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Vertigo and Dizziness

A Case-based Study

Ambar Chakravarty

2nd Edition

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COMMENTARY 3

Definitions and Classification of Vestibular Disorders

CLASSIFICATION

The classification of vestibular disorders remained at its infancy till almost a decade ago. In 2006, The Bárány Society had a meeting in Uppsala and it was decided to classify the vestibular disorders in a systematic and scientific manner. Until then, no attempt had been made to classify these disorders as a whole. Some diagnostic criteria existed about some common disorders like Ménière's disease, benign paroxysmal positional vertigo, and a few others but there was no comprehensive classification scheme available to incorporate all the vestibular disorders under a single diagnostic scheme. This was perhaps due to a lack of communication between the basic scientists, neurologists, and otolaryngologists. It was first decided to classify and describe the common signs and symptoms, which are encountered in vestibular disorders. In a Bárány Society meeting in Kyoto 2008, a draft for the classification of vestibular symptoms was accepted. This divided vestibular symptoms into four broad categories: (1) Vertigo, (2) Dizziness, (3) Vestibulo-visual symptoms, and (4) Postural symptoms. These four major categories of symptoms were subdivided into further subdivisions and given a number to go with it somewhat akin to International Classification of Headache Disorders.

Along with **Box 1** a short but accurate description of the underlying condition was given and in this manner a method of describing all the major vestibular symptoms which we encounter in clinical practice was developed. Thus each symptom had a definite place with an appropriate number to go with it in the classification table. It was soon realized that a real patient in clinical practice could have more than one symptom. This could easily be incorporated as a vestibular disorder having presented with more than one symptom. After having achieved a reproducible classification of the vestibular symptoms it was felt to incorporate all the features of vestibular disorders under one classification scheme. The Bárány Society formed subcommittees headed by an international expert to further subclassify the major groups of vestibular disorders. This would have at least three members from three subcontinents and included a neurologist, an otolaryngologist, and a basic scientist. The recommendations would be reviewed by the Bárány Society and was subjected to criticism and modification by other experts in the field. This created a flexible and open system and left ample scope for modifications. The Bárány Society recommended a four layer classification scheme:

- The *first* layer mainly deals with the clinical signs and symptoms. This was more or less covered in the earlier scheme but did not include cardinal signs like nystagmus, which need to be incorporated.

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BOX 1: Classification of vestibular symptoms.

- *Vertigo*:
  - Spontaneous vertigo 1.1
  - Spinning 1.1.1
  - Nonspinning 1.1.2
  - Triggered vertigo 1.2
  - Subdivided into positional, head motion, visually induced, sound-induced, Valsalva-induced, orthostatic, and others (spinning or nonspinning)
- *Dizziness*:
  - Spontaneous dizziness 2.1
  - Triggered dizziness 2.2
  - Subdivided into positional, head motion, visually induced, sound-induced, Valsalva-induced, orthostatic, and others
- *Vestibulo-visual symptoms*:
  - External vertigo 3.1
  - Oscillopsia 3.2
  - Visual lag 3.3
  - Visual tilt 3.4
  - Movement induced blur 3.5
- *Postural symptoms*:
  - Unsteadiness 4.1
  - Directional pulsion 4.2
  - Balance associated near fall 4.3
  - Balance associated fall 4.4

- The *second* layer is to consider the different types of syndromes. This could be an acute vestibular syndrome as could happen in a peripheral vestibular disorder like vestibular neuritis or a more sinister central cause like a cerebellar infarct. The second type of syndrome which may be encountered is an episodic recurrent form as evident in vestibular migraine or Ménière's disease. Here patients may be symptomatic periodically but may be totally normal in between attacks. The third type of syndrome would be where the symptoms would be longstanding and chronic and there would be no asymptomatic spells after the onset of the illness. This is typical of certain chronic cerebellar lesions and bilateral vestibular failure.
- The *third* layer is the most important and subdivided into A and B categories.
  - Type A is mainly concerned with the different types of diseases, which can lead to the vestibular symptoms. These would use terminology which is already in use in the vestibular literature but would incorporate new terms with proper explanations if required. Four subcommittees catering to Ménière's disease, benign paroxysmal positional vertigo, vestibular migraine, and behavioral neuro-otological conditions have already been formed. Here the experts will analyze the data available and formulate definite and probable criteria for diagnosis of these diverse conditions. Some of the recommendations of

