Current Concepts of the Vestibular System Reviewed: 1. The Role of the Vestibulospinal System in Postural Control

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This paper reviews the research findings that support the presence of vestibulospinal reflexes in corrections for head and body instability. Studies of the importance of labyrinthine inputs to the central nervous system organization of eye, head, and body movements demonstrate that the vestibular nuclei are more than a simple relay station for labyrinthine activity. At all levels of the vestibular system beyond the primary vestibular afferents, parallel processing of labyrinthine signals occurs with input from other sensory systems. Thus, output of the vestibular nuclear complex (VNC) is not equivalent to the labyrinthine input. It is the VNC output that influences motor behavior. Various sensory inputs are available to the nervous system to detect and correct postural instability. Most notably, vestibular, visual, and proprioceptive signals contribute significantly to the stabilizing responses in humans. The intent of this paper is to review experimental results rather than to discuss treatment interventions. Wherever possible, conclusions are drawn as to the clinical implications of current research findings.

The vestibular system has been understood to control head and eye movements in space. One of the reasons for this understanding has been the fact that direct stimulation of the labyrinths affects neck and eye muscle reflexes. Although the neuronal organization and very possibly the muscles involved in reflex responses as well as the more complex postural reactions may differ (Keshner & Peterson, 1987), an examination of the reflex responses give some indication of how central nervous system pathways are organized.

Postural Reflexes

Vestibulocollis Reflexes

Vestibular and cervical reflexes stabilize the head and neck by exciting the neck muscles in opposition to head and body movements (Outerbridge & Melvill Jones, 1971). The vestibular-neck reflex, or vestibulocollis reflex (VCR), aligns the head with respect to the gravitational vertical. For example, a patient leaning forward over a walker uses this reflex to lift her head as she ambulates so that her gaze is stabilized and oriented to the vertical position in space. Damage to the labyrinth impairs the VCR and causes an unstable, oscillating head (Schor, 1974). The cervicocollic reflex (CCR) aligns the head with respect to the position of the body. In both decerebrate and alert cats, the CCR was initiated by the cervical proprioceptors; its actions complemented those of the VCR during movement (Peterson, Goldberg, Bilotto, & Fuller, 1985). If the body is stationary and the head moves, the VCR and CCR work jointly to stabilize the head (Peterson et al., 1985). For example, a quadriplegic patient in a wheelchair depends on both the VCR and the CCR to maintain body stability while performing a full range of controlled head movements. If the body and head are equally free to move, then the effect of the two reflexes on the neck and head is cancelled, so that voluntary actions of the limbs can perform without the interference of these reflexes (Roberts, 1973). With loss of labyrinthine input, head stability is recovered by co-contraction of the neck muscles to increase the gain of the CCR and passive muscle stiffness (Allum & Pfaltz, 1985; Baker, Goldberg, Peterson, & Schor, 1982).

Isolated stimulation of the labyrinthine or cervical proprioceptors does not effectively stabilize head movement in humans. Combined semicircular canal and otolith inputs (Anderson, Soechting, & Terzuolo, 1977; Money & Scott, 1962), inertial and viscoelastic properties of the head and neck (Bizzi, Dev, Morasso, & Polit, 1978; Viviani & Berthoz, 1975), and voluntary processes (Guitton, Kearney, Werely, & Peterson, 1986) are believed to be responsible for producing head stabilization across many stimulus frequencies.
For example, in a study of neck muscle responses in alert cats during whole-body rotations, the rotations stimulated only the vestibular labyrinths. Electromyograms (EMGs) revealed a specific direction of rotation that produced a maximal excitation of each muscle (Keshner et al., 1986). During a voluntary head-tracking task in the same animal, the muscles were maximally activated around different axes of motion, which suggests that the motor programs for the voluntary and reflex head movements varied in their spatial properties (Keshner & Peterson, 1987). In humans, head position was measured during rotations of the body while the head was voluntarily stabilized or during a mental arithmetic task so that there was no interference by the voluntary processes. Results indicated that head stabilization was produced by responses with longer latency periods (150 ms) than those of the VCR latency periods (50 to 70 ms) (Guitton et al.).

