Parallel Ascending Vestibular Pathways

Anatomical Localization and Functional Specialization

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Information from the vestibular nuclei ascending through the brainstem to the oculomotor and trochlear nuclei (NIII, NIV), the interstitial nucleus of Cajal (INC), the pretectum, or thalamus, is thought to be distributed in at least five different pathways. They include the medial longitudinal fasciculus (MLF), the ascending tract of Deiters (ATD), possibly the brachium conjuctivum (BC), the crossing ventral tegmental tracts (CVTT), and a recently observed ipsilateral pathway close to the medial lemniscus, possibly the equivalent of the ipsilateral vestibulo-thalamic tract (IVTT). This short review describes the location of these ascending tracts, their function with respect to ocular motor control and perception, and their clinical relevance. There is evidence that the MLF carries mainly information from the canals to NIII, NIV, INC, and possibly the thalamus, whereas otolith signals may ascend in the CVTT, along with excitatory anterior canal connections. The evidence for BC as a specific vestibulo-oculomotor pathway is weak and could be the result of the initial observations of CVTT. The ATD carries mostly ipsilateral otolithic information to the medial and inferior recti subgroups in NIII and the Edinger-Westphal nucleus. In the rostral pons an ipsilateral vestibular pathway was seen lying close to the medial lemniscus. This anatomical projection could be the equivalent of the IVTT, bypassing the ocular motor centers and projecting to the thalamus. The IVTT mediates perception of verticality and may be part of a fast three-neuron vestibulo-thalamo-cortical pathway, which provides the multisensory cortical system for spatial orientation and self-motion-perception with information about head acceleration.

Key words: **medial longitudinal fasciculus; brachium conjuctivum; ascending tract of Deiters; crossing ventral tegmental tract; ipsilateral vestibulo-thalamic tract; Y-group; subjective visual vertical; monkey; human**

Introduction

Vestibular projections arising from the semicircular canals and the otoliths are processed in a brainstem network to subserve ocular motor functions (e.g., the vestibular-ocular reflex and eye coordination in roll plane) and a thalamocortical network to contribute to multisensory perceptive functions (e.g., spatial orientation and motion perception).

The vestibulo-ocular reflex (VOR) is generated by a fast three-neuron link between vestibular receptors, secondary neurons in the vestibular nuclei, and eye-muscle motoneurons. A major pathway carrying these signals is the medial longitudinal fasciculus (MLF), and

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many studies have provided data concerning canal signals in this tract.[1–3] However, the pathways by which otolith signals reach the oculomotor neurons are not so clear.[4] Stimulation of otolith nerves evoked postsynaptic potentials in most extraocular motoneurons.[5,6] Only the utriculus has a direct monosynaptic and a disynaptic input to abducens motoneurons.[7,8] The VOR is supported by several other vestibulo-oculomotor pathways, some of which are secondary, such as the ascending tract of Deiters (ATD), and others of which are nonsecondary, for example the pathway from the dorsal Y-group, which uses a pathway crossing in the basal pontine tegmentum to project to upward-moving eye-muscle motoneurons. There is evidence for otolith and canal signal input to integration centers for eye–head coordination in the roll plane, namely the interstitial nucleus of Cajal (INC) in the midbrain tegmentum.[9]

The route of vestibular projections from the brainstem to the thalamus, and the contribution of otolith and canal signals to it, has not yet been defined in detail. The ascending tract of Deiters and the crossing ventral tegmental tract (CVTT) have been suggested as possible routes for ipsilateral and contralateral vestibulo-thalamic projections, respectively[10,11] In this article we review the anatomy of the ascending vestibular pathways, with the addition of some new observations, and relate the data to their function and clinical significance.

Methods

In this article ascending vestibular pathways in monkeys have been visualized using tritiated leucine autoradiography, a highly selective tract-tracing method.[12] The major advantage of this technique is that [^{3}H]-leucine is only taken up by somata and not by axons; therefore unlike more popular tract tracers (wheatgerm horseradish peroxidase complex, phaseolus agglutinin, or cholera toxin subunit B), there is no spurious labeling due to uptake of the tracer by fibers of passage at the injection site, for example in the adjacent brachium conjuctivum (BC). One disadvantage of autoradiography is that it is not as sensitive as some more modern methods; therefore, weak projections may remain undetected. Unlike proline, leucine is not transported transynaptically.

