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Review Article

Proposed Mechanistic Theories Pertaining to Sleep Walking: A Critical Review

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Abstract

Introduction: Sleep walking cases have been reported by the Clinicians over last few years, however the exact mechanistic theory knowledge still lacks. We tried to critically evaluate the possible mechanism behind sleep walking in the current review.

Methods and Results: In current review, critical analysis of individual studies was conducted evaluating credibility of experiments leading to a final opinion pertaining to mechanism of Sleep Walking. Possible overlaps among different mechanisms were also identified to provide robust conclusion.

Conclusion: Multiple studies provide strong evidence of dissociated state: Processes I and II synchronization in Sleep Walking cases. Process I originate from sensory motor areas and have an upward movement via subcortical neuronal projections towards cortex. Overall process of synchronization is pretty fast. Process II however is slow in nature and waves originating in this are of low amplitude and of short duration.

Keywords: *Sleep Walking; Sleep Disorders; Sleep Walking Treatment; Sleep Walking Pathophysiology*

Abbreviations

ADHD: Attention-deficit Hyperactivity Disorder; EDS: Excessive Day Time Sleepiness; ESS: Epworth Sleepiness Scale; FDG: Fluorine-18-flourodeoxyglucose; PET: Positron Emission Tomography; PSG: Polysomnography; PSQ: Pediatric Sleep Questionnaire; NREM: Non Rapid Eye Movement; SPECT: Single Photon Emission Computed Tomography

Introduction

Sleep walking is a complex phenomenon involving activation/deactivation of different parts of brain. It involves hyperarousal of certain regions of brain during which actual sleep walking episode takes place. However, it has been reported that patients with sleep walking will not be able to remember activities they performed during episode. Hence it gives an understanding to mechanistic process of sleep walking that it is quite complex and needs deep understanding. For explanation of mechanism behind sleep walking, multiple theories

have been proposed. Serotonergic hypothesis of sleep walking was highlighted by different groups [1-3]. Other scientists have worked on excessive day time sleepiness being associated with mechanism of Sleep Walking [4]. Hypnotics have also been found to have association with induction of Sleep walking [5,6]. Migraine and other pain conditions can also been linked to sleep walking [7]. Their have been strong family trends being found in sleep walking patients with evidence of both maternal and paternal family history of sleep walking.

One of the most important aspects pertaining to sleep walking is based on theory of two dissociated states [8]. This mechanistic process seems of key importance and somehow other theories are linked to this process. In the current review, individual theories have been critically analyzed with a special focus on theory of two dissociated states.

Proposed mechanistic theories pertaining to sleep walking

Serotonergic hypothesis of sleep walking [1-3]

Sleep walking is associated with slow wave sleep arousal accompanied by physical actitivites like walking and alterted consiouness. Sleep disordered breathing may have a mechanistic connection with occurrence of sleep walking.It is being proposed here that serotonergic neuronal pathway may be involved in process of sleep walking as these neurons respond to decearsed oxygen levels secodnary to sleep disordered breathing and can lead to hyper excitation of motor neurons resulting in hyperarousal state. In a study conducted by Edmund G Cape and Barbara E Jones, serotogenetic injections were given in 24 male wister rats at region corresponding to cholinergic basalis neurons. EEG results in serotonin injected group revealed an increase in delta activity of these animals. The delta waves activity was linked to wakefulness like behavior with opening of eyes in rats associated with a confused behavior. This finding is similar to human sleep walking which is hyperarousal state during slow wave sleep [1].

Excessive day time sleepiness may be associated with mechanism of sleep walking [4]

In a retrospective study of 140 human subjects (70 with sleep walking history and 70 controls) association of EDS (Excessive day time sleepiness) with sleep walking disorder was evaluated using Epworth Sleepiness scale (ESS). To rule out other etiopathological causes overnight PSG in 70 patients with sleep walking presentation was performed. Further, ESS scoring based sleep variables were evaluated to find out association between sleep walking and other sleep disorders (cases 32 sleep walking patients with day time sleepiness and control group 38 sleep walking patients without day time sleepiness). Sleep walking patients (n = 70) scored greater than 10 in ESS and mean ESS scores in this group was significantly high when compared to control group (n = 70, p value = 0.0003) (Figure 1A and 1B).

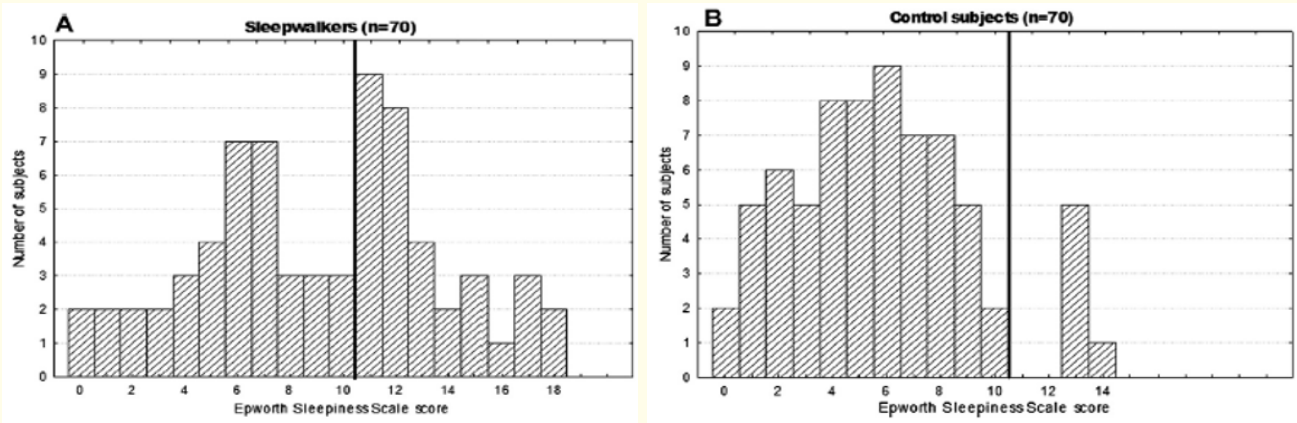


Figure 1A and 1B: *ESS scores details in cases (70 sleepwalking patients A) versus controls (70 adults B). Mean ESS scores in this group was significantly high when compared to control group (n = 70, p value = 0.0003) [4].*

Current study provides evidence pertaining to excessive day time sleepiness in absence of other sleep disorders can be linked towards mechanism of Sleep walking. As indicated by results, mean ESS levels of sleep walking patients were highly statistically significant (p = 0.0003) compared to controls. Scientific group has also evaluated other parameters like overnight PSG to exclude other sleep related disorders.