Vestibular and Limb Reflexes

Convergence of vestibular and somatosensory inputs onto the vestibulospinal and reticulospinal neurons (Brink, Hirai, & Wilson, 1980) occurs at the level of the vestibular nuclear complex (VNC) and at the level of the spinal interneurons (Wilson, Ezure, & Timerick, 1984). Convergence of afferent inputs may occur at the motoneuron level as well (Brink et al.). These combined signals can initiate a series of interspinal reflexes that act to align the body segments (Roberts, 1973; Wilson et al., 1984). The specific influence of the labyrinthine signals on head and limb movements has been studied in the cat and the monkey. With the head position fixed relative to the body and the canals stimulated with either angular rotation in the horizontal plane or with electrical stimulation applied to individual canal nerve trunks, the animal compensates with neck and eye muscle movement away from the direction of stimulation (Suzuki & Cohen, 1964). Electrical stimulation also results in reciprocal limb movements so that the body is pushed into a vertical position by the extension of the opposite limb (Cohen, Tokumasu, & Goto, 1966).

Utricular and saccular signals are used for long-term stabilization of head position; canal signals are not involved. This functional separation between otolith and canal signals was demonstrated by Money and Scott (1962) when they plugged the semicircular canals in cats. The cats showed no change in control of head position. Subsequent removal of the otoliths produced bizarre head positions in the same animals. Otolith signals in cats, monkeys, and humans also stimulated limb flexion and extension patterns that restored the head to a stable vertical position in response to horizontal or vertical accelerations, with or without vision (Melvill Jones & Watt, 1971; Watt, 1976; Wicke & Oman, 1982) and in response to gravity (Roberts, 1973; Schor, 1974). Thus, converging vestibular and spinal inputs either combine or cancel their effects so that the animal can attain a position of optimum stability and orientation in space.

Functional Anatomy of the Vestibulospinal System

Neural structures responsible for the reflexes observed in the above studies can be traced from the peripheral vestibular afferents to the descending central pathways that compose the vestibulospinal system, as described below.

Vestibular afferents. The bony labyrinths in the inner ear contain several fluid-filled membranous sensory end-organs, which comprise three mutually perpendicular semicircular canals (vertical anterior, vertical posterior, and horizontal or lateral) and two mutually perpendicular otoliths (utricle and saccule) (see Figure 1). This arrangement permits the most efficient signal detection in three-dimensional space. The three canals communicate with the utricle, which communicates with the saccule. The sensory areas of the canals and otoliths are called the ampullae and maculae, respectively.

The ampullae and maculae support cilia, which are hair cells that act as mechanoreceptors. When acceleration ceases so that the animal moves at a constant velocity, ampullar cilia uncurl within the fluid medium. Therefore, their time constant, representing the rate at which they return to their resting discharge level, lasts only as long as the accelerative force, resulting in a phasic signal. Within the maculae, the mass of the statoconium membrane prevents the cilia from returning to their resting position when stimulated by gravity. Thus these cells have a longer time constant. The behavioral effect of the longer time constant is a tonic signal that represents head position in relation to gravity (Nashner, 1972).

The short and long time constants as well as the orientation of the cilia in the maculae result in a peripheral vestibular apparatus sensitive to both acceleration and gravity. The fluid mechanics of the system transduce the acceleration information to a velocity signal. This is analogous to a mathematical integration of acceleration to velocity so that the neural code signals head movement velocity. Within the VNC, the velocity signal can be mathematically integrated again to yield a position signal (Correa & Guedry, 1978), which is conveyed to the extraocular and spinal motoneurons as well as to higher centers.

The otoliths have both static and dynamic receptors that respond to low-frequency stimulation. The otolith output is the sum of the gravity and acceleration signals plus the rate of change of this sum. The
Figure 1. The orientation of the semicircular canals and otoliths in the inner ear. From Three Unpublished Drawings of the Anatomy of the Human Ear by M. Brodell (with P. D. Malone, S. R. Guild, & S. J. Crowe), 1946, Philadelphia: W. B. Saunders. The superior, lateral, and posterior canals communicate directly with the utricle. Primary innervations of the end-organs are identified. In humans, each canal is perpendicular to the other two. The lateral canal is inclined about 30° from the horizontal head plane; the superior and posterior canals are roughly in vertical planes of the head. The posterior and superior canals of one ear are oriented approximately 45° from the sagittal plane of the head.

canals are the phasic receptors of the labyrinths and combine the measures of angular velocity and angular acceleration of the skull at high frequencies of stimulation. The canals and the otoliths are all necessary to detect the extent and direction of linear movement of the head, the direction of the vertical, and the initiation of functional postural compensations (Young & Meiry, 1968).