The ascending pathways from the vestibular nuclei were traced using autoradiography. Macaque monkeys received [^{3}H]-leucine injections into the vestibular complex (H33, H19, and A4–90, *M. mulatta*; A83-B1 *M. fascicularis*) under general anesthesia and aseptic conditions. All experimental procedures conformed to the state and university regulations on Laboratory Animal Care, including the Principles of Laboratory Animal Care (NIH Publication 85–23, Revised 1985), and were approved by their Animal Care Officers and Institutional Animal Care and Use Committees. After a survival time of 2–3 weeks, the animals were sacrificed with an overdose of Nembutal (80 mg/kg body weight) and transcardially perfused with 0.9% saline (35°C) followed by 2% paraformaldehyde with 1% glutaraldehyde in 0.1 M phosphate buffer solution (pH 7.4). The brains were cut at 40 μm on a freezing microtome. The sections were mounted and treated with NTB-2, nuclear track emulsion exposure of 4 or 8 weeks, developed in Kodak D-19 developer, and counter-stained with cresyl violet. For further details see Wasicky and colleagues (2004).[13]

The sections were analyzed under the microscope using bright- and dark-field illumination. Brightness and contrast were enhanced as needed, and maximum intensity projections were generated for visualization. All images taken were converted into gray-scale mode (Adobe Photoshop) and then arranged and labeled with drawing software (CorelDraw).

Results

The injection site of H33 lay in superior vestibular nucleus (SVN) and medial vestibular nucleus (MVN), mainly in the magnocellular

