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Neurological conditions in Charaka Indriya Sthana - An explorative study

Abstract

Ayurveda is a traditional Indian system of medicine and 'Charaka samhita' has been the most popular referral treatise for Ayurvedic academicians, clinicians and researchers all over the world. 'Indriya sthana' is one among the 8 sections of 'Charaka samhita' and it comprises of 12 chapters which deals with prognostication of life expectancy based on 'Arishta lakshanas' (fatal signs and symptoms which indicates imminent death). Arishta lakshanas are the fatal signs which manifests in a diseased person before death. Various neurological conditions are mentioned throughout 'Charaka Indriya sthana' in a scattered way. The present study attempts to screen various references pertaining to neurological conditions of 'Charaka Indriya sthana' and explore their rationality, clinical significance and prognostic importance in present era. Various references related to neurological conditions like, 'Neuropathies', 'Neuro-ophthalmological disorders', 'Neurocognitive disorders', 'Neuromuscular disorders', 'Neurodegenerative disorders', 'Lower motor neuron syndromes', 'Movement disorders' and 'Demyelinating disorders' are mentioned in 'Charaka Indriya sthana'. The neurological conditions mentioned in 'Charaka Indriya sthana' are characterized by poor prognosis, irreversible pathology, progressive in nature and commonly found in dying patients or at the end-of-life stages. It seems that neurological conditions mentioned in 'Charaka Indriya sthana' have clinical applicability and prognostic significance in present era also. Further studies are required to substantiate the clinical findings mentioned in 'Charaka Indriya sthana'.

Keywords: demyelinating disorders, neurocognitive disorders, neurodegenerative disorders, neuromuscular disorders, neuro-ophthalmological disorders, neurovascular disorders

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Abbreviations: DPN, diabetic polyneuropathy; TLE, temporal lobe epilepsy; PWS, prader-willi syndrome; PD, parkinson's disease; DLB, dementia with Lewy bodies; AVH, auditory verbal hallucinations; NES, non-epileptic seizures; BHS, breath-holding spells; GTCS, generalized tonic clonic seizures; SUDEP, sudden unexpected death in epilepsy; EEG, electroencephalogram; SE, Status epilepticus; PME, progressive myoclonic epilepsy; PMA, progressive myoclonic ataxia; CNS, central nervous system; RLS, restless legs syndrome; TS, tourette's syndrome; SCI, spinal cord injury; LMN, lower motor neuron; UMN, upper motor neuron; FNP, Facial nerve palsy; MS, Multiple sclerosis; GBS, guallian-Barre syndrome; MG, myasthenia gravis; CVA, cerebrovascular accident; NMO, neuromyelitis optica; PRS, parry romberg syndrome; BAD, brachial amyotrophic diplegia; LAD, leg amyotrophic diplegia; NNMD, neurological and neuromuscular disorders; NMD, neuromuscular disease; DMD, duchenne muscular dystrophy; DM 1, myotonic dystrophy type 1; ALS, amyotrophic lateral sclerosis; HD, huntington's disease; SD, seborrheic dermatitis; PSP, Progressive supranuclear palsy; MSA, multiple system atrophy; CBD, corticbasal degeneration; AIDS, acquired immunodeficiency syndrome; EP, exploratory procedures; ABS, anton Babinski syndrome; AIWS, Alice in wonderland syndrome; VPD, visual perceptual distortions; RHS, ramsay Hunt syndrome; PTSD, post traumatic stress disorder; HS, horner syndrome; CPEO, chronic progressive external ophthalmoplegia; MFS, miller fisher syndrome; SCD, spino cerebellar degernation; AD, alzheimer's disease or alzheimer's dementia; ADHD, attention-deficit/hyperactivity disorder; PRES, Posterior reversible encephalopathy syndrome; SOL, space occupying lesion; SSPE, subacute sclerosing panencephalitis; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus; SMA, Spinal muscular atrophy; CMT, charcot Marie Tooth disease; DHS, dropped head syndrome; CIDP, Chronic inflammatory demyelinating polyneuropathy; LEMS, lambert-Eaton myasthenia syndrome; FSHD, facioscapulohumeral dystrophy; INEM, isolated neck extensor myopathy; MND, motor neuron disease; PBP, Progressive bulbar palsy; PLS, primary lateral sclerosis; OD, oropharyngeal dysphaiga; LNS, lesch-Nyhan syndrome; REM, rapid eye movement; OGC, occulogyric crisis; NMS, Neuroleptic malignant syndrome; SC, sydenham's chorea; FOSMN, Facial onset sensory and motor neuronopathy; TIA, transient ischemic attack; FND, functional neurological disorder; RCVS, reversible cerebral vasoconstrictive syndrome

Introduction

Ayurveda is a traditional Indian system of medicine which has great antiquity dating back to about 5000 years BC. It has stood the test of time for four millennia or more.[1] *Charaka samhita* is the most authoritative and comprehensive compendium of Ayurvedic knowledge. This treatise is considered as the oldest available literature of Ayurveda.[2] Charaka samhita has 8 sections (sthana) which contain a total number of 120 chapters. It is estimated to be redacted almost contemporaneously with Aristotle and it is the most popular and referral text book of medicine for Ayurvedic academicians, clinicians and researchers all over the world.[3] 'Indriya sthana' is one among the 8 sections of Charaka samhita.

'Indriya sthana' of 'Charaka samhita' comprises of 12 chapters which deals with prognostication of life expectancy based on 'Arishta lakshanas' (fatal signs and symptoms which indicates imminent death). Arishta lakshanas are the fatal signs which definitely occurs in

a diseased person (sometimes even in healthy persons also) just before death. Physician should be alert to identify the arishta lakshanas whenever they manifest, incorporate them in calculating remaining life expectancy and also in clinical decision making.[4] 'Charaka Indriya sthana' holds ample of references of various clinical conditions which are having poor prognosis, fatal and generally seen in patients who are at the end-of-life stages. Various neurological conditions are mentioned throughout 'Charaka Indriya sthana' in a scattered way.[5–16] The present study attempts to screen various references pertaining to neurological conditions of 'Charaka Indriya sthana' and explore their rationality, clinical significance and prognostic importance in present era. The neurological conditions (except 'Dementia', 'Delirium' and neuropsychiatric conditions) of 'Charaka Indriya sthana' have been explored in the following sections (Table 1).

Table 1 Neurological conditions in *Charaka indriya sthana*

References in *Charaka indriya sthana*	Relevant neurological conditions
Neuropathies	
'Swedanubandha sweda stambho vaa' (Ch. I. 3 / 4)	Hyperhidrosis; Anhidrosis; Dyshidrosis;
'Kesha lomaani – na chet vedayeyu' (Ch. I. 3 / 6)	Hypoestheisa; Hypoalgesia; Peripheral neuropathy; Diabetic polyneuropathy (DPN);
Seizures & Movement disorders:	
'Asattama pashyati – badhyate' (Ch. I. 5 / 21)	Epilepsy; Temporal lobe epilepsy (TLE); Advanced dementia;
'Hrasvam cha ya – punarvasu' (Ch. I. 7 / 25)	Status epilepticus (SE); Non-epileptic seizures (NES); Sudden unexpected death in epilepsy (SUDEP); Generalized tonic-clonic seizures (GTCS); Cortical myoclonus;
'Ghattayajjanuna – na sa jeevati' (Ch. I. 8 / 17)	Movement disorders; Cerebrovascular events; Hyperactive subtype of delirium; Cortical myoclonus; SE;
'Muhurhasan – na sa jeevati' (Ch. I. 8 / 20)	Restless legs syndrome (RLS); Sensory aspects of movement disorders; Psychogenic movement disorders;
'Shiro vikshipate – chyuta bandhana' (Ch. I. 8 / 26)	Versive seizures; Post-traumatic syringomyelia; Spinal cord injury (SCI) at the level of cervical spine; Cervical dystonia with focal hyperhidroiss;
Paralysis / Plegia / Paresis:	
'Gulpha chyuta' (Ch. I. 3 / 5)	Foot drop due to central or peripheral causes;
'Bhru chyuta' (Ch. I. 3 / 5)	Horner syndrome; Facial nerve palsy (FNP); Bell's palsy;
'Vyavrutta murdha – preta stathaiva sa' (Ch. I. 7 / 29)	Idiopathic facial nerve palsy;
'Bhruvau yasya chyutau – jeevitam' (Ch. I. 10 / 6)	Multiple sclerosis (MS); Cerebrovascular accidents (CVA); Neuromyelitis optica (NMO); Aneurysms;
'Varna bhedhena – vyakhyaataa' (Ch. I. 1 / 10)	Hemiplegia; Paraplegia; FNP; Bell's palsy
'Stabdhaa nishchetana – visarpini' (Ch. I. 8 / 14)	Neurological and neuromuscular disorders (NNMD); Amyotrophic lateral sclerosis (ALS); Progressive bulbar palsy (PBP);
'Bhedham swaro' (Ch. I. 12 / 51)	Dysphonia due to recurrent laryngeal nerve involvement; Vocal cord paralysis;
Parkinsonism:	
'Pakshmaani jataabadhaani' (Ch. I. 3 / 6)	Seborrheic dermatitis (SD) in Parkinson's disease (PD);
'Shareera kampa – na jeevati' (Ch. I. 11 / 10)	PD; Ataxia; Atypical parkinsonian syndromes;
'Gomaya choornaabham – tasya jeevitam' (Ch. I. 12 / 3)	PD; Immunocompromised state associated with PD and SD;
'Nikashanniva – vasati maanava' (Ch. I. 12 / 4)	Parkinsonian gait;
Agnosia & Perceptual disorders or abnormalities:	
'Sameepe chakshushe – kaalachodita' (Ch. I. 11 / 18-19)	Agnosia; Somato-sensory impairment;
'Aahvayastham – na pashyati' (Ch. I. 11 / 21)	Prosopagnosia; Visual agnosia; Delirium;
'Jale suvimale – parimuchyate' (Ch. I. 4 / 9)	Visual perceptual distortions (VPDs); Alice in wonderland syndrome (AIWS);
'Viparyayena – gataayushaam' (Ch. I. 4 / 21)	Anosmia, Parosmia, Cacosmia & Dysosmia;
'Yo rasanna vijaanaati – kushalo naram' (Ch. I. 4 / 22)	Ageusia, Hypogeusia, Pargeusia, Dysguesia & Phantogeusia;
'Ushnaan sheetaan – manyate' (Ch. I. 4 / 23)	Lack or impaired discriminative tactile sensation;
'Shabda sparsha – pratikarma pravruttishu' (Ch. I. 12 / 58)	Age related sensory impairment; Hallucinations; Organic psychosis;