Vestibular efferents. Our understanding of the role of the vestibular efferents is still limited. The vestibular efferent cells originate from a column of cells between the abducens and superior vestibular nuclei in the brain stem, and they synapse on the cilia in the vestibular end-organs. Electrical stimulation studies in monkeys (Goldberg & Fernandez, 1980) have demonstrated that the efferents transmit an excitatory stimulus bilaterally to irregularly discharging vestibular afferents. These afferents tend to be of large diameter (i.e., fast conduction velocities) and tend to innervate small receptor fields in the VNC (Goldberg & Fernandez, 1979). A burst of efferent activity as a result of self-initiated motion or behavioral arousal was demonstrated in the dogfish (Roberts & Russell, 1972) and the toadfish (Highstein & Baker, 1985). Efferent activation was not dependent upon activation of the afferent bundle, but acted instead as a feed-forward, or descending, command to the sensory end-organs (Goldberg & Fernandez, 1980; Highstein & Baker). By electrically stimulating the efferent pathways, researchers found that the purpose of this descending command was to alter the magnitude of the response of labyrinthine cilia (Goldberg & Fernandez, 1980). The efferent system appeared to support the resting discharge level of the vestibular afferents.
Figure 2. Interrelations of various neural structures associated with the vestibular system. Identified structures play major roles in the voluntary and reflex behaviors initiated by vestibular stimulation. Not shown are the multiple inputs from other sensory pathways (e.g., retinal, somatosensory) that influence output from the vestibular nuclei. Functionally, the efferent vestibular system would prevent saturation to maintain an accurate signal from the canals during high-velocity head rotations (Goldberg & Fernandez, 1980) and would prevent a loss of signals from cilia inhibited by the direction of head motion (Highstein & Baker).

Vestibular nuclei. The output of the VNC and the input from the vestibular end-organs are not equivalent. The afferent labyrinthine signal ascends the eighth cranial nerve to the VNC and to the flocculonodular lobe of the cerebellum. Although most input to the VNC comes from the vestibular nerve (Carleton & Carpenter, 1983), the VNC also receives inputs from the contralateral VNC and several other sensory systems (e.g., somatic, visual, auditory) (Keller & Precht, 1979; Petrosini, Trotani, & Zannoni, 1982; Tickle & Schneider, 1982) via the cerebellum, the brain stem nuclei, and the spinal cord (see Figure 2). The signals from each of the vestibular end-organs (semicircular canals and otoliths) have differing distributions among the vestibular nuclei (Gacek, 1969; Stein & Carpenter, 1967). Damage to another area could be manifested as vestibular dysfunction through this sensory convergence, even if the patient has normal labyrinthine input. For example, patients with damage to cervical dorsal roots exhibit nystagmus, ataxia, and falling (Cope & Ryan, 1959), which are the same symptoms found in patients with labyrinthine deficits.

Spinal pathways. The output from the VNC is more widely distributed than is the output from any other special sensory system. Input and output relations of the vestibular system are illustrated in Figure 2 (for a discussion of their relation to oculomotor control and spatial orientation, see the next paper, Cohen & Kesner, 1989, pp. 331-338). The VNC influences spinal motoneurons primarily through the lateral vestibulospinal tract (LVST), the medial vestibulospinal tract (MVST) travelling in the medial longitudinal fasciculus, and the reticulospinal tracts (Carleton & Carpenter, 1983; Peterson & Fukushima, 1982; Wilson & Yoshida, 1969). Originating in the VNC, both the MVST and LVST receive somatosensory input from dorsal root afferents in the central cervical...
nucleus as well as visual and cerebellar signals that have been received by the VNC (Peterson, Fukushima, Hirai, Schor, & Wilson, 1980; Wilson & Maeda, 1974). The reticulospinal tracts receive inputs from the vestibular labyrinths via projections from the VNC and the cerebellum (Peterson & Coulter, 1977; Peterson et al., 1980). Anatomical and physiological studies in cats, rabbits, and monkeys have shown that the LVST originates exclusively from the lateral vestibular nucleus and extends ipsilaterally throughout the length of the spinal cord, where it synapses directly with alpha and gamma motoneurons in medial portions of the anterior horn (Carleton & Carpenter, 1983; Peterson et al., 1980). Many of the LVST neurons have axonal branches that project to both the cervical and lumbosacral areas of the spinal cord, where they have the potential to influence muscles located in different parts of the body (Abzug, Maeda, Peterson, & Wilson, 1974; Shinoda, Ohgaki, & Futami, 1986). The MVST is a bilateral tract that originates from the medial and inferior vestibular nuclei and projects as far as the midthoracic region in the cat but more caudally within the thoracic cord of the rabbit (Akaike & Westerman, 1973). MVST neurons are mostly inhibitory and terminate monosynaptically on the upper cervical and thoracic motoneurons (Pompeiano, 1972; Wilson, Yoshida, & Schor, 1970). Medial and lateral reticulospinal tract neurons are primarily ipsilateral in the cat and extend to all levels of the spinal cord (Peterson & Fukushima, 1982).

