VESTIBULAR CONTROL OF POSTURE AND GAIT

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GENERAL ORGANIZATION OF POSTURAL ACTIVITY

Postural control is determined by multiple factors.

Postural control, which is the regulation of the activities of the various agonist and antagonist muscles for attaining or maintaining a postural position, is organized at more than one level (see 30 for a review). At segmentary (or spinal) level, it is largely automatized. It depends on information from various sources (cutaneous, muscular and joint proprioceptive afferents) which is integrated by spinal motoneurons and which triggers reflex postural adjustments. These pluri- and intersegmentary reflexes, which are fundamental in organizing motor activities, only explain part of the origin of postural and balancing reactions.

A spinal animal cannot take an antigravity position typical of the species and remains incapable of righting itself; its movements are rudimentary and imperfect. To this elementary spinal level, supraspinal influences are constantly added, determined in part by sensory inputs of diverse origins. All of the sensorimotor systems which participate in detecting the position of the body and its various parts in space, as well as its orientation and the direction of its movements (somesthetic, musculo-articular proprioceptive, visual, auditory, visceral and vestibular information) are integrated by central nervous structures, mainly located in the brain stem. A thalamic animal, unlike a spinal preparation, has static and dynamic postural reactions quite comparable to an animal retaining an intact cerebral cortex. It holds itself erect against gravity and its reactions of righting, of head stabilization and achieving equilibrium are present as normal; one fundamental role is of course played by the labyrinth and visual afferents which participate in maintaining posture and in producing adequate postural readjustments. However, the vestibulospinal fibers, transmitting the tonic and phasic nerve messages necessary for effecting these movements, still stereotyped, are only one of the pathways contributing to postural control. Other influences, notably from the red nucleus, the reticular formation and the cerebellum, make important contributions to carrying out postural activities and regulating locomotor programs. On this background of postural activity regulated by multiple sensorimotor systems are organized the voluntary movements, i.e. the deliberate actions which displace the body and its parts in space. Depending on which motor behavior is predicted and in which

environmental context it would take place, the nature of the preparatory postural activities (anticipated responses) and those associated with movements (compensatory responses) may change radically. Such modifications show that the functional connections of the neuronal networks regulating motor programs for keeping or recovering balance are extremely flexible, and do not support a reflex or predetermined view of postural activity.

The dual nature of postural adjustments, stereotyped yet functionally flexible, is exemplified in man by the observations of Nashner and his group (55, 56, 35). While a subject stands on a platform, his posture is perturbed by sudden forward or backward movements of the platform. These movements induce a sequence of muscular contractions in accordance with a particular, precise space-time organization. A backward platform movement, for example, will unbalance the subject forward. Compensatory postural responses for maintaining equilibrium are here expressed by a rapid contraction of the ankle extensors (gastrocnemius), then the thigh and back extensors (Fig. 1A). This distal to proximal sequence
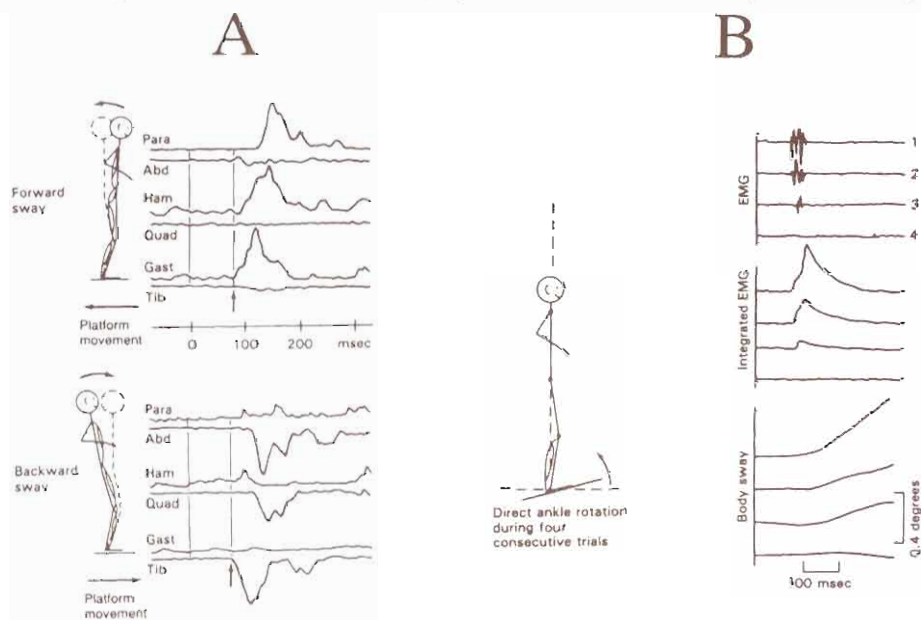

Fig. 1 – *Compensatory postural reactions in response to rapid movements of the support.*

A. Space-time organization of muscular responses when the basis of support (platform) moves backward (upper diagrams) or forward (lower diagrams). The postural reactions evoked are compensatory, aimed to reestablish equilibrium. They follow a distal to proximal sequence of activation of the extensor muscles (support moving backwards) or flexor muscles (support moving forward) (from Horak and Nashner, 35, adapted by Ghez, 30). Electromyographic records, integrated and rectified from the following muscles: paraspinal (Para), abdominal (Abd), hamstring (Ham), quadriceps (Quad), gastrocnemius (Gast) and tibialis anterior (Tib).

B. Muscular responses evoked by a stretching of the ankle extensor muscles (Gast) identical to the one in A, but obtained by an unexpected upward movement of the platform. EMG records in the gastrocnemius muscle in the course of 4 successive trials. The muscle response, similar to that obtained in A, is not compensatory: in fact it increases the dysequilibrium. Diminishing rapidly, it disappears on the fourth trial and is even replaced by compensatory activity in the ankle flexor tibialis anterior. (Adapted from Nashner, 55, modified by Ghez, 30).

of muscular activation results from the bringing into play of proprioceptive afferents initiated by the stretching of the ankle extensors, of labyrinth information resulting from head motion, and of visual cues triggered by the displacement of the visual environment on the peripheral retina (retinal slip). Because the information provided by these various sensory modalities is complementary, it elicits the appropriate compensatory postural adjustments. A similar stretching of the ankle extensors can also be obtained by upward platform rotation (Fig. 1B). In this situation, the sequence of muscle activation is the same but it results in increased postural unbalance. However, after a few trials the subject manages to stabilize his posture by suppressing this inappropriate activation of the extensors and by producing a compensatory contraction of the antagonist flexor muscles of the ankle (tibialis anterior). The environmental context where the activities take place can also induce profound modifications in postural behavior (cf. the concepts of «hip strategy» and of «ankle strategy» dependent on the type of support, wide or narrow, as described by Nashner). These data demonstrate that the subject's prior experience, his training and his history can profoundly modify very automatized postural patterns.

