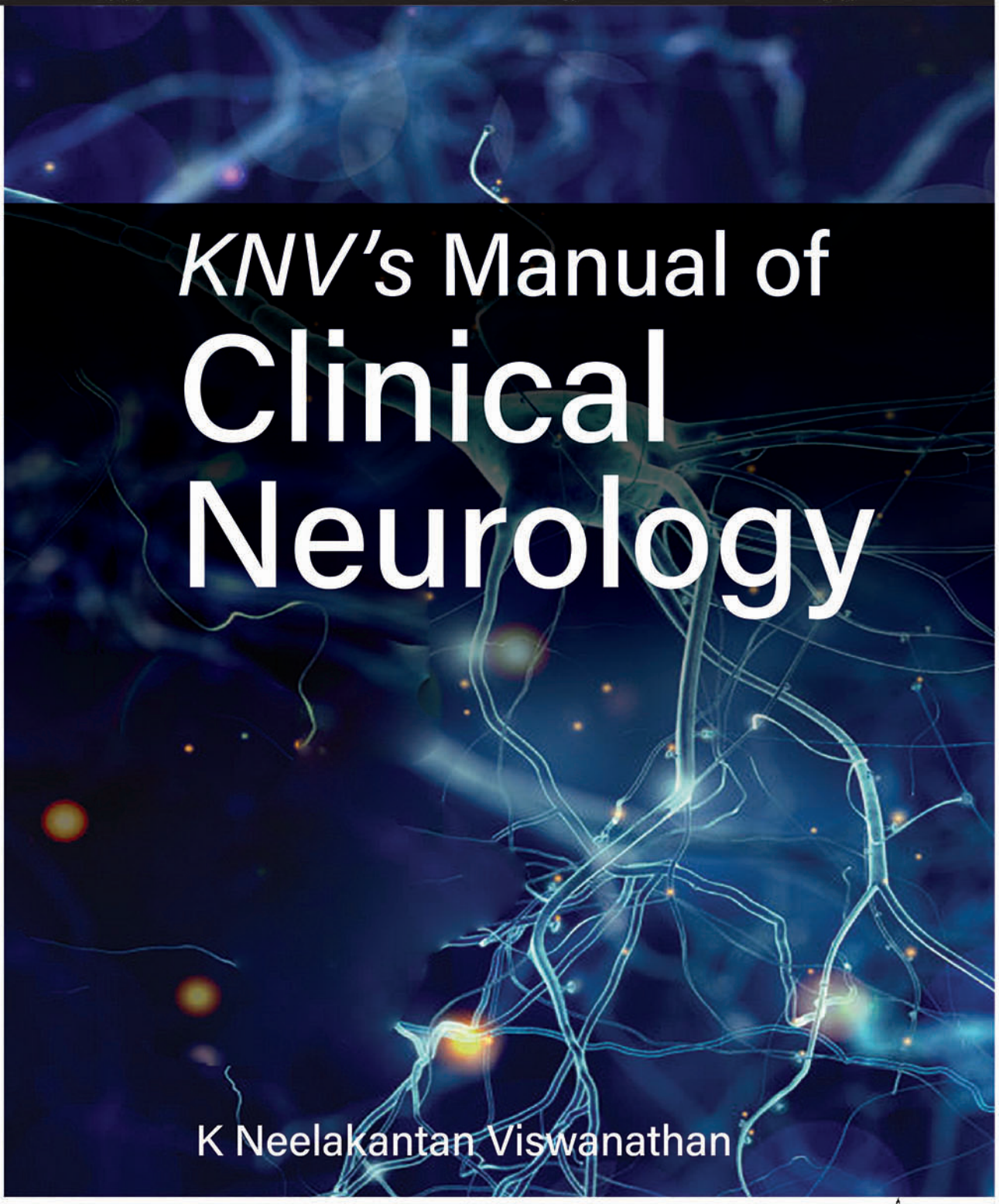
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KNV's Manual of Clinical Neurology

K Neelakantan Viswanathan

Forewords
KR Sethuraman
Ashok Kumar Das

JAYPEE

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CHAPTER 4

Nystagmus

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Nystagmus is defined as rhythmic involuntary movement of the eyes either at rest or precipitated by eye movements and continues to be present for some time after the eyes have stopped moving. It is almost always bilateral and unilateralism is rare. It may be jerky or pendular.