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the committees formed earlier like vestibular migraine are already available. The recommendations for behavioral neuro-otological conditions are still under evolution.
    - Type B considers the anatomical, physiological, etiologic, and genetic basis of the vestibular disorders. This may be the most incomplete of all the layers described as new information from genetic and biochemical studies are constantly pouring in and existing concepts are continually being modified. The characteristic of this modern classification is that it is not a fixed one but an evolving one. There is a lot of scope to modify specifics as and when we find relevant information, which needs to be incorporated.
- Finally (*fourth* layer) all modern classification systems are concerned with disability and handicap. The International Classification of Vestibular Disorders also aims to emphasize the impact the disease is having on the affected person and the disability and handicap, which it may cause.

DEFINITION

In 2009, a set of five definitions for five common symptoms mentioned by patients have been suggested. These include:

1. "*Dizziness* is the sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion. This includes sensations sometimes referred to as giddiness, lightheadedness, or nonspecific dizziness but does not include vertigo.
2. *Presyncope* (also near syncope or faintness) is the sensation of impending loss of consciousness. This sensation may or may not be followed by syncope. When patients report lightheadedness, it should be classified as presyncope, dizziness, or both.
3. *Syncope* (also faint) is transient loss of consciousness due to transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. Syncope usually leads to loss of postural control and falling.
4. *Vertigo* is the sensation of self-motion (of head body) when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head motion.
5. *Unsteadiness* is the feeling of being unstable while sitting, standing, or walking without a particular directional preference. This sensation has previously been called disequilibrium or imbalance."

Many clinicians try to differentiate between sensation of self-motion with surrounding remaining still and sensation of surround rotation or spin with self-remaining still. There is essentially no physiological difference between the two but it is easier for patients to make this differentiation.

Recent attempts have been to formulate an algorithmic approach to patients attending the emergency room (ER) or even a clinic and abbreviated as TiTrATE, an acronym standing for timing, triggers, and targated bedside examination. The protocol is summarized in **Flowchart 1**.

