibrium	Frontal dysequilibrium Thalamic astasia Subcortical dysequilibrium Cerebellar ataxia Sensory dysequilibrium Pusher syndrome	Frontal lobes Ventrolateral thalamus Vestibular nuclei Midline cerebellum and fastigial nucleus Peripheral visual, vestibular and somatosensory systems Spinal cord
Start hesitation and freezing	Gait ignition failure Magnetic gait Freezing gait Akinetic gait Slipping clutch syndrome	Frontal lobes Basal ganglia Dorsolateral midbrain locomotor region
Short, shuffling steps and en bloc turning, with or without festination	Parkinsonian gait Lower-half parkinsonism Frontal gait disorder Marche á petits pas Gait apraxia Magnetic gait	Basal ganglia Frontal lobes
Choreic gait	Hyperkinetic gait	Basal ganglia
Dystonic gait		Basal ganglia
Hemiparetic gait	Hemiplegic gait Circumducting gait	Supraspinal pyramidal tract lesion
Spastic gait	Scissoring gait Paraplegic gait	Thoracic or cervical spinal cord damage
Ataxic gait	Sensory ataxia	Peripheral nerves

Gait abnormality or sign	Related terms	Sites of pathology
	Spinal ataxia Cerebellar ataxia Reeling, lurching gait	Dorsal spinal columns and spinocerebellar pathways Cerebellum Brainstem motor nuclei Spinal cord
Myopathic gait	Lordotic-waddling gait Waddling gait	Hip girdle weakness
Steppage gait	Bilateral foot drop	Bilateral weakness of muscles innervated by the peroneal nerves
Ataxic gait	Sensory ataxia Spinal ataxia Cerebellar ataxia Reeling, lurching gait	Peripheral nerves Dorsal spinal columns and spinocerebellar pathways Cerebellum Brainstem motor nuclei Spinal cord
Antalgic gait	Hyperesthetic gait Limping gait Arthritic gait	Foot, spine, pelvis or lower extremity
Hysterical gait	Psychiatric astasia-abasia	

A big advantage of this gait classification scheme is that it forces clinicians to analyze gait in terms of the anatomy and physiology of locomotion. The chore of memorizing the large, redundant and variably-defined terminology of gait disorders (Table 2) is largely avoided. However, a few terms or concepts are worth emphasizing because they are used broadly and commonly by clinicians.

Cautious gait is a compensatory pattern of walking in which patients walk in a slow, guarded or restrained manner, similar to the way any of us would walk when negotiating a slippery surface or threatening environment. Patients exhibit slightly stooped posture, reduced arm swing, increased time with both feet on the floor (double-limb support), loss of the normal heel-toe sequence of foot-floor contact during stance phase, slightly widened base, and reduced hip and knee rotations, all of which are commensurate with the patient's reduced stride and gait velocity. This pattern of walking is a nonspecific compensatory response that is produced by most causes of impaired locomotion. The features of cautious gait frequently dominate the gait pattern of patients with mild neurological impairment, obscuring the more diagnostic signs of underlying pathology.

Dysequilibrium is a disturbance of postural control and balance. Severe dysequilibrium produces a staggering, wide-based gait, particularly when the disturbance is acute. Mild or chronic dysequilibrium is often associated with a predominantly slow, cautious gait. Dysequilibrium can result from damage at any level of the neuraxis. Consequently, there is sensory dysequilibrium (loss or conflict among vestibular, somatosensory and visual feedback), caused by disturbances of primary sensory pathways and central dysequilibrium due to lesions in the central nervous system. Dr. Kattah has already discussed the main types of sensory dysequilibrium, emerging from somatosensory, vestibular and visual impairment.