PENDULAR NYSTAGMUS

Horizontal oscillations, of equal amplitude, to either side of the midline.

- *Congenital*: This is inherited as an X-linked recessive disorder but may be autosomal dominant at times. It persists lifelong and is rapid and horizontal.
- *Spasmus nutans*: This appears between 4 months and 1 year of age. It occurs in children who live in houses with very poor lighting facilities. Vitamin D deficiency usually coexists. It is monocular and is associated with titubation. Head tilting may also occur. By about 2 years of age, it is known to disappear spontaneously.
- *Ocular nystagmus*: This is pendular and rapid and occurs in children soon after birth representing an attempt to project the vision on the retina outside the macula and is due to corneal opacities, cataracts, or chorioretinitis. Miner's nystagmus is an example in adults. Titubation and levator palpebrae spasm are associated features.

JERKY NYSTAGMUS

This nystagmus of unequal amplitude shows a slow phase in one particular direction of gaze and a rapid phase in the opposite direction. The nystagmus is described with reference to the direction of the rapid phase.

Nystagmoid/Paretic Jerks

When testing for nystagmus, while looking to the extremes of gaze, nystagmoid jerks occur owing to fatigue of ocular muscles. This is because the *binocular vision is lost* at the extremes of gaze and the eyes try to compensate for the same. Hence *to test for nystagmus, the examiner should maintain a finger distance of 1 m (or 2 feet) from the patient's eyes and not take the finger to the extremes*. Jerky nystagmus is also seen in a paretic eye, when it is unilateral or in conjugate gaze palsy, when it is bilateral. In some myasthenic patients, progressive diplopia with weakness of ocular muscles follows paretic jerks, in trying to gaze in a sustained manner.

Vestibular Nystagmus

Disturbance of the action of vestibular organs or their connections to the opposite abducens nucleus, as labyrinthitis, causes this. Phasic nystagmus is encountered. This condition

causes the slow phase in the direction of endolymph movement and recovery is characterized by a rapid phase. Vertigo is a very common accompaniment, accentuated by head and eye movements in the direction of the rapid phase. Vestibular nystagmus may be precipitated by turning the patient around in a Bárány chair or by applying a weak galvanic current to a particular side.

Caloric testing is done by irrigating the external auditory meatus with a few milliliters of cold water.

If the vestibular apparatus is damaged completely, there is *NO* response.

A lesion affecting the supranuclear connections that control eye movements will produce tonic deviation of the eyes to the same side, *unassociated with nystagmus*.

Irritative lesions of the vestibular apparatus will *accentuate the nystagmus to the opposite side*.

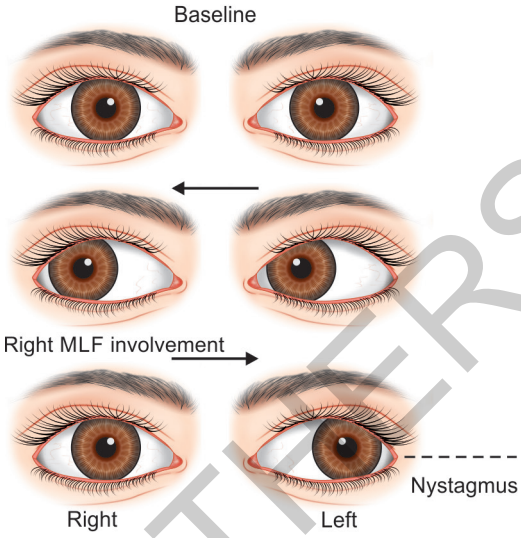


FIG. 1: Medial longitudinal fasciculus (MLF) lesion —Harris' sign.

Alexander's Law

The nystagmus of labyrinthine or vestibular origin increases in amplitude on looking toward the fast phase (the healthy side).

Cerebellar Lesions

Usually nystagmus results from dysfunction of cerebellar connections in the brainstem rather than the cerebellum itself. It is a phasic nystagmus with the fast component in the direction of gaze and slow component in the resting position. (Slow movements of increased amplitude occur in the direction of lesion). *Extensive degeneration of cerebellar hemispheres can exist without nystagmus*.