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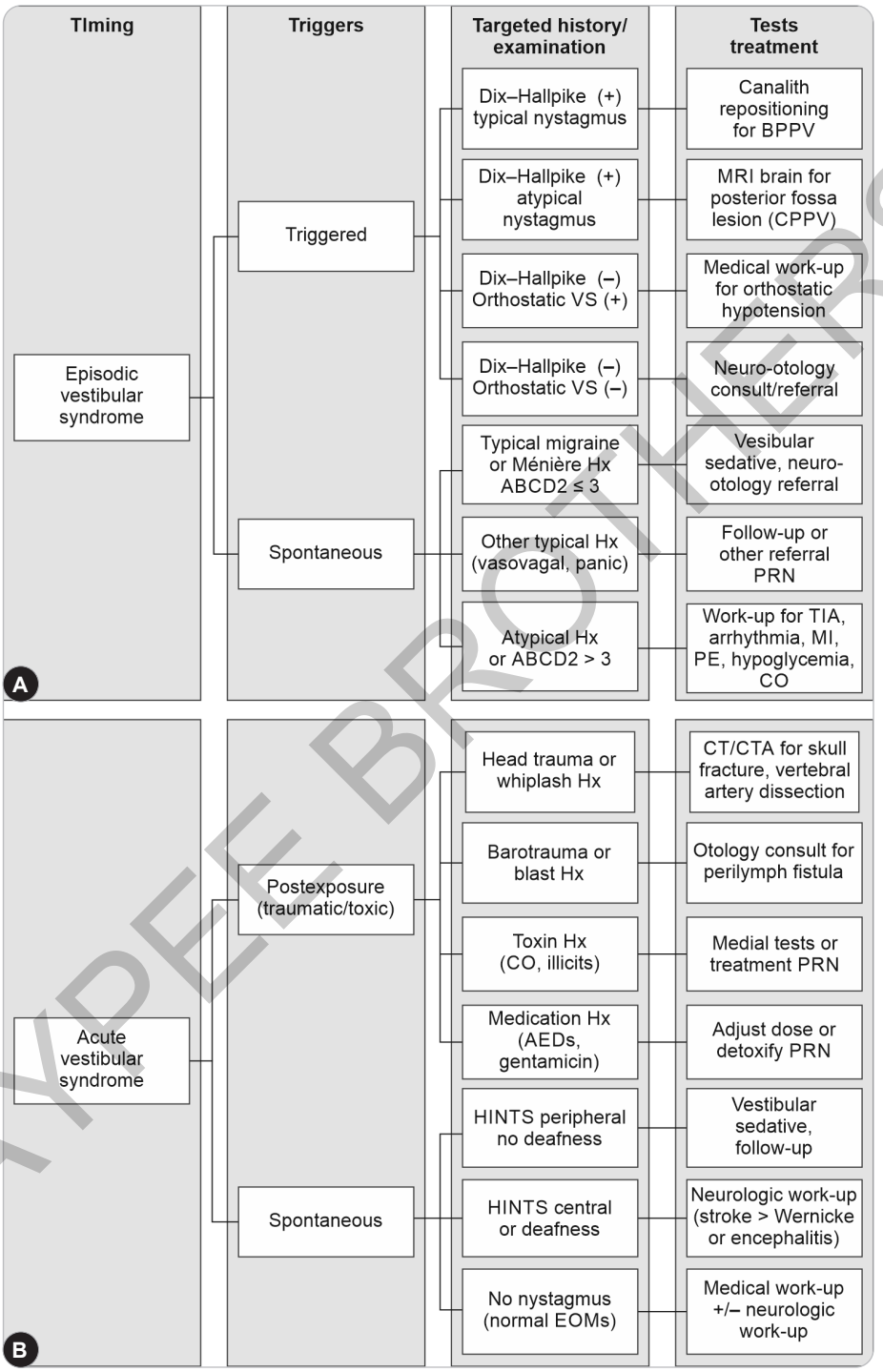

FLOWCHARTS 1A AND B: TiTrATE (timing, triggers, and targeted bedside eye examinations) protocol.
[AED: antiepileptic drug; BPPV: benign paroxysmal positional vertigo; CO: carbon monoxide; CPPV: central paroxysmal positional vertigo; CT: computed tomography; CTA: computed tomography angiography; EOM: extraocular movement; HINTS: head impulse-nystagmus-test of skew; Hx: history; MI: myocardial infarction; PE: pulmonary embolus; PRN: pro re nata (as needed); TIA: transient ischemic attack; VS: vital signs]

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CONCLUDING REMARKS

The Bárány Society along with other relevant associations and scientific bodies is working on a comprehensive and systematic classification system for the vestibular disorders. The framework in the form of different layers has already been laid out and will take its final shape in the next few years as more information flows in and controversies are resolved. Meanwhile we can use the resources already available and also contribute to the evolving system with useful relevant information.

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1. Bisdorff AR, Stabb JP, Newman-Toker D. Overview of the International Classification of Vestibular Disorders. Neurol Clin. 2015;33:541-50.
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CASE 1 What is not Vertigo?

CASE HISTORY

A 45-year-old healthy lady was traveling in a crowded bus and had been standing for a while. She started feeling unwell and felt hot and suffocated. Soon she started feeling light headed. There was no spinning but her vision got blurred. She started sweating. She looked for a place to sit but could not find any. She collapsed on the floor and blacked out. Fellow passengers splashed water on her face and she regained consciousness in a couple of minutes. On regaining consciousness, she felt very weak but had no headache nor had any nausea or vomiting. Fellow passengers got her down in the next bus stop and took her home, the address of which she could tell them correctly.

