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ALZHEIMER'S DISEASE - A BASIC AYURVEDIC APPROACH*

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-Susruta Samhita

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CONTENTS

Editorial 02

Alzheimer's Disease - a basic ayurvedic approach 03
Dr. M. R. Vasudevan Namboodiri

Dementing Illnesses 05
Dr. P. S. Mathuranath, DM

Understanding a person or self- Realization 16
Dr. A. Raghunathan, Dr. K. Sundaran

Schizophrenia in Indian Society 19
Dr. G. V. Ramana, Dr. H. K. Gupta

From Pune..... 24
Dr. Anuradha Haridas Benkar

Ayurvedic Understanding of Sleep Disorders 27
V. S. Sreelakshmi

Mandookaparni (Centella asiatica. Linn) 30
Baby Joseph and Sophy Paul

Editor's Page

Mind Your Words

"Mind your words" is an utterance of disharmony. Usually it comes from emotion, not from thought or mind – the connecting unit of human body to nature – is in a subtle state of existence. Everybody knows it is there; but no body knows where it is. Some says it is in between palate and mid scalp; some says it is in the middle part of the body- heart. Heart and mind are used as synonyms. Heart is taken as the symbol of love – the most beautiful feeling of the mind.

According to Indian Philosophy, mind and word are interdependent. Word also has a subtle state of existence. Controlling of word in turn controls mind and similarly controlled mind controls word. This control is not only for happiness through meditation but for worldly pleasures also. Word and heart are fast bound. Words are used as blessings; they are heart some. When words came as curse, they are heart breaking. Words of congratulations can be hearty. Verses of texts are by-hearted. Words of intimacy are expressed in a heart to heart talk. Thus words are the expressions of state of mind.

Acharya Vagbhata while saluting '*Apoorva Vaidya*' – the Almighty – praises Him as who cures the mental problem like '*Raga*' etc. *Raga, Dwesha, Moha, Mada, Lobha, Ahankara* etc are mental diseases. They are the products of *Mano-doshas- Rajas* and *Tamas*. Thought, discretion, assumption, understanding and desire are the fields of mind and control of sense organs to the function of mind. Ayurveda has designated the qualities of mind as *Anu* and *Eka*. There is only one mind and it is very minute. Oneness of mind shows that it can be attached to one thing or one sense organ at a time. Mind works in high speed beyond the imagination; hence uninterrupted simultaneousness is felt in perceptions. Mind is so minute that anatomical tracing cannot reveal it. Over powering the backward pulling of mind, sense organs run to the fields in pursuit of worldly pleasures and meet with positive and negative reciprocations. These reciprocations result in mental diseases like *Raga* (lust), *Dwesha* (hatred), *Moha* (frustration) etc. These mental outcomes are highly poisonous. They inactivate the power of mind to control the sense organs. They will be tempted to eat the forbidden fruits. The mind oscillates between happiness and sorrow, success and failure, fulfillment and frustration etc. There is not even a single moment of stand stillness of mind in the life span of a common man. *Apoorva Vaidya* is capable of detoxifying the mind and body. Salutation to that Almighty by *Acharya Vagbhata* in the first verse of *Ashtanga Hrudaya* initiates the learner to be free from the above said mental toxicity. Mind kept under the control of wisdom will be capable of understanding '*Sastra*'. His words will be meaningful and will console the listener. He can discharge the duty of a good Physician with cool mind. Wisdom, courage and spiritual awareness will make the mind strong and controlled. A sound mind in a sound body – they are interdependent. A Physician himself should be healthy by mind and body.

Thus comes the Renaissance.

Dr. K. Unnikrishna Pillai

ALZHEIMER'S DISEASE - A BASIC AYURVEDIC APPROACH*

**Dr. M. R. VASUDEVAN NAMBOODIRI

Alzheimer's disease is a progressive neuro-degenerative disorder in which a gradual decline in the memory along with at least one other area of higher intellectual function. The domains affected are cognition, daily functioning and behavior. The cognition includes memory, orientation - judgement etc.

Dhee, Dhrithi and Smrithi are the most important cognitive faculties that determines the physical, psychological and social well being of an individual. These are essential for the proper functioning of the *budhi, indriya* and *manah.* Deterioration (*vibhramsa*) of the above said factors creates imbalance to the homeostasis.

Chakrapani on commenting *Charakacharya,* explains *dheevibhramsa, dhrithi bhramsa* and *smruthi bhramsa.*

धीविभ्रंश - ... हितेऽहितमहिते वा गितमिति या बुद्धिः स बुद्धिभ्रंशः ।

धृतिभ्रंश - ... मनोनियमं कर्तुमशक्त्र धृतिः स्वकर्मभ्रष्टा भवतीत्यर्थः ।

स्मृतिभ्रंशं - ... तत्वजानस्य शिष्टानां स्मर्तव्यच्चेन संमतस्य यदऽस्मरणं ।

THRIDOSHAS AND COGNITIVE FUNCTIONS

Doshas plays vital role in maintaining cognitive functions. Any factors that impairs the *sareerika bhavas* will affect the *manasikabhavas* also.

Vata regulates the proper functioning of *budhi, indriya* and *manah,* while *pitta* enhances *medha* and *kapha;* nurture *dhee, dhrithi* and *smrithi.* Thus the normalcy of *thridoshas* is essential for maintaining the orchestra of cognitive speciality tuned.

AGING

Nature is never stable, changes are inevitable *(परिणामः पुनः काल उच्यते).* Thus the equilibrium of *doshas* may not be maintained as such during different stages of life, giving rise to inevitable changes in various physiological phenomenon of body. *Susruthacharya* explains this very well, as tabulated below.

Period of human life span	Faculty undergoing physiological *kshaya*
10 years	*Balya*
20 years	*Vridhi*
30 years	*Prabha*
40 years	*Medha*
50 years	*Twak*
60 years	*Sukla*
70 years	*Akshi*
80 years	*Smrithi*
90 years	*Manas*
100 years	*Sarvendriyani*

Thus it is clear that *smrithi kshaya* can be normally

*Paper presented in the Scientific Session, Nagarjuna Merit Evening 2007.

** Principal, Govt. Ayurveda College Thiruvananthapuram.

anticipated after the age of 80 years and that does not mean that it is irrevocable.

WHEN IT BECOMES A PROBLEM ?

Problem arises when the normal aging process starts very much early or when the degree of aging process worsens and manifest in such a way that it hinders the normal daily living of the individual.

ANY *NIDANA* ?

No clear *nidanas* for Alzheimer's disease are available in ayurvedic texts. Early neuro degeneration may be considered as *akalaja swabhavika roga* and for which the cause in *pranjaparadha.*

Increased life expectancy and increased contribution of the older age group in the population density is one of the cause of increased incidence of A.D. Apart from this, life style disorders (hypertension etc.), stress, *vegarodhas,* pollution of *desa, kala, vayu, jala, ahithaahara upayoga,* all adds to the dramatic rise of AD incidence. Radiation exposure is another alarming factor of this era and need further studies.

SAMANYA SAMPRAPTHI

Unhealthy life styles and disease causing factors, polluted environment, *prakruthi* of the individual etc. all acts in summation leading to *roga sampraptHi.*

Samanya nidanas as explained lead to *doshadushti* which inturn causes *dathu dushti.* Minor symptoms of the disease starts manifesting at this stage onwards. The vitiated *dhathus* inturn vitiate the s*rothases* causing the recurrence of the pathophysiology. Since *dhathus* are the basic structural units and *doshas* maintain physiological functioning, their impairments give rise to secondary diseases. All these lead to early onset of aging process.

When these pathological changes happens during *vardhakya,* many more factors come into play as *srothokharathwam, agni mandyam, ahara sara agiranavaigunyam* and *upachaya apachaya asamatha.* These factors results in *rasadhathukshaya.* The defect in *rasadhathu* results in the defective formation of *utharothara dhathus.* And finally resulting in *ojakshayam and vata vridhi.*

The cumulative effect of all these process are accelerated in aging process.

GENERAL *DOSHA STHITHI* IN DEGENERATIVE DISEASES

Pitta due to its *theekshna, ushna, laghu gunas* and *vata* to its *rooksha, laghu, sookshma gunas,* plays a key role in almost all degenerative conditions. But as the degenerative process progresses, a stage occurs where the *soumya bhavas* get very much destructed and hence extreme *vata prakopa* happens. When the degenerative process become generalized as in the case of *vardhakya avastha,* the *vata kopa* will be generalized. The impact of this vitiated *vata* on *agni* causes *vishamagni* resulting in *Rasadhathwagni mandhya.* Defective transformation of *rasadhathu* causes *malarupa kapha vridhi* which inturn causes *agni vaishamya* and this terms a vicious cycle- explaining the recurrence of the pathophysiology. Thus in effect, there is a *vata kapha* vitiated situation.

PROGNOSIS OF *VATA KAPHA JANYA ROGAS*

Generally, *vatakapha janya rogas* are considered to have bad prognosis. Some of the facts behind it are as follows.

- Defective *dhathu* transformation due to *srothorodha* caused by the vitiated *kapha.*
- Defective *dhathu* formation due to *vishamagni* caused by vitiated *vata.*
- *Agnimandya,* as *kapha* and *vata* over power and inhibit *pitta* from its normal functioning.

To..... P.No. 13

DEMENTING ILLNESSES*

**Dr. P. S. MATHURANATH, DM

The developing world accounts for more than 50% of the global elderly population. Large populations and increasing average life expectancy of individuals at birth, will only increase these numbers in the coming years. By the year 2020, approximately 70% of the world's elderly population will be in developing countries, with India accounting for 14.2% of them. Only a few population-based studies on dementia are available. They indicate that, the prevalence of dementia in the population aged 65 years or more varies from 1.36 to 3.5% and incidence is about 4.7/1000 person years. With an elderly population of nearly 71 million, it follows that even by conservative estimates there are between 0.97 to 2.5 million people with dementia in India and that 0.33 million are added to this every year.

Clinically evident cognitive impairment occurs in about 10% and subtle cognitive decline on neuropsychological testing in 66% of all normal older people above the age of 65 years. This is often known as age-associated memory impairment (AAMI) and is defined by the consensus guideline as a decline of more than one standard deviation in any area of cognitive functioning in comparison with age matched controls[12] in the presents of normal activities of daily living. Mild cognitive impairment (MCI) is an evolving concept where cognitive changes in the elderly are assumed to be the pathological precursor to dementia. It is characterized by subjective memory complaints with demonstrable memory impairment but preserved general intellectual and other cognitive functions on cognitive testing and an intact ability to perform activities of daily living. Although these concepts need further refinements they remain clinically useful as they provide a window of opportunity for effective early interventions. Dementia, on the other hand, represents a progressive decline in the cognitive and intellectual functions of the individual (involving at least two cognitive domains) significant enough to affect their day-to-day activities in the absence of delirium. This chapter gives a brief overview of the clinical aspects of dementia taking Alzheimer's disease (AD) as a prototype, describes some of the dementing illnesses with an emphasis on the Indian context and briefly provides a diagnostic approach to patients presenting with dementia.