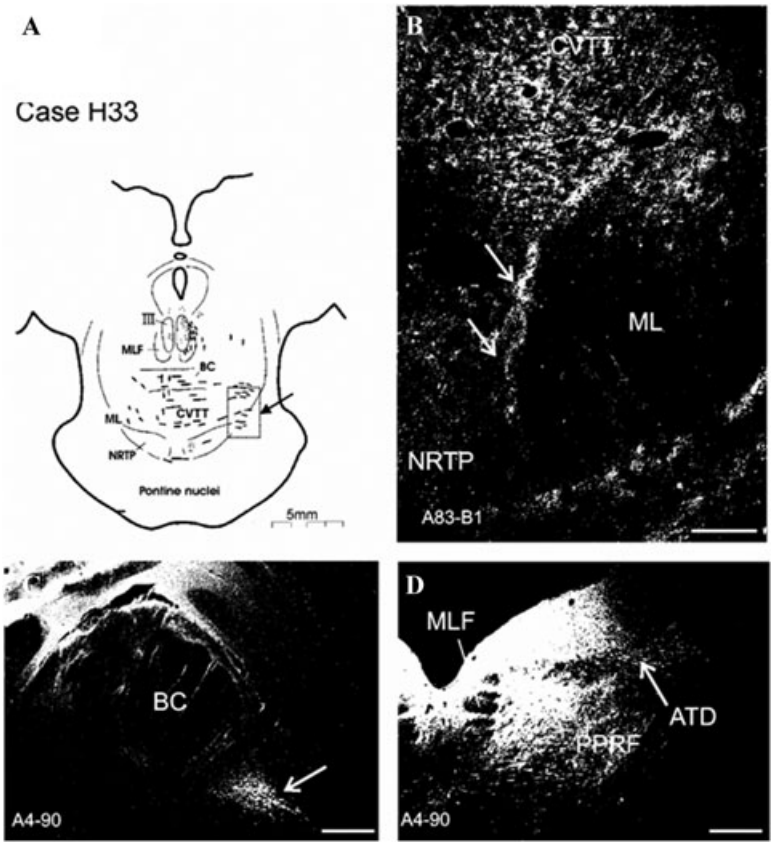

Figure 1. Labeled pathways in the brainstem after leucine injections into vestibular complex. (**A**) Labeled pathways at the level of NIII: note fibers crossing in BC, CVTT, and those adjacent to ML. (**B**) Photomicrograph of the area shown in A. Arrows indicate vestibular fibers ipsilateral to the injection lying mainly adjacent to ML, possibly IVTT. (**C**) After injection into the Y-group a group of ascending fibers (arrow) lie ventral to BC but not in it. (**D**) The ATD and the MLF are indicated by arrows: the fibers crossing the midline are related to efferents of the cochlear nucleus, which was also included in the leucine uptake area. Calibration bars 250 μm.

subdivision, whereas that of H19 lay more lateral and dorsal in SVN and lateral vestibular nucleus (LVN). The injections in cases A4–90 and 83-B1 were centred on the Y-group, and in the latter case included dorsal SVN. Case 83-B1 also involved the cerebellar nuclei to a minor extent, but their involvement in case H33 is unclear. An autoradiographic analysis of the brainstem sections showed that the following ascending pathways were labeled:

Medial longitudinal fasciculus was labeled ipsilaterally and strongly in H33 (Fig. 1A), weakly both ipsi- and contralaterally in H19 (not shown[13]), and only contralateral in A83-B1. No MLF labeling was seen from the Y-group injection in A4–90 (Fig. 1D).

Ascending tract of Deiters was strongly labeled after the H19 injection into lateral vestibular complex, and axon terminals were found over the ipsilateral medial and inferior rectus oculomotor subgroups and the Edinger-Westphal nucleus. The ATD was more weakly labeled in A4–90 (Fig. 1D).

Crossing ventral tegmental tracts were most strongly labeled from H33, H19,

and to some extent in A4–90. However the fibers are difficult to see, perhaps due to their small diameter and scattered arrangement.

Brachium conjuctivum was slightly labeled in 83-B1 and H33. In both of these cases some cerebellar nuclei uptake may have occurred.

The **ipsilateral vestibulo-thalamic tract** was only clearly labeled in 83-B1. The whole bundle of fibers (Fig. 1C, arrow) in the caudal pons ventral to BC did not all cross the midline. A subset was seen to collect along the border of the ipsilateral medial lemniscus (ML) just lateral to nucleus reticularis tegmenti pontis (NRTP) (see arrows, Fig. 1B). A few fibers lay inside the ML. The labeled fibers moved laterally with the ML but could not be followed further rostrally for technical reasons.

Discussion

The vestibular system processes information by means of a multilevel sensorimotor network that subserves ocular motor, postural (not discussed here), and perceptual functions. This functional dichotomy is reflected by parallel vestibular pathways that target 1) the nuclei of the ocular motor system and 2) the multisensory thalamic and cortical areas involved in motion perception and spatial orientation. The current knowledge on ascending vestibular pathways comes both from tracer injection studies in several species, including the primate, and from clinical lesion studies showing vestibular dysfunction by ophthalmological and psychophysical measurements. These two perspectives have not been systematically brought together, although this "translational" approach seems most promising for the following reasons. The anatomical view to the ascending vestibular pathways is capable of characterizing the origin of the vestibular input (canals and otolith organs) and the target structures (including ocular motor centers and thalamus). The clinical view includes functional aspects related to these pathways. The sensitive clinical signs of unilateral brainstem damage involving vestibular pathways are: 1) Pathological ocular torsion (OT) and skew deviation (SD), indicating imbalance of vestibular input to the midbrain integration centers for eye movements in the roll plane (vestibular–ocular-motor dysfunction).[9] 2) Deviation of the perceived visual vertical, which indicates an imbalance of vestibular input from the vestibular endorgans (mostly otoliths) to the multisensory thalamocortical areas (vestibular–perceptive dysfunction).[9] Lesions at different brainstem levels and localizations show a highly characteristic "fingerprint" of vestibular–ocularmotor and vestibular–perceptive dysfunction, which can be used for functional "pathway mapping."

In the following we will characterize four ascending vestibular pathways on the basis of combined anatomical and clinical evidence and speculate on their functional relevance (Fig. 2).