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Table Continued...

References in *Charaka indriya sthana*	Relevant neurological conditions
Neuro ophthalmological conditions:	
'Ati pravishthe' (Ch. I. 3 / 6)	Horner's syndrome;
'Ati jihme' (Ch. I. 3 / 6)	Strabismus; Squint;
'Ati mukta bandhane' (Ch. I. 3 / 6)	Ptosis; Ophthalmoplegia; Myasthenia gravis; Cranial nerve (III, IV & VI) palsy; Guillain-Barre syndrome (GBS);
'Satata unmeshite' (Ch. I. 3 / 6)	Facial palsy;
'Satata nimeshite' (Ch. I. 3 / 6)	Ptosis; Horner's syndrome; Myasthenia gravis; Cranial nerve (III) palsy; Ocular myopathies;
'Vibhranta drishtike' (Ch. I. 3 / 6)	Strabismus; MS; Vitamin B_{12} deficiency; Nystagmus; Neurodegenerative disorders;
'Vyasta drishtike' (Ch. I. 3 / 6)	MS; Seizures; Neurodegenerative disorders;
'Heena drishtike' (Ch. I. 3 / 6)	Neuro-ophthalmologic diseases; Neurovascular pathology;
Neuromuscular conditions:	
'Tatra prakruta – prashastam' (Ch. I. 1 / 15&16)	Lower motor neuron (LMN) lesions; ALS; Myasthenia gravis (MG);
'Na bhibharti shiro – mumurshata' (Ch. I. 8 / 22)	Neuromuscular disorders (NMDs);
'Na upaiti kantham ahaaro – pariheeyate' (Ch. I. 8 / 25)	NMDs; Oropharayngeal dysphagia; Dystonia;
'Hasta paadam mukham – na jeevati' (Ch. I. 11 / 12)	NMDs; Myopathies; Neuropathies; Muscular dystrophies;
'Peyam paatum – na sa jeevati' (Ch. I. 9 / 11)	Dysphagia in NMDs; Dehydration due to dysphagia;
Others:	
'Stabhyete pratibuddhasya – asamshayam' (Ch. I. 5 / 24)	Tetanus;
'Hanu manya graha – parivarjayet' (Ch. I. 6 / 20)	Tetanus;
'Uthapyamaana – vikathana' (Ch. I. 7 / 19)	Pre syncope; Classic orthostatic hypotension;
'Urdhwagre nayane – na sa jeevati' (Ch. I. 7 / 27)	Hypoxic encephalopathy; Oculogyric crisis (OGC); Movement disorder emergencies;
'Aayamya utpaatitaan – naativartate' (Ch. I. 8 / 8)	Syringomyelia; Silent neuropathy; Facial onset sensory and motor neuronopathy syndrome (FOSMN);
'Yam naram sahasaa – tasya manyate' (Ch. I. 9 / 15)	MS; Alzheimer's disease (AD); Dementia of Lewy bodies (DLB); Reversible cerebral vasoconstriction syndrome (RCVS); Transient ischemic attack (TIA); Functional neurological disorders (FND);
'Pindike shithilikrutya – jeevitam' (Ch. I. 10 / 5)	Neurosyphilis;
'Ahaasya haasi – na sa jeevati' (Ch. I. 11 / 20)	Cerebrovascular diseases; Advanced dementia; Delirium;

(Ch. I. XX / YY): Ch - Charaka samhita; I - Indriya sthana; XX - Chapter number; YY - Verse number;

Review methodology

Ayurvedic literature regarding 'Indriya sthana' has been collected from Charaka samhita, including 'Ayurveda dipika' commentary by Chakrapani. Electronic databases 'Google search' and 'Google scholar search' have been searched to find out the relevant studies and reviews published till 'June 2020', irrespective of their appearance / publication year. The key words used for searching were, 'Charka samhita', 'Indriya sthana', 'Charaka indriya sthana', 'Neuromuscular disorders', 'Neuro-ophthalmological disorders', 'Neurocognitive disorders', 'Demyelinating disorders', 'Neurodegenerative disorders', 'Agnosia', 'Perceptual distortions', 'Visual illusions', 'Parkinsonism', 'Tetanus', 'Cerebrovascular diseases', 'Neuropathy', 'Seizures', 'Epilepsy', 'Lower motor neuron syndrome', 'Neurological conditions', 'Paralysis', 'Plegia', 'Paresis' and other relevant terms. Abstracts and full text, open access articles in English language were only considered. 'Dementia', 'Delirium' and other 'Neuropsychiatric conditions' were excluded from present study.

Neurological conditions

Various references related to neurological conditions (except neuropsychiatric conditions) like, 'Neuropathies', 'Neuro-ophthalmological disorders', 'Neuromuscular disorders', 'Neurodegenerative disorders', 'Lower motor neuron syndromes', and 'Demyelinating disorders' are mentioned in 'Charaka Indriya sthana' (Table 1).

Neuropathies

'Swedanubandha sweda stambho vaa' (Verse 4)[7]

'Swedanubandha' denotes 'Hyperhidrosis' or excessive sweating whereas 'Sweda stambha' denotes 'Anhidrosis' or absence of sweating. Enhanced sweating (hyperhidrosis), can be generalized or focal. Secondary hyperhidrosis develops due to dysfunction of the central or peripheral nervous system. Secondary focal hyperhidrosis is the result of central or peripheral neuronal

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defects. Peripheral causes for secondary focal hyperhidrosis are neuropathies (diabetic neuropathy). Sweating may be peripherally increased (swedanubandha) at the onset of a polyneuropathy and may disappear (sweda stambha) as nerve damage progresses. In cerebral infarctions or haemorrhages, hyperhidrosis (swedanubandha) occur contralaterally to the lesion. In spinal lesions, sweating is reduced or completely absent (sweda stambha) ipsilaterally by that compensatory hyperhidrosis (swedanubandha) develops on the remaining body parts. In posttraumatic syringomyelia, hyperhidrosis (swedanubandha) in the affected area may be one of the first symptoms. In Harlequin syndrome and Frey's syndromes special forms of focal hyperhidrosis occurs. Harlequin syndrome is characterized by erythema and hyperhidrosis (swedanubandha) develop unilaterally with contralateral anhidrosis (sweda stambha). In Frey's syndrome focal gustatory sweating occurs (swedanubandha).[17] Anhidrosis (sweda stambha) can be the presenting symptom in a wide variety of conditions such as leprosy, diabetic neuropathy, thyroid dysfunction, Sjögren syndrome, ectodermal dysplasia, and autoimmune diseases etc.[18] Ross syndrome is a progressive degenerative disorder and anhidrosis is one of the characteristic features of this syndrome. Anhidrosis (sweda stambha) may be accompanied by compensatory hyperhidrosis (swedanubandha) and other alterations of the autonomic nervous system in Ross syndrome.[19] It is evident that the above verse denotes hyperhidrosis and anhidrosis which manifests due to various underlying neurological or neurovascular pathology.

