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Disconjugate Eye Movements

Dominik Straumann

Neurology Department, Zurich University Hospital, Zurich, Switzerland

Abstract

To foveate targets in different depths, the movements of the two eyes must be disconjugate. Fine measurements of eye rotations about the three principal axes have demonstrated that disconjugate eye movements may appear not only in the horizontal, but also in the vertical and torsional directions. In the presence of visual targets, disconjugate eye movements are driven by the vergence system, but they may also appear during vestibular stimulation. Disconjugate eye movements are highly adaptable by visual disparities, but under normal condition the effects of adaptation only persist when one eye is covered. Finally, disorders of the brainstem and cerebellum may lead to abnormal disconjugate eye movements that are often specific for the topography of the lesion. This chapter reviews the literature on the phenomenology of disconjugate eye movements over the last 15 years.

The goal of normal disconjugate eye movements is to direct the corresponding retinal points of the two eyes to a visual object that is nearer or farther than the previous object. Such vergence movements can also be smooth when the object of interest moves slowly in depth. Both disparity and accommodation-vergence synkinesis can drive vergence movements. Recently, it has also been shown that perceived depth alone elicits vergence eye movements [1].

Geometrically, binocular movements are disconjugate, if amplitude and/or direction are unequal for both eyes. Considering the full kinematics of eye rotations, the term ‘direction’ includes ocular rotation about the line of sight, which is an important degree of freedom to ensure extrafoveal retinal correspondence. If one takes into account the rigid geometric specifications for 3-D – i.e. horizontal, vertical, and torsional – binocular rotations, it is not surprising that normal eye movements are generally disconjugate when subjects view near targets. As we shall see, even eye movements for foveation of targets at infinity exhibit some disconjugacy due to neural and mechanical factors.

This paper reviews the literature on the phenomenology, including pathophenomenology, of disconjugate eye movements over the last 15 years.

Horizontal Vergence Movements

Under natural viewing conditions, horizontal vergence movements are usually dysmetric, i.e. moving gaze from a near to a far target leads to excessive convergence, and moving gaze from a far to a near target to insufficient convergence [2]. The degree of this physiological vergence weakness can be reduced by increased attention [3] and instruction [4], but vergence is always less precise than version [5]. In subjects with strong monocular preference, vergence movements are typically associated with small horizontal saccades [6].

Upon symmetric step stimulation with horizontal disparity, convergence is usually faster than divergence [7]. While the dynamics of convergence movements is independent of target location, divergence movements become faster the closer the initial target is to the eyes [8]. When visual feedback is eliminated during vergence, the position trajectories are step-like, not smooth. This open-loop response consists of a pulse-like or transient component and a step-like or sustained component [9, 10]. While both components are adaptable, only the pulse-like component influences the dynamics of the adapted vergence response [11]. Experiments eliciting vergence movements by velocity steps of horizontal disparities suggest that the vergence open-loop response may originate from monocular visual pathways [12].

Disparity-driven convergence eye movements frequently show large asymmetries, which vary from trial to trial and are usually compensated in the later phase of the convergence movement [13]. While this later phase probably uses visual feedback, the initial phase seems to be preprogrammed [14]. The occasional appearance of two closely spaced high-velocity vergence movements in response to disparity supports the notion that the initial vergence component is evoked by an internal, not visual, feedback mechanism that is switched on and off, analogous to the saccadic system [15, 16].

Small or large dichoptic displays that are counterphasically oscillated in the horizontal direction elicit dynamic convergence/divergence [17]. Brief horizontal (or vertical) disparity steps of 2° or less evoke short-latency vergence movements, which are enhanced when the stimulus is presented shortly after a saccade [18–20]. Similar vergence movements with short latencies are also driven by radial flow [21]. When vergence movements with or without accompanying saccades are elicited with a gap period before target onset, vergence latency decreases significantly [22].

Vertical Vergence Movements

A vertical prism placed in front of one eye induces divergent eye movements in the vertical direction. Training with vertical prisms can increase the vertical fusional amplitude, predominantly by enhancing the motor, not the sensory component [23]. The motor capability to fuse vertical disparities increases with convergence. This increase is due to the motor component, while the sensory component for far and near viewing is practically the same [24]. Similarly, skew deviation associated with static counterroll (intorting eye hypertropic) increases with convergence [25].