DEMENTING ILLNESSES

Causes of dementia are many. They could be classified in many ways. The important conditions classified clinico-etiologically into difference groups helps in planning the treatment and providing the prognosis of a particular condition.

*Paper presented in the Sceintific Session, Nagarjuna Merit Evening 2007.

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Group I consists of diseases with neuronal degeneration. Some have dementia as the core feature, like AD, and others have it as a co morbid condition. All diseases in this group, generally occur in the elderly, and are slowly progressive. Group II consists of the Vascular Dementias. Group III includes dementia as a result of other secondary causes. The list is quite long and yet not comprehensive. Group IV is made up of the overlap syndromes with the pathologies from group I and/or Group II coexisting. It is important to recognize this group since intervention can mitigate the disease and its also of importance to researchers as they can dilute or confound the purer groups.

ALZHEIMER'S DISEASE

The most widely used criteria for clinical definition of AD was introduced by the NINC: DS-ADRDA, which classifies AD into definite, probable and possible.

CLINICAL FEATURES OF ALZHEIMER'S DISEASE

Gradual onset and progression is considered typical. In mild stages (early AD), forgetfulness, characterized by repetition of questions and misplacement of objects, constitutes the core feature. Additionally, temporal and spatial disorientation and impaired calculation may also be present. Social skills and self-care are well preserved. In moderate stages, memory is preserved only for highly learned materials. There is disorientation and compromise of language functions. Behavioural changes, e.g., delusions or wandering, are often present. Instrumental activities of daily living are severely compromised, while activities of daily living require supervision. In advanced stages, the patient is totally dependent for everything and often bed-ridden and mute. Average duration from onset to death is about 8-10 years. Clinical variants in the presentation of AD include patients with progressive visual dysfunction, progressive bi-parietal syndrome and progressive aphasia, all of whom go on to develop the typical clinical syndrome and histopathological features of AD. Behavioural changes can occur in 88% of AD patients and are usually seen in advanced stages of the disease. Important amongst them are apathy (72%), agitation (60%), and anxiety (48%). They are more amenable to treatment than the cognitive symptoms.

NEUROPSYCHOLOGICAL PROFILE OF ALZHEIMER'S DISEASE

Profound amnesia involving episodic memory both verbal and visual is characteristic. There is an early impairment of the sense of time and place- As the disease advances, immediate recall also gets affected. Finally, there is a complete failure of registration or new learning. In contrast, semantic memory, i.e. factual information learnt in the distant past, and procedural memory are usually preserved until the late stages of the illness. With advancing disease, visuospatial skills and ideational and ideomotor praxis also gets affected. Language and most frontal executive functions are involved only in the late stages of illness.

RISK FACTORS FOR ALZHEIMER'S DISEASE

The four main risk factors for AD are age, gender, education and Apo E e4 allele. The prevalence, as well as the incidence, of AD double with every 5 years increase in age. Data from most studies, including Indian studies, suggest that the prevalence of AD rises with age. The incidence of AD increases from 1.2% at 65 years to 63.5% at 90 years and above. Prevalence of all dementias in women, were higher when compared with that in men in the studies from South India (3.7% vs. 3.4% in rural Tamil Nadu and 3.5% vs. 2.8% in Kerala). It is generally acknowledged, that women are at greater risk

than men for AD as was also seen in Kerala (1.8% as against 0.73% in men). Thus, in general, the pattern found in other parts of the world is also seen in India.

Several studies on prevalent patients show relationship between low education and dementia in general and AD in particular while studies on incident patients do not. In India, prevalence rates of dementia (and AD) in the illiterates as compared to literates in rural north India (1.65% (0.74%) vs. 1.24% (0.29%)) and in Tamil Nadu (3.5% vs. 4.5%) also showed no statistically significant difference. So far, no study on the Indian population has found any association between education and dementia. Carrying the Apo E e4 allele increases the risk by two to four folds in Caucasians and Chinese but not in Africans and Hispanics.

Other factors investigated include head-injury (positive association in some but not in other studies), family history (positive association in some but not in other studies), homocystine, hypothyroidism, and factors affecting cerebral micro and macrovasculature. Interestingly, some factors such as ApoE e2 allele, nonsteroidal anti-inflammatory drugs, estrogen replacement therapy; and high education and challenging occupationts have been reported to offer some protection against AD.

FRONTO-TEMPORAL DEMENTIA

Frontotemporal dementias (FTD) accounts for up to 10 percent of all dementing disorders and the vast majority of early onset (<65 years) dementia. Pick's Disease, is now considered a histopathological variant of frontotemporal dementia. FTD is a heterogeneous condition consisting of different clinical and histopathological entities. A plethora of terms, including 'Pick-Complex' and 'Frontotemporal Lobar Degeneration' have also been used for the FTD syndromes as elaborated elsewhere. Three distinct clinical syndromes are recognized under FTD frontal variant of frontotemporal temporal dementia (fvFTD) progressive non-fluent aphasia (PNA) and semantic dementia (SD). fvFTD is primarily a behavioural syndrome and the latter two conditions constitute the progressive aphasic syndromes. The topographical distribution of atrophy determines the clinical syndrome although the underlying histopathological changes can be similar in all the three conditions.

CLINICAL FEATURES OF FTD

FTD often begins earlier than AD, the mean age of onset is between 50-60 years although earlier onset in fourth decade has been documented, especially in familial cases. In general, it is also known to progress more rapidly than AD. Clinical features of FTD can often accompany other neurological manifestations such as Parkinsonism, amyotrophy, etc., especially in familial cases, suggesting an overlap syndrome.

Early clinical features of fvFTD are behavioural abnormalities and include early loss of insight, disinhibition, impulsivity and risk-taking behaviour, apathy, reduced empathy and sympathy, poor self-care, mood fluctuations, stereotypic behaviour, mental rigidity and eating preferences (often an obsession for sweets). As the disease advances, there is impairment of language functions and semantic memory. Unlike AD, impairment of new learning abilities, immediate recall (episodic memory) and visuospatial abilities occurs only with advanced disease. Initial description of the progressive aphasic syndrome was provided by Mesulam et al who called them as Primary Progressive Aphasia. The clinical syndrome of PNA consists of an insidious onset, slowly progressive word-finding difficulty, phonological paraphasia, impaired syntax construction and telegraphic speech. Later, dysarthria and frank mutism can occur. Over time most of these patients develop frank dementia with behavioural abnormalities similar to that of fvFTD. SD is also referred to as progressive fluent aphasia and temporal variant

of frontotemporal dementia. In sharp contrast to PNA, they have superficially normal speech with preserved syntactic construction. Word-finding pauses and semantic errors characterize the early stages and as the disease evolves there is degradation of semantic knowledge and in most cases frank dementia with behavioural changes. These patients also show poor recall of events taken place years ago in their personal life, much in contrast to AD. As with PNA, patients with SD have well preserved new learning abilities and recall of the same.

NEUROPSYCHOLOGY IN FTD

Neuropsychological testing in FTD is of immense value in differentiating FTD from AD, especially in the early stages. Impairment on tests of verbal fluency (especially letter fluency), attention and vigilance (such as digit span) and planning and executions and intact performance on tests of orientation, new learning, its immediate and delayed recall, and visuospatial perception, typically distinguish early FTD from early AD. Distinctive pattern of neuropsychiatric involvement on the Neuropsychiatric Inventory Is also a useful marker of early FTD. Neuropsychological testing and interpretation of results in moderately or severely advanced FTD is more difficult since attention, which is required for attempting any neuropsychological test, is markedly impaired in these patients. Some simple neuropsychological screening instruments such as the Addenbrooke's Cognitive Examination and the Frontal Assessment Battery have been found to be helpful in bed- side clinical differentiation of AD and FTD.

Formal language testing using aphasia batteries provides information to qualify the language abnormalities and classify the type of progressive aphasia. Detailed language and semantic testing is required for quanlifying the deficits and follow-up evaluation of patients. Patients with early PNA show impairment on tests for reception of grammar, syntax construction and complex tests of reading and writing and are usually intact on simple comprehension. On the contrary, patients with early SD show impairment on tests of semantic processing such as naming objects to description, matching pictures to words, sorting pictures into different categories etc.

Detailed published reports of FTD from India are almost nonexistent. In the memory and neurobehavioural clinic at SCTIMST, over past 3 years, we have seen about 35 cases of FTD of which 5 patients have been PNA and the rest are fvFTD. Yasha et al reported an unusual, pathologically proven, familial case of a man of 45 years presenting with dysarthria, bradykinesia, acalculia, and myoclonus followed a little later by dementia with a similar illness in his mother and sister. Autopsy of his brain showed frontal and temporal atrophy, with neuronal loss, gliosis, ballooned neurons and focal microvauolation. Immunohistochemistry showed numerous granular, neuronal, cytoplasmic 'tau' positive inclusions in the midbrain, putamen, frontal and temporal neocortex. Immunochemistry for Ubiquitin and Prion protein were negative.

DEMENTIA WITH LEWY BODIES

Dementia with Lewy bodies (DLB) is also known as Diffuse Lewy Body dementia, senile dementia of Lewy body type and Lewy body variant of AD and accounts for 12% to 36% of all cases of dementia, which are diagnosed as AD, in the west.

CLINICAL FEATURES AND NIEUROPSYCHOLOG IN DLB

DLB is a prototype of a mixed cortical and subcortical dementia. It may present as parkinsonism, dementia or both, depending on whether the Lewy body pathology begins in the mid-brain, the cortex or both together. When the initial symptoms are parkinsonian, it is often difficult to distinguish DLB from idiopathic PD in its early stages. Bradykinesia, resting tremors, parkinsonian gait, falls and occasionally subtle abnormalities of ocular pursuits are all seen in DLB. Many of these features are known to respond, at

least partially, to Levodopa. Neurapsychiatric manifestations of (vivid visual) hallucinations and psychosis (delusions and depression), that shows profound day-to-day fluctuations, dominate when the onset is with dementia. Other cognitive changes of behavioural signs of frontal lobe dysfunction, episodic memory impairment, language impairment and focal cortical afflictions producing apraxia or agnosia soon follow. In addition to fluctuations stated above, another feature of diagnostic significance is the sensitivity to neuroleptic agents, which is seen in almost all patients. Caution should be exercised in interpreting the significance of hallucinations and falls, since in some cases they may be secondary to Levodopa exposure and not due to the disease per se. Clinical diagnostic criteria for DLB have been prescribed by the consortium on DLB international workshop in 1996[72] The clinical features that help in distinguishing DLB from Parkinson's Disease and AD include early symmetrical but subtle extrapyramidal symptoms and signs, vivid visual hallucinations, fluctuating confusions and psychosis, depression, myoclonus and unexplained falls.