This was a retrospective study which adds to major limitation of study. Sample size was also limited considering an observational study. Further, both subjective and objective parameters should have been evaluated to rule out other causes of excessive day time sleepiness.

Zolpidem induced sleep walking [5,6]

In a case report a patient with a history of road side accident post -surgery was put on conservative management. Later patient reported episodes of insomnia based on which he was prescribed 0.5 mg alprazolam however no benefit was felt by patient and he discontinued drug of his own. He started Zolpidem 10 mg at night (self-medication) without any prescription and after 15 days family reported sleep walking episodes and reported same to hospital. Patient was reexamined and history pertaining to sleep walking was taken.

It was confirmed that patient had episodes of sleep walking and based on available literature and consensus among the team Zolpidem which has been reported to induce sleep walking was stopped (De challenge process). After stopping Zolpidem no sleep walking episodes were reported by patient's family. This case report provides a clinically significant finding however only one case was reported and hence requires more data on post marketing use of zolpidem and subsequent sleep walking episodes.

In another case study similar reporting was presneted. A 44 year patient with history of insomnia was put of Zolidem 10 mg and after 2 weeks multiple episodes of sleep walking including driving were reproted. Zolpidem was stopped immediately and patient had improvement in sleep walking. Post 2 months of Zolpidem treatment PET CT brain using 18 FDG was performed.Another PET CT was done after 10 mg of Zolpidem treatment on the next day after 1 hour of drug administration (Figure 2).

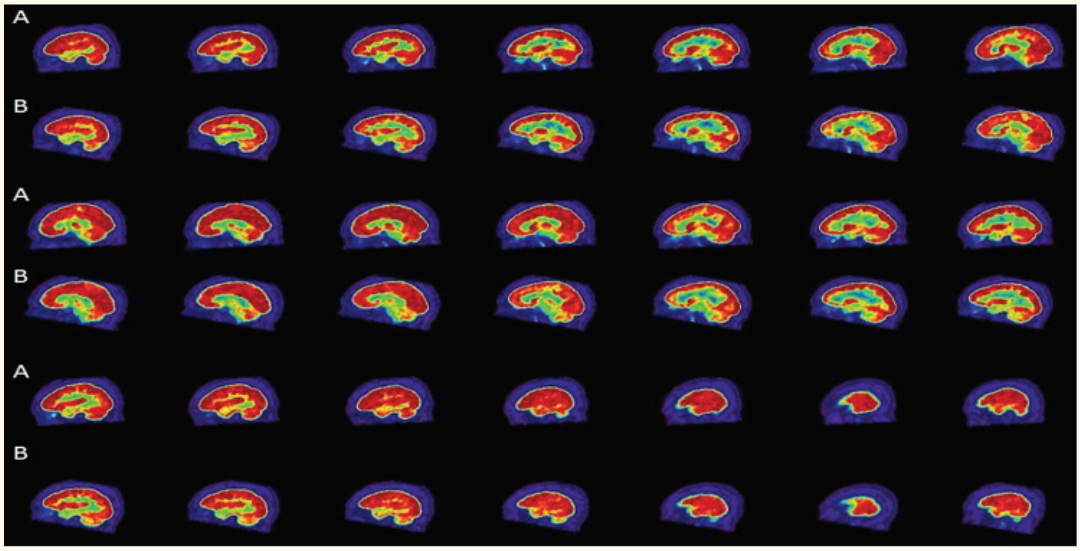


Figure 2: *A) PET CT brain was performed after 2 months of stopping of Zolpidem treatment. B) PET CT brain in the same patient post reintroduction of Zolpidem 10 mg single dose. (PET CT was performed 1 hour after oral intake of Zolpidem) [6].*

No statistically significant difference was observed in both PET CT findings based on software based statistical analysis. These finding are suggestive of post wash out period of 2 months there should have been appropriate number of dosages of Zolpidem to maintain a threshold serum level which would be required to reflect some changes onto the PET CT brain. Further there is an opportunity to investigate more robust biomarkers and imaging techniques which can reflect appropriate findings altering the brain physiology post zolpidem administration.