Vertical fusion is accompanied by conjugate torsion toward the higher eye [26], a pattern that qualitatively resembles the one seen in patients with dissociated vertical deviation [27]. Whether the binocular torsion associated with vertical fusion is mediated by the superior oblique muscles (SO) [26] or is of central origin [28] remains to be answered. 3-D eye movement trajectories during vertical fusion suggest that patients with congenital trochlear nerve palsy use predominantly the vertical recti, while patients with acquired trochlear nerve palsy show various patterns of vertical and oblique eye muscle activations [29].

Dichoptic counterphasic oscillation of displays in the vertical direction elicits vertical vergence [30]. These movements show increased gain and reduced phase lag with larger stimulus diameter, which contrasts horizontal dichoptic display oscillation, in which display diameter is less important [31].

Cyclovergence

Spontaneous fluctuation of torsional eye position is generally conjugate, i.e. cyclovergence is considerably more stable than cycloversion [32]. Opposite cyclorotation of the images presented to the two eyes evokes static cyclovergence, which adds to the eye position-dependent cyclovergence [33]. The latter results from the outward rotations of Listing's planes during convergence (see below).

Dynamic cyclovergence can be elicited by fusible visual patterns projected to each eye separately and oscillated out of phase in the frontal plane [34, 35]. The gain of dynamic cyclovergence is highest for low frequencies and low amplitudes and therefore is appropriate to correct for drifts in binocular stereoscopic alignment, which are both slow and small [35]. Occlusion of the central area does not influence the gain of cyclovergence, although the gain of cycloversion decreases [36].

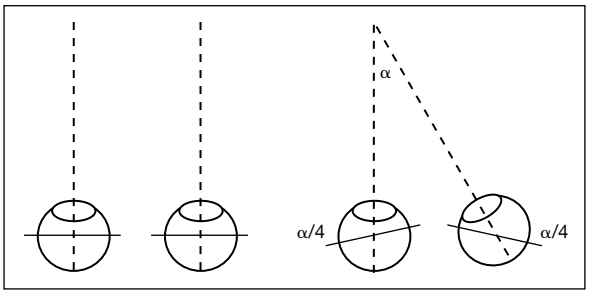

Fig. 1. Top view of binocular Listing's planes during far (left) and near (right) viewing. Each Listing's plane rotates temporally by a quarter of the vergence angle (α).

Listing's Law during Convergence

For the following considerations, eye positions need to be described three-dimensionally with a horizontal, vertical, and torsional component. Since rotations are noncommutative, the most convenient conventions, such as rotation vectors or quaternion vectors, express every eye position as a single axis rotation from a reference position. Accordingly, vergence is then defined as the rotation that transforms the left eye position into the right eye position [37].

Rotation or quaternion vectors hold a specific 3-D orientation in the head. Listing's law states that, in the absence of dynamic vestibular stimulation, these 3-D vectors all lie in one plane, so-called Listing's plane. In the absence of convergence, the Listing's planes of the two eyes are relatively parallel and oriented approximately frontal. With convergence the planes rotate outward [38–41], i.e. they 'swing out like saloon doors' [42]. In other words, the primary positions of the two eyes diverge during convergence [43]. Among the cited studies, the angle of the outward rotation of the Listing's planes varies considerably and amounts roughly to about 1/4 (range: 0.16–0.43) of the convergence angle (fig. 1).

An explanation of why the Listing's planes rotate outward during convergence has to consider both visual and motor variables [44]. Tweed [45] proposed a most compelling hypothesis that is based on an optimal compromise between visual and motor variables: The visual variable is the maximal alignment of images in the visual plane on the two retinas irrespective of gaze direction; the motor variable is to keep rotation about the line of sight as close as possible to the zero vergence primary position. Since the amount of cyclovergence varies with gaze elevation when the Listing's planes are rotated outward, stereograms that critically depend on the relative torsional orientation of the two retinas are only visible at a specific gaze elevation [46]. Hence, ocular motor control plays an important role in depth vision.