VASCULAR DEMENTIA

In India, Vascular dementia is thought to account for 22 to 57% of all dementias. Western literature suggests that cerebrovascular diseases account for 10% to 30% of referrals for dementia. In the past, the concept of VaD had been one of a dementia syndrome due to small or large brain infarcts. While the infarct concept in the genesis of VaD remains important, interaction between any other form of CVD and behavioural impairments have received sparse attention in the study of the etiology for VaD. Added to this is the fact that a cerebrovascular event, may be responsible for merely unmasking an underlying dementia by disturbing the compensated state of brain functioning. In the light of all these considerations, investigators have now broadened the concept of VaD as a syndrome of behavioural impairments with varied manifestations due to vascular factors of diverse causes, affecting the brain and introduced the concept of Vascular Cognitive Impairment. The importance of early diagnosis of VaD lies in the fact that unlike AD or FTD, disease modifying treatments available for cerebral vascular disease (CVD) are applicable to VaD.

CLINICAL FEATURES OF VASCULAR DEMENTIA

Considering the broad concept of vascular dementia certain syndromes can be defined under it. All of these syndromes share certain common clinical features. The core feature is the presence of dementia in the presence of cerebral vascular disease, in the form of clinical or radiological evidence of stroke or ischemic lesions involving the brain and a relationship between the cerebrovascular disease and dementia. While it is difficult to accurately judge the presence of such a relationship in a given case, certain broad guidelines such as, onset of dementia within 3 months of a recognized stroke, history of abrupt deterioration in cognitive functions, or fluctuating step-wise deterioration of cognitive deficits, are helpful. Many types of clinical presentations of VaD have been described. From a practical point of view, five clinical presentations can be recognized in vascular dementia: a multi-infarct state. Binswanger's dementia with a steadily progressive cognitive deterioration, multiple subcortical lacunar infractions causing dementia with or without history of strokes, arteriolopathic form of VaD (as seen in Cerebral Autosomal Dominant Arteriolopathy with Subcortical Ischemic Leukoencephalopathy (CADASIL) or amyloid angiopathy}; mixed dementia in which VaD is associated with other pathologies, Little or no published, data is available on VaD from India.

DEMENTING DISEASES WITH EXTRAPYRAMIDAL MANIFESTATIONS

Huntington's Disease (HD) has been reported in India from 1955 onward. Vas et al found HD in 1 percent of the demented patients seen in Mumbai in a recent epidemiological survey. Two clinico-

genetic studies, one from north and one from south have suggested similar clinical profiles in patients with HD across the country. Saleem et a1 however, suggested that there may be multiple origins of mutation in different parts of the country. Murgod et a1 found that only 11.5% patients presented with psychiatric symptoms. But as the disease progressed behavioural disturbances were noticed in nearly 66% depression in 65.4%, dementia in 50% and occupational disability in 84.6%. The predominant behavioural symptom consists of a frontal behavioural syndrome.

Progressive Supra Nuclear Palsy (PSP) is another degenerative disorder characterised by trunkal dystonia, vertical gaze palsy and Parkinsonian features, in which dementia is common in late stages of the disease. The dementia is of the subcortical variety with behavioural disturbance and mild cognitive impairment. Suresh and Rao reported 14 patients with PSP between 1975 and 1987. Cognitive dysfunction was seen in half their cohort in the form of change in personality, slow thinking, and inability to acquire and use new knowledge. CT scan showed midbrain, pontine and subcortical atrophy with lateral ventricular enlargement.

Patients with Parkinson's Disease show some degree of cognitive impairment, especially on formal neuropsychological testing. But the true incidence of dementia ranges from I S to 25%. Later age at onset, depression, and severe akinesia are considered risk factors for dementia, which is relatively uncommon in those in whom tremor predominates. The dementia is essentially of the subcortical variety and significant disorientation, dysphasia, dyspraxia and impaired new learning should raise doubts about the diagnosis of PD. Nagaraja et a1 examined 48 patients with idiopathic PD three months after stopping drugs and found that the incidence of dementia was as high as 58% (28/48). The severity of dementia did not correlate with the physical disability. Another Indian study assessed the cognitive functions of 21 patients with early onset of Parkinson's Disease (EOPD) (<41 years). They too reported a high rate of frontal and temporal lobes dysfunction.

In Corticobasal Ganglia Degeneration (CBD), dementia is not an early or prominent sign. Asymmetrical dystonia, limb apraxia, action myoclonus, cortical sensory loss and alien hand are more prominent. Dementia often appears with acalculia and visuospatial disorder. Atypical cases with dementia as the presenting feature and mimicking Frontotemporal dementia are also documented. There are no published reports from India, but anecdotal accounts are available from various Movement Disorder clinics in the country.

OTHER CAUSES OF DEMENTIA

These are a group of disorders in which dementia is a part of the symptom complex, and often a presenting manifestation. Reference will be made to only those where relevant Indian data is available. They constitute an important group since they are fully or partially reversible if diagnosed early. The prevalence of potentially reversible dementia ranges from 6% to 32% in various studies. Most of these studies have included pseudodementia due to depression as a reversible dementia. In a meta-analysis of 32 studies (2889 subjects), Clarified et a1 found that the commonest reversible causes were drugs (28.2%), depression (26.2%) arid metabolic (15.5 %).

PSEUDODEMENTIA

Geriatric population receives drugs for many reasons. Anticholinergic agents, long- acting benzodiazepines, barbiturates and anaesthetic agents can all worsen the cognitive status of elderly subjects. Other drugs such as DOPA agonists, phenothiazines, MAO inhibitors and other neuroleptic agents can also transiently affect the cognition of these subjects. Caution should be exercised in prescribing these agents in this population. Furthermore, these drugs should be carefully withdrawn from elderly patients who

present with cognitive symptoms and they should be observed for a period of time before investigating for other causes of dementia. Depression is a common comorbidity in the elderly. It can mimic dementia as well as be a comorbid condition with it, often masking it. The clinical features that helps differentiate depression from dementia are the mood state, fluctuations in cognitive abilities, patient's unwillingness to attempt cognitive testing, and the presence of risk factors for dementia. Delirium is another condition likely to be mistaken for dementia because of the neuropsychiatric features. Acute nature, fluctuating course, associated autonomic features and history of obvious precipitating factors help distinguish delirium from dementia. It is important to be aware that patients with dementia can develop delirium in situations of an acute illness or on exposure to certain drugs that are active in the CNS.

INFECTIONS

Acquired immunodeficiency Syndrome (HIV) has affected millions in India and is on the rise. The nervous system is the most frequent target and a variety of intracranial complications of AIDS can cause impaired cognition, altered sensorium and even dementia by a variety of mechanisms. These syndrome complexes are often termed as HIV encephalopathy, HIV Associated Dementia (HAD) or AIDS- dementia complex. HAD causes progressive subcortical dementia, which begins with loss of memory, poor concentration and reduced intellect, and is followed by prominent behavioural changes, motor disabilities and profound dementia. The CSF examination reveals increased proteins and a mild pleocytosis. It is stated that HAD occurs in 15-20% of untreated AIDS patients but the figure varies with the diagnostic criteria and assessment methods and can be as high as 66%. Reports of HAD in Indian patients with CNS involvement are few. Being a late complication of HIV infection, HAD may be seen less often in India as patients with HIV infection die early in the disease from other causes.

Subacute Sclerosing Panencephalitis (SSPE) is a progressive, dementing disorder of children and young adults caused as a sequel of measles and still very much evident in India. A national registry with the Neurovirology Department of NIMHANS and a state registry with a two-year follow-up (1999-2001) recorded 49 cases of SSPE in Kerala giving an incidence of I per million/year. Subacute progressive dementia (100%), myoclonic jerks and a typical EEG with long interval periodic complexes are its hallmarks. The disease, with rare exception, is fatal as there is no specific treatment.

High prevalence of neurocysticercosis in India has made it one of the important infectious causes of dementia. The parenchymatous and meningitic forms can both cause dementia. In the latter variety, which is more common, multiple cysticerci in all stages of evolution enter the brain causing seizures, dementia, visual disorder, paralysis and raised intracranial pressure. Most of such presentations have been reported from India and China.

Neurosyphilis is on the wane in India as elsewhere in the world. The major dementing illnesses are General Paralysis of the Insane (GPI) and meningoencephalitis. Cognitive failure appears approximately 15 to 20 years after the original infection. In GPI, impaired intellect, failing memory, and poor concentration constitutes the 'simple dementing type' as opposed to the grandiose, agitated and psychotic form.

Creutzfeldt-Jacob Disease (CJD) is a transmissible disease due to abnormal Prion protein. The disease is invariably fatal. No accurate data is available from India on its prevalence. However, in an effort to assess the burden of the disease, a National Registry was set up which gathered 78 cases between 1968 to 2000 giving a figure of 0.07 per million population.

METABOLIC & NUTRITIONAL DISORDERS

Vitamin B12 and Folate Deficiency have a fairly well established association with neuropsychiatric manifestations, including dementia. Wadia et al

found that 18.2% of the 168 patients with Vitamin B 12 deficiency had neuropsychiatric disorders in the form of confusion/hallucination (3.5%), depression (5.3%), impaired memory (9.4%) or a score of 21 or less on the MMSE (8.9%). Other studies have reported that affective disorder is more common in those with folate than in those with Vitamin B 12 deficiency.

OTHER CAUSES OF DEMENTIA

It is not uncommon in clinical practice in India to find ataxic and epileptic diseases having dementia as a comorbid condition. They often include neurodegenerative as well as neurometabolic diseases. Dentato Rubro Pallido Luysian Atrophy (DRPLA) and Spinocerbellar ataxia type-2 and type-7 are the degenerative ataxia syndromes associated with dementia reported from India. The dementia mainly manifests as frontal subcortical signs with prominent behavioural and personality changes and is prominent in cases with an early onset of the disease. Lafora body disease is another disorder that is well documented in India and has a know association with dementia. The other diseases include neuronal ceroid lipofusinosis. Wilson's disease and mitochondrial encephalopathies.