Disorders of posture and balance: diversity of ataxias.

The functional importance of the captors and central nervous structures responsible for postural and balacing reactions has been brought out by animal experiments and stressed in various pathologies affecting posture.

Cerebellar ataxia, observed in numerous cerebellar syndromes, includes a group of clinical signs resulting from serious deficiencies in muscle tone and in postural and locomotor equilibrium. In particular, hypotonia is observed in muscle extensors and a paradoxal exageration of neck and vestibular reflexes appears, accompanied by an «alpha» type rigidity due to the deficit of inhibitory influences from the Purkinje cells on the vestibulospinal neurons of the lateral (Deiters') vestibular nucleus. The most intense postural and locomotor deficits result from disorders affecting the paleocerebellum. The base of support of these cerebellar patients is wider, their gait is vacillating and accompanied by falls to the side of the lesion, and reflexes for maintaining posture and balance are extremely attenuated or even absent.

Friedrich's ataxia results from lesions involving the posterior spinal cord and the cerebellar bundles. Like pure cerebellar ataxia, it is revealed by postural and locomotor deficits which are not accentuated by deprivation of visual information.

Tabetic ataxia mainly originates in proprioceptive sensory fiber lesions. These afferents of muscular origin normally participate in the regulation of muscle tone and the development of postural adjustment reflexes looping at the segmentary and supra-spinal levels. Therefore it is not surprising that this type of ataxia is revealed by postural and locomotor disorders which are greatly aggravated in the absence of vision.

Labyrinthic ataxia is observed after a vestibular system lesion. A unilateral lesion

is accompanied by major deficits involving posture, locomotor equilibrium and oculomotor control. In most species, an asymmetry in distribution of postural tone is seen, characterized by flexion of the ipsilateral limbs and extension of the limbs contralateral to the lesion. A marked tilt of the head toward the lesioned side is present, resulting from ipsilateral increase and contralateral decrease of muscle tone in the head extensors. Lateral head tilt is also accompanied by a downward rotation of the head. Standing is impossible during the first days following the lesion, and the base of support remains widened for a longer time. Walking, with frequent falls to the side of the lesion, shows a typical ipsilateral deviation. Righting, Magnus reflexes and the dynamic postural reactions are absent. Stabilizing the head in space is defective. An intense ocular nystagmus beats (quick phase) toward the intact side. These deficits, further accentuated in eyes closed and dark conditions, diminish progressively over time, exhibiting a time constant of about 4 to 5 weeks in most species.

VESTIBULAR SYSTEM: ANATOMY AND FUNCTION

Peripheral vestibular system.

Figure 2 A-B illustrates the location of the peripheral vestibular system and the organization of the sensory receptors of the semicircular canals (SCC) and of the otolith (utricle and saccule) system (see 40). The vestibular receptors make up a complete inertia center, sensitive to angular accelerations (canals), linear accelerations and gravity (otolith organs), which has very low response thresholds (0.1 to 0.2 deg/s^2 for the canals, 10^{-3} to 5.10^{-3} g for the otoliths).

The afferent fibers in contact with the vestibular receptors have their cell bodies located in Scarpa's ganglion. The axons, 12.000 in the cat, make up the vestibular nerve, which is divided into three branches: superior, inferior and posterior.

The primary vestibular afferents in the cat have a mean spontaneous activity of 35 imp./sec for fibers of canal origin (90 imp./sec in the monkey), and of 30 imp./sec for fibers of otolith origin (60 imp./sec in the monkey). The studies of Goldberg and Fernandez (see 31) showed that the canal afferents transmitted to central structures messages related to angular acceleration at low stimulus frequencies (less than 0.025 Hz) and to velocity at higher frequencies. Macular afferents originating in the utricle and the saccule transmit information on the position of the head in space and on the acceleration of movements, although certain units do show sensitivity to velocity.

Central vestibular nuclei.

The vestibular nuclear complex is composed of four principal nuclei (superior, medial, lateral, descending) constituting one of the largest nervous structures in the brainstem. Recent three-dimensional reconstruction of the vestibular nuclear

complex from serially sectioned brains in humans showed that each VN had typical shape and structural characteristics (13). Associated cell groups having either direct (group y) or indirect (groups x and z) relationships with the VN should also be included but, as a rule, only those groups receiving afferents from the vestibular nerve are considered as belonging to the VN complex.

1. *Topography and cytoarchitectonic organization of the vestibular nuclei.* — Gerrits (29) published the most recent synthesis on the topography and cytoarchitecture of the VN complex in humans. We shall give below a summary of his paper.

Superior vestibular nucleus (SVN): First described by von Bechterew in 1885, this nucleus is located under the lateral floor of the fourth ventricle, bordered dorsally by the superior cerebellar peduncle and laterally by the juxtarestiform body, situated dorsal to the medial vestibular nucleus. A great many fibers originating in the cerebellum pass through the SVN. It is composed of small and medium-sized neurons in the periphery and of larger cells in the central part of the nucleus.

Medial Vestibular Nucleus (MVN): The MVN is one of the two largest VN, with the descending nucleus. It has been divided into a parvo-cellular, a magnocellular and a caudal section, in the rabbit as in the cat. This three-part division seems far less clear in man (29). The parvocellular section starts at the caudal pole of the SVN and continues caudally where the size of the neurons increases. The magnocellular section, known in literature under several names, shows large neurons suggesting a relation with the part of the Deiters' nucleus containing the giant cells (15, 67). However, this was not suggested from experimental anatomical studies (60, 61) and it was also found that acetylcholine esterase reactivity was greater in this section as compared to the nearly negative reactivity encountered in the lateral nucleus. The caudal section of the MVN contains small and medium-sized neurons.

Lateral vestibular nucleus (LVN): Described by Deiters in 1865, it contains numerous giant cells (50-100 µm) even larger than those of the magnocellular section of the MVN, less rounded and with their nucleus in a rather eccentric position. This nucleus is bordered ventrally by the descending nucleus, the vestibular nerve and its interstitial nucleus over almost its entire length. The observations by Gerrits (29) are at variance with the results of earlier studies in man (67), particularly with regard to the inclusion of the magnocellular section of the MVN in the LVN, which is not suggested by Gerrits.

Spinal or descending vestibular nucleus (DVN): First described as the spinal component of the vestibular nerve, this structure was named «descending nucleus» by Ramon y Cajal (64) in 1896. Bordered laterally throughout its length by the inferior cerebellar peduncle, its main anatomical characteristic is the presence of longitudinally oriented fiber bundles, allowing to delineate its rostral pole from the LVN which has transversaly oriented fibers. The bundles present in the DVN are composed of fibres originating in the fastigial nucleus, the flocullus, the uvula, the nodulus and the caudal vermis, and of olivocerebellar climbing fibres.