Descending signals with the greatest influence on the final motor output are those that are transmitted through monosynaptic connections, which permit rapid and unaltered transmissions of information from the VNC to the target motoneurons. The LVST excites ipsilateral neck, trunk, and some knee and ankle extensors (gastrocnemius, soleus, and hamstring) monosynaptically (Pompeiano, 1972; Wilson & Yoshida, 1969). Contralateral extensors are excited by the same stimulus via commissural interneurons, which results in a bilateral excitation of extensors and sometimes, through reciprocal inhibition, bilateral inhibition of flexor motoneurons. Polysynaptic excitatory signals to limb motoneurons are widespread, but less so to neck and trunk extensor motoneurons.

The MVST is concerned only with axial motoneurons. Bilateral monosynaptic inhibitory connections to neck motoneurons (Wilson & Yoshida, 1969) and polysynaptic connections to thoracic motoneurons (Wilson et al., 1970) have been identified by electrical stimulation studies in the cat. The MVST also provides monosynaptic excitation of contralateral neck motoneurons and bilateral excitation of lower cervical motoneurons (Carleton & Carpenter, 1983). The medial and lateral reticulospinal tracts produce monosynaptic excitation of the flexor and extensor motoneurons of the neck, trunk, and limbs as well as monosynaptic inhibition of neck motoneurons (Peterson, Pitts, Fukushima, & Mackel, 1978; Wilson & Yoshida, 1969). The axial motoneurons, especially those of the neck, are the principal targets of the reticulospinal excitatory system (Peterson & Fukushima, 1982).

**Body Sway Stabilization**

Most models of limb reflex responses to changes in vertical orientation have been based on the results of experiments on decerebrate and physically restrained animals and have therefore applied the physiology of the reflex pathway to a complex motor process. Unfortunately, the literature on therapeutic interventions has been strongly influenced by this approach (Bobath, 1976; Fiorentino, 1973). Recent studies have added data on quiet standing in humans. Normal subjects and subjects with peripheral vestibular deficits were observed. In all of the studies we will mention, subjects stood on a platform that delivered either a brief anterior or posterior translation disturbance or a rotational stimulus at the ankle joint in either a plantar or dorsiflexion direction (see Figure 3). EMG showed activity of the lower limb and cervical muscles.

There is a consensus in the literature that the responses to whole-body destabilization are in-

![Figure 3](http://ajot.aota.org/pdfaccess.ashx?url=/data/journals/ajot/930342/)

Figure 3. Subject standing on a moving platform. A: Anterior translation of the platform resulted in a forward motion of the lower limbs from the body's center of mass and increased plantar flexion of the ankle. B: Dorsiflexion rotations of the platform produced an increased dorsiflexion angle of the ankle.
fluenced by visual, proprioceptive, and vestibular input. Researchers do not agree, however, on the relative importance of each of these pathways. The earliest studies suggested that proprioceptive input from the ankle was primarily responsible for initiating postural reactions (Nashner, 1977). Ankle muscles that received stretch inputs from the linear and rotational platform motions demonstrated a pattern of EMG activity that started in the lower limb within 90 to 120 ms of platform displacement and then appeared to radiate upward as the body restabilized. When the muscles of the neck, lumbar spine, and abdominals were also recorded during platform translations (Keshner, Woollacott, & Debu, 1988), they were found to respond as early as the stretched ankle muscles, which suggests that parallel pathways activated the sway-stabilizing response of the whole body.