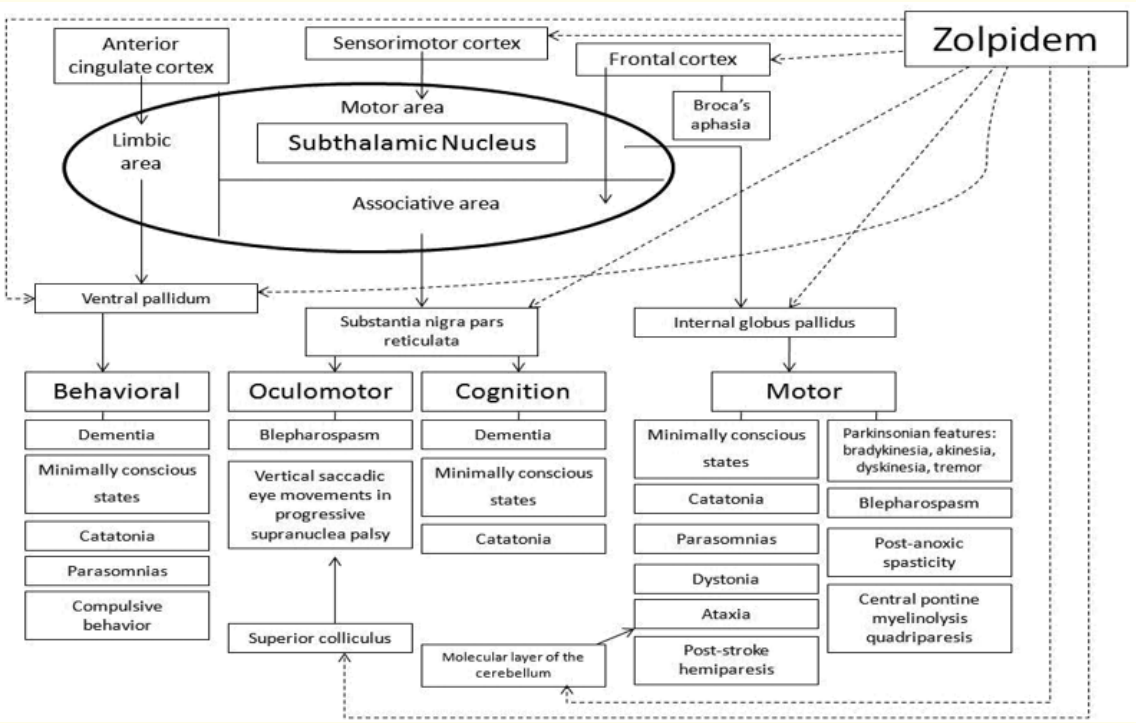


Figure 3: *Describes GABAA receptor sites involved at multiple levels. Zolpidem is benzodiazepine receptor agonist. For purpose of sleep walking, area of interest is Internal globus pallidus which can lead parasomnias [6].*

Role of chronic headache and migraine in sleep walking mechanism [7]

Lopez and his team evaluated role of Chronic painful conditions as pathophysiological factor in Sleep walking in a retrospective case control study (n = 100 Sleep walking patients and n = 100 normal adults). Team further investigated painful stimuli perception during injurious sleep walking in patients. It was observed that patients with chronic headache and migraine had statistically significant linkage with sleep walking. Approximately 78% of forty seven patients who had experienced injurious sleep walking episodes did not recall pain sensation during these incidences.

This study provides an evidence of association between the chronic pain conditions specifically headache (Odds Ratio 3.8) and migraine (Odds ratio 10.04) and sleep walking NREM parasomnia. This study also reported analgesia during sleep walking episodes as one of the key aspects to be studied in detail to establish mechanistic action between the two. However, as this study was a retrospective observation there is possibility of recall bias and pain stimulus sensitivity was not measured. Further, PSG was not performed for all patients to rule out other sleep disorders like sleep apnoea.

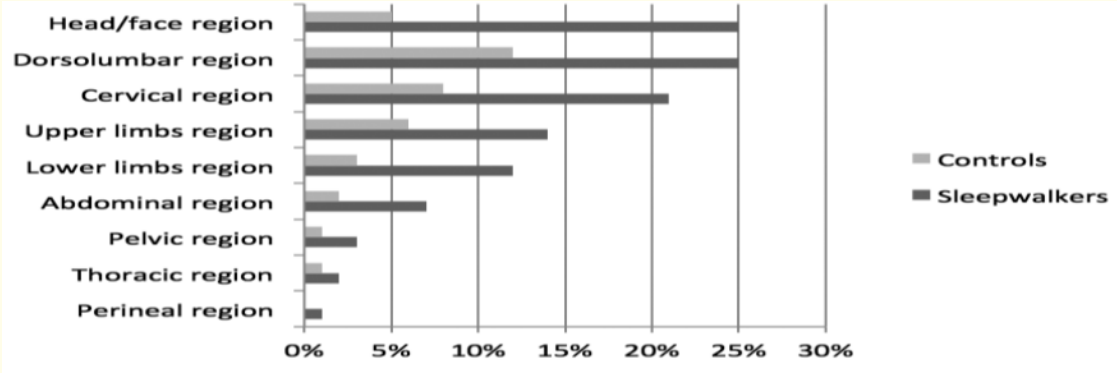


Figure 4: *Describes various pain locations in Sleep Walking Patients as compared to controls [7].*

Sleep deprivation and sleep walking association

Dang-Vu., *et al.* [9] performed SPECT study in overnight sleep deprived sleep walking patients (n = 11) to evaluate perfusion patterns during wake period as compared to controls (n = 12). At baseline, perfusion patterns in both groups were similar. Post 1 night of sleep deprivation of sleep walking cases, perfusion decreased significantly (p value < 0.05) at inferior temporal gyrus level bilaterally as indicated by figure 5.

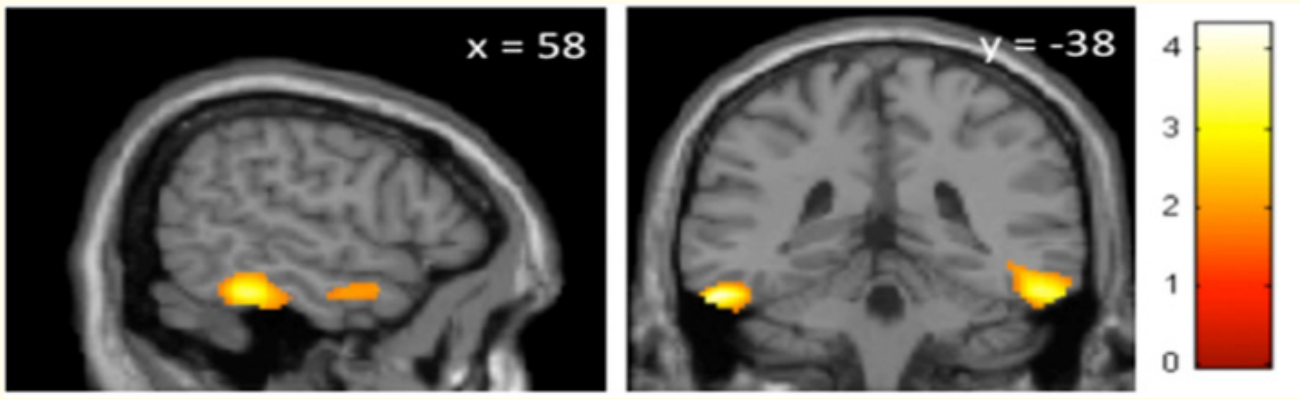


Figure 5: *Describes SPECT based perfusion study in overnight sleep deprived sleep walking patients. Figure indicates decreased bilateral perfusion (p value < 0.05) in inferior temporal gyrus region in these cases (n = 11) and compared to controls (n = 12). Left panel (sagittal section) and Right (coronal section) [9].*

Current study provides evidence to involvement of neural mechanisms in sleep walking patients during wake period and these can act as biomarker to predict probability of sleep walking in patients with chronic sleep deprivation. Perfusion differences post overnight sleep deprivation also indicates role of homeostatic pressure in alteration of neural signaling. Bigger challenge with this kind of study is case to case clinical variation being reflected in functional neuroimaging.