'Tasya chet kesha lomaani aayamyamaanaani praluchyeranna chet vedayeyu tam paraasuriti vidyaat' (Verse 6).[7]

The above verse denotes decreased or absence of sensitivity to pain (na chet vedayeyu). The person is unable to feel pain when the hair is plucked out or pulled. In most patients with peripheral neuropathy, loss of sensation (na chet vedayeyu) is directly attributable to kind, severity, and distributed loss of these sensory receptors, nerve fibers, or neurons.[20] Diabetes causes a wide variety of acute or chronic, focal or diffuse neuropathy syndromes. The commonest is DPN (Diabetic polyneuropathy) and other patterns of nerve injury include diabetic autonomic neuropathy, cranial neuropathy, mononeuritis multiplex, mononeuropathy, radiculoplexus neuropathies, and diabetic neuropathic cachexia. Patients describe a range of sensory symptoms like loss of pain sensation (na chet vedayeyu) or "Novocain-like" insensitivity, tingling, "pins and needles" sensation, burning, "electric shocks", and allodynia, or hyperalgesia.[21] The above verse may also denote 'Painless - DPN.[22] Large fiber neuropathy manifests with the loss of joint position, vibration sense and sensory ataxia, whereas small fiber neuropathy manifests with the impairment of pain, temperature and autonomic functions. Sensory symptoms such as numbness and loss of sensation in hands and feet (na chet vedayeyu) are considered as negative symptoms of peripheral neuropathy.[23] The above verse indicates peripheral neuropathy due to various underlying pathological conditions.

Seizures & movement disorders

'Asattama pashyati ya shrunotyapyasata svanaan bahun bahuvidhaan jagruta soapasmaarena badhyate' (Verse 21)[19]

Patient with 'Temporal lobe epilepsy' (TLE) may present with a dreamy state, delusions with anxiety, complex audiovisual hallucinations, elementary auditory hallucinations (shrunotyapyasata svanaan), and metamorphopsia (due to a large lateral temporal lobe lesion followed by ipsilateral hippocampal sclerosis).[24] Though visual hallucinations (asattama pashyati) associated with seizures are simple in nature, complex, formed visual hallucinations (asattama pashyati) are also seen as ictal, peri-ictal, and intra-ictal phenomena. Complex visual hallucinations (asattama pashyati) due to seizures are thought to require the involvement of the visual association cortex.[25] Hallucinations are associated with a wide range of medical conditions like thyroid disease, Hashimoto disease, deficiencies in D and B12 vitamins, Prader–Willi syndrome (PWS), autoimmune disorders, acquired immunodeficiency disorders such as HIV/Acquired immunodeficiency syndrome (AIDS), narcolepsy, tumors, traumatic brain injuries, epilepsy, neurodegenerative conditions, Parkinson's disease (PD), dementia with Lewy bodies (DLB) and cardiovascular events may also cause hallucinations in which brainstem regions, temporal, occipital, or temporo-parietal pathways are involved.[26] AVH (auditory verbal hallucinations) (shrunotyapyasata svanaan) can be seen in TLE. As a neurological disorder, epilepsy can create the biological threshold under which hallucinatory symptoms develop due to neurological abnormalities.[27] The above verse denotes visual (asattama pashyati) and auditory hallucinations (shrunotyapyasata svanaan) in a patient of epilepsy due to organic brain pathology.

'Hrasvam cha ya prashvasati vyaviddham spandate cha ya mrutameva tamaatreyo vyachachakshe punarvasu' (Verse 25).[11]

The above verse denotes 'Non epileptic seizures' (NES) such as 'Breath-holding spells (BHS) or 'Syncope' etc conditions.[28] Fast and shallow breathing along with GTCS (generalized tonic-clonic seizures) (vyaviddham spandate) can be seen in 'SUDEP' (sudden unexpected death in Epilepsy). Patients with SUDEP have shown varying patterns of respiratory and bradyarrhythmic cardiac dysfunction with profound EEG (electroencephalogram) suppression.[29] Respiratory abnormalities (hrasvam cha ya prashvasati) like apnea, oxygen desaturation, and peri-ictal hypopnea etc. Peri-ictal respiratory dysfunction is common in individuals with epilepsy. Extreme hypoventilation (hrasvam cha ya prashvasati) in the presence of chest wall movements due to paradoxical breathing, airway obstruction, and shallow breathing can be found in SUDEP cases.[30] SE (Status epilepticus) (vyaviddham spandate) is the most common form of SE. Myoclonic SE (vyaviddham spandate) presents as a bilateral massive myoclonus that follows severe hypoxic-ischemic insult, viral encephalitis, and prion disease etc is associated with poor prognosis.[31] Focal cortical myoclonus (vyaviddham spandate) almost always points to an underlining lesion of the sensori-motor cortex, which produces hyperexcitability (e.g. vascular, inflammatory or neoplastic). Posthypoxic myoclonus (Lance–Adams syndrome), progressive myoclonic epilepsies (PMEs), progressive myoclonic ataxias (PMAs) and neurodegenerative diseases are the examples of 'Multifocal cortical myoclonus'. Metabolic derangements such as renal failure, hepatic failure, respiratory failure (hrasvam cha ya prashvasati), glycaemic disturbances, electrolytic disturbances, hyperthyroidism, metabolic alkalosis or acidosis, vitamin E deficiency, Hashimoto encephalopathy and hypoxia etc may cause symptomatic myoclonus.[32] It is evident that the above verse denotes conditions like SUDEP or NES or Cortical myoclonus.

'Ghattayajjanuna janu paadaaudyumna paatayan yoapasyati muhurvaktramaaturo na sa jeevati' (Verse 17)[12]

The above verse indicates various abnormal involuntary movements of the body (ghattayajjanuna janu paadaaudyumna paatayan) and face (muhurvaktram). Movement disorders with stroke occur due to the lesions of small vessel cerebro-vascular disease in the middle or posterior cerebral artery territories. Hemorrhagic strokes appear to be more likely to lead to movement disorders than ischemic ones. Hemiballism or hemichorea is the most common movement

disorder found to occur after stroke. Myoclonus can be seen followed by a stroke in numerous brain regions such as frontoparietal lobes, basal ganglia, midbrain, pons, and cerebellum.[33] The above verse denote various 'Hyperkinetic movements' such as dystonia, chorea, athetosis, myoclonus, tremor, tics, and stereotypies etc due to an underlying neurological disease.[34] As with dementia patients, patients with movement disorders are also predisposed to delirium in the presence of comorbid medical illness (e.g., infections, stroke, and cardiovascular events).[35] Hyperactive delirium is characterized by (motor) agitation, restlessness, and sometimes aggressiveness.[36] Delirium is an acute confusional state that is a life-threatening clinical syndrome and hyperactive behavior ranges (ghattayajjanuna janu paadaaudyumna paatayan) from simple restlessness to constant movement with agitation. The severe delirium classes with either hypoactive or mixed with hyperactive features psychomotor subtypes were predictive of increased risk for death.[37] Underlying etiology is the most powerful outcome predictive factor in SE patients. Many studies have found "acute symptomatic etiology", or "acute, life threatening aetiology" to be a strong predictor. SE associated with stroke and CNS (central nervous system) tumours have shown higher mortality[38] The above verse denotes various conditions like 'Movement disorders', 'Cortical myoclonus', 'SE', and 'Hyperactive subtype of Delirium' etc.

'Muhurhasan muhurkshvedan shayyaam paadena hanta yaucchai chhidraani vimrushyannaturo na sa jeevati' (Verse 20)[12]

Many basal ganglia disorders have psychiatric symptoms and movement disorders that may be bizarre or difficult to categorize (like Huntington disease, chorea-acanthocytosis, and Parkinson disease). Tremor and dystonia are the most common presentations of psychogenic movement disorders.[39] Movement disorders like Parkinson's disease, dystonia, Tourette's syndrome, restless legs syndrome, and akathisia etc are characterized by impaired motor control resulting due to dysfunction of the basal ganglia. They are also associated with behavioural (muhurhasan muhurkshvedan), psychiatric, autonomic, and other non-motor symptoms. Various sensory aspects (ucchai chhidraani vimrushyan) like 'sensory cueing' (PD), 'alleviating manoeuvres' (Dystonia), 'Premonitory urge phenomena' (Restless legs syndrome -RLS, Tics & Tourette's syndrome - TS), enhanced sensory perception (RLS), 'Self stimulatory behaviors', 'Stereotypies' and 'Proprioceptive & kinaesthetic dysfunction' etc can be seen in movement disorders.[40] RLS (shayyaam paadena hanta ya) is characterized by an urge to move the legs, usually due to feeling of uncomfortable sensations in the legs. The symptoms begin or worsen during periods of rest or inactivity and they are partially or totally relieved by movement. Depression, anxiety, sleep deprivation, mood disorders (muhurhasan muhurkshvedan), fatigue and poor concentration etc are frequently found in RLS patients.[41] The above verse denotes 'RLS with mood disorder'.