More recently, studies have measured the kinematics of the human response to platform rotations and to translations on a narrow support beam (Horak & Nashner, 1986; Keshner, Allum, & Pfaltz, 1987), and a more complex description of sway-stabilizing responses has emerged. Platform rotations were found to produce an immediate passive acceleration of the head that was sufficient to stimulate the labyrinths, which suggests that vestibular signals participate in the initiation of stabilizing responses at the ankle and head (Allum & Pfaltz, 1985). Measures of head and body angular acceleration indicated that, as a result of platform motion, forces sufficient to stimulate the semicircular canals were transmitted within 20 ms of platform displacement. Subjects exhibited an initial head extension of approximately 110 ms following platform rotations that induced dorsiflexion of the ankles. Forward flexion of the body peaked at about 160 ms after onset of platform motion (Keshner & Allum, 1986; Keshner et al., 1987). Hence, the body’s initial response to dorsiflexion rotations of the platform was an appropriate forward sway with a head extension to stabilize gaze.

While subjects stood on a narrow support beam, perturbations to that beam produced stabilizing responses that began at the hip and lower trunk and were correlated with head movements in opposition to the direction of trunk motion, as described above (Horak & Nashner, 1986). Neck extensor EMG activity was temporally correlated with the backward head pitch, which confirmed a pattern of head extension as the body accelerated forward. Ankle and neck muscle responses occurred so close in time as to exclude the possibility of a biomechanical or neural transmission ascending from one joint to the other (Keshner & Allum, 1986; Keshner et al., 1988); this strengthened the argument that labyrinthine inputs generate postural responses (Allum & Keshner, 1986).

Finally, body sway during quiet standing in sub-
jects with peripheral vestibular deficits differed little from that of normal subjects (Nashner, Black, & Wall, 1982). During platform rotations however, response amplitudes of the ankle muscle EMG curves decreased significantly in patients with both eyes opened and closed (Allum & Pfaltz, 1985; Keshner & Allum, 1986; Keshner et al., 1987). Acceleration traces of these patients were also greatly reduced, and their peak head accelerations were delayed approximately 60 ms. Functionally, the delays and reduced muscle activity produced an inability to restabilize the body after each platform rotation and a greater tendency for falling. Since falls did not occur immediately or on every rotation of the platform, the loss of the vestibular signals was probably minimally compensated by other sensory inputs and initial body position. However, compensation decreased with repeated platform disturbances, which resulted in greater instability.

These results do not dismiss the role of proprioceptive afferents in sway stabilization. Short latency (60 to 90 ms) segmental reflexes that are initiated by ankle joint afferents and that are responsible for determining the functional level of stiffness in the muscle (Allum & Mauritz, 1984) are evident in the ankle muscle EMGs. In normal subjects, however, ischemia that was produced at both ankles to block ascending proprioceptive and cutaneous inputs delayed the short latency muscle responses but had no effect on the sway-stabilizing ankle muscle responses (Berger, Dietz, & Quintern, 1984; Diener, Dichgans, Guschlbauer, & Mau, 1984). Thus, the previous hypothesis of postural synergies appearing in the lower limb, and initiated only by a pattern of ascending signals from the support surface receptors is only a partial factor in stabilization. Converging inputs from the vestibular labyrinths and segmental proprioceptors are necessary to produce a postural response that is both successful and appropriate for total body restabilization.

**Visual Contributions to Stabilizing Responses**

Visual inputs converge on the VNC and contribute to the vestibular reflexes involved in eye–head coordination. The influence of eye position has been observed in VCR activity during horizontal rotations in cats (McCrea, Yoshida, Berthoz, & Baker, 1980; Vidal, Roucoux, & Berthoz, 1982), and many vestibuloocular neurons project to the upper cervical spinal cord where the neck motoneurons are located (McCrea et al.). A cervicoocular reflex that assists in compensatory rotations of the eyes to active head movements has been recorded in labyrinthine-deficient animals (Peterson et al., 1985) and in patients with absent vestibular function (Bronstein & Hood, 1986).