Medial Longitudinal Fasciculus Lesion (Fig. 1)

Medial longitudinal fasciculus (MLF) lesion produces *internuclear ophthalmoplegia (INO)*. There is no adduction on the side of MLF lesion while the *contralateral abducting eye shows nystagmus (Harris' sign) (dissociated nystagmus)*. The MLF may also be involved by lesions of cervicomedullary junction such as syringobulbia.

One and a Half Syndrome (Fig. 2)

In this condition, the *MLF and parapontine reticular formation (PPRF)* are damaged on the same side and the eye on the side of the lesion is in the central fixed gaze, adduction is lost contralaterally, and *abduction is the only possible movement* and it *shows nystagmus*.

Iatrogenic

Nystagmus with a fast component in the direction of gaze and a slow component in the direction of rest occurs with drugs such as *anticonvulsants and alcohol*.

See-saw Nystagmus

See-saw nystagmus occurs in *parasellar lesions*. One eye goes up and intorts while the other comes down and extorts, resembling a see-saw. It may be associated with bitemporal hemianopia.

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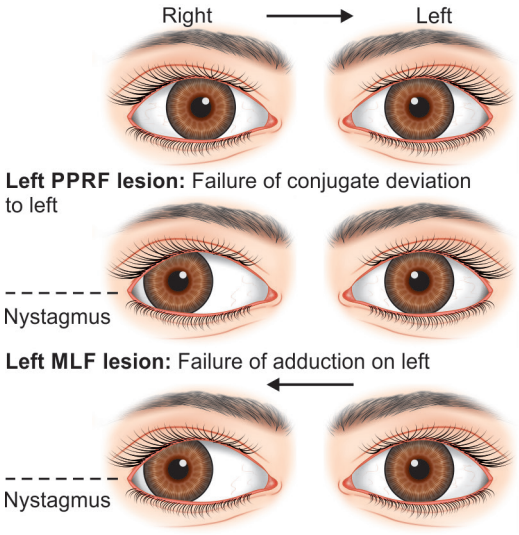


FIG. 2: One and a half syndrome (The syndrome usually results from unilateral lesion of the PPRF and MLF).

(PPRF: parapontine reticular formation; MLF: medial longitudinal fasciculus)

Retraction Nystagmus/Convergence Nystagmus

Retraction nystagmus/convergence nystagmus occurs with *lesions of the upper midbrain (tectum or pretectal region) in the area of the pineal gland*. When gaze is attempted upward in upward gaze palsy, a jerky nystagmus with fast phase convergence results. It is best visualized by seeing the patient from the sides. A rapid jerky retraction of the eyeballs into the orbit with adduction is seen, called as convergence nystagmus.

It is best seen by a slow downward rotation of an optokinetic drum or as an upward saccade from the down gaze to the primary position.

Upbeat/Downbeat Nystagmus

Lesions of anterior vermis of cerebellum produce *vertical* nystagmus, upbeat in the primary position. *Upper medullary lesions* produce *fine small amplitude upbeat nystagmus with the fast phase upward. Downbeat nystagmus with the fast phase downward*, is seen *in foramen magnum (cervicomedullary/lower medullary) lesions*. A variant of upbeat nystagmus is called "*bow-tie nystagmus*," where there are quick phases obliquely upward and horizontal components alternating sides.

Rebound Nystagmus

This is a phasic type nystagmus, horizontal, and lasts for about 15 seconds on looking laterally when the eyes get fatigued and return to the midline. Then there is nystagmus to the opposite direction also for the same time when the eyes get fatigued again returning to the midline. This is seen in degeneration of cerebellum.

Rotatory Nystagmus

This occurs in disorders of the labyrinth.

A useful rule is cerebellar lesions produce horizontal nystagmus, brainstem lesions produce vertical nystagmus and labyrinthine disorders produce rotatory nystagmus.

Optokinetic Nystagmus

When looking out of a fast-moving train, the eyes focus on several objects that move across the visual field. The movement is slow in the direction of the object and fast, when back to the resting position. This nystagmus may be induced by looking at a rotating optokinetic drum. There is usually a symmetric response. The response may be absent in lesions of optic radiation or parietal lobe. Asymmetric response is seen in brainstem lesions. The optokinetic drum may also expose patients faking to be blind, in whom nystagmus is absent.