'Shiro vikshipate krichhraan munchayitva prapaanikau lalaata sruprata swedo mumurshu chyuta bandhana' (Verse 26)[12]

Following cerebral infarctions or hemorrhages, due to the failure of cortical inhibitory centers leads to hyperhidrosis (lalaata sruprata swedo) contralaterally to the lesion. After spinal lesions, sweating is reduced ipsilaterally or may even be completely absent, so that compensatory hyperhidrosis (lalaata sruprata swedo) develops on the remaining body parts. In posttraumatic syringomyelia, hyperhidrosis (lalaata sruprata swedo) in the affected area may be one of the first symptoms. In Harlequin syndrome, hyperhidrosis (lalaata sruprata swedo) develop unilaterally. In Frey's syndrome—focal gustatory sweating may develop after surgical procedures, tumors and in patients with lesions of the parotid salivary gland with damage to the facial nerve.[42] Focal hyperhidrosis (lalaata sruprata swedo) with neck rigidity or stiffness (shiro vikshipate krichhraan) can be seen in various conditions like versive seizures, atonic seizures, occipital lobe epilepsy, spinal cord injury (SCI) at the level of cervical spine, intramedullary spinal cord tumours, and post-traumatic syringomyelia etc.[12]

Paralysis, Paresis & Plegia:

'Gulpha chyuta' (Verse 5)[7]

The above verse denotes 'Foot drop' with central or peripheral etiology. Foot drop is defined as a weak anterior tibialis muscle and is usually caused by lower motor neuron (LMN) disease. Common causes are L4-L5 radiculopathy (due to a herniated nucleus pulposus or foraminal stenosis) and peroneal peripheral neuropathy. Other causes include any axonal or demyelinating damage along the whole peripheral nervous system. Central nervous system pathology such as spinal upper motor neuron (UMN) pathology, mass lesions, anterior cerebral artery stroke, and myelopathy etc can also cause foot drop[43] Metabolic disease as well as neurodegenerative, neuromuscular and inflammatory processes can also cause foot drop.[44]

'Bhru chyuta' (Verse 5)[7]

The above verse denotes 'drooping of eyebrow'. Drooping of eye brow on the affected side can be seen in Bell's palsy.[45] The ipsilateral upper eyelid appears slightly drooped due to paresis of the Müller muscle in the patients of 'Horner's syndrome'.[46] Patients with 'Facial nerve palsy' (FNP) may typically present with brow ptosis (bhru chyuta), lagophthalmos, ectropion, and exposure keratopathy along with nasal alar collapse, nasolabial flattening, drooping of the corner of the mouth, and drooling. Systemic and metabolic disorders such as diabetes mellitus, hypertension, amyloidosis, and sarcoidosis may lead to FNP. Multiple sclerosis (MS), Guillain-Barre syndrome (GBS), myasthenia gravis (MG), and cerebrovasclar accident (CVA) comprise some neurological causes of FNP.[47]

'Vyavrutta murdha jihvaasyo bhruvau yasya cha vichyute kantakaischachita jihvaa yathaa preta stathaiva sa' (Verse 29)[11]

Facial nerve palsy (FNP) is the most common cranial nerve disease. Its idiopathic form (Bell's palsy) accounts for 60–75% of cases. Idiopathic FNP usually manifests as sudden weakness of the muscles of facial expression on one side of the face. The typical features of peripheral FNP are a lack of wrinkling of the forehead (vyavrutta murdha), low eyebrow position (eyebrow ptosis) (bhruvau yasya vichyuta), incomplete lid closure, hanging corner of the mouth (vyavrutta aasya), and a flattened nasolabial fold.[48] The most prevalent causes of secondary FNP are systemic viral infections, trauma, surgery, diabetes, local infections, tumor, immunological disorders, or drugs.[49] Melkersson-Rosenthal is a rare neuromucocutaneous syndrome characterized by recurrent facial paralysis, fissured tongue (lingua plicata) (kantakaischachita jihvaa), orofacial edema.[50] The above verse denotes FNP either primary or secondary or idiopathic in origin.

'Bhruvau yasya chyute sthaanaat antardaahashcha daaruna tasya hikkakaro roga sadyo mushnaati jeevitam' (Verse 6).[14]

Atrial pacing, aortic aneurysm (thoracic or abdominal), myocardial infarction, pericarditis, and temporal arteritis are the cardiovascular causes for persistent and intractable hiccups (hikkakaro) whereas

aneurysms (especially posterior inferior cerebellar artery), encephalitis, lateral medullary syndrome, meningitis, MS, neuromyelitis optica (NMO), neoplasms (astrocytoma, brain stem tumor), Parkinson disease, seizure, stroke, syringomyelia, and vascular malformations (cavernoma) are the central nervous system related causes for intractable hiccups (hikkakaro).[51] Bilateral ptosis (bhruvau yasya chyute sthaanaat) can be seen in midbrain infarctions. Neurogenic ptosis (bhruvau yasya chyute sthaanaat) occurs due to the damage of the oculomotor or sympathetic nerve, or central nervous system abnormalities. Other conditions that frequently affect the oculomotor nerve are diabetes, tumors, aneurysms, multiple sclerosis, and trauma.[52] Inflammatory demyelinating diseases (including NMO & MS) and acute and recurrent myelitis are the causes for intractable hiccups (hikkakaro).[53] MS is an inflammatory disease of the CNS, which is associated with demyelination and neurodegeneration. Pain has been recognised as a symptom of MS and can broadly be classified as nociceptive or neuropathic. Visceral pain (antardaahashcha daaruna) can be found in MS.[54] Dysesthesia or burning sensation or burning type of pain (antardaahashcha daaruna) can be seen in MS.[55] The above verse denote various conditions like MS, NMO, CVA, and aneurysms etc.

'Varna bhedhena glaani harsha raukshya snehaa vyakhyaataa' (Verse 10)[5]

Instantaneous manifestation of pathological features (like weakness, roughness and smoothness etc) in half of the body (either upper or lower half, right or left half, anterior or posterior half and internal or external body surfaces) should be considered as 'Arishta' (fatal and indicates imminent death). The word 'Glaani' denotes weakness or atrophy or paralysis or fatigue or exhaustion etc; the word 'Harsha' in the present context denotes 'Normal or physiological' half of the body. 'Ruksha' and 'Senha' denotes roughness or stickiness in half of the body. Half of the body or face may get affected by 'Glaani' and another half by 'Harsha' in a same patient. Similarly 'Ruksha' and 'Sneha' also affects half of the body or face.

Muscle loss (glaani) is a severe complication due to many medical conditions and nerve damage is one amongst them. Chronic constriction injury to the nerve may hamper the motor function and also leads to skeletal muscle atrophy (glaani).[56] One side of the face becomes paralyzed (for both voluntary and involuntary movement) (glaani), and the forehead is affected as much as the lower face in Bell's palsy. In facial hemiatrophy (which is not caused by cranial nerve VII lesion), there is disappearance of fat (glaani) in the dermal and subcutaneous tissues on one side of the face. Facial hemiatrophy is actually a form of lipodystrophy.[57] Parry-Romberg syndrome (PRS), which is also known as progressive hemifacial atrophy (glaani), is a rare and poorly understood condition. In PRS, the face shows unilateral, slowly progressive atrophy. Rarely, PRS can progresses and involves one half of the body.[58] A slow, lower motor neuron variant affects the arms is termed as 'Brachial amyotrophic diplegia' (BAD) and affects the legs is termed as 'Leg amyotrophic diplegia' (LAD).[59] The above verse denotes various conditions like hemiplegia, paraplegia, diplegia, facial palsy and LMN syndromes etc.

Stabdhaa nishchetana gurvi kantakopachita bhrushamshyaava shushka athava shunaa preta jihwa visarpini' (Verse 14).[12]

Pathological features like stabdhata (rigid or stiff), nishchetana (lack of movements), gurvi (heavy), kantakopachita (rough surface), shyaava (brownish discoloration), shushka (atrophy), shuna (swelling) and visarpini (protruding) of the tongue denotes imminent death as per the above verse. Various neurological and neuromuscular disorders (NNMD) can alter the tongue movements (nishchetana), thickness (shushka / shuna) and tongue pressure.[60] Reduced tongue pressure, enlarged tongue (gurvi) and bulbar muscular atrophy (shushka) are seen in patients with neuromuscular diseases (NMDs) such as Duchenne muscular dystrophy (DMD), myotonic dystrophy type 1 (DM1), and amyotrophic lateral sclerosis (ALS).[61] Various other conditions like CVA, MS, Parkinsonism, Huntington's disease (HD), Bell's palsy, and MG etc can also cause dysfunction of the tongue.[62] Damage of CN XII (hypoglossal nerve) causes tongue weakness, atrophy (shushka), fasciculations, and a deviation (stabdha / visarpini) toward the affected side when the tongue is stuck out. Mild dysarthria and loss of taste and sensation of the inside tongue can be seen in CN IX (glossopharyngeal nerve) and X (vagus nerve) palsy, and the deviation of the tongue on protrusion (visarpini) and tongue weakness are seen in CN XII palsy.[63] The above verse denotes various conditions like NMDs or cranial nerve palsies.