The absence or presence of visual inputs does not alter the EMG responses to whole-body destabiliza-
tion, except during the initial period of compensation in labyrinthine-deficient animals and humans (Keshner & Allum, 1986; Lacour & Xerri, 1980). Inappropriate or unexpected visual inputs, however, are destabilizing. Changes in the size of the EMG response and increased body sway can be induced by modifying the visual input such that the visual scene appears to move in the same direction as the moving body, rather than, as in normal experience, in the opposite direction. Similar changes can be induced by moving the visual scene at a different velocity than that of the subject (Nashner & Berthoz, 1978; Wicke & Oman, 1982). Loss of peripheral vestibular inputs magnifies the changes in EMG and sway elicited by the abnormal visual stimuli (Lee & Aronson, 1977). Unexpected feedback interferes with the subject’s ability to respond appropriately. Therefore, therapy should aim to eliminate extraneous stimuli and to assist patients in planning for a changing visual scene. Patients with visual deficits or who have difficulty compensating for unexpected or novel inputs would benefit from a stable, unchanging environment in the early stages of motor learning.

**Vestibulospinal Reflex Modifiers**

The cerebellum and reticular formation are two of the higher motor centers that have reciprocal connections with the VNC and have been implicated in the control of postural reflexes (Peterson & Fukushima, 1982; Shimazu & Smith, 1971). Areas in the parietal lobe also receive both semicircular canal and cervical proprioceptive inputs (Mergner, Becker, & Deecke, 1985), but their effect on postural reflexes is indirect and not fully understood.

By modulating the descending influences on spinal motoneurons, the cerebellum may provide an error-correction mechanism for the trunk and limb movements initiated by the vestibulospinal tract that is similar to that of the vestibuloculomotor pathways (Ito, 1971; Robinson, 1976). For example, when placed on a rotating platform, subjects with damage to the anterior cerebellar lobe had normal tibialis anterior muscle response latencies, but the amplitude and duration of their responses were 2 to 3 times greater than normal (Diener, Dichgans, Bacher, & Guschlbauer, 1984). Also, the response amplitudes of these subjects failed to decrease over trials, unlike the responses of normal subjects. In monkeys, removal of portions of the cerebellum (i.e., nodulus and uvula or flocculus) produced disequilibrium and head oscillations (Dow, 1938). When these ablations were performed after bilateral labyrinthectomy, no postural disturbances occurred. Thus the cerebellum may influence the vestibulospinal reflexes, but as a modifier and a comparator rather than as an initiating of these reflexes (Robinson).

The reticular formation receives input from various sources that could influence the control of posture, including vestibular, somatosensory, cortical, and cerebellar signals (Magni & Willis, 1964; Orlovsky, 1970; Pompeiano & Barnes, 1971). The vestibulospinal pathways participate in the transmission of sensory input to the motor nuclei underlying the vestibulocollic reflex (Peterson et al., 1980). When the LVST and MVST were damaged in cats (Bilotto, Goldberg, Peterson, & Wilson, 1982), the vestibulocollic reflex was not abolished, presumably as a result of these reticulospinal tract signals—another indication that stimulation of peripheral vestibular afferents does not directly correlate with an observed postural response. Tecto-reticulo-spinal neurons in the cat discharge in relation to active eye-head orienting toward novel stimuli and have been found to terminate in the premotor areas on the lower brain stem (Grantyn & Berthoz, 1985). Reticulospinal neuron signals control phasic cervical muscle activity and also project to structures controlling ocular and facial movements (Grantyn & Berthoz, 1987).

Results from studies on the influence of the visual scene on the organization of a postural response suggest a predictive, or cognitive, component in the planning of responses to destabilization. Keshner (1983) studied the role of expectation in the organization of the equilibrium reaction in children aged 4 to 6 years. The equilibrium reaction is assumed to be a compensatory reaction to the labyrinthine and proprioceptive stimuli arising from whole-body tilt in space (Martin, 1965). Subjects initiated their equilibrium responses in advance of the tilt stimulus, however, when either direction of tilt or onset of tilt or both was known. The equilibrium response occurred in reaction to the tilt stimulus only when both direction and onset were known (see Figure 4). The strong influence of instructional information on this response implies involvement of higher levels of the nervous system in the planning and production of postural responses.