OVERLAP SYNDROME

Dementia due to vascular disease (VaD) may coexist with that due to AD. This is seen not infrequently when a CVA is superimposed on a clinical picture and history suggestive of AD. A detailed history often provides evidence of the temporal relationship between the CVA and the cognitive defect. Such a condition is often called mixed dementia. The other overlap syndromes, are mainly under the rubric of familial dementia and involve the overlap of Parkinsonian or amyotrophic features over frontotemporal dementia.

APPROACH TO THE DIAGNOSIS OF DEMENTIA

When evaluating demented patients, an acute onset and/or a rapid progression should raise the possibility of a reversible cause. A careful exposure history will determine the possible role of drugs, metals or toxins. Physical examination may reveal focal deficits in cases of intracranial mass lesions and spasticity or ataxia of the lower limbs if hydrocephalus is present. Coexistence of dementia with peripheral neuropathy, asterixis, and myoclonus should prompt the search for a reversible cause. Focal abnormalities in EEG suggest an intracranial mass lesion while generalized abnormality suggests toxic-metabolic encephalopathy. A complete laboratory evaluation including blood counts, ESR, electrolytes, blood urea nitrogen, blood sugars, liver, renal and thyroid functions tests, serum calcium and phosphorus, B12, folate, copper and ceruloplasmin levels, VDRL, CSF study and neuroimaging are important components in the evaluation of any dementia.

Structural neuroimaging is mainly useful in vascular dementia. Functional neuroimaging such as Single Photon Emission CT (SPECT) studies reveal asymmetric hypoperfusion in the frontal and temporal regions in FTD, symmetric parietal and medial temporal regions in AD and DLB and patchy hypoperfusion in VaD, in early stages of the disease. Presence of diffuse slowing and triphasic waves in EEG is suggestive of a diagnosis of metabolic or toxic encephalopathy. Periodic complexes in EEG are a useful are marker for the diagnosis of SSEP and C1D. CSF biochemistry and cytology is abnormal in patients with dementia due to infections diseases. Detection of protein 14-3-3 in CSF is a useful marker for CJD.

Acknowledgments

The author wishes to thank Professor NH Wadia (Jaslok Hospital & Research Center, Mumbai) and Professor CJ Vas (Medical College Hospital, Goa) for providing some of the data presented in this chapter. Sincere thanks are also due to colleagues in the CBNC, SCTIMST for all their help and support in compiling this chapter.

A.D. basic ayurvedic Approach ..... from P.No. 4

- *Rasa* which act against *vata kapha rogas* is not available, which again affects the prognosis.

To sum up as per ayurvedic view, the pathological changes in AD is produced when external causative factors are superimposed on the already existing inevitable physiological changes of aging. And the facts to be considered are, age of different organs need not coincide with the chronological age and degeneration is deterioration and loss of specialized functions of cells, tissues and organs.

MANAGEMENT

While considering the degenerative pathology of the nervous tissue and the *vardhakya avastha* of the patient, we are forced to understand and accept the fact that it is less likely to reverse the pathology to normalcy through treatment. And hence the main objective of treatment is to assure and attempt in order to alleviate the diseases progression and to manage the secondary disease caused by the ongoing side by side pathologies, due to defective *dhathu* transformation and unhealthy *dhathu* formation.

While considering the palliative approach, the most effective treatment modality may be *Rasayana.*

The *Sodhana* and *peyadikramas* conducted prior to the *rasayana* therapy itself has some rejuvenating effects as evident from the *srothonirmalatha, dhathusudhi, kayagni deepthi, Dhathwagni deepthi*-which are the results of *samyak sodhana.*

WHY *RASAYANA* ?

Rasayana is considered as an appropriate choice because of the following facts.

It Corrects the structural deformity of the *dhathus,* there by normalizing their functions.

Prevents the instinct of recurrence.

Prevents occurrence of secondary diseases.

Prevents early aging process.

Rasayana oushadhas are not having a common *rasa, Veerya* or *guna* but all are *deepana* and *srothosodhana.*

Normal functioning of *srothas (srotho soushtava)* depends on healthy *dhathus* and *srothosoushtava* is highly essential to convey the nutrients to each and every cell.

Sodhana therapy acts at the level of *doshas,* but *rasayana* drugs acts still deeper that is at the level of *dhatus.*

Hence in deep seated diseases, where severe vitiation of *dhathus* occurs, such as AD, *rasayana chikitsa* in the regime of choice.

***Rasayana* therapy - How it works ?**

The possible mechanisms of action of *rasayana* drugs may be-

Neutriceutical action - Nourishes and maintains the cell life.

Regenerative action- Encourages the growth of new cells.

Immunomodulatory action - prevents recurrent infections, expelling the damaged cells.

Antioxidant action- Eliminates the toxic metabolites and pollutants.

Adaptogenic - Maintains the balance between mind and body.

Thus it may be postulated that *rasayana* drugs acts at the subcellular level.

Thus the approach to the patient considering the *rasayana* therapy as a main treatment modality can be through-

- *Pachana/Rookshana*
- *Deepana*
- *Snehapana*
- *Sodhana*
- *Keraliya Kriyakarma* (like *Sirodhara, Thalapothichil* etc.)
- and finally *rasayana*

PREVENTIVE ASPECTS

As far as the preventive aspects of a CNS degenerative condition like AD is considered, life style modifications including *ahara* and *vihara* are

NAGARJUNA MERIT EVENING 2007

essential. *Susruthacharya* explains the similarity of brain matter with solid *gritha* in consistency and no wonder, the importance of *Ksheera* and *grutha* in preventing CNS degeneration is evident from the indication of *Ksheera* and *grutha* for daily usage by the old age group in classics. It is now established that fat soluble materials alone can cross blood brain barriers and nourish brain. *Satwik* diet is another essential food discipline. Brain exercise through constant study, mutual discussion, learning other disciplines etc. keeps the brain matter healthy and fit and endow one with intelligence and memory.

To conclude, the pathology of AD is not happening by a single day. The unnatural early onset of aging process can be prevented by healthy living and thinking. The best method for this is explained well in the *rithucharya* and *dinacharya, achara rasayana* etc.

Panchakarma procedures and *medhya rasayanas* have a lot to do with the prevention of disease progression. The criteria for assessing the health status of a society not only depends on the density of old age group in that population but also on the mental and social well being of the group.

AWARDEES WITH DIGNITORIES



Padmasree. Dr. K. Rajagopalan, Kollam, Dr. M. R. Vasudevan Namboodiri, Principal, Govt. Ayurveda College, Thiruvananthapuram, Dr. K. Sankaran, Director, Ayurveda Medical Education, Govt. of Kerala, Dr. S. Vidyadharan, Principal, Sree Narayana Institute of Ayurvedic Studies & Research, Puthur, Kollam, Dr. R. Sreekumar, General Secretary, Akhila Kerala Govt. Ayurveda College Adyapaka Samghatana, Dr. Sandeep D. Nair, General Secretary, P. G. Students Association, Govt. AVC, TVM (Sitting) & Dr C. Manoj Kumar, Deputy Manager, Ayurveda, Nagarjua (Standing back) with the winners of NAGARJUNA MERIT AWARD 2007.

NAGARJUNA MERIT EVENING 2007

Nagarjuna Research Foundation is the organ of Nagarjuna Ayurvedic Group to advance the cause of Ayurveda through scientific research, dissemination of information and promotion of herbal and medicinal gardens. N R F has been regularly conducting Merit Evening to honour the first rank holders in B.A.M.S. examination every year from the Universities of Kerala. The Merit Award (ASHTAVAIDYAN BRAHMASREE ARYAN MOOSS MEMMORIAL) consists of cash price and a plaque. As a matter of motivation to students NRF is very particular to organize Merit Evening in one of the Ayurveda Colleges in Kerala in a rotational manner. Inter disciplinary seminar on academic and clinical interest is also a part of the function.

NRF Merit Evening 2007 had organized in Government Ayurveda College, Thiruvananthapuram on 24th November 2007. Padmasree Dr. K. Rajagopalan, the colossal presence among Ayurvedic scholars, academicians and physicians gifted the function presenting the award. Dr.Anoopa Sivapal (Kerala University), Dr. A. J. Asitha (M. G. University), Dr. S. Ramya (Calicut University) & Dr. S. Soniyaraj (Kannur University) received the awards. Dr. K. Sankaran, Director, Ayurveda Medical Education, government of Kerala, in his key-note address admired the contributions of Nagarjuna Ayurvedic Group for their research and development activities and shared the reminiscence of his personal experiences in this connection.

Dr. M.R. Vasudevan Namboodiri, Principal, Government Ayurveda College, Thiruvananthapuram presided over the function. Dr. R. Sreekumar, Professor & HOD, Roganidana, Government Ayurveda College, Thiruvananthapuram & General Secretary Akhila Kerala Govt. Ayurveda College Adhyapaka Samghatana and Dr. Sandeep D. Nair, General Secretary. Post Graduate Students Association felicitated.

An inter disciplinary seminar on Alzheimer's Disease was also organized. Dr.K.Vidyadharan, former principal, Government Ayurveda College, Thiruvananthapuram and Principal, Sree Narayana Institute for Ayurvedic Studies & Research, Kollam was the moderator. Dr. M. R. Vasudevan Namboodiri, Principal, Government Ayurveda College, Thiruvananthapuram presented his paper on basic Ayurvedic approach to Alzheimer's Disease. The modern concept was presented by Dr. P. S. Mathuranath, DNB(MED) DM (NEURO), Professor of Neurology, Sree Chitra Thirunal Institute for medical sciences And Associate Technology, Thiruvananthapuram. The rich gathering of UG & PG students and teachers interacted with the faculty.

Dr.C.Manoj Kumar, Manager (Ayurveda), Nagarjuna Ayurvedic Group, welcomed the gathering and Mr. H. Krishnakumar, General Secretary, College union, rendered vote of thanks.

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UNDERSTANDING A PERSON OR SELF- REALIZATION

*Dr. A. RAGHUNATHAN, **Dr. K. SUNDARAN

INTRODUCTION

Person in Indian Philosophical descriptions found by the name *purusha,* is the matter of discussion while encountering the substratum of sensuacis power. In the older texts of Ayurveda like *Charakasamhita, purusha* is emphasized as the conglomeration of 24 factors *(chaturvimshatika)* or six factors viz, five *mahabootas* and the *Atman*, the unique being cause of sensuousness. The atman alone is also reputed as *purusha* depicted *chetanaadhatu.* But practically, *purusha* in the eyes of a physician is different. He is the *chikitsadhikrita purusha* whom a physician has to treat. That *purusha* is the physiological body appendaged with d*hatu, upadhatu* and *mala.*

Therefore, in short, one ayurvedic physician has to consider not the philosophical body regarding the treatment but the physiological body as the material for his field of deeds. This will be very clear regarding the objectives of treatment highlighted in ayurvedic texts.