Associated cell groups: These include a compact group of small cells located dorsolateral to the LVN, capping the restiform body, originally described in the
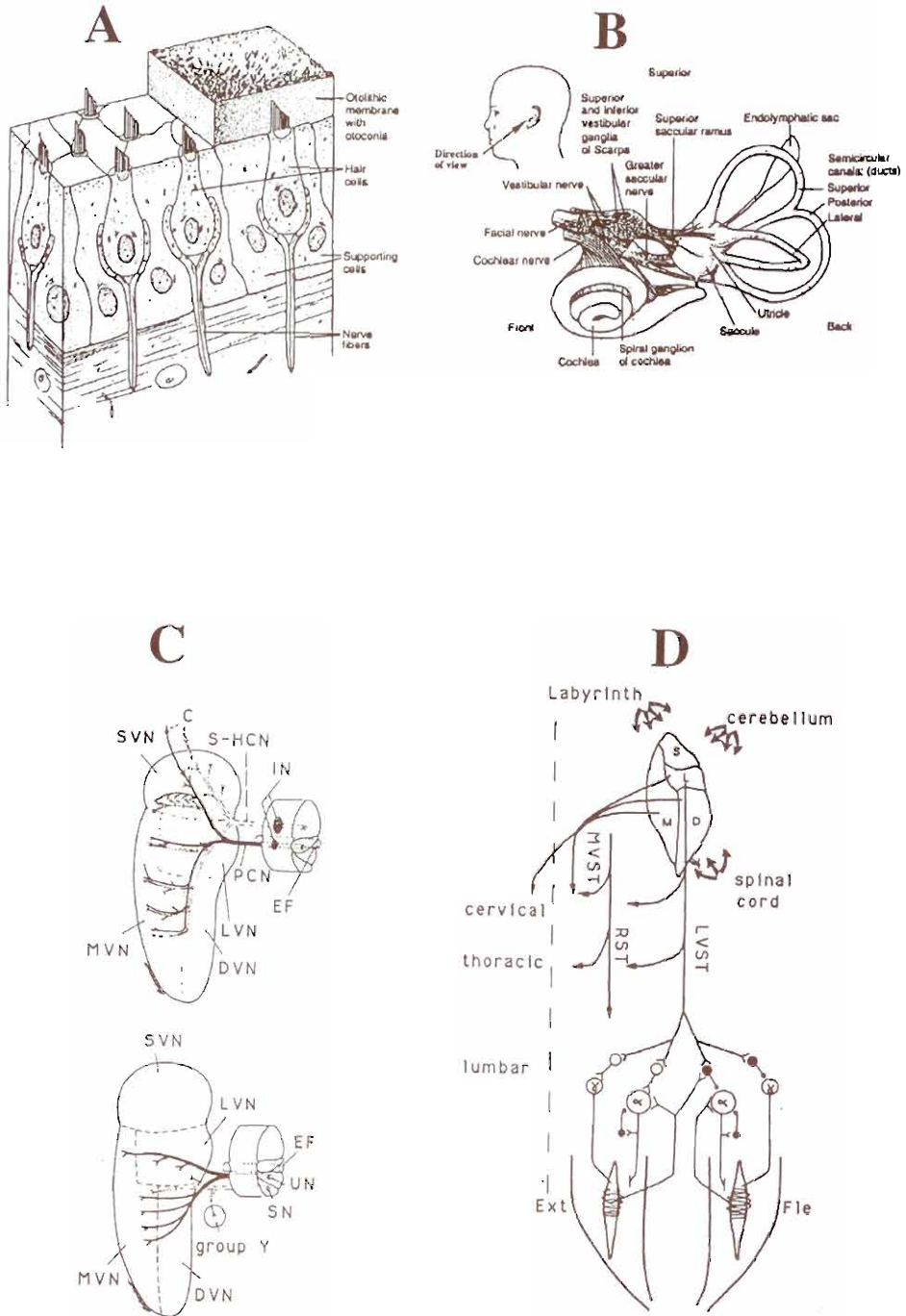
Fig. 2

cat by Brodal and Pompeiano (15) as group *y*. This group is well developed in most mammals, including human beings, and is considered as belonging to the VN complex, since it receives afferents from the vestibular nerve. The interstitial nucleus of the vestibular nerve (IN) has been first described by Cajal (64) as clusters of large neurons. Many of these cells are giant, especially in humans, suggesting a close relation with the LVN. Other cell groups, not receiving primary vestibular afferents, have been described by Pompeiano and Brodal (61, 62).

2. *Vestibular nuclei afferents.* — *Labyrinth afferents*: Golgi studies have shown that the primary vestibular afferents from the otolith and SCC systems divide into two branches, an ascending branch terminating in the SVN after passing through the LVN and a descending branch reaching the DVN, the MVN and group *y* (see 18). Rather controversial results have been reported for the LVN, with absence of degeneration in light microscopy (41) but the presence of terminal degeneration in electron microscopic studies.

Silver degeneration and autoradiographic methods were used to investigate the projection of the primary vestibular afferents to the vestibular nuclear complex. A summary of these results in cat and monkey has been recently presented by Carpenter (19), pointing to a massive ipsilateral projection in parts of each VN, except in the dorsal part of the LVN, the caudal and medial sections of the MVN, the peripheral portion of the SVN and the cell groups *f*, *x* and *z*. According to Raymond et al. (65), primary vestibular projections have aspartate/glutamate as their excitatory neurotransmitter. A segregation between canal and otolith inputs was demonstrated, with utricle afferents projecting in the rostroventral section of the LVN, saccule inputs reaching also the LVN, group *y* and the lateral part of the DVN, and canal information projecting in the SVN and the MVN (Fig. 2C: cf. 28, 57). These neuroanatomical findings are corroborated by electrophysiological investigations showing neuronal responses to canal stimulation (angular acceleration), mainly in the rostral section of the vestibular nuclear complex while otolith stimulation (gravity and linear acceleration) elicits responses most frequently in the central and caudal parts of the VN (see 76, also 68 and 39).

Fig. 2 – *Anatomical and physiological organization of the vestibular system.*

A. Structural organization of the utricular macula. The sensory cells are sensitive to head position (gravity vector) and to linear accelerations (modified by Kelly, 40).

B. Peripheral vestibular system, composed of the 3 semicircular canals and of the otolith system (utricle and saccule). These vestibular receptors transmit nerve messages indicating the head's position and its movements in space, mediated by the afferent fibers whose soma are located in Scarpa's ganglion. The axons of these primary vestibular afferents make up the various branches of the vestibular nerve (adapted from Kelly, 40).

C. Projection sites of the primary vestibular afferents originating in the semicircular canals (upper diagram) and the otolith organs (lower diagram) (adapted from Gacek, 28). SVN, MVN, LVN, DVN: superior, medial, lateral and descending vestibular nuclei. PCN: posterior canal nerve; S-HCN; superior and horizontal canal nerves; IN: Interstitial nucleus of the vestibular nerve; EF: efferent fibres; SN: saccular nerve; UN: utricular nerve; Y: group Y; C: cerebellum.