Restabilization of the body after platform perturbations may also be due to a central motor program that produces a net effect of agonist and antagonist forces rather than due to the result of stretch reflexes ascending along one side of the body. Overlapping bursts of EMG activity for ankle and neck muscles on both sides of a joint have been recorded for a single direction of platform rotation for velocities ranging from 12 degrees/s to 80 degrees/s with platform excursions of 1 to 5 degrees (Allum & Budinggen, 1979; Allum & Pfaltz, 1985). This temporal coincidence of the joint antagonists (e.g., tibialis anterior and soleus or neck extensors and flexors) suggests that a central coactivation command (Fel'dman, 1981) rather than a reciprocal stretch reflex has been initiated.
The American Journal of Occupational Therapy

Summary

General principles of treatment may be derived from the research data. When treating a patient with postural instability, the therapist should consider several interventions, including (a) assisting the patient in planning a motor response pattern through prior information about the task demands; (b) supporting the motor response through graded stimulation of functionally opposed muscles surrounding an individual joint or of muscles participating at opposite ends of the body (i.e., neck extensors with ankle flexors); (c) enhancing the feedback loop by intensifying a specific sensory input, or supplying cognitive input by discussing the patient’s expected and actual response; and (d) limiting interference of conflicting stimuli by having the patient practice a simple postural task within a structured environment. By evaluating the functional status of the individual sensory systems and the patient’s ability to process motor commands, the therapist can determine how these techniques could be combined and when task demands should be increased.

All head movements generate vestibular signals. Most, if not all, body movements generate head movements due to the inertial forces acting on the head atop the flexible neck. Descending signals from higher centers also influence the control of head and body movements through expectation of the consequences of actions. Many signals—vestibular, visual, auditory, and somatosensory—converge at the VNC, the output of which reflects the combination of these inputs. Loss of any one of the many pathways to this center changes the nature of the response to destabilization.

Motor behaviors that are highly dependent on specific labyrinthine signals, such as eye movements, are impaired following loss of peripheral vestibular inputs, but postural responses are less dependent on these specific labyrinthine inputs. Stabilizing reactions at the neck and ankle, for example, are altered when peripheral vestibular inputs are absent, but the remaining EMG responses are still sufficient to maintain an upright position. In situations where other sensory information is also unreliable (e.g., conflicting visual inputs), peripheral vestibular deficits create greater instability. Without labyrinthine signals, postural instability becomes worse with repetition of a destabilizing stimulus, so that a successful attempt by a patient on a task presented for the first time may not carry over through several repetitions.

Given the complex processes involved in producing a simple response, no single stimulus, such as spinning for labyrinthine input or vibration of individual ankle muscles, will elicit normal postural behavior in patients. Furthermore, results of tests for behaviors assumed to be mediated by labyrinthine signals, such as nystagmus and disequilibrium, cannot serve as the sole diagnoses of suspected vestibular dysfunction. Therapists should consider treating patients with postural disorders in a highly consistent environment, rather than in a clinic where the arrangement of the equipment and the visual and auditory stimuli change daily. A consistent environment would provide patients with reliable and predictable feedback while they practice correct postural behaviors. Once pos-

![Figure 4](https://example.com/figure4.png)

Figure 4. Proportion of responses that appeared in a predictive or reactive mode for each age group within each instructional set. Numbers under each bar indicate the age groups (4-, 5-, and 6-year-olds). Responses during the warning interval are predictive of the tilt stimulus; responses during the tilt interval are in reaction to the tilting of the platform. COCD = knowledge of the onset and direction of tilt. COUD = knowledge of the onset, but uncertain direction of tilt. UOCD = uncertain onset, but knowledge of direction of tilt. UOUD = uncertain of both onset and direction of tilt.

From these data we can conclude that early postural reactions occur at several segments of the body following a disturbance to a supporting surface. Studies showed that postural reactions were initiated not only by the proprioceptive afferents that were directly stimulated by a change in the support surface, but also by vestibular, visual, and even tactile afferents that were stimulated by a mechanically transmitted disturbance to head and body position. Postural stabilization appears to be the net effect of simultaneous, but opposing, forces at a single joint (agonist and antagonist muscles) that have been modulated by the initial position at that joint and the task of regaining stability. Any alterations of the expected sensory feedback, either through experimental manipulation of the environment or through a sensory deficit, will impair the performer’s ability to regain stability.
cultural reactions are learned, the patient could be introduced to variable disturbances of equilibrium and to more dynamic environments.

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