Karyam dhathu samyam (the objective of treatment is homeostasis of bodily humours).

Dhathusamya kriya chokta thanthraou asya prayojanam (the benefit of this scientific branch is to establish the activities that lead in the homeostasis.)

To preserve *dhatus* (bodily parts) in the normal state as per the viewpoint of preventive aspect of the science whereas these are to be normalized from the abnormal states in the viewpoint of curable aspect. Both these aspects are coming under the responsibilities of a physician as per the prime objective of Ayurveda.

SCOPE OF EXAMINATIONS

To evaluate the condition of *dhatus* some examinations are highlighted by *acharyas. Dasavidha pareeksha* (tenfold examination) is one among them. This ten fold examination is not merely the examination of ten aspects of a patient. It is really the examination of the former mentioned physiological body (*chikitsadhikrita purusha*). Out of ten only *vikrititah pareeksha* (examination of abnormality in a patient) using the methodology of *nidaana panchaka* is applicable to a patient. Other nine are to be applied on both normal as well as abnormal persons. Therefore, this method of examination can be applied not only to investigate the condition of *dhatus* of a second person but also to examine, self.

1. *PRAKRUTI*
   (investigation on constitution innate nature)
2. *VIKRUTI*
   (investigation on abnormal constitution)
3. *SARA* (investigation on body excellence)

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4. *SATVA* (investigation on mental nature on behavioural nature).
5. *SAMHANA* (investigation on physical statima)
6. *SATMYA* (investigation on body accostamation)
7. *PRAMANA* (investigation on body measureness)
8. *AHARASAKTI* (investigation on digestive capacity)
9. *VYAYAMASAKTI* (investigation on physical education).
10. *VAYAS* (investigation on age)

Most of the examination methods attributed in Indian sciences are for self-analysis also, leading a person for Self-realization as its final goal. *Bharttruhari's* verse of self-realization is very practical. In this contexts the benefit of this scientific branch is to establish the activities that lead to the homeostasis.

The *dhatus* (bodily parts) are normal in the viewpoint of preventive aspect of the science whereas these are to be normalized from the abnormal states in the viewpoint of curable aspect. Both these aspects are coming under the responsibilities of a physician as per the prime objective of Ayurveda.

kakaalah, kaanimitrani
kodesah kwou vyavyaou ?
kaschaham kaacha me saktih
iti chityam muhurmuhuh

The meaning of this verse may resumed as follows.

Every individual should be cautious regarding the time factor (positive and negative time of a person, atmosphere, self-physical age etc...) site factor (site of action of an individual, positive and negative influences, own physical body condition etc.) status of own friendly circle, debits and credits and finally the fact of "whom am I" and "my own capacity regarding the current issue".

TWELVE ASPECTS OF *SATVA PAREEKSHA*

To analyze self or *satva* of an individual a readily available methodology is there in the final two chapters of *Srimad bhagavatgeetha.* In these two chapters twelve aspects in connection with *Satva* are highlighted with three varieties as per the dominance of cosmic properties *(triguna).* The twelve aspects are either the behaviour patterns or attitude versions of an individual (see table). For instance self-intention of people is analysed first, which can be seen in three types usually. Some persons will be interested to do work for the accomplishment of pleasure of own intellect and mind e.g.:- construction of a School, Public Service Schemes etc., without any monitory benefit. Some may do such works with an intention to increase their fame, reputation or to accomplish their own ego. The former category comes under *Satvika* and the latter under *rajasika sradhaa.* The third one, *taamasika* category will be indulged to construct monuments or memorials of dead people as a matter of traditional trial.

Likewise, the second aspect i.e., food habit-also varies into three types as per *triguna* influence. *Satvika* people desire nutritious, salubrious and palatable food, *rajasa* people take acrid hot, much spicy food and the last type prefer decayed, worthless food articles.

Thus, all the twelve aspects are categorized and that will be still practical if we do properly take the message of the verses from the texts.

For current use, we have to compile all the characteristic features of these twelve aspects first in a table and arrange them into a good format. Then modify them in appropriate with

our own individual concerns. Then count the scores from each one among these twelve aspects. Count the final score and realize the position of us in the network of *trigunas.*

Understanding an individual or oneself is for what purpose? This is meant to correct oneself and reach higher in the field of personal skills. Higher levels in common use is nothing but the attainment of more *Sattvaguna* inside us. If an individual scored less points from *Sattvaguna* and more points from other two *gunas*, that shows he has to improve a lot. On the endeavour of improvements he has to understand his position first and then has to avoid possible *rajasika* and *tamasika* aspects. If he succeed in that naturally, he will be attaining more points from *Sattvaguna.* By the principle of

"Sarvadaa sarvabhaavaanam saamaanyam vridhikaranam".

More scoring of *Sattvaguna* naturally lead one to higher levels. Therefore, the understanding of twelve aspects is not only for self-realization but for self-correction also.

Out of these twelve aspects *sraddha* and *tapas* have more concern with the psychiatric treatment. Generally, one of the duties of physician is the education of patient regarding his illness and its countering ways. This will be more intensified regarding a psychiatric physician. Ayurvedic texts also hold this supreme remedy *(Paramoushadhi)* in the field of psychiatry.

"Dhee dhairyaadmaadi vijnanam manodoshoushadham param"

The psychological measures we have to concentrate are much obvious as described in *bhootagraha chikitsa.* This has to be applied in all psychiatric cases indeed.

These are

Tapah	-	Discipline
Seelah	-	Behavioral attributes
Samaadhanah	-	Consolation
Jnanaah	-	Awareness
Daanah	-	Support
Dayaa	-	Empathy

TAPAS AND *SEELA* IN CURRENT PRACTICE

As we already noted *Tapas* is one of the aspects to improve our own mental ability. That *Tapas* is not meant for penance or asterism but for discipline as sensual meaning. It is not a hypothesis but a fact that the *Tapas* is certainly indicative of the meaning discipline in Indian literature in most of the places. Evidence for the fact is seen in the description of *Bhagavatgeetha* itself, where *Tapas* is divided into three as follows.

First division *sareerika Tapas* includes the affinity to worship the respectful persons in the society, cleanliness, straight-forwardness, celibacy and nonviolence.

Vachika Tapas, the second discipline comprises of the use of words that are true, desirable and positive; the practice of enhanting hymns and reading texts is also included in it.

Manasika tapas, the last division is indicative for pleasant nature in mind, soft nature in behavior, practice of silence, as well as self control and purity even for thoughts (*Bhagavat geeta* 17/14/-16)

These three are nothing but physical discipline, verbal discipline and mental discipline.

Let us see the taste of disciplinary practice. How we can make a person disciplined for various means. In fact, these means we do experience from our childhood as the instruction and indications by parents, elders and teachers. For instance if we in our childhood abuse our tongue, our parents might have corrected that by scolding, beating or advising us properly.

How we can learn to dress properly? Behind that lot of turmoil of our parents will have there. Thus by lots of training we became cultured or disciplined is most which is attained by the

To..... P.No. 26

SCHIZOPHRENIA IN INDIAN SOCIETY

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INTRODUCTION

About 1% of population worldwide is suffering from schizophrenia. There are number of studies conducted earlier to estimate the incidence rate, prevalence rates of schizophrenia and to assess the burden of this illness on the society. Studies conducted to assess distribution of incidences in relation to age sex and other factors has helped for formulating an aetiological hypothesis for the disease.

In India, the first large scale study on prevalence of this disease was conducted by Indian Council Of Medical Research , Department of Science and Technology (ICMR-DST) at four centers in 1987. The studies examined population of varying sizes, ranging from 327 to 1,01,229. The reported prevalence rates for schizophrenia was estimated to vary from 0.9 to 7.2 per 1000 (urban 2.5 and rural 3.6).

Some of the specific observations made on Schizophrenia patterns in India are

- higher incidence
- increased rates among men compared with the women
- better prognosis

The features of *Unmada* resemble with the features of schizophrenia in contemporary medicine. In this paper, attempt is made to present the aetiological factors and assessment parameters in more practicable forms.

UNMADA (SCHIZOPHRENIA?) IN AYURVEDA

Definition

Unmada or mental aberration is that condition where in mind decision, orientation , responsiveness, memory, desire, habits and emberment, psychomotor activity and conduct are astrayed. As per Ayurveda, *Unmada* is broadly classified into six categories, 1) *Vataja Unmada,* 2) *Pittaja Unmada,* 3) *Kaphaja Unmada,* 4) *Tridoshaja Unmada,* 5) *Aadija Unmada,* 6) *Vishaja Unmada.*

***Nidana* (Aetiology):** *Samanya* and *Visesa nidana* are separately described for *Dosaja Unmada* and *Agantu Unmada.* The different aetiological factors can be classified into different categories like factors related to *Ahara* (food) and *Vihara* (psycho-behavioural activities), those affecting bodily *Dosa* like *Vata, Pitta* and *Kapha* and *Manasa Dosa* viz., *Rajas* and *Tamas*, those which are *Sannikrista* (immediate/recent) and *Viprakrista* (remote, *Bhahya* (external) and *Abhyantara* (internal), etc., for facilitating better management. It is also necessary to consider those factors which have been termed as *Apathya* (contra-indications) in *Unmada* while studying its aetiology.

Samanya Nidana

- *Bhiruta* (timidity)
- *Upaklista Satva* (agitated/disturbed mental state).

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- *Vyadhivegasasamubhrama* (perturbation due to severity of disease).
- *Abhighatabhyahata* (mental disturbance caused due to trauma).
- *Devagurudvijapradharshana* (disrespect to gods / teachers/twice born learned)
- *Pujavyatikrama* (deviation from the method of worship)
- *Visamasarera Cesta* (faulty bodily activity)
- *Utsannadosa* (exceedingly emaciated body)
- *Samalovikrtopahita Anuchitaviruddha Aharase* va (intake of unhygienic or unclean / sate or spoilt / adulterated/ incongruous food).

ETIOLOGY / RISK FACTORS FOR SCHIZOPHRENIA

Exact etiology of Schizophrenia is unknown, however several suspected theories have been proposed; these include the followings:

1. **Genetics:** A family history of schizophrenia is the most firmly established risk factor for this disorder. About 10% of first degree relatives (and 3% of second degree relatives and 2% of third degree relatives) of schizophrenia patients-have schizophrenia as compared with the 1% prevalence rate in the general population[5]. The concordance rate for monozygotic twins is 46% and for dizygotic twins 17%. If one parent has schizophrenia, the chances of the child developing schizophrenia are 10-14%. However, if both parents have schizophrenia, the chance of the child developing schizophrenia increase to about 46%.