'Bhedham swaro' (Verse 51)[16]

The above verse denotes dysphonia (bhedham swaro) due to neurological causes. Vocal cord paralysis may be partial or complete, caused by damage to the recurrent laryngeal nerve; a dysphonia arises from the incomplete glottic closure or irregular vibration of the vocal cords. The majority of vocal cord paralyses are due to trauma in the region of the vagus nerve or the recurrent laryngeal nerve.[64]

Parkinsonism

'Pakshmaani jataabadhaani' (Verse 6).[7]

Skin disorders, such as seborrheic dermatitis (SD) and hyperhidrosis, are well recognized and frequent nonmotor symptoms of PD. Seborrheic facies presents as typical sharply demarcated red patches and plaques with greasy scales (jataabadhaani) in areas that have an increased density of sebaceous glands such as the scalp, face, hairline, eyebrow, glabella, nasolabial folds, ears, upper trunk, and flexures. It usually affects multiple body areas, most commonly face, scalp, chest, arms and legs.[65] The above verse denotes a condition of excessive greasiness or seborrhea which leads to matting of eye lashes. Matting of eye lashes can be seen in the patients of PD due to seborrhea.

'Shareera kampa sammoho gatirvachanameva cha mattasyeva upalabhyante yasya maasam na jeevati' (Verse 10).[15]

Parkinsonism is defined as a hypokinetic syndrome and is characterized by the presence of resting tremor (kampa), muscular rigidity, bradykinesia or akinesia, and postural instability (sammoho gati / mattasyeva). Patients may present with a parkinsonian syndrome that has atypical features like early dementia (sammoho), frequent falls (mattasyeva gati), ocular dysmotility, prominent dysautonomia, or ataxia (mattasyeva gati). Progressive supranuclear palsy (PSP), multiple system atrophy (MSA), corticobasal degeneration (CBD), and DLB, as well as other rarer causes are called as atypical parkinsonian syndromes. Gait in PSP is characteristically stiff, broad based, with knees extended and arms abducted. It is often described as clumsy like a "drunken sailor" or "dancing bear" (mattasyeva gati). Bulbar features such as progressive dysarthria that is often spastic or hypernasal, hypokinetic, and monotonous. Speech can be slow and can include stuttering, echolalia, and occasional involuntary vocalizations (sammoho vachanam). Cognitive decline (sammoho)

and dementia occur with disease progression. A frontal subcortical dementia is typical, with slowed processing, or bradyphrenia, reduced verbal fluency, and executive dysfunction (mattasyeva / sammoho gati & vachanam).[66] The above verse denotes various conditions like 'Atypical parkinsonian syndromes', 'Advanced dementia', 'Delirium' and 'Ataxia' etc.

'Gomaya choornaabham choornam murdhani jaayate sasneham bhrashyate chaiva masaantam tasya jeevitam' (Verse 3).[16]

SD is a common benign inflammatory condition of the skin which mainly affects lipid-rich regions of the head (murdhani jaayate) and trunk. SD is caused by over proliferation of the lipophilic (sasneham) fungus *Malassezia*. PD and SD are strongly associated. SD occurs mainly in lipid-rich skin regions (sasneham), especially the face, trunk and scalp (murdhani jaayate). The strong epidemiological association between PD and SD suggests these two diseases share risk factors or underlying mechanisms. The best established shared risk factor is AIDS (masaantam tasya jeevitam), which greatly increases the risk of both parkinsonism and SD.[67] The above verse denotes an immunocompromised condition associated with SD and PD.

'Nikashanniva ya padau chyutaamsa paridhaavativikrutya na sa loke asmin chiram vasati maanava' (Verse 4).[16]

Shuffling gait, impaired balance, and freezing of gait (nikashanniva ya padau) are main motor dysfunctions shown in patients with PD. Postural control and gait dysfunction such as slowing of gait (nikashanniva ya padau), reduced arm swing, shorter step length, postural instability, and loss of disassociated arm and trunk movements during gait can be seen in PD.[68] Stooped posture is an abnormal posture marked by shoulder dropping (chyutaamsa) and head bowing. Gait festination is one of the characteristic features of PD characterized by quicker (paridhaavati) and shorter steps.[69] The above verse indicates parkinsonian gait.

Agnosia & perceptual abnormalities

'Sameepe chakshusho krutvaa mrugayet angulikaramsmayate chaapi kaalaandha urdhwagaanimishekshanashayanaadaasanaatangaat kaashtaat kudyaadapi vaasanmrugayate kinchit sa muhyan kaalachodita' (Verse 18-19)[15]

The above verse denotes somatosensory impairment with gaze dysfunction due to an underlying neurological pathology. Asomatognosia is a condition in which patient fees that, parts of his or her body are "missing". Asomatognosia may be modified by touching (mrugayet angulikaram) the missing body part or by looking at it (sameepe chakshusho krutvaa). Somatosensory impairments (sa muhyan) range from primary deficits in tactile detection, perception of features, haptic object recognition and bodily experiences. A higher level in the hierarchical processing of somatosensory input concerns the discrimination of the haptic features of an object (texture, substance, size, shape, weight and the hardness of a stimulus) (impaired recognition of haptic features of an object) (shayanaadaasanaatangaat kaashtaat kudyaadapi va asanmrugayate). Amorphognosia, Ahylognosia, Asomatognosia (mrugayet angulikaram), Somatoparaphrenia, Autotopagnosia, finger agnosia (unable to identify his own fingers) (mrugayet angulikaram), tactile apraxia etc are some somatosessory impairments (shayanaadaasanaatangaat kaashtaat kudyaadapi va asanmrugayate) seen in stroke patients. Different types of hand movements (exploratory procedures or EPs) (mrugayet) are used to discriminate a particular dimension of a stimulus. The ability to perform EPs (mrugayet) needed for haptic object recognition can be selectively disturbed (asanmrugayate kinchit sa muhyan) in stroke patients. Vertical gaze palsy or paresis involving both upward (urdhwagekshana) and downward gaze with lid retraction (animishekshana) has been seen in stroke patients. Cortical blindness (kaalaandha) results in binocular vision loss due to insult to the occipital cortex. Anton-Babinski syndrome (Anton syndrome or ABS) is visual anosognosia (denial of loss of vision) associated with confabulation (defined as the emergence of memories of events and experiences which never took place) in the setting of obvious visual loss (kaalaandha) and cortical blindness. The above verse denotes various conditions like somatosensory impairments with gaze dysfunction commonly seen in stroke patients, dementia, neuropathies, spinal cord injuries, ABS and delirium etc.[15]

'Aahvayastham sameepastham swajanam janameva va maha mohavruta mana pashyannapi na pashyati' (Verse 21).[15]

The above verse denotes acute confusional state or inability to identify the people. Face recognition is generally effortless and rapid. Subjects with prosopagnosia, however, cannot recognize (pashyannapi na pashyati) that they have seen a face before, an impairment that affects both faces well known to them (swajanam) and also those recently encountered (janameva va). This is not due to more general problems with vision (pashyannapi na pashyati), object recognition, or memory. Patients with prosopagnosia realize that a face is a face and not a jeep or a wood, but simply cannot say whether they have seen it before or whose face it is (pashyannapi na pashyati).[70] Prosopagnosia may results from genetic, developmental and acquired brain injury (involving occipital-temporal and anterior temporal regions). Patients with autism, Williams's syndrome, schizophrenia, age-related cognitive decline and dementia may also suffer with face processing and social cognitive deficits.[71] Acute confusional state (maha mohavruta mana) or delirium is a clinical syndrome characterized by disturbed consciousness, cognitive function, and perception (maha mohavruta mana). It is often associated with serious adverse outcomes such as death, dementia, and also the need for long-term patient care.[72]

'Jale suvimale jaalamajaalavatate nara sthite gacchati va drushtvaa jeevitaat parimuchyate' (Verse 9).[8]

Patients with visual hallucinations and illusions secondary to degenerative eye disease have reported remarkably stereotyped experiences. 'Tessellopsia' is the term used for the patients who visualizes regular, repeating patterns such as brickwork, lattices, netting (jaalamajaalavatate), mosaics, chequer boards, wallpaper, grids, fences, roof-tiles, crazy paving and cobwebs (jaalamajaalavatate).[73] Alice in Wonderland syndrome (AIWS) is a perceptual disorder characterized by distortions of visual perception (metamorphopsias), the body schema, and the experience of time. It is characterized by derealization, depersonalization, hyperschematia, hyposchematia, and somatopsychic duality, as well as illusory changes in the size, distance, or position of stationary objects in the visual field, illusory feelings of levitation and illusory alterations in the sense of the passage of time. 'Kinetopsia' is a form of visual illusion in which stationary objects are visualized as moving (sthite gacchati va).[74] The above verse denotes visual illusions or visual perceptual distortions (VPD) due to various neurological, neuro ophthalmological or ophthalmological disorders.