D. Organization of the vestibulospinal pathways. Influences exerted by the medial and lateral vestibulospinal tracts (MVST and LVST) and the reticulospinal tracts (RST) on the α-and γ-motoneurons controlling extensor (Ext) and flexor (Fle) muscles of neck, back and limbs. Excitatory and inhibitory spinal interneurons are shown as open and filled circles, respectively. Convergence of inputs from the labyrinth, the cerebellum and the spinal cord on the medial (M), lateral (L), descending (D) and superior (S) vestibular nuclei is illustrated.

Commissural afferents (crossed vestibulo-vestibular connections): In contrast with the primary vestibular afferents (and also cerebellar inputs) which are purely ipsilateral, a massive commissural system interconnects the VN (63), allowing the VN to integrate the vestibular inputs on both sides. Commissural fibres were evidenced in lesion experiments and studies using retrograde axonal HRP transport. They interconnect principally parts of different VN (19), suggesting a less specific, more divergent projection, with a given neuron able to influence other secondary vestibular cells located in various parts of the VN complex on the contralateral side. The only exception is the LVN which has no commissural system in the cat (63), as well as in the rabbit and the monkey (37). The commissural connections would have inhibitory effects on canal-related neurons. By contrast, crossed afferents (but not commissural) from the reticular formation were found to exert excitatory influences on otolith-related units located in the VN.

Intrinsic afferents (ispilateral vestibular connections): These are ipsilateral connections between one nucleus and another in the same vestibular complex. They include reciprocal liaisons between the SVN, the MVN and the DVN and also the connections from group *y* onto these nuclei. The LVN would receive projections from the MVN.

Cerebellar afferents: The cerebellovestibular projections have long been known but recent studies have produced more precise data, using retrograde transport of HRP and autoradiographic techniques (2, 22). Afferents to the VN arise from the vestibulocerebellum, the cerebellar vermis and the fastigial nuclei. The Purkinje cells of the B-zone in the anterior lobe of the cerebellar cortex project on the LVN, those of the A2-zone on the MVN. The caudal part of the vermis (lobules 8, 9 and 10) influences the entire vestibular nuclear complex. A bundle of fibers originating in the flocculus also projects to all the VN, except for the LVN. Projections from the nodulus and uvula reach the MVN and the DVN. These cerebellar influences are supplemented by ipsi- and contralateral fastigio-vestibular projections to the DVN and the MVN. Other afferents, originating in the inferior olive in particular, have been shown at the LVN level. These would be either collaterals of olivocerebellar climbing fibers or direct olivovestibular projections.

Spinal cord afferents: Important spinovestibular projections, direct or originating in collaterals of axons in the spinocerebellar tracts, have been shown with neuroanatomic degeneration methods (16, 62). They project mainly to the dorsal parts of the LVN, the MVN, the DVN, and to group *x*. These influences would originate in the lumbar spinal cord. More recently, it was distinguished an ascending medial spino-vestibular system, connecting the cervical levels with the MVN (magnocellular part), and an ascending lateral spino-vestibular system originating at thoracic and lumbar levels and impinging more specifically the DVN, the LVN and the group *x*.

Other afferents: the vestibular nuclear complex receives afferents from other nervous structures such as the interstitial nucleus of Cajal (INC), the thalamus, the raphe nuclei, the reticular formation and the prepositus hypoglossal nucleus (PHN). Projections from the parietal cortex have been described recently (25).

Many reciprocal connections were evidenced between these structures, and notably between the PHN and the VN (53).

One can schematically consider here two main groups of neuronal signals converging on the VN. A first group is related to visual detection of ego- and exocentric motions and includes messages elicited by the displacement of the images on the retina (retinal slip). Such signals relay in various nervous structures: the optic tract and the accessory optic nuclei, the inferior olive and the cerebellum, the nucleus reticularis tegmentis ponti (NRTP) and/or the PHN (49). Neuronal responses were recorded in the MVN during rotatory optokinetic stimulation in the horizontal plane (74) and during linear optokinetic stimulation in the vertical plane (4). A second group is more specifically related to nervous signals specifying gaze direction. The experimental arguments supporting this view that VN activity depends on orienting behavior are presented in the following section. The reciprocal connections between the PHN and the VN on one hand, the INC and the VN on the other hand would play a major functional role in elaborating eye position signals in the horizontal (6) and vertical (26) planes.

VESTIBULAR NUCLEI AND POSTURAL CONTROL

The efferent connections between the VN complex and spinal levels constitute the anatomic foundation for the vestibulospinal reflexes and, in large measure, for postural control of the head and limbs. The vestibular nuclear complex is not only involved in these compensatory reflexes but also in eye-head coordination and orienting behavior, in close relationship with the reticulospinal and the tecto-bulbospinal systems.

Vestibular control of the head-neck system.

This is mainly effected by the medial vestibulospinal tract (MVST) composed of fibers arising principally in the MVN and the DVN (61). Slight contributions from the SVN and the LVN have been reported, at least in the rabbit. Via the medial longitudinal fasciculus (MLF), this tract distributes bilaterally at the cervical and upper thoracic levels (cf. Fig. 2 D). The axons of these vestibulospinal neurons, which conduct fairly rapidly (60 m/sec average), terminate in Rexed's laminae VII and VIII. These vestibulospinal pathways, with nearly exclusive control over neck muscles, exert facilitatory and inhibitory influences on the spinal motoneurons according to a very complex pattern (Fig. 3: cf. 75, 77, 18).

Control over the postural activity of the neck and back is also ensured by the lateral vestibulospinal tract (LVST). The LVST has long been thought to originate only in the LVN, from giant Deiters' cells and from smaller neurons, although a slight contribution does come from the DVN (61). It therefore transmits mainly information from the saccule and the utricle (Fig. 2 C, D). Certain of its fibers reach as far as the lumbosacral level, but the majority terminate more rostrally.
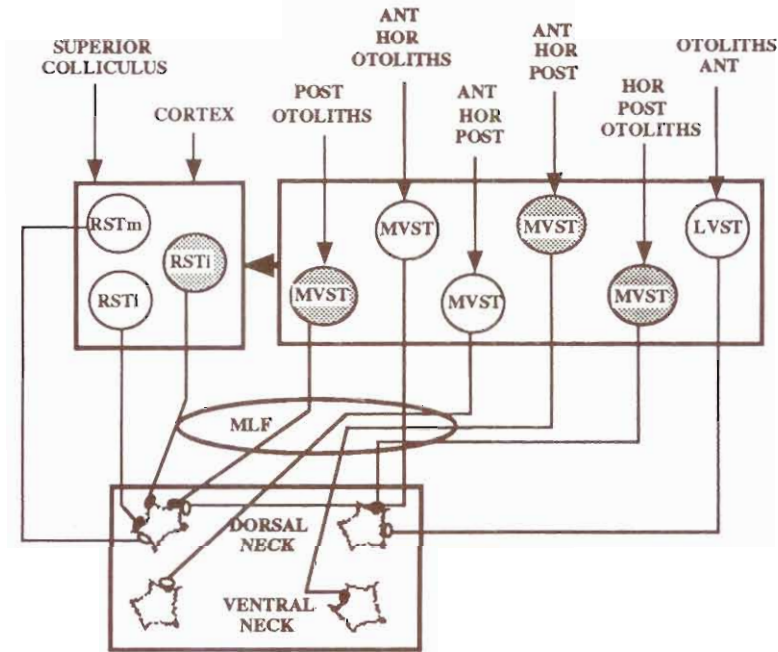
Fig. 3 – *Vestibular control of the head-neck system.*