GRADING OF NYSTAGMUS

I. Nystagmus with a fast phase to one direction, on looking toward that direction

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II. Nystagmus with a fast phase to one direction, on looking straight ahead
III. Nystagmus with a fast phase to one direction, on looking toward the opposite direction

Ocular Bobbing

There is brisk downward with slow upward movement of eyes. There is *loss of horizontal movements* and it is seen in *midbrain, pontine lesions, and cerebellar* hemorrhage.

Reverse ocular bobbing is of *metabolic* cause, but is unreliable.

Ocular Dipping

There is slow downward followed by brisk upward movement of eyes (the opposite of ocular bobbing). The *horizontal movements are normal*. It occurs in *diffuse anoxic cortical damage* and in *drug intoxication*.

Reverse ocular dipping: Occurs in *pontine infarct*, but is unreliable.

Nystagmoid jerks, in any direction, *in an unconscious patient* indicate a *severe pontine lesion*.

Periodic Alternating Nystagmus

There is horizontal conjugate deviation to either sides, alternating every 2 minutes, occurring in hypoxic encephalopathy, other unconsciousness or vegetative states. It is also seen in lesions of the lower brainstem or cerebellum.

Ping-pong Gaze

Ping-pong gaze consists of horizontal conjugate deviation alternating every few seconds, seen in *metabolic encephalopathy, bilateral cerebral hemispheric dysfunction,* and *toxin ingestion*.

Bruns Nystagmus

Bruns nystagmus consists of coarse movements of the eyes in one particular direction and fine movements in the other direction. It is indicative of a lesion in the cerebellopontine angle.

Windmill Nystagmus

Windmill nystagmus occurs in blind patients. The vertical plane oscillations, which are repetitive, alternate with those of the horizontal plane.

Ocular Flutter

Ocular flutter consists of quick to and fro horizontal saccades of decreasing amplitude, around a fixation point. There is no inter saccadic interval. It is seen in *cerebellar* disease or PPRF involvement. Unidirectional ocular flutter is found in postinfectious states. A mother and daughter who had small saccadic oscillations of eyes and hand tremors were coined to have an entity called *microsaccadic oscillations* and tremors in upper limbs abbreviated as microsaccadic oscillations with limb tremors (μSOLT). It is familial and described recently.

Ocular Dysmetria

Ocular dysmetria resembles an ocular flutter but the eyes move the point of fixation. The fixation saccades are one or more in number.

Opsoclonus (Saccadomania)

Opsoclonus (saccadomania) resembles ocular flutter but is chaotic, consisting of large amplitude saccades in any direction, and is indicative of diffuse bilateral *brainstem or cerebellar dysfunction*. It does not disappear in sleep or on eye closure. It may also occur in locked-in syndrome, viral, toxic, metabolic, vascular, and demyelinating lesions. Opsoclonus myoclonus (Dancing eyes dancing feet) syndrome may occur as a *paraneoplastic phenomenon* secondary to neuroblastoma in children.

Idiopathic Microsaccadic Opsoclonus

Idiopathic microsaccadic opsoclonus has been described recently.

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Oscillopsia

Oscillopsia is an illusion in which the patient feels that objects that are stationary in the visual field are oscillating in a haphazard manner. They may be jiggling, jumping, or moving like jelly. It is usually due to a vestibular lesion causing loss of vestibulo-ocular reflex.

Voluntary Nystagmus

Voluntary nystagmus is also known as psychogenic flutter. It consists of bursts of horizontal, vertical, or torsional oscillations in normal individuals disappearing on opening the eyes. It, usually, cannot be sustained for >30 seconds.

Monocular Eye Movements

Monocular eye movements are small, intermittent, rapid monocular, horizontal, vertical, or torsional movements seen in *pontine/midbrain* destructive lesions.

Koerber–Salus–Elschnig Syndrome

Koerber–Salus–Elschnig syndrome is encountered in dorsal midbrain or Sylvian aqueductal lesions. It should be differentiated from Parinaud syndrome, the terms of both of which are often used interchangeably. Parinaud syndrome is paralysis of upward gaze while Koerber–Salus–Elschnig syndrome is nystagmus which is jerky and irregular with convergence and retraction of the eye into the orbit, on upward gaze.