Gray matter volume decrease in patients with sleep walking [10]

Using 3 Tesla MRI techniques in 28 human subjects (14 diagnosed Sleep walkers and 14 healthy controls), association of Grey matter volume decrease in specific areas of brain was studied. Significant reduction ($p < 0.001$) in grey matter volume was observed in areas of dorsal posterior cingulate cortex (left) and posterior mid-cingulate cortex. These regions in brain have been hypothesized to be responsible for neurophysiological mechanisms involved in activation states responsible for sleep walking and other NREM parasomnias in humans.

However exact mechanism for this decrease in volume of referred brain regions is still not clear. The patients enrolled in this study had long history of sleep walking (19 years and above), hence these findings cannot clearly explain whether reduction in volume of brain regions is secondary to sleep walking pathology or vice versa which makes it key limitation of study with respect to providing a neuronal mechanism of sleep walking. Future prospective longitudinal studies can provide great vision into underlying mechanisms of sleep walking.

CNS stimulants and sleep walking association [11]

In children with behavioral and neuropsychiatric disorders multiple treatment options are available. CNS stimulants like Amphetamine in combination and singly have been used quite frequently in management of ADHD. Key adverse effects of these drugs are decreased total sleep time and increment in sleep onset latency. Sleep walking secondary to stimulant drugs has not been frequently reported. Pinanaka and his team reported 2 pediatric cases of sleep walking (13 year girl and 8 year boy). Both patients were on ADHD treatment and in due course they were prescribed Amphetamine based CNS stimulants in age/weight proportionate doses. Girl had a family history of sleep walking (father sleepwalker). In this case, there were two incidences of sleep walking which occurred post 1 week of amphetamine treatment. Out of these episodes 1 was associated with only sleep walking while another had elements of sleep walking and sleep eating. Patient was not able to recall both incidences.

In second case, patient was put on amphetamine based CNS stimulants for ADHD and post 1 month of treatment; multiple episodes of sleep walking were reported by patient's parents. Considering safety aspects Amphetamine based CNS stimulants were stopped and boy did not report sleep walking episodes on follow up for ADHD treatment.

Current case study analysis is very important from clinical point of view along with future research exploration. In case one i.e. 13 year old girl with family history of sleep walking making her prone to NREM parasomnias. However in second case there was no family history of sleep walking and stopping CNS stimulants eventually helped him to get rid of sleep walking. Thus it is evident that CNS stimulants by virtue of some mechanisms derange the excitation-inhibition system of neurons at central level and can lead to sleep walking. However exact mechanism is still unknown and requires robust clinical trials for evidence.

Family patterns of sleep walking in sleep walking patients [12]

In a study, conducted at Stanford University Sleep Medicine center patterns of occurrence of sleep walking in seven families who were related to fifty one sleep walking patients visiting Sleep centre over last 5 years was studied.Out of these seven families, 34 sleep walking cases were confirmed. These indentified patients had either parental or maternal family history of disease. For evaluation Pediatric Sleepiness Scale (PSS) was administered and adults received Epworth Sleepiness Scale (ESS). All patients were subjected to overnight full PSG as well. Clinical examination included Craniofacial examination pertaining to ENT, dental and other related aspects in all patients.

One of the important points to be mentioned here is out of 34 sleep walking patients, 33 had history of disturbed breathing during sleep. On further evaluation, it was found that all these 33 patients had some sort of craniofacial anatomical challenges which were later corrected and patients improved sleep quality at night.It was further observed, post correction of craniofacial abnormalities elements, all 33 cases of sleep walking had significant improvement in sleep walking episodes.