2. **Sex:** Lifetime risk for schizophrenia is generally assumed to be same in males and females. There is general agreement that onset is later in female than in male patients, in males onset is generally in the early to mid-twenties whereas the majority of the studies find that the mean age of onset in females is four to five years later. Some studies have indicated that male patient have poor outcome than female patients. But this may be explained by the earlier age of onset in males as early age of onset in itself is a predictor of poor outcome.

3. **Environmental Factors:** The fact that only approximately half instead of all monozygotic twins to patients with schizophrenia themselves develop schizophrenia, indicates that non genetic factors are important in the aetiology of this disease, as monozygotic twins are genetically identical. Most of these possible risk factors are believed to act through mechanism affecting the development of the brain during embryonic and fetal life.

4. **Urban birth and urban upbringing:** It was found that the prevalence of schizophrenia is higher in urban than in rural areas even urban birth not only urban residence at the time of onset, has been found to be a risk factor for schizophrenia. But the cause of this is unknown.

5. **Season of birth:** An excess of individuals born during winter and spring moth among schizophrenia epidemiology.

   In a large study, McGrath et al compared rates of schizophrenia in individuals born in the southern hemisphere to those born in the northern hemisphere who had later immigrated to Australia. They found an excess both in those born in the Northern hemisphere in March to April, as well as in those born in the Southern hemisphere winter (July to September).

6. **Biochemical Hypothesis:** Schizophrenia is presently thought to be probably due to a functional increase of dopamine at the post synaptic receptor, though other neurotransmitters like serotonin (especially 5-HT receptors), GABA and acetyl choline are also presumably involved.

7. **Influenza and schizophrenia:** At present influenza is probably one of the most plausible environmental risk factors in schizophrenia epidemiology. It should be noted that our knowledge is not sufficient to justify any particular warnings or risk estimates for women who are exposed to influenza during mid trimester pregnancy. Also other infections have been suggested as possible risk factors for schizophrenia, these include various infections during fetal life or early child hood transmitted through cats, or other household pets, or CNS infections, eg. meningitis during early childhood.

8. **Obstetric complications:** Obstetric complications have been associated with increased schizophrenia in a large number of studies[15]. A recent meta analyses of the existing studies[16], concluded that children exposed to obstetric complications were twice as likely to develop schizophrenia. The specific nature of complications that particularly increase schizophrenia risk has not been identified without certainly, one specific factor rhesus incompatibility, have been suggested but this would only apply to minority of cases. In summary, the literature suggest that a causal role of obstetric complications in schizophrenia is very likely but more research is needed to clarify the specific nature of this relationship.

9. **Social Class:** It has been an early observation that the prevalence of severe mental disorders including schizophrenia was higher in those of lower social class compared with high social class, these findings are largely excepted but the explanation for this is the subject of a continuing debate.

10. **Stress:** Increased number of stressful life events before the onset or relapse probably as a triggering effect on the onset of schizophrenia, in a vulnerable person eg, genetic vulnerability and stress vulnerability hypothesis.

Samprapti (Pathogenesis)

The excited *Dosha* of an individual with *Alpa satva* (weak psyche) mounts upwards through *Manovahasrota* vitiate *Hrudaya* the seat of *Buddhi* and disturb *Manas* which is located between *Sira* and *Talu* and there by occlude *Manovahasrota*. As a result, *Citta* is disturbed. This in turn causes impairment of *Buddhi*. Due to this, the individual loses of the power of discrimination and indulges in faulty activities. This disturbed state is referred to as *Unmada.*

Samanya Purvarupa (General premonitory features)

1. *Sirasahsunyata* (emptiness in the head)
2. *Cakshorakulata* (restlessness of the eyes)
3 *Karnasvana* (sound in the ears)
4 *Uchvasadhikya* (hurried breathing)
5. *Asyasamsravanam* (dribbling of saliva)
6. *Arocak* (tastelessness)
7. *Avipaka* (indigestion)
8. *Anannabhilasa* (aversion to food)
9. *Hrdgraha* (catching pain in the heart region)
10. *Asthana Ayasa Sanmohadhyana Udvega*

(Fatigue confusion/mental absorption/anxiety out of context).

11. *Atyutsaha* (over enthusiasm).
12. *Satatamlomaharsah* (continued horripilation)
13. *Jvaraschabhikshnam* (frequent fever)
14. *Unmattachitatvam* (Crazy mind)
15. *Udarditatvan* (pain in the upper half of the body)
16. *Arditakritkaranam Cha Vyadheh* (appearance of features presented in facial paralysis (bell's palsy).
17. *Svapne Chabhikshnam Bhranta Chalitanavasthita Rupanidarsanam* (frequently dreaming of roving / moving / shaking forms).
18. *Svapne Tilapoedakachakradhi-Rohanam Bharamaschakragatosya Va* (dreaming of oneself mounting on the wheels of an oil press).
19. *Svapne Vatakundalikabhischonomathanam Majjanam Kulusamanbha Samavarta* (dreaming of oneself being churned by whirl-wind sunk in the whirlpools and dirty waters.
20. *Svapne kalusabhojanam* (dreaming of eating dirty things).
21. *Chaksusah Apasarpanam* (retraction of the eye balls).
22. *Gatranamapakarsanam* (emaciation).

The prodromal features reflect the involvement of the individual's psychological physiological and social aspects. It is interesting to note that many of the *Purvarupa*, appear in dreams. Therefore, it would be worthwhile studying the dreams of even normal subjects so that it will become possible to detect the persons who might be vulnerable to get *unmada.* A thorough knowledge of *Samanyapurvarupa* would also aid forecasting the type of *Unmada* that one might get even in the absence of *Visesapurvarupas* and help in taking preventive measures. It is however necessary to consider examples appropriate to contemporary life in place of classical ones, while understanding the *Purvarupa* of *Unmada.*

Laksana (Clinical signs and symptoms)

1 *Dhivibhrama* (impaired intellect)
2 *Satvaparipla* (crazy / fickle mind)
3 *Paryakuladrusti* (agitated look)
4 *Adhiarata* (lack of courage)
5. *Abaddhavatktva* (innocent speech)
6. *Hrdayasunyata* (vacuity in the heart)
7. *Na Sukham / Dukham / Dharmam / Santim / Acaram Vindati* (does not understand pleasure / pain / righteousness/peace / conduct).
8. *Apsta Smrti / Buddhi / Sajna* (deprived of memory / decision/orientation and responsiveness).
9. *Acintitarambha* (aimless activity)
10. *Dehadukha Sukha Bhrasto Bhrastasarathivadratha Bbramati* (forgetting pleasure or pain, wanders like a chariot without a charioteer)
11. *Vijnanabhrama* (distored understanding).

It will be clear from the above, that after *sthanasamsraya* the involvement of *Manas* in relation to the individual's psychological and social behaviour will be complete at the stage of the onset of *Rupa* than the *Purvarupa* stage.

Clinical features and Diagnostic criteria for Schizophrenia

Characteristic symptoms	ICD-10	DSM-IV
At least one feature of	Thought echo, thought insertion/ withdrawal / broadcast	Bizarre delusions
	Passivity, delusional perception	Third person auditory hallucinations
	Third person auditory hallucination, running commentary	Running commentary
	Persistent bizarre delusions	
Two or more features of	Persistent hallucinations	Delusions
	Thought disorder	Hallucinations
	Catatonic behaviour	Disorganized speech
	Negative symptoms	Grossly disorganized behaviour
	Significant behaviour change	Negative symptoms
Duration	More than 1month	1 month of characteristics symptoms
		With 6 months of social / Occupational dysfunction
Excl: Criteria	Mood disorders	Schizoaffective or mood disorders
	Schizoaffective disorder Overt brain disease	Direct consequences of substances use or general medical condition
	Drug intoxication or withdrawal	Pervasive developmental disorders

Conclusion

It should hopefully be clear from this review that schizophrenia is a disorder of major public health concern, of both genetic and environmental origin. It is a disorder where much research still remains to he conducted before we think of possible prevention. Future studies in schizophrenia have to include both genetic and environmental risk factors, which will help to apply the available epidemiological evidences for effective management of the disease.

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From Pune.....
Dr. Anuradha Haridas Benkar

My dear friends of Ayurveda fraternity,

My husband, Dr. Haridas Benkar and myself are running ***Sree Samarth Hospital*** at ***Chandoli Rajguru Nagar, Pune***. We have been looking for an authentic destination for getting expertise in *Panchakarma* treatments to meet the increasing need of treatment modalities in curative as well as preventive health care. Our visit to www.nagarjun.com opened the doors of **Nagarjua Ayurvedic Institute** before us. I came alone for the first time to Nagarjuna Ayurvedic Institute, Kalady, Eranakulam Dist, Kerala on 31st January 2008. All the arrangements for my safe arrival were done by the Institute well in advance. My purpose was to get training on the practical applications of Panchakarma and Kerala special treatments for curative as well as preventive health care. I got informative study materials compiled from the classics and Kerala texts covering introduction to contributions of ancient Kerala physicians to the practical applications of Panchakarma, Practical hand book on 20 types of treatments and Treatment protocols of Old age care & Alcohol de - addiction.

Theory classes in tutorial system and practical sessions under the supervision of institute faculty were highly informative and interesting. All the treatment procedures were doing in more scientific and effective way than what we are doing in North India. I learnt the seven positions for *Abhyanga*, scientific procedures for *Pizhichil(dhara)*, *shashtika Pinda Sweda, Patra Potala Sweda, Dhanyamla Dhara, Talam* etc. In addition to the classes from in house faculty, the institute arranged interactions with senior reputed faculty from the Ayurveda College.

The opportunities I got to attend the out patient and in patient department with the senior Physicians also added knowledge and confidence to my career. Patients with all types of ailments are approaching the hospital and getting cured by pure Ayurvedic medicines and treatments. I got chance to attend a big medical camp organized by Nagarjuna Ayurvedic Center and a visit to the Government Ayurveda college. Every where large number of patients are getting cured with Ayurveda. I met with so many foreign students also in the campus. The atmosphere was conducive for study, training and comfortable stay.

I have sincere thanks to the Director, Dean of studies, Chief Physician, Course co coordinator and all the faculty members and staff of the institute for informative and loving experience in Kerala.

Nagarjuna Ayurvedic institute authorities assured to guide me for setting up and running smoothly a treatment centre attached to our hospital in Pune. Nagarjuna Ayurvedic Group has a wide marketing network through out India and their special formulations also will be available to us.