Medial Longitudinal Fasciculus

The secondary *vertical* canal vestibular projections to trochlear nucleus (NIV) and oculomotor nucleus (NIII) from medial vestibular nucleus, pars magnocellularis (MVNm) utilize the MLF, and were labeled in these experiments. The cells of origin can be classified as "*posterior canal* vestibulo-oculomotor," "*posterior canal* vestibulo-oculomotor-spinal," and "*anterior canal* vestibulo-oculomotor-spinal." The category *anterior canal* vestibulo-oculomotor where not found in this MVN region; but they have been found in SVN and utilize the CVTT (see below) to reach NIII. The excitatory oculomotor afferents in MVNm use the contralateral MLF, whereas the inhibitory vertical canal secondary vestibular neurons use the ipsilateral MLF.[2,3] These secondary vestibular cells lie in the central magnocellular region of the superior vestibular nucleus.[2] Therefore, anatomically the MLF represents a VOR

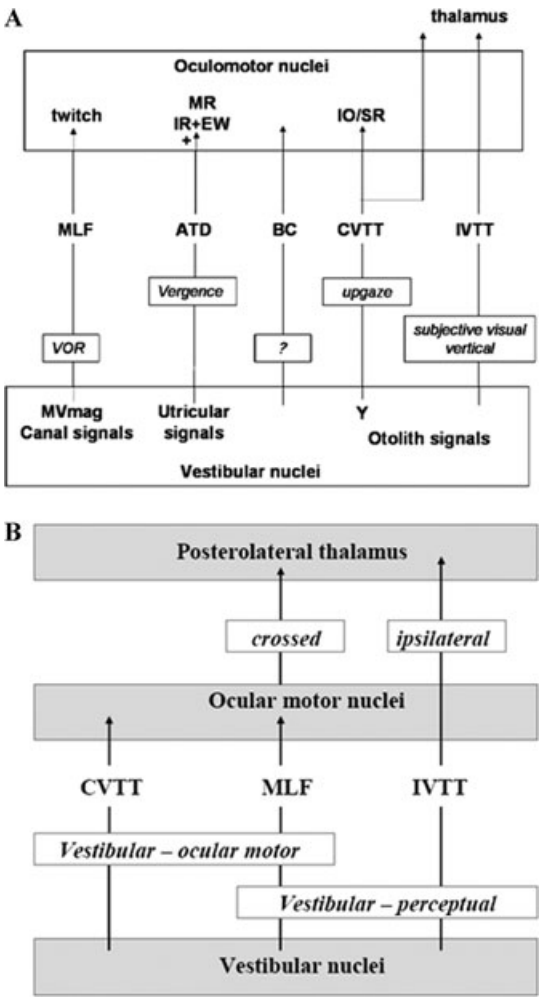

Figure 2. (**A**) Ascending parallel vestibular pathways based on anatomical labeling studies. (**B**) A map of vestibular pathways based on evidence from clinical neuro-opthalmological and psychophysical examination and lesional studies.

pathway, and dysfunction of the vertical VOR has been described clinically with unilateral MLF lesions.[14] A recent study in patients with unilateral internuclear ophthalmoplegia (INO) due to circumscribed lesions of the MLF additionally showed contralesional ocular tilt reaction (i.e., subjective visual vertical [SVV] deviation, OT, SD) in more than 90% of cases (INO plus syndrome).[15] This indicates that the MLF also carries vestibular–ocularmotor information to the rostal midbrain tegmental centers for eye–head coordination (INC) and vestibular–perceptive information to supratentorial centers (e.g., the posterolateral thalamus). Indeed, a vestibular projection through the MLF to the INC as well as sparse fibers ascending up to the thalamus have been seen in primates.[11,16] The concept of canal signals subserving vestibular-perceptive function is in agreement with experimental data in the monkey, which show convergence of information from the vertical canals and otoliths at the level of the vestibular nuclei and the posterolateral thalamus to segregate perception of tilt and translation (graviceptive information).[17] In conclusion, we propose that the MLF includes crossed excitatory vestibulo-ocularmotor pathways (VOR, eye–head coordination in roll) and crossed vestibulo-perceptive pathways (motion perception, verticality) (Fig. 2B).