Diagram of the organization of the vestibulocollic reflexes. White circles stand for groups of excitatory neurons and shaded circles for groups of inhibitory neurons (adapted from Wilson and Peterson, 75). MVST and LVST: vestibular neurons receiving canal (Ant, Hor, Post) and otolith input, then projecting on neck motoneurons via the medial and lateral vestibulospinal tracts; MLF: medial longitudinal fasciculus: RST: reticulospinal tract.

This distribution is not random. In fact, Deiters' nucleus is organized somatopically according to a dorsoventral gradient: cells located in the dorsocaudal portion project to the lumbar level while cells in the central region project to the thoracic level and those of the rostroventral region send out axons to the cervical level (61). An even more complex scheme seems to exist if one considers the fact that collaterals of a single axon reach different medullary levels (1). The LVST are constituted by rapidly conducting fibers (90 m/sec) which terminate ipsilaterally on the interneurons of Rexed's laminae VII to IX. Connections with neck motoneurons are monosynaptic, at least in the cat. The influences exerted by the LVST facilitate the α and γ- motoneurons of the extensor muscles (59) and have opposite effects on the flexor muscles. Latencies of less than 2.5 msec have been observed at the level of neck muscle motoneurons after electrical stimulation of the vestibular nerve.

Figure 3 summarizes the organization of the connections involved in postural control of the head (vestibulocollic reflexes, see 77). At the level of the dorsal neck motoneurons (MN), the LVST transmits excitatory influences originating in the anterior canal and the ipsilateral otolith receptors. In parallel, the influence

of the MSVT is exerted through the mediation of excitatory fibers, which transmit the excitation of the contralateral anterior and horizontal canals, and the mediation of inhibitory fibers which transmit the inhibition of the ipsilateral horizontal canal and the bilateral inhibition of the posterior canal and the otoliths. Projections to the ventral neck MN seem to pass only via the MVST. The stimulation of the three semi-circular canals is the source of an ipsilateral inhibition and a contralateral activation of the MN of the flexor muscles (27). The participation of vestibulocollic reflexes of otolith origin to head stabilization in space has long been known (71), but the respective contribution of the utricle and saccule to eye and head (gaze) stabilization has been demonstrated in a more recent past (77). The investigations we have conducted in our laboratory have been devoted particularly to the description of the dynamic properties of the otolith-eye and -head reflexes in the three dimensions (X, Y, Z) of space (46, 42, 10, 11). We have clearly demonstrated the functional involvement of otolith inputs in eye-head coordination and their predominant contribution in the high range of motion, at stimulus frequencies above 0.5 Hz.

Vestibular control of back and limbs.

Control of axial muscles and postural adjustments of the limbs are effected with the mediation of the vestibulospinal pathways described above (LVST and MVST). Animal experiments in our laboratory have pointed to the major contribution of the otolith inputs in elaborating such compensatory motor reactions and muscular synergies for maintaining equilibrium. We have described the spatio-temporal properties of muscle responses to otolith stimulation in the vertical plane in the monkey (45) and in the cat (46, 11) as well as the motor deficits induced by the lesion of the vestibular system (10). Recent experiments in the cat (48) have nicely shown that the cortical-induced postural changes of the limbs were mediated through the Deiters' nucleus, suggesting again a participation of the LVST in the limb postural adjustments.

Vestibular control of back and limbs may be also effected by means of a third contingent of fibers forming the caudal vestibulospinal tract (CVST: cf. 58). The CVST originates in the caudal pole of the medial and descending nuclei and in group *f*. The axons forming the CVST reach all way to the lumbar level and conduct slowly: 12 m/sec average. Its functional properties have not as yet been clearly defined. Moreover, recent studies in the cat and the monkey did not confirm this third tract (see 19).

Coordination of head, back and limb musculature could be performed by single vestibulospinal neurons acting on different motoneuronal pools located at different spinal levels. In fact, single physiologically-identified lateral and medial vestibulospinal tract axons in the cat were found to have multiple axon collaterals at different segments in the cervical cord (69). Up to seven collaterals were given off from the stems of MVST and LVST axons, with similar morphological characteristics showing restricted rostrocaudal extent (0.2 mm - 2.1 mm) contrasting with wide

spreads in the mediolateral and dorsoventral directions. These anatomical data suggest that single LVST and MVST axons may control excitability of multiple dorsal axial muscles. Moreover, some axon collaterals at the cervical segments were identified as branching of LVST axons reaching the thoracic and lumbar levels (1, 69), indicating that single vestibulospinal neurons may coordinate head, trunk and limb muscle activity.

Vestibular control of eye-head coordination.

Another very stimulating result, now well documented in animal experiments, is the recent discovery of branching patterns from secondary vestibular neurons, giving rise to terminal arborizations at the oculomotor and spinal levels. A single secondary vestibular neuron can constitute a common structural element involved in both vestibulospinal and vestibulo-ocular pathways. Using intracellular injections of HRP, McCrea et al. (54) and Berthoz et al. (8) showed that around 70% of the neurons located in the MVN in the cat had branching axons reaching the contralateral abducens nucleus and the spinal cord. Such branchings have been seen for posterior and anterior SCC-related secondary vestibular neurons. In the rabbit, 20% of vestibular units identified as preoculomotor neurons send axon collaterals to the spinal cord. Via these extensive branching patterns, a given sensory signal would be economically distributed to multiple target structures involved in orienting behavior or eye-head coordination process (32, also 6).

Neuronal activity of cells within the VN complex seems closely related with orienting mechanisms. It was shown that neurons located in the pontine RF and discharging in relation with eye movements (phasic excitatory and inhibitory neurons) project to the VN (see 7). 40 to 50% of secondary vestibular neurons exhibit neuronal modulations during ocular saccades, most of them showing an inhibition of their firing rate. Moreover, around 20% of the secondary vestibular cells are influenced by eye position signals. Eccentricity of the eyes to the right, for example, induces an increase of the resting discharge of neurons located in the VN on the left side, which in turn produces an increase of the EMG activity of dorsal neck muscles on the right side. Such a correlation between eye position in space and vestibular cell discharge was found for eye deviations in the horizontal (8, 9) and vertical (38) planes. It is commonly admitted that the nervous messages specifying gaze direction and influencing VN activity are made of oculomotor efferent copy signals. However, neuronal modulations in the VN were shown associated with passive eye displacements, suggesting that proprioceptive afferents from the extraocular muscles which project onto the VN (3) could also intervene.