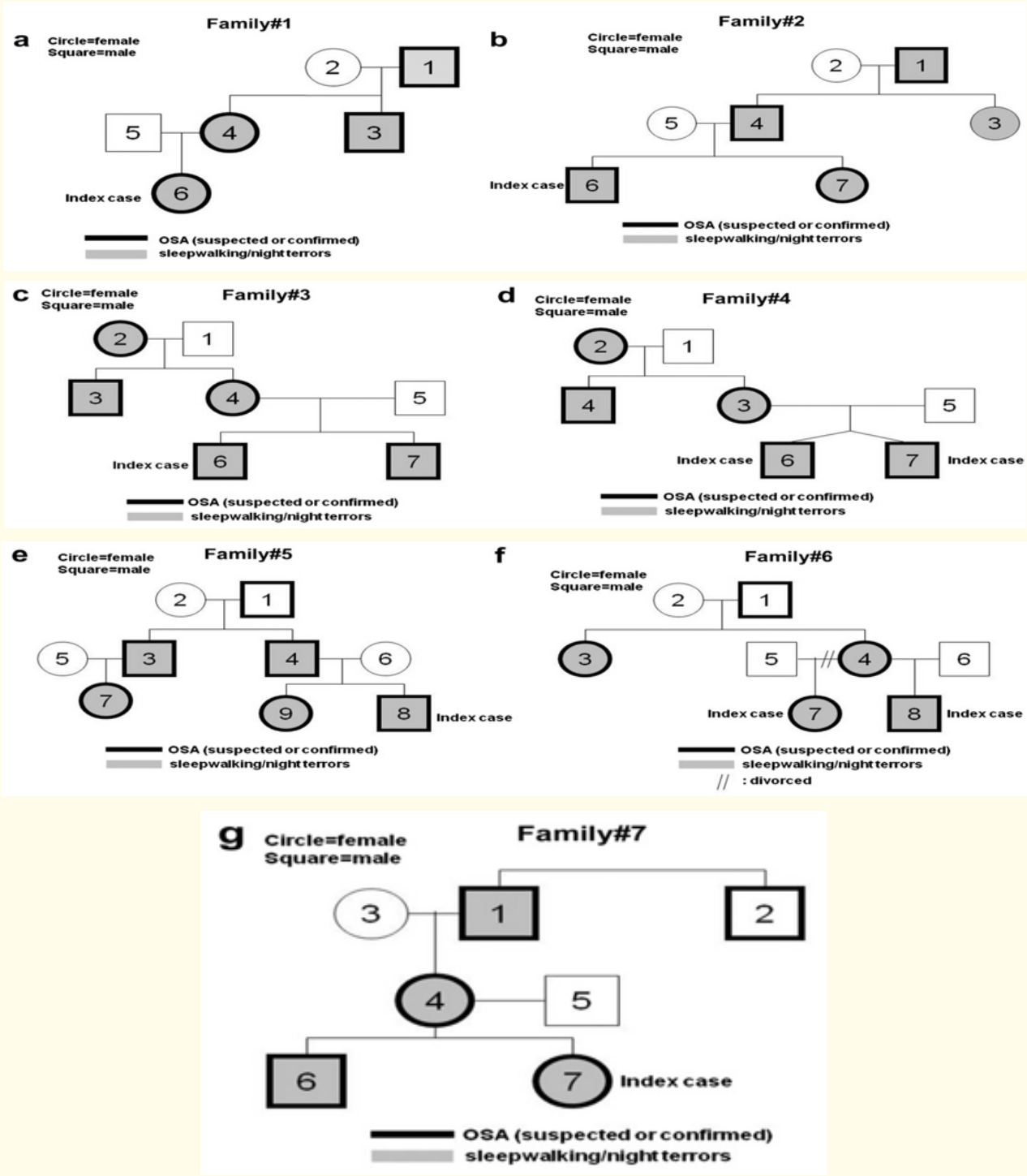


Figure 6: *Representing various 7 families and their respective family members with Sleep walking issues (Square: Representing Males, Circle: Females, Dark-filled color: Sleep walkers and Black frame: Sleep Deprived Breathing [12].*

Current study provides an important aspect of NREM parasomnias specially sleep walking. Based on findings, genetic predisposition of sleep walking in patients has been linked to family history of sleep walking. Another important takeaway from this study is disordered sleep breathing can lead to development of sleep walking and other parasomnias. In all cases of sleep walking, sleep breathing disorder was observed primarily secondary to anatomical deformities. Once these deformities were corrected patients stopped reporting episodes of sleep walking (based on a 12 month follow up post surgery, ortho-dental corrections etc). Biggest drawback of this study is lack of statistical analysis of data and moreover results are much more clinical examination driven which can have subjective difference in interpretation.

Sleep walking is based on theory pertaining to two dissociated states [8]

Sleep has been earlier described as a state in which whole brain gets into complete deactivation. As this branch of sleep medicine got evolved, new insights were brought into consideration based on multiple studies in animal and human population. Currently, Sleep is understood as a complex phenomenon which may involve certain parts of brain in active state and others in depressed or deactivated condition. It is proposed that an overall equilibrium is required in these two states, absence of which can lead to different types of sleep disorders like NREM parasomnias.

Sleep walking is a NREM type parasomnia which involves a dissociated state, in which patterns of both sleep and wakefulness can be observed. In the depolarization state, neuron firing is extensive and persistent till the time hyperpolarization is achieved and neurons move to an off state. In NREM stage of sleep, there is some evidence of thalamo-cortical neurons to get into a state of deactivation post depolarization. Studies have indicated that cortical regions are supposed to be putting up brakes to activation states and bringing up off states i.e. slow waves induction and also sustaining them in NREM. Areas like frontal orbital cortex and anterior cingulated gyrus have been associated in slow wave generation process.

It has been postulated that slow wave synchronization process involves two steps: process 1 leading to type 1 slow waves and process 2 leading to type 2 slow waves.

(Figure 7)

Process I originates from sensory motor areas and have an upward movement via subcortical neuronal projections towards cortex. The slow waves originating in this manner are of large amplitude and of longer duration. Overall process of synchronization is pretty fast.

Process II however is slow in nature and waves originating in this are of low amplitude and of short duration. These originate from different areas and movement is horizontal.

The theory of two dissociated states fits very well in explaining the mechanism of sleep walking. In initial phase of slow waves, regions involved are frontal cortex, however in later part of slow wave occipital, temporal and parietal regions are more active evident from different studies. Thus theory of two distinct dissociated process -Synchronization I and Synchronization II seems one of the most compelling mechanistic technique leading to sleep walking.

Rationale pertaining to dissociated states mechanism leading to nrem parasomnia sleep walking [13-17]

Desjardins., *et al.* [16] conducted brain perfusion pattern analysis study using SPECT imaging technique on 20 subjects (10 sleep walkers and 10 normal adults). Both cases and controls were subjected to overnight sleep deprivation. The contrast medium was administered to patients through intravenous route after 2minutes of slow sleep wave initiation and similarly in resting state wakefulness.