'Viparyayena yo vidyaat gandhaanaam saadhvasaadhutaam na va taan sarvasho vidyaat tam vidyaat gataayushaam' (Verse 21).[8]

Olfactory distortions can occur in any of three ways, decreased sensitivity (hyposmia, anosmia) and two types of distortion (dysosmia)

(vidyaat gandhaanaam saadhvasaadhutaam), distorted quality of an odorant stimulation (troposmia) (viparyayena) and perceived odor when no odorant is present (phantosmia, hallucination) (na va taan sarvasho vidyaat).[75] Olfactory dysfunction can be caused by various etiologic factors. The two main types of olfactory dysfunctions are, 'conductive losses' (losses secondary to obstruction of the nasal airflow to the olfactory cleft) and 'sensory/neural losses' (losses secondary to damage or dysfunction of the olfactory nerves due to various conditions such as head trauma, toxins, congenital disorders, dementia, Alzheimer's disease, and multiple sclerosis).[76] The above verse denotes olfactory distortions due to 'sensory / neuronal losses'.

'Yo rasannavijaanaati na vaa jaanaati tatvatmukhapaakaadrute pakvam tamaahu kushala naram' (Verse 22).[8]

Pathology of the gustatory pathway might lead to the alteration of taste: partial loss (hypogeusia), complete loss (ageusia) (rasan na vijaanaati), or a sensation of altered taste (dysgeusia) (na vaa jaanaati tatvata). Taste distortion with stimulus (parageusia) or without stimulus (phantgeusia) also can occur. Various causes like viral, bacterial, fungal, or parasitic infections of the oral and hypopharyngeal mucosa, ageing process, poor oral hygiene, neoplasms, injury to the chorda tympani, chronic otitis media, Bell's palsy, Ramsay Hunt Syndrome (RHS), Lyme disease, and pathological involvement of cranial nerves IX and X etc can cause distortions in taste perception.[76]

'Ushnaan sheetaan kharaan shlakshaan mrudunapi cha daarunaan sprushyaan sprushtvaa tato anyatvam mumurshasteshu manyate' (Verse 23).[8]

It is known that roughness-smoothness (ruksha - snigdha), hardness-softness (daaruna - mrudu), stickiness-slipperiness (shlakshna) and warm-cold (ushna - sheeeta) are predominant perceptual dimensions in macro, micro and nano texture perception. Elder people have shown significantly reduced fine texture discrimination ability (sprushyaan sprushtvaa tato anyatvam).[77] Both decreased sensation and sensory perversions may result from the pathology of sensory receptors, peripheral nerves, dorsal root, dorsal root ganglia, dorsal columns, spinal cord, spinothalamic tracts, brain stem, thalamus and parietal cortex. Compressive and anoxic lesions affect proprioception, discriminative tactile sensation (sprushyaan sprushtvaa tato anyatvam), and fast and slow pain.[78] Tactile discrimination declines with age due to the decreased nerve conduction velocity, decreased density of Meissner's and Pacinian corpuscles, and gray matter changes within the central nervous system, and is also associated with cognitive decline.[79] The above verse denotes impaired discriminative tactile sensation due to an underlying neurological pathology.

'Shabda sparsha raso rupam gandha cheshta vichintitam utpadyante ashubhaaneva pratikarma pravruttishu' (Verse 58).[16]

The above verse denotes age related sensory impairment or hallucinations or organic psychosis or advanced stages of neurodegenerative conditions. Age related decline of the five classical senses (vision, smell, hearing, touch, and taste) poses significant burdens on older adults. Aging has long been associated with decline in sensory function, a critical component of the health. Individual sensory impairments are common such as hearing loss (shabda), vision impairment (rupa), deficits in smell (gandha) and taste (rasa) and impairment of the sense of touch (sparsha) is noted in older adults. Multiple sensory impairments and cognitive decline (cheshta vichintitam) are associated with increased mortality risk.[79]

Hallucinations (utpadyante ashubhaaneva) are perceptions in the absence of an external stimulus. Hallucinations can be seen in various conditions (utpadyante ashubhaaneva) such as damage to the peripheral sensory pathways, thyroid function, Hashimoto disease, deficiencies in D and B12 vitamins, PWS, autoimmune disorders, HIV/AIDS, narcolepsy, neurological conditions such as tumors, traumatic brain injuries, epilepsy, cardiovascular events involving the brainstem regions and temporal, occipital, or temporo-parietal pathways, neurodegenerative conditions such as PD, DLB, psychotic disorders such as schizophrenia, schizotypal personality traits, schizoaffective disorders, bipolar affective disorder, personality disorders, post traumatic stress disorder (PTSD), anorexia and bulimia nervosa etc.[80]

Neuro-ophthalmological conditions

'Ati pravishthe' (Verse 6)[7]

The word 'Ati pravishthe' denotes 'Enophthalmos'. Unilateral ptosis can be seen in 'Horner syndrome' (HS). The combination of the upper eyelid ptosis and the lower eyelid elevation narrows the palpebral fissure, giving rise to an apparent enophthalmos.[81] Enophthalmos can be defined as a relative, posterior displacement (ati pravishthe) of a normal-sized globe in relation to the bony orbital margin. Enophthalmos can be caused by various conditions such as maxillary sinus disease, silent sinus syndrome, maxillary hypoplasia, orbital bony defects, Paget's disease, senile enophthalmos, lipodystrophy, Scleroderma, Parry-Romberg syndrome, orbital varix, Blue rubber bleb naevus syndrome, metastasis, restrictive myopathy, Wegener's granulomatosis, tuberculosis, Sarcoidosis, pseudoenophthalmos and idiopathic reasons. [82]

'Ati jihme' (Verse 6)[7]

The word 'Ati jihme' denotes 'Strabismus or squint'. Strabismus is a frequent ocular disorder characterized by a misalignment (ati jihme) of the visual axes (convergent or divergent, horizontal or vertical, with variable angles of deviation). Binocular vision may also vary greatly. Strabismus must be considered as possibly resulting from abnormal genetic or acquired factors, anatomical or functional abnormalities, in the sensory or the motor systems, both peripherally or in the brain itself. Strabismus occurs due to various causes such as abnormal weakness of extraocular muscles (muscular dystrophies, myopathies, myasthenia or abnormal muscular pulleys), acquired nerve palsies, abnormal development of visual paths, and genetic causes.[83]

'Ati mukta bandhane' (Verse 6)[7]

The word 'Ati mukta bandhane' denotes 'Ptosis' or 'Ophthalmoplegia' or 'Strabismus'. Chronic progressive external ophthalmoplegia (CPEO) is the most common disease of mitochondrial myopathies, which is clinically characterized by bilateral ptosis (ati mukta bandhane) and limitation of eye movements.[84] MG is an autoimmune disorder of the neuromuscular junction characterized by fatigability and fluctuating weakness of voluntary muscles, diplopia, palpebral ptosis (ati mukta bandhane), dysphagia, dyspnea, or limb weakness. Miller Fisher Syndrome (MFS) is an immune-mediated neuropathy presenting with ophthalmoplegia (ati mukta bandhane), ataxia, and areflexia.[85] Ptosis (ati mukta bandhane) is most commonly the result of age-related stretching and dehiscence of the levator aponeurosis. Other causes may be neurogenic (e.g., third-nerve palsy, MG and HS), traumatic, congenital, mechanical (eyelid tumours) or myogenic.[86]

'Satatam unmeshite' (Verse 6)[7]

The word 'Satatam unmeshite' denotes 'Lagophthalmos'. Lagophthalmos is the incomplete or defective closure of the eyelids

(satatam unmeshite). The inability to blink and effectively close the eyes leads to exposure keratitis. The main causes of lagophthalmos are facial nerve paralysis (paralytic lagophthalmos), trauma or surgery (cicatricial lagophthalmos) or during sleep (nocturnal lagophthalmos). The main cause for paralytic lagophthalmos is Bell's palsy but it may be secondary to trauma, infections, tumors, and many other conditions.[87]

'Satatam nimeshite' (Verse 6)[7]

The word 'Satatam nimeshite' denotes 'Ptosis'. Drooping of the upper eyelid (upper eyelid ptosis) may be minimal, moderate, or severe, covering the pupil entirely (nimeshitam). Ptosis can be unilateral or bilateral and it can be congenital or acquired. Ptosis may be due to myogenic, neurogenic, aponeurotic, mechanical or traumatic causes. Ptosis may be associated with various other conditions such as immunological, degenerative, or hereditary disorders, tumors, or infections.[88] The above verse denotes bilateral ptosis due to various neuromuscular conditions.