Vestibular influences on neck, back and limb muscles are also exerted through reticulospinal pathways which will not be described here. But it must be reminded that recent studies combining electrophysiological recordings and HRP stainings have described reticulospinal neurons (RSN) controlling eye-head synergy for orienting in the cat (33, 34). Both reticulospinal and vestibulospinal pathways would be therefore implicated in eye-head coordination by means of a combination of stabilizing

reflexes and of orienting movements. Neuronal networks subserving these two functions interact and have some common elements. Vestibulo-ocular and vestibulospinal reflexes are inhibited or suppressed during voluntary movements. Such suppression, which is required during orienting behavior, could result from cortical and/or reticular influences on the VN complex.

Effects of vestibular lesions on posture and gait.

The unilateral or bilateral ataxia resulting from peripheral receptor lesion (labyrinthectomy) or vestibular nerve lesion (vestibular neurectomy) has been studied thoroughly in experiments on animals of various species. This ataxia, including a static vestibular syndrome and a phasic vestibular syndrome, is classic (cf. 70 for a review) and bears witness to the functional role of the SCC and otolith receptors in postural control, balance, orientation in space and stabilization of gaze (43, 44, 10, 23, 70, 78). Recent observations of spacenauts during orbital flights (21) in microgravity demonstrate, in particular, the important role of the afferents originating in the utricular and saccular maculae in maintaining tonic postural activity in the antigravity muscles (Fig. 4). Weightlessness creates a functional deafferentation of the vestibular nuclei comparable in some respects to the deafferentation resulting from pathological disorders or experimental lesions in the labyrinth receptors.
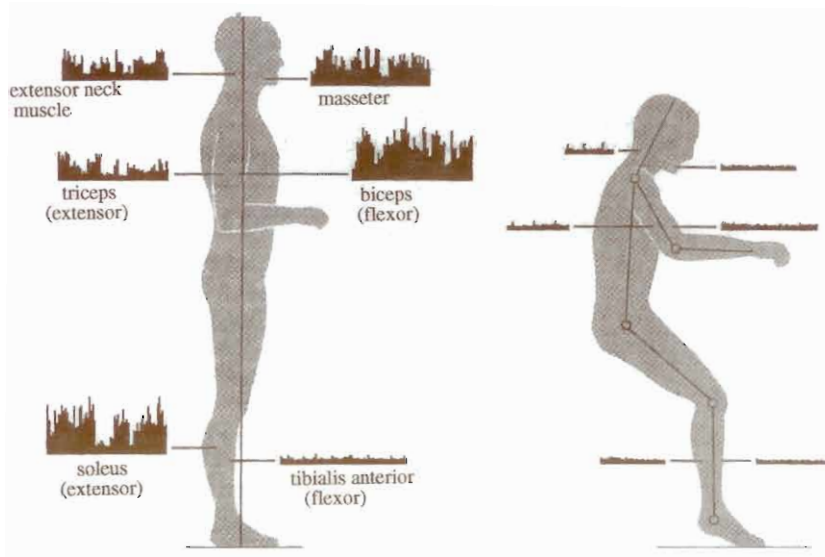

Fig. 4 – *Influence of gravity on postural activity in man.*

Activity of different muscle groups, including the muscles which support the body against gravity, while standing on earth (A) and resting in weightlessness (B) (adapted from Lestienne, 47).

2.

However, experimental findings are scarce for the postural effects of central vestibular lesions involving either the vestibular nuclei as a whole or, more specifically, a single vestibular nucleus. It seems however that compensation of unilateral vestibular deficits requires the functional integrity of the VN complex located on the side of the lesion (36). Partial studies in the cat (17, 20) and more systematic studies in monkey (72) have led to formulation of a few notions tending to clarify somewhat the respective role of the different VN in postural equilibration. The various clinical pictures seen following specific vestibular nuclei lesions in monkey are shown in Figure 5.

1. *Lateral vestibular nucleus lesions.* — In monkeys, a unilateral lesion in the ventral part of Deiters' nucleus results in postural deficits more severe than those
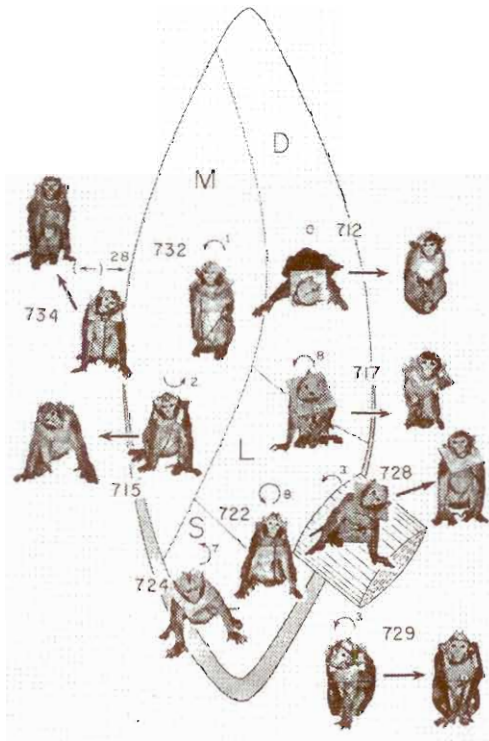

Fig. 5 – *Postural effects of vestibular nuclei lesions in the monkey.*

Photographs showing postural deficits at head and body levels observed after unilateral lesion of different zones in the vestibular nuclear complex, in the medial (M), descending (D), superior (S) and lateral (L) vestibular nuclei in the monkey. Lesions were on the left side in each animal, which place on the chart indicates the location of the lesion within the VN. Small arrows on the figures show direction of spontaneous vestibular nystagmus and duration (in days) in the light. The large arrows represent the effects of a labyrinthectomy performed on the opposite (right) side after compensation for the postural deficits occasioned by the first VN lesion. A second photograph of the same animal shows the postural syndrome resulting from this second lesion (adapted from Uemura and Cohen, 72).

resulting from a simple labyrinthectomy. They are comparable to those following a unilateral vestibular neurectomy. A marked tilt of the head to the lesioned side and a considerable asymmetry in the limbs, shown by a flexion of the ipsilateral and an extension of the contralateral limbs, appear, with repeated falls toward the side of the lesion when movements are attempted. A horizontal spontaneous nystagmus with a slight rotatory component beats toward the intact side (quick phase), lasting for 3 days in the light, one week in darkness.