QUESTIONS

1. What is the likely diagnosis? Why this is not dizziness or vertigo?
2. What is the likely pathophysiology?
3. Would you advice any test to confirm the diagnosis?
4. What are the other causes of postural dizziness?
5. Can vestibular disease cause postural symptoms?
6. What is vestibular syncope?

DISCUSSION

The constellation of symptoms of lightheadedness, blurred vision, sweating, and then blacking out in this lady developing in a crowded place, suggests the diagnosis of a vasovagal or better termed neurally mediated syncope or neurocardiogenic syncope. Examination at that stage would have revealed bradycardia and a low blood pressure (BP). This is an extremely common condition across all ages and assumption of a supine posture usually relieves the symptoms. This is not dizziness as there had been no disturbed or impaired spatial orientation; only she had blurring of vision, sweating, and an impending sense loss of consciousness; she did black out and fell. There had been no false or distorted sense of motion at any stage to suggest vertigo. Patients with vertigo, however severe that may be, rarely lose consciousness.

The actual loss of consciousness itself is commonly associated with amnesia. Bystanders will often describe the patient as being quite pale and ashen in complexion with profuse diaphoresis and dilated pupils. There may on occasion be urinary (and rarely fecal) incontinence. Often the most disturbing and anxiety provoking sign during an event will be a tonic-clonic convulsive muscular motions. The convulsive activity that may accompany neurocardiogenic syncope occurs because the anoxic threshold of the brain has been exceeded, and this produces a form of acute "decortication", resulting in cortical disinhibition with excessive tonic brainstem motor activity. This process is not unique to neurocardiogenic syncope, rather it may be seen in any hypotensive event. Detailed observations of tilt-induced convulsive syncope have allowed for a specific sequence of movements to be described. With the onset of profound hypotension, the patient will display a startled appearance as well

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as cessation of any other activity in progress (e.g., speech). This is followed by a sudden loss of postural tone after which is noted extension and tonic contraction of the legs. There is usually a simultaneous extension of the arms in adduction. The head elevates and is thrown backwards, after which there are small short, jerking movements of the arms. This form of muscle activity usually will not follow the classic sequence of increasing and decreasing amplitude that is a distinguishing feature of the tonic-clonic grand mal seizure activity seen in epilepsy. In the majority of patients with convulsive syncope the ictal phase itself is quite brief, followed by a rapid recovery.

Our current understanding of the pathophysiology of neurocardiogenic syncope is however complex. The laboratory evaluation of such subjects is usually carried out by passive tilting on a tilt table where the subject's tolerance to orthostatic stress is tested by keeping him tilted at 60° upright position with continued monitoring of the physiologic parameters.

In subjects who after a while develop presyncopal symptoms or actually blacks out, a series of vascular events have been documented. It is found that initially there is rise of heart rate (HR) and BP but after a while there is sudden fall of BP with bradycardia. This would suggest an initial sympathetic overactivity in these subjects. Experimental studies in animals have also confirmed these findings where following an acute hemorrhage, initial tachycardia is often interrupted by periods of bradycardia as seen in humans with vasovagal syncope. In such animals an excess of afferent discharge through the unmyelinated cardiac C fibers has been demonstrated and currently it is thought that the primary fault in patients with vasovagal syncope lies in the cardiac mechanoreceptors, which send abnormal amount of signals to the vasomotor center causing an initial excessive sympathetic drive with increase in HR and force of ventricular contraction and peripheral vasoconstriction. However, after a while, probably this sympathetic drive is suddenly withdrawn, especially in the periphery but not on the heart, causing vasodilatation and fall in BP and venous return to the heart. The result is that, cardiac filling falls and relatively empty ventricles continue to contract vigorously under influence of the still continuing heightened sympathetic activity on the heart. This results in parasympathetic stimulation leading to bradycardia.

This theory had been challenged by some who demonstrated that head-up tilt was able to provoke neurocardiogenic hypotension in many patients who had previously undergone cardiac transplantation where near total denervation of the heart occurs during the procedure. However in such hearts as well, C fibers activity has been demonstrated in the aorta and pulmonary arteries. Hence, the aforementioned observations do not entirely exclude a cardiac contribution to the process causing neurocardiogenic hypotension and bradycardia.