'Vibhranta drishtike' (Verse 6)[7]

The word 'Vibhranta drishtike' denotes 'Nystagmus'. Nystagmus is an involuntary oscillation (vibhranta drishtike) of one or both eyes about one or more axes. Nystagmus may be divided into one of three categories, physiological (e.g., optokinetic, vestibular and end point), congenital or infantile and acquired (due to neurological disease). Retinal disorders, inner ear conditions, lesions of the vestibulo-cerebellum (flocculus, parafloccculus, nodulus and uvula) and medulla, cerebellar disease, parasellar lesions of the optic chiasm (e.g. pituitary tumours), alcohol, lesions of medial longitudinal fasciculus, multiple sclerosis, brainstem and cerebellar disease, oculopalatal myoclonus, Wipple's disease and disease of central myelin etc various conditions can cause nystagmus.[89]

'Vyasta drishtike' (Verse 6)[7]

The word 'Vyasta drishtike' denotes 'oscillopsia' or 'abnormal saccadic eye movements'. Abnormal eye movements or abnormal saccadic eye movements are seen in various conditions like PD, cerebellar ataxia, Spinocerebellar degeneration (SCD), PSP, HD, Alzheimer's disease (AD), Attention deficit hyperactivity disorder (ADHD), MS and various other neurodegenerative disorders.[90] Various abnormal saccadic eye movements such as 'Hypometric saccades', 'Prolonged saccade latencies', 'Reduced peak velocities', and 'Disorganized visual scanning' etc have been noted in neurodegenerative disorders.[91]

'Heena drishtike' (Verse 6)[7]

The word 'Heena drishtike' denotes 'reduced or diminished vision'. The diseases of anterior visual pathway are more common than cortical vision loss (heena drishtike). Neuro-ophthalmologic diseases can occur due to damage at any location from optic disc, optic nerve, optic chiasm, optic tract to optic radiation and occipital cortex. Conditions like optic neuritis, multiple sclerosis, neuromyelitis optica, tubercular meningitis, optic neuropathy (demyelinating, ischemic, toxic and infectious causes), cavernous sinus thrombosis, cryptococcal meningitis, malignant infiltration of optic nerve, Crouzon's syndrome, occipital gliosis, idiopathic intracranial hypertension, pituitary adenoma, acute disseminated encephalomyelitis, posterior reversible encephalopathy syndrome (PRES), space occupying lesions (SOL), stroke and other neurovascular pathology, and subacute scelrosing panencephalitis (SSPE) can leads to vision impairment (heena drishtike).[92]

Neuromuscular conditions:

'Tatra prakruti vaikarikanam swaranam aashu abhinirvrutti swaranam ekatvam ekasya chanekatvam aprashastam' (Verse 15&16)[5]

According to the above verse, sudden or abrupt change of voice denotes inauspiciousness and also indicates imminent death. The term dysphonia is used to describe any impairment of the voice (alteration in the sound of the voice with hoarseness, restriction of vocal performance, or strained vocalization) (vaikarikanam swaranam). The causes of dysphonia are diverse such as acute or chronic laryngitis, functional dysphonia, benign and malignant tumors, vocal cord paralysis, presbyphonia, psychogenic factors, Tuberculosis, Rheumatoid arthritis (RA), Systemic lupus erythematosus (SLE), Wegener disease, Sarcoidosis, and Amyloidosis etc.[93]

The most common NMDs are acquired peripheral neuropathies. Other acquired NMDs include ALS, poliomyelitis, GBS, MG, and Polymyositis. Hereditary NMDs include spinal muscular atrophy (SMA), Charcot Marie Tooth disease (CMT), congenital myasthenia, and DMD. Strained and strangled quality of speech, reduced rate, low pitch, imprecise consonant pronunciation, vowel distortion, and breaks in pitch etc are seen in 'Spastic dysarthria' due to UMN pathology. LMN dysfunction creates 'Flaccid dysarthria' in which speech has a nasal or wet quality, pitch and intensity are monotone, phrases abnormally short and audible inspiration (vaikarikanam swaranam).[94 & 95] The above verse denotes voice impairment due to NMDs.

'Na bhibharti shiro greevam na prushtam bharamatmana na hanu pindamaasya stamaturasya mumurshata' (Verse 22).[12]

The above verse denotes various conditions like NMDs, LMN syndromes and myopathies. The dropped head syndrome (DHS), also known as the floppy head syndrome (na bhibharti shiro greevam) is characterized by weakness of neck extensor muscles against gravity with or without weakness of neck flexor muscles (na bhibharti shiro greevam). Neurological causes of DHS includes ALS, PD, MSA, cervical dystonia, postpolio syndrome, cervical myelopathy, chronic inflammatory demyelinating polyneuropathy (CIDP), neuromuscular causes include MG, Lambert-Eaton myasthenia syndrome (LEMS), muscular causes includes primary inflammatory such as polymyositis, scleromyositis, isolated inflammatory axial myopathy, nemaline myopathy, mitochondrial myopathy, congenital myopathy, facioscapulohumeral dystrophy (FSHD), and isolated neck extensor myopathy (INEM).[96] The predominant clinical features of SMA are muscle weakness and atrophy. Weakness is usually symmetric with proximal muscles more affected than distal groups. SMA is characterized by symmetrical, proximal predominant extremity weakness that also affects axial (na prushtam), intercostal, and bulbar musculature.[97] Dysphagia (i.e., swallowing impairment) (na hanu pindamaasya), is one of the most common and devastating symptoms affecting individuals with motor neuron disease (MND), particularly when bulbar muscles are involved, as in progressive bulbar palsy (PBP), primary lateral sclerosis (PLS) and ALS.[98]

'Na upaiti kantham ahaaro jihva kanthamupaiti cha ayushyantam gate jantorbalam cha pariheeyate' (Verse 25).[12]

The above verse denotes 'Oropharyngeal dysphagia' (OD). Dysphagia or swallowing disorder (na upaiti kantham ahaaro) is characterized by the dysfunction of one or more parts of the swallowing apparatus. The swallowing apparatus begins with the mouth and includes the lips, the tongue, the oral cavity, the pharynx,

the airway, and the esophagus and its sphincters, both upper and lower. Various causes like stroke, PD, ALS, MS, AD, PBP, poliomyelitis, polymyositis, muscular dystrophies, myopathies, and local structural reasons can leads to OD.[99] Intermittent or sustained severe involuntary tongue protrusion (jihva kantham na upaiti cha) can be seen in patients with dystonia. Causes include neuroacanthocytosis, neurodegenerative diseases, Lesch-Nyhan syndrome (LNS) and post-anoxic and tardive dystonia.[100]

'Hasta paadam mukham chobhe viseshadyasya sushyata shuyate va vina dehaat sa cha mamsam na jeevati' (Verse 12).[15]

The above verse denotes 'Myopathies' or 'Neuropathies' or NMDs. Anatomical distribution, patterns of weakness, focal wasting (hasta paadam mukham cha sushyate) and hypertrophy (hasta paadam mukham cha shuyate) of muscle groups (arms versus legs - hasta paadam, proximal versus distal and symmetrical versus asymmetrical) are important features while evaluating NMDs.[95]

'Peyam paatum na shaknoti kanthasya cha mukhasya cha urasashcha visushkatvadyo naro na sa jeevati' (Verse 11) [13]

The above verse denotes OD associated with dehydration and Dysphagia is common in dementia and patients of show signs of liquid aspiration (peyam paatum na shaknoti). Dysphagia progresses with increasing cognitive impairment.[101] In dysphagic patients, dehydration (kanthasya cha mukhasya cha urasashcha visushkatvat) is frequent and often accelerated as a result of limited fluid intake. Dysphagia is associated with malnutrition, dehydration (kanthasya cha mukhasya cha urasashcha visushkatvat), pneumonia, reduced functional outcome, and mortality (naro na sa jeevati).[102] A major cause of dysphagia in adults is CVA. Swallowing dysfunction often occurs in patients with PD. Dysphagia may also be a symptom MG, LMN diseases, ALS, MS, HD, AD, laryngeal nerve injury and various other causes.[103]

Other neurological conditions

'Stabhyate pratibuddhasya hanu manye tatha akshinee yasya tam bahirayamo gruheetvaa hanta asamshayam' (Verse 24).[9]

The above verse denotes 'Tetanus' or 'Dystonia'. Sleep disturbances are found in various focal dystonias such as impaired sleep efficiency and quality, reduced REM (rapid eye movement) sleep, increased awakenings, and an increase in abnormal movements prior to awakening (stabhyate pratibuddhasya).[104] An acute dystonic reaction is characterized by involuntary contractions of muscles of the extremities, face, neck (manya), abdomen, pelvis, or larynx that lead to abnormal movements or postures (stabhyate & bahirayama). 'Buccolingual crisis' is characterized by trismus (lock jaw) (stabhyate hanu), risus sardonicus, dysarthira, dysphagia, tongue protrusion and grimacing. 'Ocurlogyric crisis' (OGC) is characterized by spasm of the extraocular muscles (eyes deviate upward) (stabhyate akshini) and 'Torticolic crisis' is characterized by abnormal asymmetric head or neck position (stabhyate manya). 'Ophisthotonic crisis' is defined as characteristic flexion posturing with arching of the back (bahirayama).[105] Trismus (stabhyate hanu), ophisthotonus (bahirayama) and neck stiffness (stabhyate manya) can be seen in 'Tetanus'.[106]