The clinical features, in detail, that help to distinguish peripheral nystagmus and nystagmus of central origin are given in **Table 1**.

Nystagmus may also be induced by Valsalva maneuver or hyperventilation. It can also occur during epilepsy.

Lid Nystagmus

Lid nystagmus is eyelid twitching that occurs simultaneously with the quick phase of horizontal nystagmus on lateral gaze. When it is brought about by convergence, it is called *"Pick sign."* The site of lesion is the lateral medulla or cerebellum.

*The classification of nystagmus by the systems classification (as per electrooculography) into vestibular, gaze-evoked and visual stabilization is beyond the scope of this manual and the reader is referred to clinical texts of Neurology for details of the same.

TABLE 1: Differences between peripheral (labyrinthine dysfunction) and central nystagmus (brainstem and cerebellar dysfunctions).

Clinical feature	Central nystagmus	Peripheral nystagmus
Nystagmus direction	Uni, bi, or multidirectional	Unidirectional
Only horizontal nystagmus without torsion	Commonly seen	Not common
Vertical nystagmus and pure torsional nystagmus	Seen	Not seen
Fixing the vision	Inhibition of nystagmus does not occur; it may be worsened	Nystagmus inhibited
Etiology	Trauma, toxins, ischemia, inflammation of labyrinth, Meniere's disease	Demyelination, vascular lesions, tumors

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KNV's Manual of Clinical Neurology

Salient Features

- This manual provides an essential knowledge of Clinical Neurology for the exam-going postgraduates in Medicine.
- In the present era of modern technologies that have made tremendous strides, this manual strives to inculcate the basic, time-honored methods of history taking and clinical examination into the minds of students to deduce a logical diagnosis, thus serving as a very useful revision tool.
- The basis and interpretation of each clinical sign are explained in detail.
- Applied Neuroanatomy and Neurophysiology have been incorporated for better understanding.
- The highlights are self-explanatory figures.
- The questions in Clinical Neurology such as "is there a lesion; where is the lesion; what is the lesion; why is the lesion, and what is to be done" find suitable answers.
- Important topics in Neurology have been discussed with basic investigative procedures as well as CT/MRI.
- Mnemonics form the highlight of this manual, which are given in color or in italics each topic, to enable students to recapitulate the subject with ease.
- Selected topics will also be of use to the undergraduates.
- Recent advances in the therapy have been briefly touched upon.
- Several syndromes mentioned in the manual come under "nice to know" category.

K Neelakantan Viswanathan MBBS (JIPMER) DNB (MED) FRCP (London) FRCP (Glasgow) FACTM AB (US) FACP (US) FCCP FIACM is currently a senior Professor of Medicine at Sri Manakula Vinayagar Medical College & Hospital, Puducherry, India. He had a brilliant academic career during his student days and has worked in premier medical institutions such as JIPMER, NIMHANS, SRMC and RI and Madras Medical College including a stint overseas. He has a vast teaching experience in Clinical Medicine for more than three and a half decades and has been an examiner for several undergraduate and postgraduate examinations at various Indian and Malaysian Universities. His inclination for Neurology began right from his undergraduate days in Medicine, at JIPMER under Professor S Chandrasekhar, and his residency training in Neurology at NIMHANS, Bengaluru, later under stalwarts like Professor M Gourie Devi and her colleagues further kindled his interest in Neurology and passion for the subject. He is an excellent teacher with immaculate clinical acumen. He has guided several postgraduates in their thesis work.

He has published more than 50 scientific and research articles in national and international journals including Cochrane Database of Systematic Reviews and has contributed to several chapters in Kumar and Clark's Clinical Medicine, Conn's Current Therapy, Bates' Guide to Physical Diagnosis and History Taking, Medicine Update, and Progress in Medicine at APICON. He often has been an invited speaker at conferences in India and abroad. In addition, he is an International Advisory Board Member for Kumar and Clark's Clinical Medicine for the last 15 years and has recently been inducted into the International Advisory Board for Davidson's Principles and Practice of Medicine. He has received EMINENT PHYSICIAN award from API City Physicians' Association, Chennai, for his outstanding contribution in expanding the frontiers of medical education. He plays an important role in shaping the cutting-edge postgraduate teaching programs in Internal Medicine.

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