The cause of the sudden sympathetic withdrawal is not known. Recent experimental evidences (from hemorrhagic shock models) point to alterations in the central nervous system (CNS) levels of serotonin, to be the signal initiating sympathetic withdrawal. A significant surge in the CNS serotonin level seems to be the precipitating factor. This idea has led to some success in use of selective serotonin reuptake inhibitors (SSRIs) in the treatment of intractable cardio-neurogenic syncopes. Serotonin probably inhibits sympathetic neural outflow while it increases adrenal sympathetic stimulation. There are three main serotonin [5-hydroxytryptamine (5-HT)] receptors—5-HT1, 5-HT2, and 5-HT3. The inhibitory

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effect of serotonin has been found to be beneficial in treatment only with the reuptake at 5-HT1 and 5-HT2 receptors and not with inhibition at 5-HT3 receptors, which may precipitate syncope. Clinical trials however failed to show any significant benefit with use of any pharmacologic agents like beta blockers, SSRI, and fludrocortisones. Only midodrine, an alpha adrenergic blocker, appears to have an edge over other agents by causing arteriolar constriction and decreasing venous pooling.

Other studies have demonstrated a sympathoadrenal imbalance between levels of epinephrine and norepinephrine, which may also contribute to hemodynamic derangements in precipitating syncope.

Transcranial Doppler studies have demonstrated cerebral vasoconstriction and not vasodilatation during tilt-table induced syncopal episodes suggesting a role of the CNS in the genesis of neurocardiogenic syncopes.

Diagnosis of vasovagal syncope is essentially clinical but confirmation may be done with head-up tilt table test (HUTT) if facilities are available.

Head-up Tilt Table Test

Head-up tilt table test has been used to evaluate the predisposition to neurocardiogenic syncope. The upright posture on the tilt table results in peripheral venous pooling, which decreases venous return to the heart. Reflex tachycardia and an increase in left ventricular pressure presumably stimulate cardiac C fibers, resulting in hypotension and/or bradycardia, which in turn activate the vasodepressor region to trigger the neurocardiogenic reflex.

Tilt table test is carried out using a specially designed tilting table with a foot board. Initially the patient rests in the supine position for a minimum period of about 25 minutes so as to stabilize the cardiovascular system. The patient is gradually tilted in the vertical angle ranging from 60° to 90° for a variable time period (15–45 min) and the hemodynamic changes are recorded. If the test fails to reproduce the syncopal attack, pharmacological agents are used and the test is repeated. These agents either augment or mediate the Bezold–Jarisch reflex and induce vasovagal syncope. Isoprenaline is the most commonly used drug. The protocols for the HUTT vary from center to center and hence the sensitivity reported in the literature varies widely from 30 to 85%. The pooled data however indicate that the sensitivity of the test is around 60% and the specificity is between 80 and 90%. The use of pharmacological agents induces syncope in 75% of the young people with negative HUTT test. Single stage isoprenaline HUTT in neurocardiogenic syncope provides a rapid method of increasing the overall test yield over the conventional two-staged HUTT protocol. This test is contraindicated in those with critical coronary and cerebral artery stenosis as well as in those with mitral and left ventricular outflow tract obstruction. Pregnant women should not undergo this test. Adverse events during test include: Hypertensive crisis, chest pain, coronary vasospasm, tachyarrhythmias, and transient asystole.

Four different types of responses may be recorded:

- *Type 1—mixed*: HR increases with HUTT and later decreases but remains above 40 beats/min or is <40 beats/min. The BP may go up initially but comes down later on or before the HR decreases.

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- *Type 2A—cardioinhibitory*: HR increases with HUTT, then decreases to <40 beats/min for >10 seconds or has asystole for >3 seconds. BP may rise initially but comes down before the HR decreases.
- *Type 2B—cardioinhibitory*: HR increases initially, then decreases to <40 beats/min for >10 seconds or has asystole for >3 seconds. BP decreases to a hypotensive level only at or after the time at which HR decreases.
- *Type 3—pure vasodepressive*: HR increases initially and decreases <10% from the peak value at the time of syncope. BP decrease accounts for the syncope.