'Hanu manya graha trishna balahraaso atimaatraya praanashcha urasi vartante yasya tam parivarjayet' (Verse 20).[10]

The above verse denotes 'Tetanus'. Trismus (hanu graha), neck stiffness (manya graha), respiratory failure, chest pain (praanashcha urasi vartante) and laryngeal spasm etc are seen in tetanus.[106] In tetanus asphyxia is the commonest cause of death resulting from laryngeal muscle spasm (and acute airway obstruction), respiratory muscle spasm (praanashcha urasi vartante) or extreme fatigue (balahraaso atimaatraya).[107]

'Uthapyamaana shayanaat pramoham yaati yo nara muhurmuhun saptaham sa jeevati vikathana' (Verse 19).[11]

The above verse denotes 'Orthostatic hypotension' or 'Presyncope' (pramoham). Classic orthostatic hypotension (uthapyamaana shayanaat pramoham yaati) is a common cause of (pre) syncope (pramoham) and it is associated with a twofold higher risk of death (saptaham sa jeevati) owing to the severity of comorbidities. Initial orthostatic hypotension, delayed blood pressure recovery and reflex mediated hypotension types of orthostasis (uthapyamaana shayanaat pramoham yaati) are more frequently associated with falls and mortality (saptaham sa jeevati).[108]

'Urdhwagre nayane yasya manye cha arata kampane balaheena pipasarta sushkasyo na sa jeevati' (Verse 27).[11]

OGC are defined as spasmodic movements of the eyeballs into a fixed position, usually upwards (urdhwagre nayane yasya). OGC can also be seen secondary to different neurological conditions such as neurotransmitter disorders, disorders affecting brainstem, multiple sclerosis and encephalitis.[109] Acute or subacute parkinsonism, parkinsonism–hyperpyrexia in PD, neuroleptic malignant syndrome (NMS), psychosis in PD, respiratory compromise in MSA, dystonic storm, tardive and neuroleptic-induced emergencies other than NMS, hemiballism, and Sydenham's chorea (SC) etc conditions can leads to movement disorder emergencies (na sa jeevati). Severe dehydration (pipasarta & sushkasya) and electrolyte disturbances can occur in dystonic storm. OGC (urdhwagre nayane yasya) can also be considered as one of the movement disorder emergencies.[110] The above verse denotes various conditions like 'Movement disorder emergencies', 'OGC', 'Dystonia', 'Tetanus' and 'Hypoxic encephalopathy' etc.

'Aayamya utpaatitaan keshaan yo naro na avabudhyate anaaturo va rogi va shadraatram naativartate' (Verse 8).[12]

Bilateral occipital neuropathy or occipital nerve compression can cause scalp numbness (na avabudhyate).[111] Peripheral neuropathy can cause numbness (na avabudhyate) and many other distressing symptoms.[112] Dissociated sensory loss in the cranial (na avabudhyate), cervical and brachial dermatomes is the classic presentation of syringomyelia and syringobulbia. FOSMN (facial onset sensory and motor neuronopathy) syndrome is a slowly progressive neurodegenerative disorder characterized by numbness in a trigeminal nerve distribution, which slowly progress to involve scalp (utpaatitaan keshaan na avabudhyate), neck, upper trunk and upper limbs sequentially.[113] In the early stages of peripheral neuropathy, patients typically present with progressive symptoms, including sensory loss (na avabudhyate), numbness, and pain sensations in distal limbs later the numbness may extend proximally. AIDS, carcinoma (paraneoplastic syndromes), chronic liver disease, critical illness neuropathy, diabetes mellitus, end-stage renal disease, hypothyroidism, leprosy, lyme disease, lymphoma, amyloidosis, porphyria, vitamin B_6 & B_{12} deficiency, genetic disorders, toxins and idiopathic conditions etc can leads to peripheral neuropathy.[114] The above verse indicates sensory neuropathy or peripheral neuropathy due to various underlying disease conditions.

'Yam naram sahasaa rogo durbalam parimunchati samshaya praaptamaatreyo jeevitam tasya manyate' (Verse 15).[13]

Transient ischemic attacks (TIA) are episodic usually lasting a few minutes to a few hours leaving no residual signs and symptoms (parimunchati) between attacks. 'Stroke in evolution' or 'progressing stroke' or 'incomplete stroke' may progress further causing major stroke or it may improve temporarily and reappear later often with widespread involvement (samshaya praaptam). A pattern of waxing and waning of signs and symptoms (sahasaa rogo parimunchati) that occurs over hours to days with an incomplete recovery in the above said conditions (samshaya praaptam).[115] Functional neurological disorders (FND) present acutely, particularly with dissociative seizures (resembling epilepsy) or persistent weakness (durbalam) resembling a stroke. Waxing & waning or fluctuating course (sahasaa rogo parimunchati) is characteristic feature of dissociative seizures and stroke like episodes.[116] MS is an intermittent disease with relapses and remissions (sahasaa rogo parimunchati) and having variable prognosis (samshaya praaptam).[117] Reversible cerebral vasoconstriction syndrome (RCVS) is a transient and fully reversible (sahasaa rogo parimunchati) cerebral intracranial arteriopathy. Clinically this entity presents acutely, with severe waxing and waning headaches ("thunderclap") and occasional fluctuating (samshaya praaptam) neurological signs.[118] Waxing and waning episodes (sahasaa rogo parimunchati) of behavioral inconsistency, incoherent speech and variable attention etc are seen in DLB and AD.[119] The above verse denotes various neurological conditions which are characterized by remissions and relapses or fluctuating course.

'Pindike shithilikrutya jihmeekrutya cha naasikaam vayu sharire vicharan sadyo mushnaati jeevitam' (Verse 5).[14]

The above verse denotes 'Neurosyphilis'. Syphilis may also be associated with myalgias, and clinical muscle disease (pindike shithilikrutya) has been reported in a few cases. Muscle atrophy or wasting (pindike shithilikrutya) can be seen in infectious myositis also.[120] Spastic weakness of the legs, sensory loss, and muscle atrophy (pindike shithilikrutya) can be seen in 'Meningovascular syphilis' or 'Syphilitic meningomyelitis'.[121] Destruction of the bridge of the nose and sequestration of the bone and other nasal deformities (jihmeekrutya cha naasikaam) are seen in syphilis.[122]

'Ahaasya haasi sammuhyan praledhi dashanachhadau sheetapadakarochwaso yo naro na sa jeevati' (Verse 20)[15]

Adequate hydration is essential for normal brain function and dehydration (praledhi dashanachhada) induces cognitive deterioration (sammuhya). Severe dehydration (praledhi dashanachhada) leads to cognitive dysfunction, delirium (sammuhya), coma (sammuhya) and risk factor for stroke. Dehydration is a predisposing factor for confusion (sammuhya) in long-term care residents. Plasma hypertonicity, a marker of dehydration may precipitate cerebral ischemic events in susceptible elderly individuals.[123] Cerebrovascular diseases encompass a wide range of conditions that interfere with normal cerebral circulation by causing changes of blood vessels, blood components, and hemodynamics. They include several causes of vascular brain injuries such as intracranial atherosclerosis, aneurysms, vasculitis, vascular spasm, vascular malformations, chronic cerebral hypoperfusion, infarction, and haemorrhages. Cerebrovascular diseases are associated with cognitive impairment (sammuhya), dementia, depression, and anxiety. Various behavioural (ahaasya haasi) and cognitive defects (sammuhya) are found in cerebrovascular diseases.[124] The above verse denotes various conditions like 'Advanced demenita', 'Delirium', 'Cerebrovascular diseases', and 'Shock' (sheeta paada kara) etc.

Conclusion

Charaka indriya sthana deals with conditions which are fatal, having poor prognosis and commonly seen in end-of-life stages. Various references pertaining to neurological conditions explained in 'Charaka indriya sthana' have been explored in the present article. Conditions like 'Neuropathies', 'Epilepsy', 'Movement disorders', 'Agnosia', 'Perceptual distortions', 'Cerebrovascular diseases', 'Neuromuscular disorders', 'Neuro-ophthalmological disorders', 'Neurodegenerative conditions' and 'Demyelinating disorders' etc are mentioned in Charaka indriya sthana. It seems that clinical findings mentioned in 'Charaka indriya sthana' are having the potential of clinical applicability and prognostic significance in present era also. Further studies are required to substantiate the clinical findings mentioned in 'Charaka indriya sthana'.

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Conflicts of interest

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