Compensation for postural, locomotor and oculomotor deficits is already well advanced by the 15th day postlesion and practically complete 40 days after the lesion. Then a labyrinthectomy on the contralateral side will produce a postural syndrome which is less severe, although it is the mirror image of the one observed after the initial lesion (Bechterew's phenomenon, see 5; cf. case 728 in Fig. 5).

2. *Descending vestibular nucleus lesions.* — A lesion affecting the rostral portion of the DVN and impinging on the most ventral parts of the LVN (case 717) induces postural deficits comparable to those described above. A cephalic nystagmus is added to this static postural syndrome. Sitting up is possible one day after the lesion. The spontaneous nystagmus beating toward the intact side shows a strong upward rotatory and vertical component. It persists for 8 days in the light. Compensation for these deficits takes a little longer than for a lesion of the vestibular nerve or of the LVN (about 70 days). A contralateral labyrinthectomy in an animal which has compensated for such lesions also results in the appearance of a Bechterew's phenomenon of slight intensity.

Lesions in the central and caudal parts of the DVN produce the most severe deficits (case 712). The animal presents an intense rotation of the head toward the side of the lesion, accompanied by a rotation of the spinal column toward the intact side. Limbs are flexed on the ipsilateral side, extended on the contralateral side. Standing is impossible, as is sitting up for the first 5 days after lesion. Severe dysequilibrium and a tendency to frequent falls toward the side of the lesion are observed. No spontaneous nystagmus is present in the light. These postural and motor deficits regress over 2 to 3 months. A contralateral labyrinthectomy causes only slight mirror image deficits.

3. *Superior vestibular nucleus lesions.* — The postural syndrome resulting from near-total lesion in the SVN is the opposite of that usually observed after a unilateral vestibular system lesion (case 724). A head tilt, a tendency to fall on the side contralateral to the lesion, and a contralateral ocular nystagmus, rotatory and upwards which persists for 7 days in the light, are observed. This unusual syndrome was also reported following SVN lesion in other experiments (52).

A more extended lesion in the SVN impinging on the dorsal part of the LVN (case 722) has few postural repercussions. Uemura and Cohen (72) interpreted this absence of a postural syndrome as perhaps due to the opposite deficits induced by the LVN and the SVN lesions cancelling each other out. Ocular nystagmus, however, is of the same type. It is present for 8 days in the light and for one month in the dark.

4. *Medial vestibular nucleus lesions.* — Generally, postural deficits resulting

from damage to the MVN are not very severe when the lesioned zones are in the rostral part of the nucleus (case 732). Spontaneous ocular nystagmus remains correspondingly weak and disappears rapidly. With these lesions the head is not bent and no tendency to fall is seen. However, slight postural disturbances do appear when the MVN lesion extends laterally to the LVN (case 715). In this case, Bechterew's phenomenon is observed consecutive to destruction of the contralateral labyrinth.

Lesions located more medially, principally in the reticular formation and in the prepositus hypoglossal nucleus, produce a classic, but not severe, postural syndrome, accompanied by lateral cephalic nystagmus on the ipsilateral side and by a spontaneous ocular nystagmus, also ipsilateral, present in the dark (case 734).

In conclusion, the postural effects of lesions, more or less selective, in different regions of the VN complex confirm, for the essentials, the anatomic and physiological data on the organization of primary vestibular afferent projections and on the efferent connections of these nuclei with the spinal motoneurons and the oculomotor structures.

The most severe postural deficits are seen after lesion of the lateral and caudal parts of the VN complex, including the DVN and the LVN known for their major contribution to postural control and developing vestibulospinal reflexes. The least severe deficits are observed after lesion of the rostroventral part of the complex, including the SVN and the MVN which are reputed to have a more specific role in oculomotor control. Paradoxically, postural disturbances following lesion of the rostral and central zones of the MVN are slight, although these areas are thought to be intimately connected to the cervical spinal cord motoneurons which ensure postural control of the cephalic segment.

The direction of postural disturbances (head tilt, falls, dysequilibrium) and of spontaneous vestibular nystagmus (slow phase) would seem to furnish information on the location of lesions. On the ipsilateral side, these signs would indicate unilateral VN damage mainly affecting the lateral and caudal part of the complex or the dorsal medullary reticular formation. On the contralateral side, however, they would indicate more rostral VN lesions or damage to the pontine reticular formation.

POSTURAL CONTROL: A FUNCTIONAL COUPLING OF MULTIPLE SENSORIMOTOR SYSTEMS

The righting reflexes, the static postural adjustments, as well as the reactions involved in dynamic equilibration, call on more than one sensory modality and bring into play zones of convergence which integrate sensory information from various sources. Taken together, these processes which maintain static and dynamic posture demonstrate the existence of close cooperation between the various functional modules involved in stabilizing the eye, the head and the body in space. Two examples will illustrate this. The first is based on recent experimental data

collected in our laboratory, demonstrating the functional role in head posture control of the neuronal processes of visuo-otolith integration at the level of the Deiters' nucleus in the alert cat. The second describes earlier observations showing the coupling of neck and labyrinth reflexes for achieving the postural control of the cephalic segment.

Visuo-otolith interactions: neuronal correlates in the VN.

The first phenomenological description by Mach (49) of the feeling of motion induced in an immobile observer by a visual scene rotating about a vertical axis contributed to bring to light the kinesthetic properties of visual input. This illusive feeling of motion, described in the rotatory plane (circular vection) and in the linear plane (linear vection), are often accompanied by changes in the orientation of the subjective vertical and by postural modifications. Since the postural adjustments induced are in the opposite direction to the effects perceived, they would be compensatory. Generally, the effects of a visual stimulus on perception and movement are related to the spatial frequency of this stimulus and its velocity, with a preponderance of effects in the slow range of motion.

Functional cooperation between the vestibular and the visual modules is expressed by postural reactions which are better adapted, in terms of gain and phase, when the two corresponding types of sensory input (visual and labyrinth) are present in conjunction and congruence (56, 45, 73). Numerous experimental studies have shown that optokinetic and vestibular stimulations in opposite directions induce similar neuronal modulations at the level of second order vestibular cells. During rotatory stimulation, visual and vestibular inputs combine their properties to produce a measure of head angular velocity in a wider frequency range (74). Studies in our laboratory, using linear stimulation in the alert cat, further demonstrated a synergistic and functional convergence of otolith and visual influences. A single neuron can code both the real and the apparent movements of an animal, and this convergence of the two sensory modalities results in improved neuronal coding of motion velocity over the whole range of stimulus frequencies (Fig. 6B) (79, 80, 4). In parallel, vestibulocollic relexes show increased gain and a phase closely related to the velocity of motion (Fig. 6A), whatever the direction (X, Y or Z) of motion would be (11).