Ya, my training in Nagarjuna Ayurvedic Institute instilled real confidence to start authentic Panchakarma center at our place in Pune.

With love and regards

Dr. Anuradha Haridas Benkar

The Convocation.....

OUT GOING BATCH THERAPIST TRAINEES



Dr. C. Retnakaran, Principal, Govt. Ayurveda College, Thripunithura, presenting the certificate.
Dr. C. S. Krishnakumar, Director, Dr. K. Krishnan Nampoothiri, Chief Phycisian, NAC Kalady,
Dr. S. Dilip Kumar, Dean of Studies, Dr. Jeevan (Germany) look on..

Doctors Orientation Programme on Panchakarma & Kerala Special Treatments



Dr. Anuradha Haridas Benkar receiving the Certificate from
Dr. C. S. Krishnakumar, Director, Nagarjuna Ayurvedic Institute, Kalady.
Dr. K. Krishnan Namboodiri, Chief Physician, Nagarjuna Ayurvedic Centre Kalady also seen.

Articles on ***Twak Pareeksha*** and ***Role of Food in Healthcare*** will be continued in next issue - Editor.

Understanding a person ..... from P.No. 18

induction of various techniques or *upayas* in ayurvedic forms.

In Indian psychiatry a group of *upayas* is highlighted which are psychotherapeutic teachings what we currently say. Treatment through disciplinary proudness *(tapas)* is performed by as above means. To induce good behavioral methods *(seela)* also, *upayas* are essential.

upayas are the tools for *Satvajaya chikitsa* as *Acharya Charaka* mentioned *Satvajaya is upayabhiplutam (cha.vim.8/87).*

CONCLUSION

The study materials described in Ayurveda to understand an aflicted (a patient who is with abnormal humours or person who is normal as both are the subjects of this Science) are for self recognition also. The twelve factors description in *bhagavatgeeta* to assess the psychic disposition can be used for self recognition and there for self pacification physicians have responsibility not only to guide but also to teach the patients.

STATEMENT OF OWNERSHIP AND OTHER PARTICULARS ABOUT AYURVEDIC RENAISSANCE

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Publisher

AYURVEDIC UNDERSTANDING OF SLEEP DISORDERS*

V. S. Sreelakshmi**

Sleep, one of the basic and essential needs of life, has become a matter of growing concern and a field for quick paced researches in the present era. This is because even such an elementary mechanism of the body is being subjected to uncontrollable disruptions and variations, which drags the patient to a hapless crisis. This situation urged the need for extensive research work to explore the mysterious mechanism of sleep and its different pathological pathways. The scene projects a sharp contrast from the bygone period of Ayurveda, where neither the physician nor the patient might have channeled greater focus and energy for considering probably a minor symptom i.e. the sleep disorder.

CLASSICAL REFERENCES

The classical texts mainly deal with the pathologies of those symptoms, which have presented as *Swatantra rogas* (primary diseases) creating a great deal of concern for the patient. However, they are also enlisted among the symptoms of other diseases as *Partantra rogas*, (secondary diseases). For example, *sopha* is a disease dealt separately as well as a symptom of *vatika pandu*. Keeping in mind this fact, the pathologies of sleep have not been allocated an exclusive chapter. Instead, they have been mentioned at appropriate instances as mere *paratantra rogas*. Therefore, in spite of the discussions of the detailed anatomy and physiology concerned with sleep, primary disorders of sleep were of lesser prevalence as well as importance as is clear from the classics.

SLEEP PHYSIOLOGY FROM CLASSICS

An individual sleeps when the *srotas* of the body becomes obstructed by kapha and sense organs withdraw from functioning supplemented by fatigue of the body with exertion1[1]. Another postulation is that *hrudaya*, the seat of *chetana*, when subdued with *tamas* induces sleep in an individual. In fact, both the expositions can be closely linked upon close scrutiny, in the context of *swapna, manovahasrotas* has been mentioned as the site of the process of its manifestation. *Chakrapani* explains the *dasadhamani* connected to the *hrudaya* themselves as the *manovahasrotas*. Moreover, *hrudaya* has been explained to be the location of *vijnanam*, senses (*indriyas*), their objects, mind and its objects. All these facts point out that *hrudaya* is, no doubt, the seat of *nidra.Hrudaya* is the origin of the *rasavahasrotas*. The *dasadhamani* connected with *hrudaya* forms the *manovahasrotas*. Moreover, *hrudaya* itself is an abode of somatic, psychic and spiritual functions. So naturally, *nidra* being a function of *hrudaya* has two platforms namely, Somatic (*rasavahasrotas*) and Psychic (*dasasdhamanis* or *manovahasrotas*).

To brief up, sleep manifests when *tamas* and *kaphadosha* afflicts the heart and blocks the *manovahasrotas* and *rasavahasrotas* respectively.

*Price winning paper presented in INDPSYCHE 07, seminar organized by Govt. Ayurvedic Researc Institute for mental diseases, Kottakkal, Kerala

** Third professional B.A.M.S student, V.P.S.V. Ayurveda College, Kottakkal, Kerala.

CLASSIFICATION

Charaka has classified sleep into seven types. It can be established that four of them namely *sleshmodbhavam, tamodbhavam, sareerashramodhavam, manasramodbhavam* are indicative of excessive sleep. *Agantuki* is explained to be fatal and *vyadhianuvartini* denotes sleep occurring secondary to diseases. Moreover, physiological sleep (*ratriswbhavaja)* is also mentioned herewith. The three fold classification of *Sushruta* as *tamasi, vaikariki* and *swabhaviki* projects a similar idea. *Tamasi nidra* is that which occurs at the time of *pralaya* from which the individual cannot be waken with external stimuli. This may be judged as the loss of sensorial occurring prior to death. *Swabhaviki nidra* and its subdivisions viz *rajobhuyishta, tamobhuyishta* and *satwabhuyishta* again details normal sleep variations according to personality types.

When such a classification of sleep is analyzed in the wake of updated medical knowledge, it cannot be directly incorporated for categorizing disorders of sleep. Therefore, for the purpose of easier study and suggestion of management protocol, disorders of sleep may be grouped as:

- Deficient sleep
- Excessive sleep
- Others

DEFICIENT SLEEP

Deficient sleep has been explained in the classics with terms like *nidranasa, nidrabhramsa, nidtraalpah, atnidrata, aswapna, nidrabhramsa, nidrahani, jagaranam, nistandri, prajagaranam, unnidrata, vinidra* and *nidraviparyaya.* Each of them points out the different aspects of insomnia such as frequent awakening during night, early morning awakening or feeling of not having rested despite adequate duration.

The pathology underlying deficient sleep is attributed on the dual platform as mentioned earlier:

- Somatic: *vata vrudhi, pittavrudhi, kapha kshaya, abhighata*
- Psychic: *manasthapa, rajovrudhi, tamakshaya, abhighata*

A substantial decrease (*kshaya*) of a *dosha* results in the manifestation of symptoms rather than a disease. Hence the pathology usually concentrates upon *vata, pitta* and *rajovrudhi,* than *kapha* and *tama kshaya.* This mechanism can fit in cases of primary insomnia. In cases of secondary deficiencies, the same can be applied. Consider few examples along with the *srotas* involved:

DISEASE	*SROTAS*
Jwarapoorvaroopa, vata jwara, pitta jwara, vatapitta jwara, sannipata jwara	*Rasavahasrotas*
Rakta vrudhi, vatarakta, pitta visarpa, agnivisarpa	*Raktavahasrotas*
Amavata, vatika prameha	*Medovahasrotas*
Asthigatavata, vata vyadhi	*Asthivahasrotas*
Majjagatavata	*Majjavahasrotas*
Vidahyamana gulma, parswasoolaAnnavahasrotas	
Vatika trishna	*Udakavahasrotas*

Pranavahasrotas

The peculiarity of *pranavaha srotas* is quite noteworthy ion this context. For that the concept of *pranavahasrotas* has to be detailed further so as to expose its dual nature. The origins of *pranavahasrotas* are *hrudaya* and *mahasrotas. Hrudaya*, being the seat of the *manovahasrotas*, represents the psychic aspect. This forms the basis of the functions of the *pranavayu* like maintenance of intellect, sense organs, mind, etc.

obviously *mahasrotas* represents the somatic aspect. This is responsible for the functions like expectoration, sneezing, belching, respiration, swallowing of food, etc. it is evident that *pranavahasrotas* is one which links the soma and the psyche. This theory explains how peptic ulcer is pathology of the GIT elicited by purely psychogenic factors. So any disturbance in the psyche can disrupt the *mahasrotas*. This also proves how deficient sleep occurs secondary to diseases in the *mahasrotas*.

Psychic: *manasika vikaras* like *apasmara poorvaroopa, kamaja jwraa, paittika unmade, ahija unmade*, etc.

Others: *andhaputana graham, skandapasmara, naigamesha graham, rakhasa graham*, etc.

Another cause for deficiency is iatrogenic (*langhana atiyoga*) which has been uniquely mentioned in Ayurveda.

Thus the deficiency of sleep is related with diseases of any of the *dhatus* or *srotas*. So an effective treatment protocol must be formulated considering these vitiations also.

EXCESS SLEEP

Excess sleep has been assigned terms like *atinidra, bahunidrata, mahanidra*, etc. in the classics. The pathological principles, again on the dual basis is as follows:

- Somatic: *vatakshaya, kaphavrudhi, kaphaprakopa, sanniatakopa, rasa vrudhi*
- Psychic: *tamovrudhi*

This derangement accounts for any case of hypersomnia. The diseases wherein the excess of sleep occurs secondarily are enlisted as follows: *Atistoulya, kaphaja sopham, kaphaja hrudrogam, kaphaja apasmaram, kaphaja madam, kaphaja jwaram, jwara poorvaroopam, sannipata jwaram, kaphavata jwaram*, etc. these are none other than the symptoms of various disorders like hypothyroidism, cerebral tumours, encephalitis, trypanosomias, etc. which cause secondary hypersomnia. Dhwamsakam and kaphaja madatyayam also exhibit hypersomnia, which may be equate to alcohol abuse. In addition, hypersomnia due to iatrogenic causes (snehavasthi vyapad), skin diseases (*kardama visarpa*), GIT disorders (*kaphaja gulmama, kaphaja atisaram*, etc) have been proposed exclusively by Ayurveda.