MR images demonstrate that rectus pulleys in converging eyes are slightly extorted [47]. The outward rotation of Listing's plane, however, cannot be explained by this change of the rectus pulleys and therefore must be due to variations of oblique muscle innervations. The convergence-induced outward rotation of Listing's planes does not depend on whether convergence is induced by a stereogram, a horizontal prism, or an accommodative stimulus [48–50]. The orientation of Listing's planes can, however, be modified by phoria adaptation (see below).

Vergence movements with the eyes at various elevations lead to different torsional components that can be explained by the vergence-modulated orientation of Listing's plane [41]. Accordingly, during pure vergence movements with gaze elevated or depressed, the eyes rotate about an axis which is orthogonal to the gaze direction [51]. This is different from the orientation of rotation axes during saccades, which tilt in the direction of gaze by only half the gaze angle [52]. During asymmetric vergence movements, e.g. when foveating a target moving along the line of sight of one eye, monocular torsion is less stable than cyclovergence and varies between convergence and divergence [53, 54]. Pitch head impulses while the eyes are converging on a near target in front of one eye lead to torsional movement components in both the adducting and the straight ahead viewing eye [55]. This effect corresponds to a modification of ocular rotation axes due to the convergence-induced outward rotation of Listing's planes.

The Listing's planes in patients with acquired trochlear nerve palsy are not symmetric; the plane of the affected eye is rotated outward, as if this eye were converging [56]. In congenital trochlear nerve palsy, the orientation of Listing's plane of the affected eye is normal; thus, congenital trochlear nerve palsy is not due to changed function of a single extraocular eye muscle [56]. In patients with acquired or congenital trochlear nerve palsy, Listing's plane of the affected eye does not rotate temporally upon convergence. This finding suggests an important role of the SO in modifying the orientation of Listing's plane as a function of vergence [57]. In patients with acute trochlear nerve palsy, Listing's law is violated by the affected eye during downward saccades; this eye shows dynamic extorsion as a result of the missing agonistic action of the SO [58]. In patients with central abducens nerve palsy, Listing's law is violated by both eyes, while in patients with peripheral abducens nerve palsy, Listing's law is violated by the paretic eye and in the acute state only [59].

Compared to healthy subjects, patients with intermittent horizontal strabismus exhibit a similar, but more variable relation between vergence angle and angle between the Listing's planes [60]. In patients with intermittent exotropia, vertical gaze-dependent cyclovergence is increased, possibly because additional convergence is required to cancel the exodeviation between the two eyes [61].

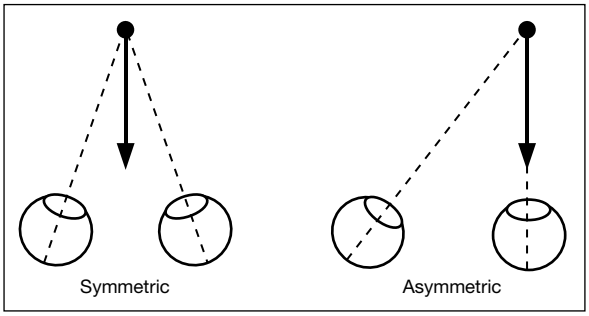

Fig. 2. Top view of both eyes during symmetric and asymmetric convergence movements. The visual target moves from far to near (arrow).

In a stereoblind patient with strabismus, the Listing's planes of the two eyes were normal in shape, i.e. relatively planar, but changed their orientation depending on which eye was fixating [62]. This effect was most probably due to accommodation-induced vergence.

Asymmetric Vergence Movements and Hering's Law

Hering's law of equal innervation implies that equal version and vergence commands are sent to both eyes and that the binocular motor output represents the sum of the two signals. The analysis of asymmetric vergence movements (fig. 2) can give some indication whether Hering's law holds [63, 64] or whether the two eyes are independently controlled, as advocated by Helmholtz [65, 66]. As we will see, there are arguments for both theories.