Postural dizziness/lightheadedness mostly results from postural drop of BP of >20 mm Hg in systolic blood pressure (SBP). The three major causes are dysautonomia (as in diabetes, MSA), drugs (any vasodilators), and low plasma volume (blood loss). An unexplained postural hypotension at any age must always be investigated for occult blood loss, especially from the gastrointestinal tract.

Spinning vertigo of brief duration from sudden posture change generally occurs in subjects with past history of benign paroxysmal positional vertigo (BPPV). A meticulous and focused history of past episode of vertigo related to head/body position change needs to be obtained. Confirmation may be by performing the Dix–Hallpike or supine roll test depending upon the semicircular canal thought to be at fault.

Vestibular Syncope

The vestibular system, together with the baroreflex pathway, plays a substantial role in maintaining cardiovascular homeostasis during motion. Neurons in the caudal part of the vestibular nuclear complex, including the inferior and medial vestibular nuclei, regulate sympathetic activity through various connections with brainstem autonomic nuclei. Given the stimulus characteristics (head motion) and short-latency action of 100–200 msec, this vestibular-driven reflex regulates autonomic activity in a feed-forward manner. In contrast, the baroreflex system operates with a feedback mechanism that controls the autonomic activity. Recent research has introduced the concept of vestibular syncope, which refers to syncope as a result of a vertigo spell. It mostly occurs during Ménière's attacks and is ascribed to erroneous otolithic information during the spell, which may affect the cardiovascular system. This is different from the Otolithic Crisis where consciousness is retained (discussed later in Cases 15 and 16). Vestibular syncope may be considered to be a form of neurally mediated syncope and can be explained due to an excessive rise in BP attributed to dyssynergia of the vestibulosympathetic reflex and baroreflex system, owing to nonphysiologic vestibular stimulation (vertigo attack). Accordingly, vestibular syncope may occur in association with other vestibular disorders, but its clinical features and relationships with underlying disorders remain to be elucidated.

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A Case-based Study

Key Features

- First case study book on vertigo and dizziness principally authored by a neurologist from India to cater to the needs of both neurologists and ENT specialists.
- A total of 58 cases discussed in an interactive manner.
- The first part of the book contains basic topics ranging from anatomy and physiology of human balance and historical approach to patients with vertigo and dizziness in an attempt to unveil the cause and level of dysfunction supplemented by a detailed discussion on bedside examination of such patients and lastly an updated chapter on modern-day computerized methods for assessment of vestibular functions.
- The second part of the book contains Case Discussions as mentioned above.
- The third part of the book principally focuses on therapeutic aspects of vestibular disorders including both principles of pharmacotherapy and detailed discussions on canalith repositioning techniques for patients with BPPV.
- All in all, a comprehensive text on vestibular disorders (both central and peripheral) suitable for general neurologists, physicians, pediatricians, ENT specialists, and trainees.

Ambar Chakravarty is an Honorary Professor and Emeritus Consultant in the Department of Neurology at the Vivekananda Institute of Medical Sciences, Kolkata, West Bengal, India. A recipient of several awards, at both national and international levels, and Lifetime Achievement Awards, Dr Chakravarty is a Fellow of the Royal Colleges of Physicians of London and Edinburg, Royal College of Physicians and Surgeons of Canada, Indian Academy of Neurology, and Indian College of Physicians. He had published extensively in national and international peer-reviewed journals on varied neurological topics ranging from headaches, epilepsy, movement disorders, ataxias, neuro-ophthalmology, neurological histories to neuroscience of art and creativity. He had been the Founder Editor of the journal of Association of Neuroscientists of Eastern India (1996–2006) and an Editor-in-Chief of Frontiers in Neurology (2012–2016). He has published 12 monographs on neurological topics and had delivered around 20 orations so far. He is a Past President of the Indian Academy of Neurology (2005–2006).

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