Shodhana atiyoga is found to cause both excess as well as deficiency of sleep. A possible explanation for this contradiction can be given by considering two stages occurring in a *shodhanakarma* –

- The period of *vega pravartana* which is a state of *dhatuprakopa*, the patient being restless and exerting a lot. The patient experiences deficiency if sleep during the same and for some period thereafter.
- The period of *glani*, which is a state of tiredness after evacuation, is completed. The patient slips into prolonged sleep automatically out of fatigue.

OTHERS

Disorders caused by suppression of urge to sleep are yawning, malaise, drowsiness, headache and heaviness in the eyes. This can be directly related to sleep wake schedule disorders like jet lag, night shifts, wherein the individual forcefully avoids sleep. Parasomnia is characterized by episodic nocturnal events occurring with sleep. Stage- 4 sleep disorders include somnambulism, pavor nocturnes, somnilogy, bruxism, eneuresis, etc. not much supporting references can be sited in the classics for such diseases.

CONCLUSION

The condition is indeed grave as even a slight disturbance in the sleep is disturbing the health conscious individuals a lot that, this leads to further aggravation of the condition. Prevalence of secondary sleep disorders is on the rise. A careful analysis of the affected *dhatus* and *srotas* is vital. Deficient understanding of the same may tarnish the wide scope of Ayurveda in this field. Hence, as mentioned earlier these theories are mandatory in chalking out an effective management strategy for sleep disorders.

MANDOOKAPARNI

(Centella asiatica. Linn)

* BABY JOSEPH AND SOPHY PAUL

Malayalam	:	***Kudakan***
Sanskrit	:	***Mandookaparni***
English	:	**Indian Pennywort**
Hindi	:	***Brahmamanduki***
Family	:	**Apiaceae**

Centella asiatica has been used as a medicinal herb for thousands of years in India, China and Indonesia. Its ability to boost memory power, heal memory problems, improve mental clarity, treat skin conditions such as leprosy, psoriasis were important reasons for its extensive use. So it has been called one of the "miracle elixirs of life" because legend has it that an ancient Chinese herbalist lived for more than 200 years as a result of using the herb.

It is also called "Brahmi" which means 'Bringing knowledge of the supreme reality' also good for revitalizing herb that strengthens nervous function and memory.

Distribution & Habitat: Mandookaparni is distributed throughout India and grown well in moist locations.

Habit and General features: It is a slender herbaceous creeping plant, stem long, prostrate, filiform with long internodes. 1-3 leaves arise from each node of the stem. Leaf has paracytic and diacytic type of stomata on both surface. Leaves orbicular- reniform crenate-dentate, base cordate and often lobed, long petioled. Flowers red, pink or white in fascicled umbels. Fruits oblong, brown, laterally compressed.

Part used: Whole plant

Ayurvedic Properties

Rasa	-	*Tiktha, Kasaya, Madhura*
Guna	-	*Laghu, Sara*
Virya	-	*Sheeta*
Vipaka	-	*Madhura*
Prabhava	-	*Medhya*
Doshagnatha	-	*Thridoshamaka*
Rogaghnata	-	*Apasmara, Unmada, Daurbalya, Agnimandhya, Grahani*
Karma	-	*Medhya, Vayasthapana, Rasayana, Swarya*

Actions and Uses : Centella asiatica is good for improving memory, tonic, nervine tonic, stomachic, carminative, antileprotic, diuretic and febrifuge. It is useful in cardiotonic, insomnia, cardiac debility, epilepsy, laryngitis, asthma, bronchitis, hiccough, abdominal disorders, leprosy, strangury and fever. It also good for skin diseases, nerves and blood abdominal disorders, dysentery.

The whole plant is alterative, cardio depressant, hypotensive, weakly sedative and tonic. It is a rejuvenating diuretic herb that clears toxins, reduces inflammations and fevers, improves healing and

* Dept. of Pharmacognosy, R & D, Nagarjuna Herbal Concentrates Ltd., Alakode, Thodupuzha

immunity, improves the memory and has a balancing effect on the nervous system. It has been suggested that regular use of the herb can rejuvenate the nervous system and it deserves attention as a possible cure for a wide range of nervous disorders including multiple sclerosis. Recent research has shown that Mandookaparni reduces scarring, improves circulatory problems in the lower limbs and speeds the healing process. It is used internally in the treatment of wounds, chronic skin conditions, venereal diseases, malaria, ulcers and nervous disorders. Externally, the herb is applied to wounds, haemorrhoides and rheumatic joints.

Historically, Mandookaparni has also been used to treat syphilis, hepatitis, stomach, ulcers, mental fatigue, epilepsy, diarrhoea, fever and asthma. Today, American and European herbalists use Centella for disorders that cause by connective tissue swelling, such as scleroderma, psoriatic arthritis, ankylosing spondilytis and rheumatoid arthritis. Recent studies suggest new applications for Centella, such as lowering high blood pressure, treating venous insufficiency, boosting memory and intelligence, easing anxiety, and speeding wound healing.

Important Formulations: *Brahmarasayanam*

Chemical constituents: Secondary metabolites like asiaticoside, Brahmoside, hydrocotylin, brahminoside, asiatic acid, medacassocide, brahmic acid, centelloside, centic acid also present.

Therapeutic evaluation: The result of Centella proved better on mentally retarded patients showed a very significant increase in both general ability and behavioural pattern when the drug was administered even for a short period of 12 weeks. It produced a significant improvement and behavioural change.

Fresh leaf extract treatment in adult rats shows enhancement of hippocampal CAS neuronal Dendritic Arborization. The adult rats (2.5 months old) were fed with 2,4 and 6 ml/kg body weight of fresh leaf extract of Centella for 2,4 and 6 weeks respectively. After the treatment period, the rats were killed. Brains were removed and hippocampal neurons were impregnated with golgi staining. Hippocampal CA3 neurons were traced and dendritic branching points (a measure of dendritic arborization) and intersections (a measure of dendritic length) were quantified. These data were compared with those of age matched control rats. The result showed a significant increase in the dendritic length (intersections and dendritic branching points along the length of both apical and basal dendrites in rats treated with 6ml/kg body weight/day of Centella for 6 weeks. However the rats treated with 2 and 4 ml/kg body weight/day for 2 and 4 weeks didn't show any significant change in hippocampal CA3 neuronal dendritic arborization. Present study reveals that constituents present in Centella asiatica fresh leaf extract has neuronal dendritic growth stimulating properties.(Gadahad M.R, Manipal Medical College)

WattanathornJ , Faculty of Medicine Khon Kaen University, Tailand studied that Centella asiatica has a reputation to restore decline cognitive function in traditional medicine. The result showed that high dose of the plant extract enhanced working memory and increased N100 component amplitude of event related potential; improvement of self rated mood were also found following the Centella asiatica treatment. Present works conclude the potential of Centella to attenuate the age related decline in cognitive function and mood disorder in the healthy elderly.

Ramanathan, Neuro Pharmacology, JSS College, Ootacamund conduct a study on Neuroprotective evaluation of standardized extract of Centella asiatica.

The effect of chloroform: methanolic (80:20) extract of Centella asiatica (CA;100 and 200 mg/kg), was evaluated on the course of free radical generation and excitotoxicity in monocodiumglutamate (MSG) treated female Sprague Dawley rats. The extract showed significant improvement in catalase, super oxide desmutase and lipid peroxides levels in hippocampus and striatum regions. Glutathione level was not altered with CA treatment. Similar observation was made with dextromethorphan. The general behaviour, locomotor activity and CAI a region of the hippocampus was significantly protected by CA indicating neuroprotective effect of CA in MSG induced excitotoxic condition. Hence it can be conducted that CA protected MSG induced neuro degeneration attributed to its antioxidant and behavioral properties. This activity of CA can be explored in epilepsy, stroke and other degenerative conditions in which the role of glutamate is known to play vital role in the pathogenesis.

Cultivation: It is propagated from seeds or stolons and can be grown on variety of soils. However moist locations are more suitable. The plant can be harvested at any time of the year and is used fresh or dried. Another report says that the dried herb quickly loses its medicinal properties and so is best used fresh.

PHARMACOGNOSY

Materials & Methods: Plant materials were collected from different parts of Kerala and Nagarjuna Herbal Garden. For Macroscopical characters Stereomicroscope is used and for microscopical studies, the compound microscope. For physical constants rotary shaker, muffle furnace, UV – cabinet, moisture balance were used.

RESULTS AND DISCUSSIONS

Macroscopic features: Small creeping herb with slender stem, rooting at nodes giving rise to thin Brownish grey roots, leaves 1 to 3 from each node, orbicular reniform, crenate , base cordate, petioles channelled with adnate stipules, flowers fascicled umbels, each carrying 3 or 4 flowers, short stalked, fruits cremocarp, ovoid, with laterally compressed seeds.

Microscopic features: Palisade two layered. Spongy parenchyma three layered with many intercellular spaces. Some containing rosette crystals of calcium oxalate. Midrib region shows 2-3 layers of collenchyma cells below the epidermis. Petiole shows 7 vascular bundles within parenchymatous zone.

PHYSICAL CONSTANT VALUES

No	PARAMETERS	VALUES
1	Foreign matter	Max 2%
2	Total ash	Max 17%
3	Acid insoluble ash	Max 5%
4	Alcohol soluble extractive	MIN 9%
5	Water soluble extractive	MIN 20%

THIN LAYER CHROMATOGRAPHY

Powder - 5 gm
Extract - Ethyl alcohol
Solvent System - Chloroform: Methanol-80:20

Rf Values	Colour in UV rays
By spraying 5 % Methanolic Sulphuric Acid	
0.72	Blue
0.85	Blue

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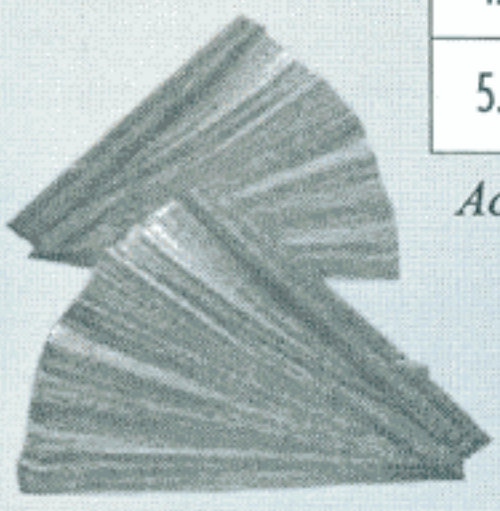
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2.	Therapist	Students out of Kerala after Plus 2	Six months
3.	Panchakarma orientation	Doctors Out of Kerala	2 weeks
4.	Therapist	International Students	6 weeks
5.	Awareness programme	International Students	2 weeks



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FOR MORE DETAILS, CONTACT.

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