During static convergence on a target in front of one eye, i.e. asymmetric convergence, only the inferior oblique muscle contracts in this eye, as demonstrated with MRI; contraction of the same muscle, apart from contractile changes in the lateral and medial rectus muscles, is also seen in the fellow eye, which is directed inward [47]. During rapid gaze shifts along the line of sight of one eye, which calls for asymmetric vergence, the horizontal peak accelerations of the two eyes are similar, despite different position trajectories [67]. This finding suggests equal saccadic pulses for each eye, according to Hering's law, together with an additional vergence signal. After human subjects were trained to have a vertical vergence component during symmetric horizontal vergence, the vertical vergence component could also be demonstrated during smooth pursuit of targets in depth both along the line of sight of one eye [68]. Thus symmetric smooth pursuit seems to be combined with vergence to produce

asymmetric slow eye movements, which speaks against monocular control of these movements.

Some subjects are able to initiate smooth asymmetrical 'saccade-free' convergence movements when changing gaze from a far to a near target [69]. Thus, during binocular viewing, the ocular motor system is able to generate eye movements that do not adhere to Hering's law of equal innervation. Similarly, the initial monocular smooth pursuit response to a target that moves in depth solely depends on target motion and is independent of the response of the other eye [70].

The firing rate of abducens motoneurons for a given eye position is higher with than without convergence, but, paradoxically, lateral rectus force (and similarly medial rectus force) is not increased [70a]. This finding still awaits an explanation. A reanalysis of single neuron recordings during eye movements that included vergence revealed that neural signals in abducens motoneurons, abducens interneurons, and medial rectus motoneurons encode the position of both eyes, not just one eye [71]. On the other hand, premotor neurons in the paramedian pontine reticular formation encode saccadic velocity signals for only one eye, not both [72]. These findings speak against a neural implementation of Hering's law.

Saccade-Associated Vergence Movements

Peak vergence velocity increases when vergence is combined with a saccade, an effect that is more pronounced in divergence than convergence [73]. Vice versa, when saccades occur with vergence movements, the peak velocity of the saccades is reduced, more prominently so with convergence than divergence [74]. These findings suggest a nonlinear interaction between conjugate and disconjugate premotor systems; the omnipause neurons probably represent the crucial neural structure for gating saccade-related horizontal vergence [75]. This would also explain why saccadic oscillations occur, when saccades end during ongoing vergence [76–78]. Note that even horizontal and vertical saccades between far targets are associated with small transient vergence components, but these are probably related to mechanical differences between adducting and abducting muscles [75, 79]. Horizontal saccades also produce small torsional transients out of Listing's plane, which are not equal in amplitude; hence, the eyes cycloverge somewhat shortly after the beginning of each saccade [80].

Saccades in patients with one deeply amblyopic eye are nonconjugate, i.e. Hering's law seems to rely on intact binocular vision [81]. Subjects with anisometropic spectacles show saccades with different amplitudes in both eyes and

asymmetric postsaccadic drift [82]. When saccades are made between targets at different distances, a presaccadic vergence movement along the isovergent line of the initial target appears [83]. This observation speaks for separate version and vergence channels contributing to fast eye displacements. A similarly strong coupling between version and vergence is found during incorrect saccades evoked by two targets appearing simultaneously in 3-D space [84]. Conversely, when targets are placed at closer distances from the eyes, no presaccadic convergence and only a small presaccadic divergence is observed, and postsaccadic vergence is usually asymmetric [85]. The latter finding speaks against a balanced interaction between the vergence and version systems during the saccade, and therefore against a Hering-type implementation of such movements. Such saccades are dominated by one eye, so that a least one of the two eyes is on target in time.

Binocular vertical displacements between near targets in front of one eye require different vertical amplitudes of each eye to maintain binocular alignment. In downward movements, a major portion of the required disconjugacy takes place during the saccades, while in upward movements the intrasaccadic portion amounts to about half [86]. Dynamic dissociations between saccadic and vergence movements can also be observed during vertical saccades between targets in the midsagittal plane at different depth [87].

Binocular Adaptation

Phoria Adaptation

Normal binocular fixation of a near target in a tertiary position requires a vertical vergence component, when eye positions are expressed in a head-fixed coordinate system. This component appears to be independent of whether subjects are viewing monocularly or binocularly [88]. Eight hours of monocular occlusion leads to excyclophoria and hyper- or hypophoria [89]. If an eye is covered and passively rotated away from the position of the fellow eye with a scleral suction lens during a few minutes, ocular misalignment persists up to 10 min or until binocular viewing is permitted [90].