These experimental findings and the modelling based on central estimation of pertinent kinematic values, such as orientation of the gravity vector and inertial acceleration, suggest that the VN are a major center for the central reconstruction of percepts of head movement in space (24) and for the regulation of postural (and oculomotor) activities for eye-head coordination. In this brain stem structure, various sub-groups of neurons could be arranged in complementary functional modules, involved in either oculocephalic stabilization functions or in orientation tasks, the two main mechanisms for gaze control.
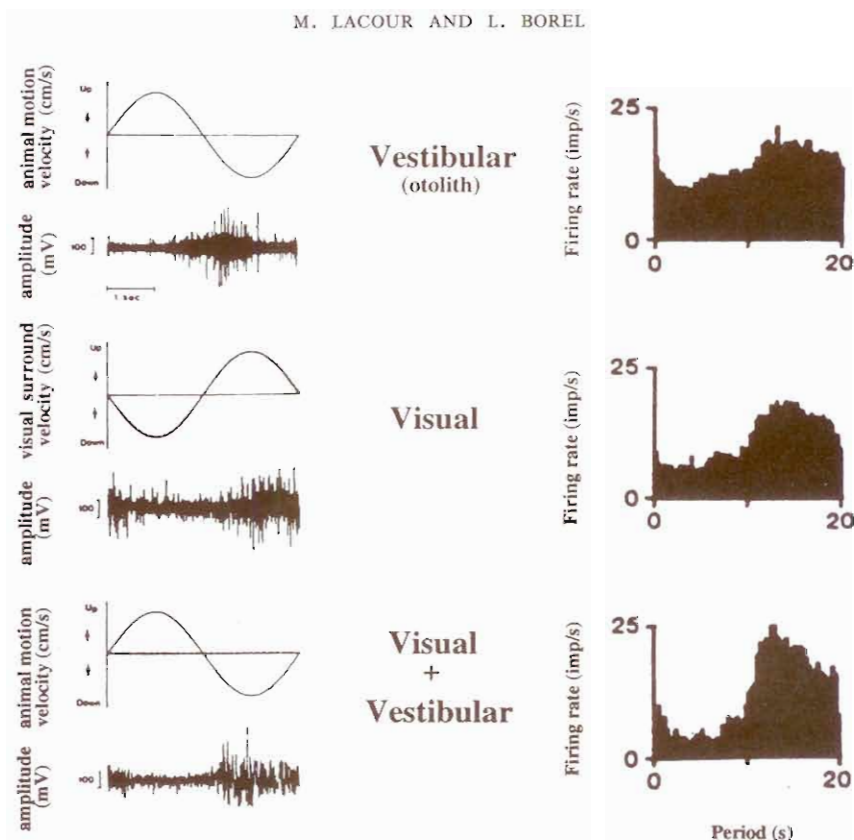
Fig. 6 – *Visuo-otolith interactions and head posture control.*

A. EMG responses evoked in the splenius capitis muscle during 3 types of stimulation — otolith, visual, and visual-otolith interaction — in the vertical plane in the alert cat. Velocity of the animal's motion (or of the visual surround) and modulation (EMG, not rectified) of neck motor responses (in mV) are shown for each of these experimental situations (stimulus frequency: 0.5 Hz).

B. Neuronal responses recorded in the lateral vestibular nucleus in the same experimental conditions as in A. Sequential histograms showing the mean modulation of the firing rate of the same single vestibular neuron (imp/sec, ordinate) during otolith, visual and visuo-otolith interaction (stimulus frequency: 0.25 Hz; abscissae).

Interaction of labyrinth and neck reflexes.

First described by Magnus (51) and later, in detail, by Roberts (66), the interaction of labyrinth and neck reflexes plays an important role in the regulation of posture and movement. Neck reflexes are easily evoked in the young child when turning or tilting the head, and they are found in adults doing tasks which require optimal postural control (Fig. 7B). They are triggered by the proprioceptive musculoarticular neck afferents looping at the segmentary level or projecting to the reticular formation and the VN. Numerous labyrinth and neck convergences have been described at the level of the vestibulospinal neurons in the cat, in particular those of the Deiters' nucleus and of the descending nucleus, after electrical (14)
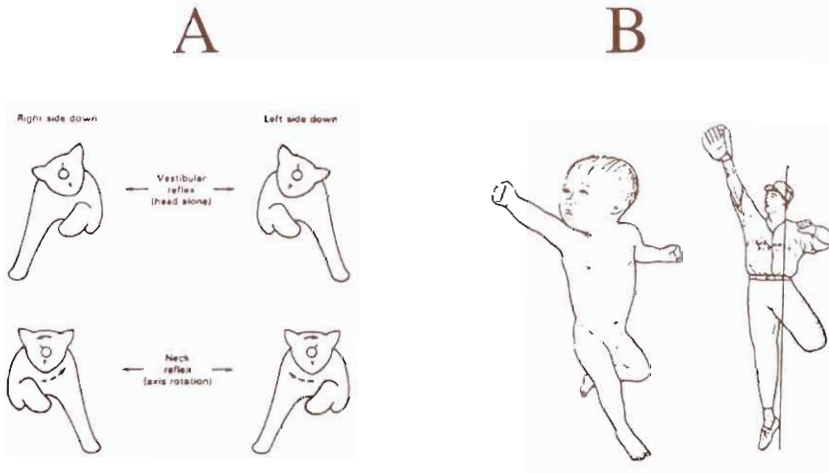
Fig. 7 – *Interaction of labyrinth and neck reflexes.*

A. Illustration of the antagonistic action of vestibular and neck reflexes on forelimb muscles in the cat (adapted from Roberts, 66, modified by Ghez 30).
B. Illustration of the neck reflexes in a very young child and in an athlete (modified by Ghez, 30).

or rotatory (12, 60) stimulation of neck afferents. These neck reflexes act not only on the muscles controlling the cephalic segment but also on the limbs.

Recent studies in the cat have demonstrated that the cervicocollic reflex (CCR) acts in perfect synergy with the vestibulocollic reflex (VCR). A summation of the compensatory effects of the CCR and VCR is observed, improving the stabilization of the cephalic segment over a very wide range of frequencies, including natural head movements (up to 5 Hz). These two subsystems which control the position of the head in space thus cooperate functionally in that major achievement: the stabilization of gaze.

In contrast, neck and labyrinth reflexes have opposite effects on limb muscles. For example, a tilt of the head toward the right in a decerebrate animal whose dorsal roots have been transsected in C1 and C2 will induce extension of the ipsilateral forelimb and flexion of the contralateral forelimb (Fig. 7 A-B: vestibular reflexes). A rotation of the body around the second cervical vertebral (neck reflexes), while functionally equivalent to the tilt noted above, does not activate the vestibular system and produces an inverse response pattern. This antagonist mode of action on the limbs by the two categories of reflex is yet another demonstration of their functional coupling in order to maintain posture and balance. Daylife activities modify constantly head position in space and induce multiple head movements which, therefore, activate neck and labyrinth reflexes. As the motor effects of these reflexes cancel out, posture remains relatively unaffected by head motion.

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