Central dysequilibrium is caused by damage to the corticobasal ganglionic thalamocortical loop (frontal and subcortical dysequilibrium) or vestibulocerebellar pathways. Following strokes, there is usually substantial improvement unless the damage is bilateral. Frontal lobe damage can cause profound dysequilibrium due to inappropriate postural synergies. Patients lean the wrong direction, in such a way that stability is impeded. For example, patients may lean backward when being helped from a chair, or they may lean away from the pivot foot when attempting to turn. Bilateral pathology is most disabling. Unilateral lesions (e.g., strokes) in the corticobasalganglionic-thalamocortical loop usually cause patients to fall backward and away from the lesion. However, patients with spatial neglect lean toward the lesion. Strokes (usually hemorrhages) in the posterior thalamus may produce the unusual "pusher syndrome" in which patients have a sense of falling toward the ipsilesional side and use the unaffected limbs to their body toward the paralyzed side.(24, 25) These patients have a disturbed perception of body orientation with respect to gravity, with the illusion that the body is tilted toward the lesion. Progressive supranuclear palsy produces bilateral destruction in the corticobasalganglionic-thalamocortical loop, MLR and cerebellar pathways, so early impairment of balance is common in this disorder, often with features of a highest level disorder, middle level disorder or both. Vestibulocerebellar dysequilibrium occurs with damage to the vestibulocerebellum and its brainstem connections. Patients veer or fall toward the

side of the cerebellar lesion. Damage to the vestibular nucleus can produce a sensation that the environment is tilted and that the body is being pulled toward the side of the lesion.

Patients with **ataxic gait** exhibit steps and postural synergies that are disturbed in amplitude and in rhythm. The erratic pattern of walking is classic in patients with damage to the cerebellum or to those areas of the brainstem, ventrolateral thalamus and frontal lobes that interact with the cerebellum. Gait is wide-based, erratic and reeling. Upper and lower limb movements are uncoordinated, and there is considerable stride-to-stride variability. Abnormal postural sway during quiet stance is present with eyes open and closed. Damage to the midline cerebellum or fastigial nucleus produces a predominantly truncal disturbance, as in the rostral vermis degeneration of chronic alcoholism. Unilateral hemispheric lesions produce ipsilateral ataxia and falling toward the lesion. Lesions in the ventrolateral thalamus can produce ataxia of the contralateral extremities (thalamic ataxia) and a tendency to fall away from the lesion or backward (subcortical dysequilibrium). Somatosensory ataxia is exhibited by patients with large-fiber sensory polyneuropathies and consists of wide-based, deliberate steps and postural dysequilibrium that are much worse when vision is limited (e.g., as in darkness). Patients with damaged dorsal columns of the spinal cord exhibit the same pattern of walking. Lesions in the anterior columns produce an uncoordinated, dysrhythmic gait due to impairment of the vestibulospinal and reticulospinal tracts. Furthermore, the ventral spinocerebellar tract is positioned in the anterolateral surface of the cord and provides feedback from the spinal locomotor network to the cerebellum. Thus, ataxia and dysequilibrium commonly coexist with spasticity in patients with spinal cord pathology and may dominate the clinical picture in some patients.

Akinetic or freezing gait. Start hesitation and freezing are signs of akinesia. Akinesia is typically associated with festination and decrement of repetitive movements.(26) Patients move their lower extremities relatively normally while seated or recumbent, but their feet appear to stick to the floor while walking. Gait is often initiated with a few delayed, aborted, shuffling steps or is tricked into action by stepping over a self-imposed obstacle (e.g., the handle of an inverted cane) or targeted spot on the floor. These tricks work best in patients with akinesia due to basal ganglia disease (e.g., Parkinson disease and progressive supranuclear palsy). Environmental distractions and obstacles exacerbate start hesitation and elicit abrupt cessation of movement, called freezing (e.g., at a doorway).