Ascending Tract of Deiters

The ATD lies lateral to MLF (Fig. 1D). The cells of origin lie in the lateral part of the vestibular complex but have so far not been recognized as a cyto-architectural or histochemical cell group. The cells were here shown to project ipsilaterally to both inferior and medial rectus subgroups of NIII as well as the Edinger–Westphal complex (accommodation). This observation has not been reported before. Furthermore it strongly supports the hypothesis of Chen-Huang and McCrea[18] that the ATD carries signals modulating vergence. In their experiments, individual ATD neurons were found to be sensitive to linear acceleration and their activity was related to viewing distance. From labeling studies it has been hypothesized that scattered ATD fibers from the rostral ocularmotor nucleus may turn laterally to reach the ipsilateral thalamus through the H1 field of Forel.[11] In cats the ATD has been reported to contribute to ipsilateral vestibulo-thalamic projections.[10]

Clinical assessment of ATD functions is limited by the fact that isolated lesions of the ATD are never observed because of damage of the adjacent MLF (see also Fig. 3).

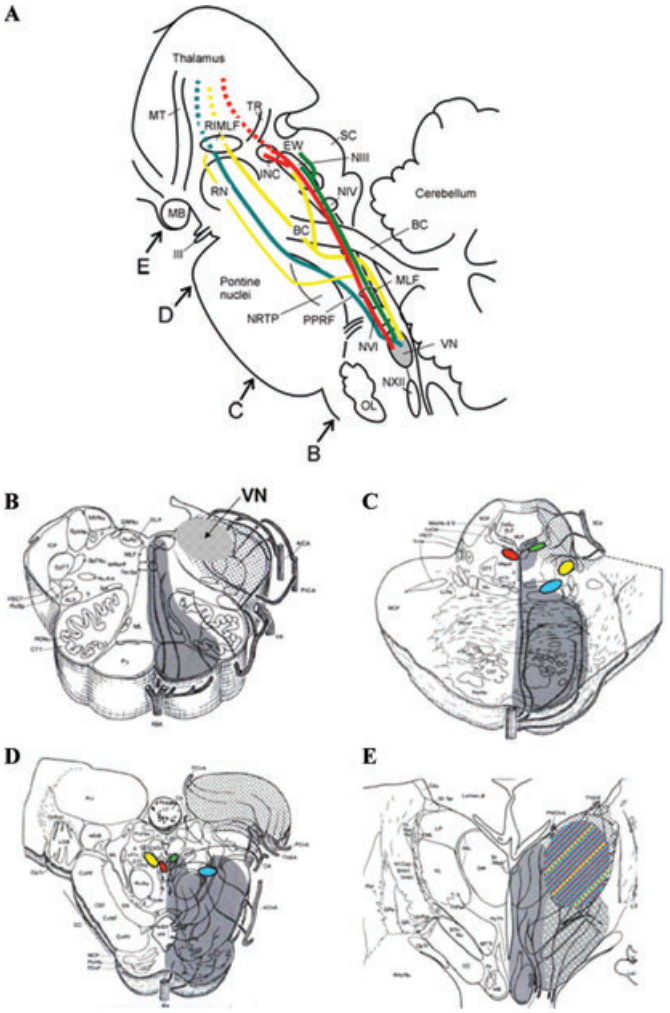

Figure 3. A map of ascending vestibular pathways in the brainstem concluded from comparative analysis of anatomical studies in the monkey and clinical lesion studies in human. (**A**) Four major parallel ascending pathways are indicated in four colors in projection to an axial brainstem view. The crossed pathway in the MLF (red), carrying mostly canal information from the medial vestibular nucleus to NIII, NIV, INC, and rostral interstitial nuclei of the medial longitudinal fascicle (ri*MLF*) and probably also to the thalamus. Note the crossed pathway of loosely scattered fibers in the ventral tegmentum, the crossing ventral tegmental tract (CVTT) (orange), coming up from the superior vestibular nucleus and the Y-group, transmitting information from the anterior canals to NIII and possibly otolith input to the thalamus. The ipsilateral ascending tract of Deiters (green), carrying otolith information to the medial rectus and EW. A newly described ipsilateral tract at the medial edge of the medial lemniscus (blue), which most likely directly travels to the posterolateral thalamus (ipsilateral vestibulo-thalamic tract, IVTT). (Dashed lines indicate putative projections concluded from clinical evidence.) (**B–E**) Transverse sections at medullary, midpontine, mesencephalic, and thalamic levels (arrows), with the ascending vestibular tracts (same colors as in **A**). The area of the posterolateral thalamus is scattered red, blue, and yellow to indicate convergence of information from different ascending pathways. Crossed pathways are depicted on the left half, uncrossed on the right half, of the slices. On the right half also the vascular territories are indicated to give information about how the ascending vestibular tracts are involved in clinical vascular brainstem syndromes. Clinically most important from this standpoint are paramedian dorsal tegmental lesions involving the MLF (giving ispilateral INO and contralateral OTR) and anteromedial lesions involving the ML (giving isolated ipsilateral subjective visual vertical tilt).

However from a clinical standpoint there is no evidence for vestibulo-perceptive information in the roll plane traveling with the ATD, as no case of ipsilateral deviation of SVV with lesions of the dorsal paramedian pontomesencephalic tegmentum has been found in case series of hundreds of patients.[9,15] It may be, that such ipsilateral SVV tilts exist but are masked by the more prominent contralateral SVV deviations caused by the concomitant MLF damage. In conclusion, the ATD seems to be mostly a vestibulo-oculomotor projection important for vergence eye movements (Fig. 2).