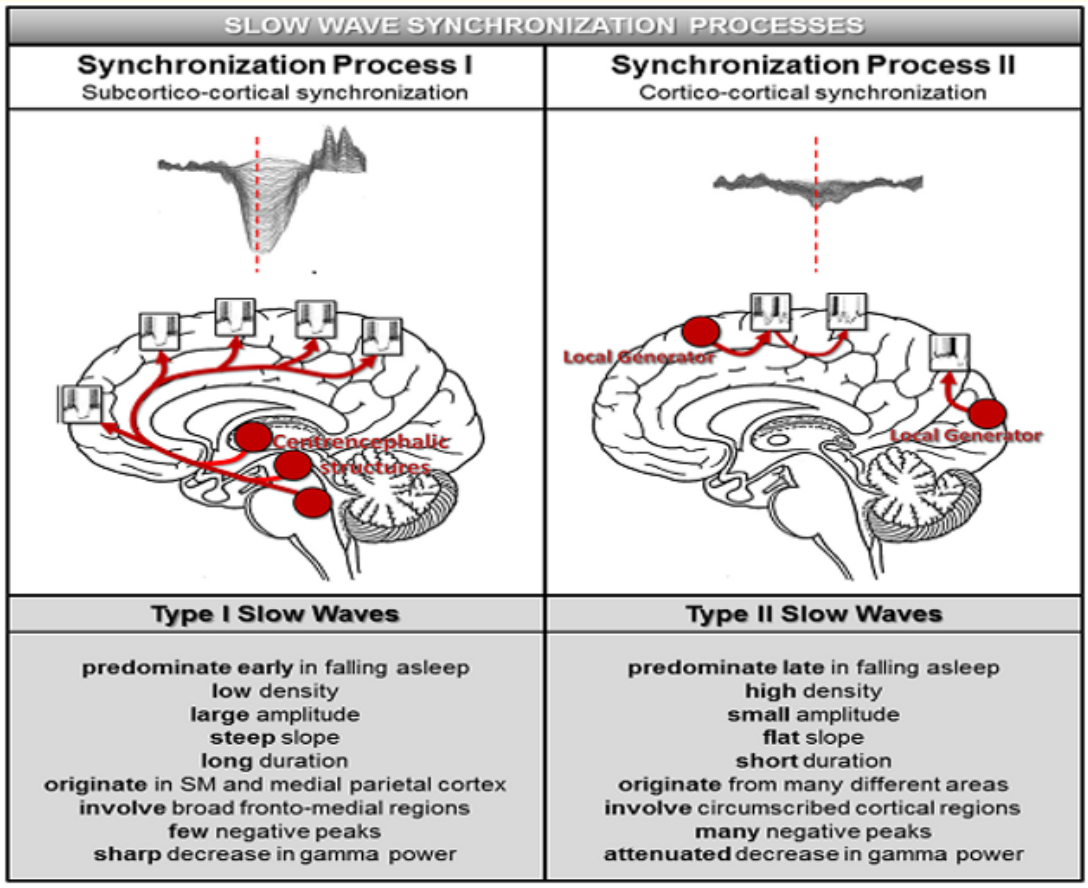


Figure 7: *Represents two synchronization processes pertaining to slow waves (Synchronization Process I and II) [8].*

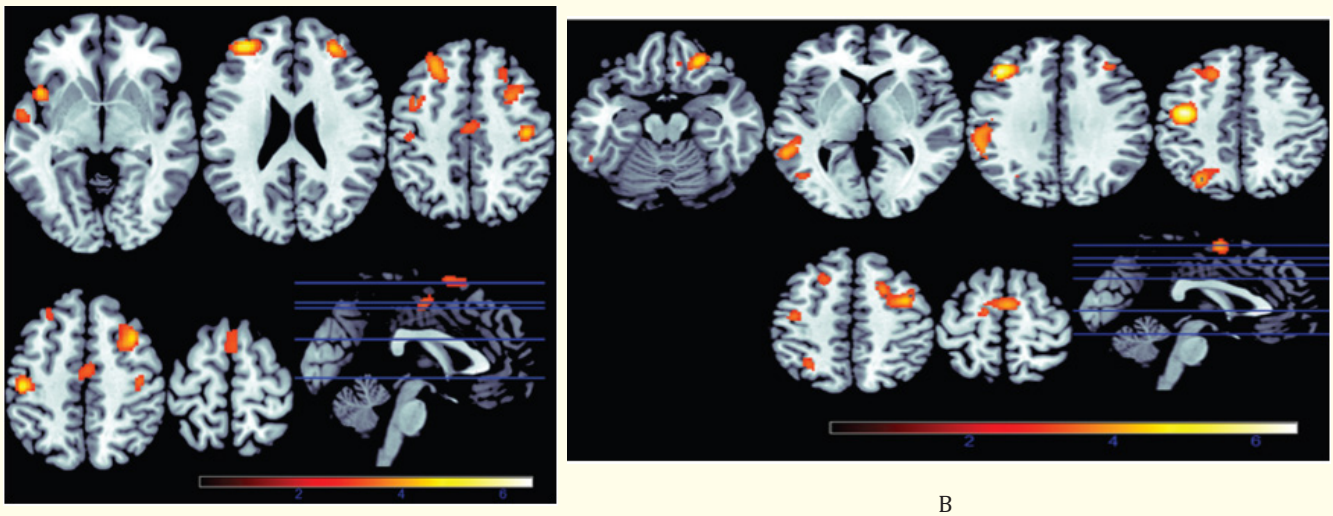
It was observed that there is decreased perfusion in multiple areas of brain (left postcentral gyrus, insula, and superior temporal gyrus) during SWS and wakefulness (temporal and parietal areas) in sleep walking patients as compared to normal controls (highly significant values refer table 1). Further there was increased perfusion in hippocampus region in sleep walking patients during resting awake state (Refer figure 8A and 8B).

Current study provides strong evidence pertaining to mechanism of dissociated process in sleep walking as indicated by highly significant decreased diffusion patterns in sleep walking patients as compared to normal adults. Decreased perfusion levels are in sink with two synchronizations process I and II described earlier. Such studies are required to be conducted in future on a much larger scale to further validate the concept.

In a SPECT based study [13] activation sites in brain during sleep walking episode in a patient were evaluated. There was an increase in blood flow by 25% in areas of anterior part of cerebellum and posterior cingulate cortex while frontal parts of brain remained deactivated, which is in alignment with dissociated process in sleep walking patients. Patient was also subjected to video polysomnography. Refer figure 9 for PSG findings.