When short-term phoria adaptation is performed with a vertical disparity at a single location, phoria becomes uniform for all gaze directions. Upon two vertical disparities at opposite gaze directions and with opposite sign, adapted phoria shows a gradient along the line between the two stimuli [91, 92]. Phoria adaptation to opposite vertical disparities is also effective along the depth axis [93] or to multiple vertical disparities at different near and far locations [94]. Human subjects are also able to adapt vertical phoria to different prism-induced vertical disparities that vary with head position [95] or with head and gaze

position [96]. When monkeys are trained to synchronize vergence eye movements in synchrony with vestibularly evoked eye movements upon pitch oscillations, these oscillations evoked vergence eye movements even in the dark [97, 98].

Adaptation to discrete increments of refraction along a horizontal prism is also possible, but adapted vergence changes only gradually when crossing the prism edges [99]. After 30–150 s of cyclovergence evoked by incyclo- or excyclodisparity, the eyes do not tort back to their previous torsional positions, even in the presence of a visual stimulus [100]. Most likely, this torsional hysteresis is the result of fast phoria adaptation.

Phoria adaptation with a vertical prism over one eye is often impaired in patients with cerebellar disease. Thus the cerebellum seems to be decisively involved in phoria adaptation [101].

Adaptation of Listing's Plane

Three days of vertical disparity with prisms induces, besides vertical phoria, reorientations of Listing's planes; Listing's plane of the higher eye is rotated up and Listing's plane of the lower eye rotated down [102]. Phoria adaptation to different cyclodisparities along the vertical axis also modifies the orientation of Listing's planes [103].

Binocular Saccade Adaptation

Intrasaccadic displacement of a visual target leads to rapid binocular saccade adaptation. If the displacement is only presented to one eye, while the target is unchanged for the other eye, short-term adjustments are again conjugate, which suggests that there is no mechanism for fast disconjugate saccade adaptation [104]. Dichoptically presented random-dot patterns with local disparities representing a 3-D object lead to immediate position-dependent saccadic disconjugacies that persist during subsequent monocular viewing [105]. Similar immediate disconjugacies of saccades can be observed when disparities are introduced by dichoptical images that differ in size [106].

Subjects with anisometropic spectacles show saccades with different amplitudes and postsaccadic drifts between both eyes, even during monocular viewing [82, 107]. Already an image size inequality of 2% leads to disconjugate horizontal and vertical saccades, which persist after a short training period when tested in the absence of normal binocular visual targets [108]. Placing an afocal magnifier in front of one eye leads to disconjugate memory-guided saccades, which outlasts the removing of the magnifier after the training period, when subjects are viewing monocularly [109, 110]. Dichoptically presented patterns that are displaced at the end of each vertical saccade induce amplitude disconjugacy, but only little disconjugate postsaccadic drift [111]. Apparently, this effect does not require foveal fusion since microstrabismic patients adapt as

well [112]. When vertical saccades are disconjugately adapted, smooth pursuit movements remain conjugate and vice versa [113]. Thus, the two classes of eye movements have separate mechanisms for binocular adaptation.

In patients with trochlear nerve palsy, saccades become more conjugate after strabismus surgery, an effect that is more pronounced in patients with congenital than in patients with acquired trochlear nerve palsy [114]. In rhesus monkeys with one surgically weakened extraocular muscle, the paretic eye shows postsaccadic drift with the normal eye viewing. Deafferenting the paretic eye leaves postsaccadic drift unchanged; thus, proprioception from the paretic eye does not play a role in the adaptation of postsaccadic drift [115]. Proprioceptive deafferentation alone impairs ocular alignment and saccade conjugacy [116].