Crossing Ventral Tegmental Tracts

Our experiments confirmed that fiber pathways originating from excitatory anterior canal neurons in the SVN[19] and in the Y-group[20] do not travel in midline fiber tracts but have axons that collect laterally beneath the BC, then cross in a broadly scattered band, CVTT. We found some fibers passed below NRTP and others within the pontine nuclei even more ventrally. No significant labeling was found within BC. The original evidence for the BC as a specific vestibulo-oculomotor pathway is based on lesion studies in rabbit,[21] and could have included or compromised the (then-unknown) CVTT. However the vestibulo-oculomotor fibers do pass through BC at right angles, turning dorsal at a sharp angle toward NIII, and innervating the upward-moving eye-muscle motoneurons, inferior oblique, and superior rectus, contralaterally.

The CVTT carries crossed anterior canal fibers from the superior vestibular nucleus to NIII supporting upward eye movements.[19] Bilateral lesions of the CVTT have been reported in the context of upbeat nystagmus, indicating an importance for the balance of upward and downward eye movements.[22] Projections through the CVTT to the thalamus have been traced anatomically.[11] SVV deviations with unilateral CVTT lesions have not been registered, but it may be that single cases of more extensive dorsal tegmental brainstem lesions, which show contralesional SVV deviation,[15] arise from a combined MLF and CVTT lesion (Fig. 2).

The Ipsilateral Vestibulo-thalamic Tract

A group of fibers at the medial edge of the medial lemniscus originates from fiber tracts ascending out of the Y-group and can be followed to the midbrain (Fig. 1C, arrow). The projections almost certainly contain otolithic signals. Although these fibers, called functionally the IVTT,[23] have not been systematically followed rostrally into the thalamus, a thalamic connection is highly likely from a clinical standpoint. In 14 patients with anteromedial pontomesencephalic infarction, an ipsilateral deviation of the SVV was observed without ocular motor signs of dysfunction.[23] The overlap zone of lesions as drawn from thin-slice MRI images was located at the medial edge of the medial lemniscus and could be followed from the level of the superior vestibular nucleus to the midbrain. The finding of purely vestibulo-perceptive dysfunction is highly suggestive of a primary uncrossed ipsilateral vestibulo-thalamic tract that bypasses the ocularmotor nuclei, reaching probably the posterolateral thalamus via the same route as the medial lemniscus.[23] The posterolateral thalamus has been shown to be the target of bilateral vestibular projections in primates[11,24,25] and humans (lesion and PET studies).[9,26] In the squirrel monkey vestibular-sensitive neurons were found in the posterolateral thalamus, which were activated with a latency of about 4 msec by electrical stimulation of the ipsilateral vestibular nucleus.[25] This indicates the existence of a direct ipsilateral vestibulo-thalamic projection. Also in humans a trisynaptic vestibulo-thalamo-cortical projection was assumed on the basis of recordings of cortical short latency vestibular potentials (10 msec) following repetitive galvanic stimulation of the ipsilateral vestibular nerve.[27,28]