Region	Cluster size (K)	x	y	z	T-value	P
Frontal cortex						
L superior frontal gyrus	439	-22	30	42	5.05	0.000
L middle frontal gyrus		-20	30	34	4.27	0.000
	125	-42	6	46	3.53	0.001
		-38	8	48	3.47	0.001
	266	-36	48	24	5.46	0.000
R superior frontal gyrus	616	34	14	54	4.64	0.000
		24	36	38	3.60	0.001
		24	26	48	3.20	0.003
R middle frontal gyrus		30	46	26	4.52	0.000
R medial frontal gyrus	119	2	-10	52	3.41	0.002
	104	2	8	68	3.34	0.002
R precentral gyrus	135	42	-16	46	4.52	0.000
Parietal cortex						
L postcentral gyrus	182	-46	-22	52	5.12	0.000
Insular cortex						
L insula	170	-44	12	-4	4.38	0.000
Temporal cortex						
L superior temporal gyrus	116	-58	-2	0	3.87	0.001

Table 1: *Regions wise perfusion reduction in sleep walkers versus controls. (P vales significant for all regions) [16].*



A B

Figures 8A and 8B: *A) Decreased perfusion in multiple areas of brain (left postcentral gyrus, insula, and superior temporal gyrus) during SWS. B) Decreased perfusion during wakefulness (temporal and parietal areas) in sleep walking patients as compared to normal controls [16].*

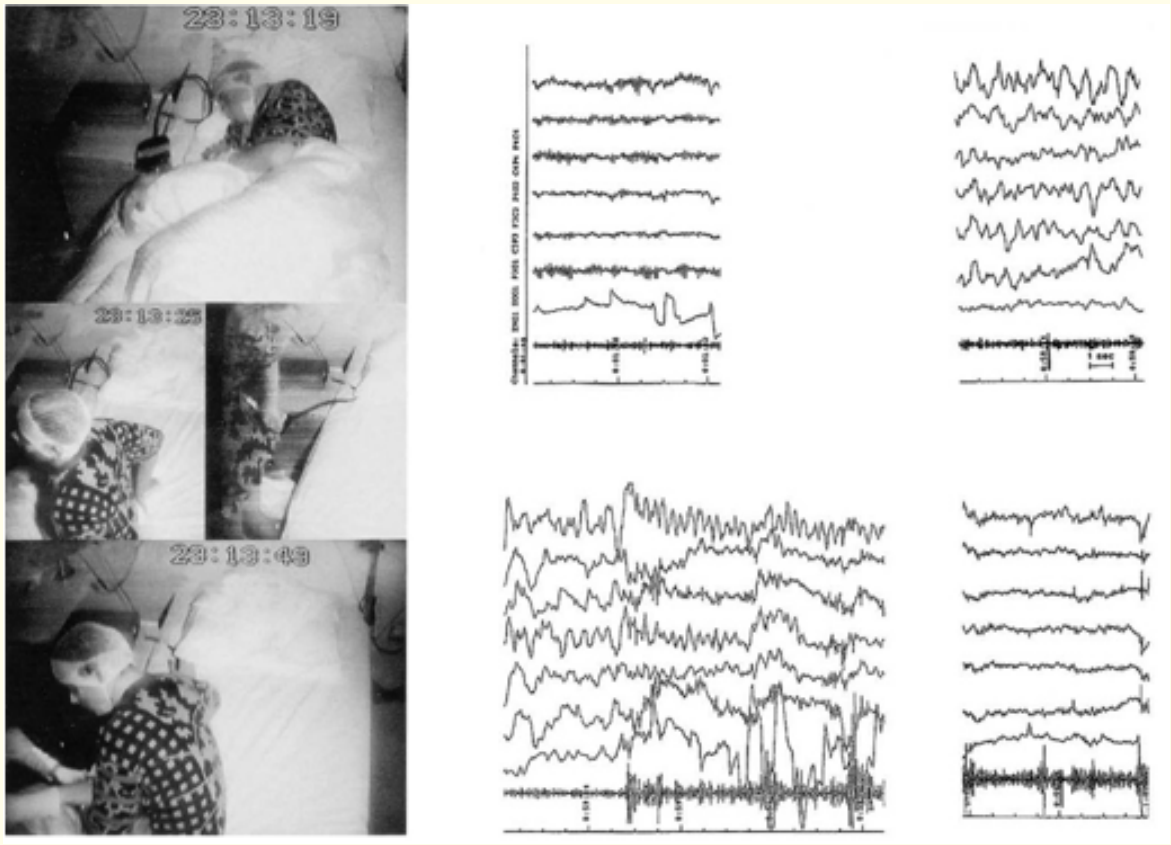


Figure 9: *Sleep Walking and corresponding PSG findings [18].*

Januszko., *et al.* [17] studied EEG patterns of 15 ICSD-2 criteria confirmed 15 sleep walking cases. For evaluation, EEG based neuroimaging was performed.20 EEG segments were taken into account of these patients. It was observed that just prior to sleep walking episode there was sudden arousal mechanism as indicating by the imaging. Results revealed significant ($p < 0.05$) activation in cingulated motor area of brain just prior to sleep walking episode. LORETA images indicating increase activation of areas like anterior cingulate cortex (red to yellow). Images were taken just prior to sleep walking episodes ($p < 0.05$) (Refer figure 10 and 11).

Current study provides evidence on activation of anterior cingulate cortex just prior to sleep walking disorder. These finding are in line with the two synchronizations process theory as well. This study presents a very good picture of various brain activation sites at time of wakefulness. Major limitation of study is low sample size and significant number of sleep walkers being involved in similar studies in future.

Castelnovo., *et al*. [14] and his team studied arousal patterns in 15 sleep walking patients and 15 healthy controls post overnight PSG in combination with high density 256 channel EEG. It was observed that sleep walkers had differences in arousal patterns and localized sleep differences based on high density EEG study. Observations made in this study were activation motor cortex and cingulated cortex in sleep walking patients as compared to healthy control group ($p < 0.05$). These findings are in alignment with the dissociated 2 synchronization processes. There should be more studies involving significant number of patients and comparable controls in future [18-28].