Disconjugate Eye Movements Evoked by Vestibular Stimulation

Vergence eye movements are elicited by linear motion in the dark with or without visual targets [117]. The gain of the translational vestibulo-ocular reflex (VOR) during heave (= up-down) and sway (= left-right) whole-body oscillation increases with increasing convergence [118, 119]. During surge (= fore-aft) oscillation, the gain of the translational VOR increases with both increasing gaze eccentricity and increasing convergence, which is qualitatively accurate for foveal stabilization of both eyes [120–122]. Such vergence responses are enhanced by the presence of visual stimuli [123]. During visual fixation upon isovergence targets along the horizontal meridian and concurrent rapid oscillations in various directions in the horizontal plane, both eyes move in the geometrically correct direction needed to stabilize the targets on the two foveae; the gain of the version component (average velocity of both eyes divided target velocity), however, amounts to only around 0.5, while the gain of the vergence component (right eye velocity minus left eye velocity) ranges around unity [124]. This finding might reflect the fact that for visual acuity it is more important to stabilize the relative orientation of the lines of sight than binocular position. Vergence also modifies the gain of the angular VOR for gaze stabilization. For example, the gain of the VOR elicited on a horizontal turntable anticipates the vergence angle by about 50 ms [125].

Ocular counterroll elicited by head or whole-body roll interferes with stereopsis. This geometric incompatibility increases further with decreasing target distance. It is therefore advantageous that ocular counterroll decreases strongly during convergence [126, 127]. In the presence of ocular counterroll, binocular movements from a far to a near target show unequal torsion; the required torsion for the undermost eye is larger than for the uppermost eye, since convergence is associated with extorsion. Such torsional disconjugacy,

however, cannot be demonstrated for divergent eye movements [128]. Static head roll also leads to excyclovergent eye positions [129]. This phenomenon can be explained by a static hysteresis that differs between the eyes contra- and ipsilateral to head roll [130]. Probably, ocular torsional hysteresis is introduced at the level of the otolith pathways because the direction-dependent torsional position lag of the eyes was related to head roll position, not eye position. Asymmetric binocular torsion evoked by hypo- or hypergravity may be a predictor for space sickness [131–133].

During position steps of head roll, the eyes show dynamic binocular counterrolling and skewing. While the gain of dynamic binocular torsion is larger in upright than in supine position, dynamic skewing is unaffected by the additional otolith input that appears in upright position [134]. Constant rotation about an off-vertical axis causes horizontal vergence movements [135]. During oscillatory head roll, the ocular rotation axes of the two eyes are convergent both in the dark and when fixating upon a far light dot; when subjects fix upon a near light dot, the convergence of binocular rotation axes exceeds the convergence of binocular positions [136]. The Bielschowsky head-tilt sign in unilateral trochlear nerve palsy, i.e. increased vertical and torsional divergence with the head tilted towards the affected eye, can be explained by inward tilt of the rotation axis of the covered eye during head oscillation about the naso-occipital axis [137]. This 'convergence' of ocular rotation axes is the result of decreased force by the SO of the covered paretic eye or, according to Hering's law, increased force parallel to the paretic SO in the covered unaffected eye. The gain of the VOR in an eye with trochlear nerve palsy is reduced in all directions, but especially towards intorsion, depression and abduction, in accordance with the 3-D pulling direction of the SO [138]. In patients with peripheral abducens nerve palsy, the gain of the horizontal VOR in the affected eye is reduced in both directions, when tested in the dark. In the light, horizontal gains normalize in patients with mild or moderate palsy [139]. The gain of the torsional VOR is reduced in both the healthy and the affected eye [140].

The orientation of ocular rotation axes as a function of eye position depends on the gain of the torsional VOR; the lower the torsional gain, the more the axes tilt with eccentric gaze position [141]. As the torsional gain decreases further with increasing convergence, average 3-D eye positions scatter closely around the temporally rotated Listing's plane, which is advantageous for binocular retinal stabilization [142]. Head roll in patients with peripheral abducens nerve palsy leads to a hyperdeviation of the ipsilateral eye, independent of which eye is affected. In patients with central abducens palsy, the same eye (healthy or affected) hyperdeviates when rolling the head to the left or the right side [143].

At low frequencies, the horizontal and vertical VOR can be cancelled by visually fixing upon head-fixed targets. During head oscillations about the

naso-occipital axis visual suppression of the elicited torsional VOR is incomplete, but the lines of sight of the two eyes remain on target [144]. If subjects during head roll fix upon head-fixed eccentric horizontal targets at near distance, the eyes also show vertical movement components, even if one eye is covered [145]. These components are required to keep the lines of sight pointed to the targets. Thus, the vergence system correctly modifies the eye movements that are not visually cancelled to prevent horizontal and vertical retinal slip in either eye.