The IVTT could be the equivalent of just such an ipsilateral vestibulo-thalamic

projection, and may represent a fast vestibular track serving as a fast transition of vestibular information to the thalamus and cortex during head or body movements, which provides the cortical multisensory network for perception of body motion and spatial orientation with information about head acceleration and position. This information would be important for distinguishing the components of self-motion in different reference frames (eye-, head-, and body-based). In this context it is of interest that most vestibular-sensitive neurons in the posterolateral thalamus show mixed otolithic and semicircular canal input, which is important for differentiating translational motion and gravitational acceleration (tilt) of the head.[24] Responses to translations to one side (activating ipsilateral otolithic afferents) and tilt to the contralateral side (activating contralateral canal afferents) add up in neuronal response of vestibular-sensitive thalamic neurons.[24] It is interesting that there is a close similarity between these clinical observations and the anatomy of parallel ascending vestibular pathways to the thalamus: information from ipsilateral otolith (e.g., IVTT) and contralateral canal projections (e.g., MLF, CVTT), which converge in the thalamus and could both functionally contribute to perception of motion and verticality (graviceptive information) (Fig. 3). The existence of crossed and uncrossed vestibular pathways to the thalamus is in accordance with earlier findings that electrical stimulation and lesions by circumscribed infarctions in the area of the posterolateral thalamus can cause ipsi- or contraversive perceptive tilts.[29]

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Graf, W.M. and K. Ezure. 1986. Morphology of vertical canal related second order vestibular neurons in the cat. *Exp. Brain Res.* **63:** 35–48.
2. Büttner-Ennever, J.A. 1992. Patterns of connectivity in the vestibular nuclei. *Ann. N. Y. Acad. Sci.* **656:** 363–378.
3. Uchino, Y., N. Hirai, S. Suzuki and S. Watanabe. 1981. Properties of secondary vestibular neurons fired by stimulation of ampullary nerve of the vertical, anterior or posterior, semicircular canals in the cat. *Brain Res.* **223:** 273–286.
4. Büttner-Ennever, J.A. 1999. A review of otolith pathways to brainstem and cerebellum. *Ann. N. Y. Acad. Sci.* **871:** 51–64.
5. Hwang, J.C. and W.F. Poon. 1975. An electrophysiological study of the sacculo-ocular pathways in cats. *Jap. J. Physiol.* **25:** 241–251.
6. Isu, N., W. Graf, H. Sato, *et al*. 2000. Sacculo-ocular reflex connectivitiy in cats. *Exp. Brain Res.* **131:** 282–268.
7. Uchino, Y., M. Sasaki, H. Sato, *et al*. 1996. Utriculoocular reflex arc of the cat. *J. Neurophysiol.* **76:** 1896–1903.
8. Lang, W. and S. Kubik. 1979. Primary vestibular afferent projections to the ipsilateral abducens nucleus in the cat. *Exp. Brain Res.* **37:** 177–181.
9. Brandt, T. and M. Dieterich. 1993. Skew deviation with ocular torsion – a vestibular brainstem sign of topographic diagnostic value. *Ann. Neurol.* **33:** 528–534.
10. Maciewicz, R., B.S. Phipps, J. Bry and S.M. Highstein. 1982. The vestibulothalamic pathway: contribution of the ascending tract of Deiters. *Brain Res.* **252:** 1–11.
11. Lang, W., J.A. Büttner-Ennever and U. Büttner. 1979. Vestibular projections to the monkey thalamus: an autoradiographic study. *Brain Res.* **177:** 3–17.
12. Künzle, H. 1989. Autoradiographie. In *Romeis Mikroskopische Technik*. P. Böck, Ed. 263–287. Urban und Schwarzenberg. München, Wien, Baltimore.
13. Wasicky, R., A.K.E. Horn and J.A. Büttner-Ennever. 2004. Twitch and non-twitch motoneuron subgroups of the medial rectus muscle in the oculomotor nucleus of monkeys receive different afferent projections. *J. Comp. Neurol.* **479:** 117–129.
14. Cremer, P.D., A.A. Migliaccio, G.M. Halmagyi and I.S. Curthoys. 1999. Vestibular-ocular reflex pathways in internuclear ophthalmoplegia. *Ann. Neurol.* **45:** 529–533.
15. Zwergal, A., C. Cnyrim, V. Arbusow, *et al*. 2008. Unilateral INO is associated with ocular tilt reaction in pontomesencephalic lesions: INO plus. *Neurology* **71:** 590–593.