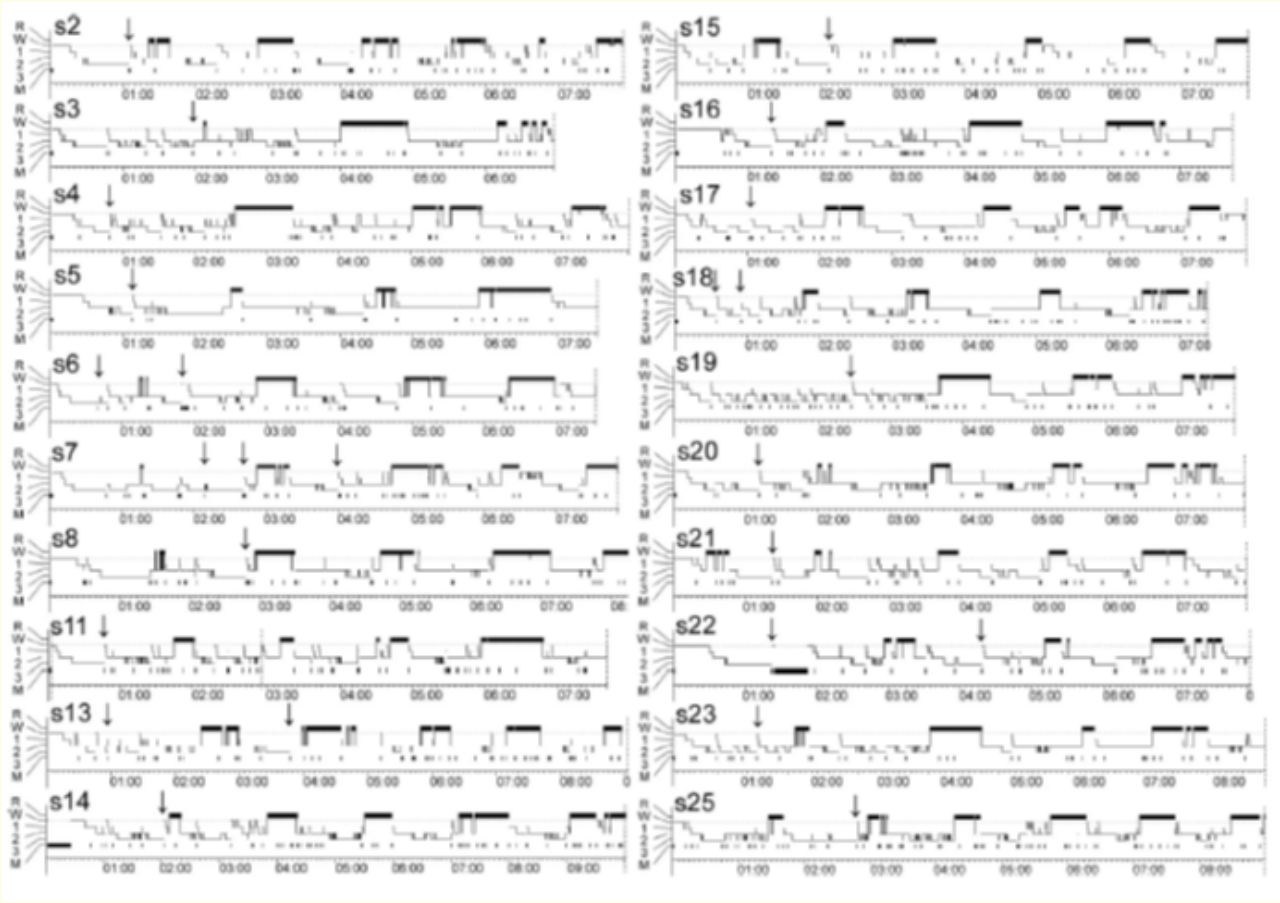


Figure 10: *Describes hypograms of patients with sleepwalking episodes (Black arrows are indicative of sleep walking episodes) [17].*

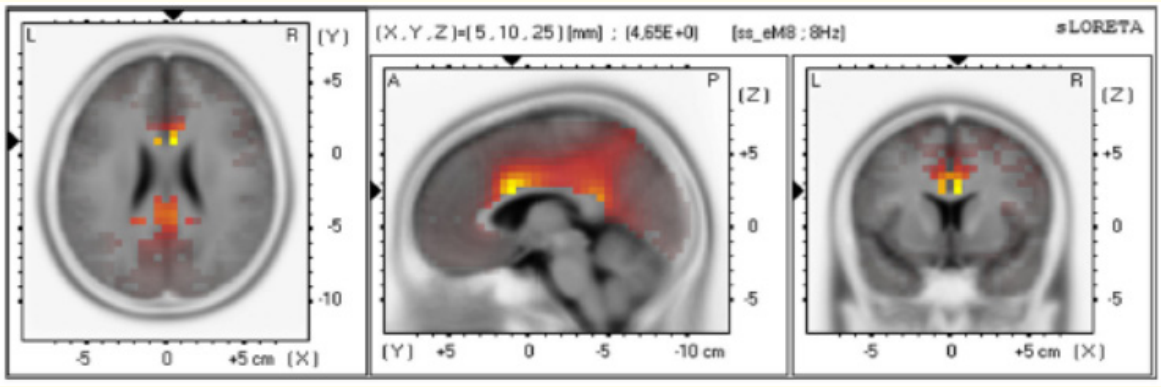


Figure 11: *Depicts LORETA images indicating increase activation of areas like anterior cingulate cortex (red to yellow). Images were taken just prior to sleep walking episodes ($p < 0.05$) [17].*

Conclusion

Multiple theories have been proposed pertaining to the mechanistic technique of sleep walking. It is a complex phenomenon involving activation/deactivation of different parts of brain. Theories like Serotonergic hypothesis, excessive day time sleepiness, use of hypnotics, migraine and genetics all have provided some evidence. One of the most important aspects pertaining to sleep walking is based on theory of two dissociated states**.** This mechanistic process seems of key importance and somehow other theories are linked to this process. Multiple studies provide strong evidence of dissociated state: Processes I and II synchronization. Process I originates from sensory motor areas and have an upward movement via subcortical neuronal projections towards cortex. The slow waves originating in this manner are of large amplitude and of longer duration. Overall process of synchronization is pretty fast. Process II however is slow in nature and waves originating in this are of low amplitude and of short duration. These originate from different areas and movement is horizontal direction. Future point of view large number of clinical trials are required to further validate the concept. This model can also act as basis of pathophysiologies for various neuropsychiatric disorders.

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