References

1. Brown TG. On the nature of the fundamental activity of the nervous centres, together with an analysis of the conditioning of rhythmic activity in progression, and a theory of evolution of function in the nervous system. J Physiol (London) 1914;48:18-46.
2. Kalb RG. Getting the spinal cord to think for itself. Arch Neurol 2003;60(6):805-808.
3. Dietz V. Spinal cord pattern generators for locomotion. Clin Neurophysiol 2003;114(8):1379-1389.
4. Holmes G. Spinal injuries of warfare. Br. Med. J. 1915;2:815-821.
5. Eidelberg E, Walden JG, Nguyen LH. Locomotor control in macaque monkeys. Brain 1981;104(Pt 4):647-663.
6. Eidelberg E, Story JL, Walden JG, Meyer BL. Anatomical correlates of return of locomotor function after partial spinal cord lesions in cats. Exp Brain Res 1981;42(1):81-88.
7. Morton SM, Bastian AJ. Cerebellar control of balance and locomotion. Neuroscientist 2004;10(3):247-259.
8. Armstrong DM. The supraspinal control of mammalian locomotion. J Physiol 1988;405:1-37.
9. Mori S, Matsuyama K, Mori F, Nakajima K. Supraspinal sites that induce locomotion in the vertebrate central nervous system. Adv Neurol 2001;87:25-40.
10. Garcia-Rill E, Homma Y, Skinner RD. Arousal mechanisms related to posture and locomotion: 1. Descending modulation. Prog Brain Res 2004;143:283-290.
11. Skinner RD, Homma Y, Garcia-Rill E. Arousal mechanisms related to posture and locomotion: 2. Ascending modulation. Prog Brain Res 2004;143:291-298.
12. Skinner RD, Kinjo N, Ishikawa Y, Biedermann JA, Garcia-Rill E. Locomotor projections from the pedunculopontine nucleus to the medioventral medulla. Neuroreport 1990;1(3-4):207-210.
13. Skinner RD, Kinjo N, Henderson V, Garcia-Rill E. Locomotor projections from the pedunculopontine nucleus to the spinal cord. Neuroreport 1990;1(3-4):183-186.
14. Garcia-Rill E, Kinjo N, Atsuta Y, Ishikawa Y, Webber M, Skinner RD. Posterior midbrain-induced locomotion. Brain Res Bull 1990;24(3):499-508.
15. Masdeu JC, Alampur U, Cavaliere R, Tavoulareas G. Astasia and gait failure with damage of the pontomesencephalic locomotor region. Ann Neurol 1994;35(5):619-621.
16. Matsumura M. The pedunculopontine tegmental nucleus and experimental parkinsonism A review. J Neurol 2005;252 Suppl 4:iv5-iv12.

17. Kawahara K, Mori S, Tomiyama T, Kanaya T. Discharges of neurons in the midpontine dorsal tegmentum of mesencephalic cat during locomotion. Brain Res 1985;341(2):377-380.
18. Drew T. Motor cortical activity during voluntary gait modifications in the cat. I. Cells related to the forelimbs. J Neurophysiol 1993;70(1):179-199.
19. Elble RJ, Hughes L, Higgins C. The syndrome of senile gait. J Neurol 1992;239(2):71-75.
20. Elble RJ, Thomas SS, Higgins C, Colliver J. Stride-dependent changes in gait of older people. J Neurol 1991;238(1):1-5.
21. Inman VT, Ralston HJ, Todd F. Human Walking. Baltimore: Williams and Wilkins, 1981.
22. Nutt JG, Marsden CD, Thompson PD. Human walking and higher-level gait disorders, particularly in the elderly. Neurology 1993;43(2):268-279.
23. Mink JW. The Basal Ganglia and involuntary movements: impaired inhibition of competing motor patterns. Arch Neurol 2003;60(10):1365-1368.
24. Karnath HO, Johannsen L, Broetz D, Kuker W. Posterior thalamic hemorrhage induces "pusher syndrome". Neurology 2005;64(6):1014-1019.
25. Saj A, Honore J, Coello Y, Rousseaux M. The visual vertical in the pusher syndrome: influence of hemispace and body position. J Neurol 2005;252(8):885-891.
26. Narabayashi H. Clinical analysis of akinesia. J Neural Transm Suppl 1980(16):129-136.