Disconjugate Eye Movements and Blinks

Initial eye movements during voluntary blinks are extorsional, downward, and inward, consistent with an early pulse-like innervation of the inferior rectus muscle [146]. Thus, during this early phase of blinking, the eyes converge and excyclodiverge. Blinks modify the kinematics and dynamics saccade-vergence and slow vergence eye movements [147, 148]. Besides mechanical factors of the eye plant, the found changes might reflect the blink-induced decrease in omnipause neuron activity.

Pathological Disconjugate Eye Movements

Normally, vergence eye movements in response to steps of a visual stimuli become slower with age, which has to be taken into account when evaluating patients with suspected vergence disorders [149].

Binocular positions in patients with cerebellar dysfunction are usually esophoric or even esotropic. In addition, there is a hypertropia that varies as a function of horizontal eye position, so-called alternating skew deviation with the abducting eye higher. The patients show both conjugate and disconjugate saccadic abnormalities that are also eye position dependent [150]. The mechanism of alternating skew deviation in patients with cerebellar disease could be due to a lost correction of changed eye muscle pulling directions, which is required when animals become frontal eyed. If, in addition, one assumes an imbalance of graviceptive-ocular pathways responding to head pitch, alternating skew deviation can be explained by this mechanism [151].

Dissociated vertical divergence (DVD) includes the following ocular motor phenomena [152]: Upon occlusion of either eye, a horizontal and cyclovertical latent nystagmus develops. This is quickly followed by cycloversion/vertical vergence, with the fixing eye intorting and tending to move downward and the covered eye extorting and moving up. Simultaneously, upward versions occur for the maintenance of fixation. This, in turn, leads to further

upward movement of the covered eye and, at the same time, to a reduction of the cyclovertical component of the latent nystagmus. Thus, a possible 'purpose' of this cycloversion and vertical vergence is to damp the cyclovertical nystagmus that occurs when one eye is covered [153]. Brodsky hypothesized that DVD is a dorsal light reflex that occurs when binocular vision is impaired in infancy [154]. Since patients with DVD only transiently perceive a tilt of the subjective visual vertical when one eye is covered, it was speculated that the cancellation of SVV tilt in these patients is the main function of DVD [155].

Binocular eye movements in patients with convergent-divergent pendular nystagmus are conjugate in the vertical direction, but phase shifted by 180° in the horizontal and torsional directions [156]. The lesion is usually localized within neural structures of the vergence system. If horizontal saccades or smooth pursuit eye movements are pathologically coupled with convergence, the abducting eye will appear paretic despite an intact abducens nerve. This so-called pseudo-abducens palsy is caused by lesions of convergence pathways near the midbrain-diencephalic junction and is frequently associated with upgaze palsy and convergence-retraction nystagmus [157]. Paramedian thalamic infarctions without involvement of the midbrain may lead to a selective bilateral pseudo-abducens palsy [158]. Convergence-retraction nystagmus, however, is due to a mesencephalic lesion [159] and represents a disorder of the vergence system [160]. Pathologically disconjugate eye movements with the vergence system intact, is typical of internuclear ophthalmoparesis [161]. Mild internuclear ophthalmoparesis, in which the adducting eye is only slightly slower than the abducting eye, is often missed by clinicians, as demonstrated by infrared oculography [162].

Ocular bobbing, which rarely appears after infratentorial lesions, but otherwise has no localizing value, may be disconjugate [163]. Disconjugate vertical and torsional ocular movements, resembling seesaw nystagmus, have been observed in a patient with locked-in syndrome after large infarction of the pons [164]. Smaller lesions in the ventral pons involving the nucleus reticularis tegmenti pontis lead to impairment of slow vergence movements to ramp targets [165]. On the other hand, fast vergence movements to step targets are affected by lesions of upper pontine nuclei [166].

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Dominik Straumann
Neurology Department
Zurich University Hospital
CH–8091 Zurich (Switzerland)
Tel. +41 44 255 4407, Fax +41 44 255 5564, E-Mail dominik@neurol.unizh.ch