16. Shiroyama, T., T. Kayahara, Y. Yasui, *et al*. 1999. Projections of the vestibular nuclei to the thalamus in the rat: a Phaseolus vulgaris leucoagglutinin study. *J. Comp. Neurol.* **407:** 318–332.
17. Green, A.M. and D.E. Angelaki. 2004. An integrative neural network for detection of inertial motion and head orientation. *J. Neurophysiol.* **92:** 905–925.
18. Chen-Huang, C. and R.A. McCrea. 1998. Viewing distance related sensory processing in the ascending tract of deiters vestibulo-ocular reflex pathway. *J. Vestib. Res.* **8:** 175–184.
19. Uchino, Y., M. Sasaki, N. Isu, *et al*. 1994. Second-order vestibular neuron morphology of the extra-Mlf anterior canal pathway in the cat. *Exp. Brain Res.* **97:** 387–396.
20. Sato Y. and T. Kawaski. 1987. Target neurons of floccular caudal zone inhibition in Y-group nucleus of vestibular nuclear complex. *J. Neurophysiol.* **57:** 460–480.
21. Yamamoto, M., I. Shimoyama and S.M. Highstein. 1978. Vestibular nucleus neurons relaying excitation from the anterior canal to the oculomotor nucleus. *Brain Res.* **148:** 31–42.
22. Pierrot-Deseilligny, C. and D. Milea. 2005. Vertical nystagmus: clinical facts and hypotheses. *Brain* **128:** 1237–1246.
23. Zwergal, A., J.A. Büttner-Ennever, T. Brandt and M. Strupp. 2008. An ipsilateral vestibulo-thalamic tract adjacent to the medial lemniscus in humans. *Brain* **131:** 2928–2935.
24. Meng, H., P.J. May, J.D. Dickman and D.E. Angelaki. 2007. Vestibular signals in the primate thalamus: properties and origins. *J. Neurosci.* **27:** 13590–13602.
25. Marlinski, V. and R.A. McCrea. 2008. Activity of ventroposterior thalamus neurons during rotation and translation in the horizontal plane in the alert squirrel monkey. *J. Neurophysiol.* **99:** 2533–2545.
26. Dieterich, M., P. Bartenstein, S. Spiegel, *et al*. 2005. Thalamic infarctions cause side-specific suppression of vestibular cortex activations. *Brain.* **128:** 2052–2067.
27. de Waele, C., P.M. Baudonniere, J.C. Lepecq, *et al*. 2001. Vestibular projections in the human cortex. *Exp. Brain Res.* **141:** 541–551.
28. Fukushima, K. 1997. Corticovestibular interactions: anatomy, electrophysiology, and functional considerations. *Exp. Brain Res.* **117:** 1–16.
29. Dieterich, M. and T. Brandt. Thalamic infarctions: differential effects on vestibular function in the roll plane (35 patients). *Neurology* **43:** 1732–1740.