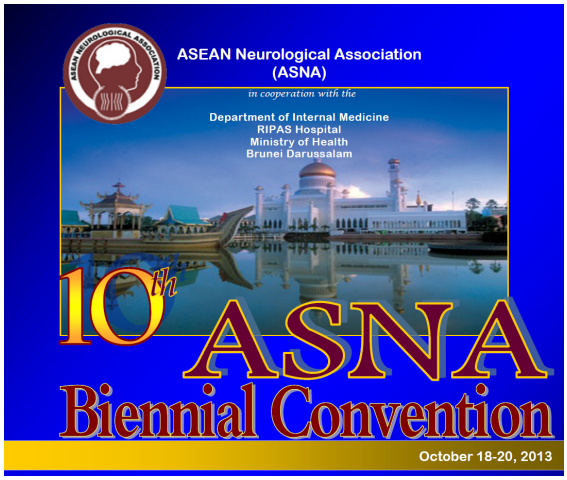
10th ASEAN Neurological Association (ASNA) Biennial Convention (Part III)



The ASEAN Neurological Association (ASNA) was formed in 1994 and at that time consisted of five member countries; Indonesia, Malaysia, Philippines, Singapore and Thailand. Presently, there are 10 members consisting of the member nations of Association of South East Asian Nations (ASEAN). The main aim of the association was to bring together experts and clinicians involved in the care of neurology from the ASEAN regions to discuss and share their knowledge and research findings. It also provide good opportunities to collaborate and form friendships. The official journal of the ASNA is the Neurology Asia which is being published quarterly and is available online from the journal website (www.neurologyasia.com).

The first ASNA convention was held in Manila, Philippines in 1995. The convention has since been organized biennially with the second convention held in Singapore (1997), followed by Chaingmai Thailand (1999), Kuala Lumpur, Malaysia (2001), Cebu, Philippines (2003), Jakarta, Indonesia (2005), Cha-am, Thailand (2007), Kuala Lumpur (2009) and Bali, Indonesia (2011).

The 10th Convention, ASNA 2013 being held in Bandar Seri Begawan Brunei Darussalam consisted of two day plenary lectures and symposium. This was preceded by a day of workshop. This convention brought together experts from member nations and also from non member nations.

Research findings were also presented as posters in the poster sessions. For the full programme and the abstracts presented, please refer to the Brunei Int Med J. 2013; 9: Supplement 1 available from the journal website at *www.bimjonline.com*.

Day 2 Convention

Symposium 7: Epilepsy: In this session, the speaker Josephine C. Gutierrez (The Philippines) spoke about the '***Diagnostic Challenge: Psychogenic Non-epileptic Seizures***', a condition that is more common than we, the non-neurologist think. Psychogenic non-epileptic seizures (PNES) are involuntary episodes of movement , sensory and behavioural abnormalities that resemble seizures but which do not result from abnormal cortical electrical discharge. As such, PNEs is commonly misdiagnosed a epilepsy and inappropriately treated. PNES is not rare and accounts for ~ 25% of referrals to

the neurologist of patients suspected to have seizures disorders. However, to complicate matter between 10 and 40% of patients with PNES have co -existing genuine epilepsy. Features that should raise suspicion of PNES includes: prolonged seizures, frequent seizures, non-stereotyped seizures, atypical seizure presentation, poor response to anti-epileptic drugs (caution autoimmune seizures) and emergency of new seizure types in patient already being treated for seizure disorder. The seismologic features, serum prolactin levels and video EEG can help to confirm the diagnosis. The mechanisms producing PNES are not fully clear, but conversion reactions or factitious psychiatric disorder appear to be important. PNEs is found mostly in patients with underlying depressive and anxiety disorders and hence can be successfully managed with psychotherapy and anti-depressants. The prognosis of PNES depends on the cause and chronicity of the symptoms. Recognition and diagnosis is essential to avoid unnecessary treatments (with anti-epileptic drugs), associated social stigma and restriction associated with epilepsies disorders. Early treatment of underlying psychiatric disorders can reduce morbidity. The second talk of the symposium was delivered by *Prof. Raymond Azman Ali* (Malaysia) on '***Treating Epilepsies with Medical Comorbidities'***, a topic that most non-neurologists not may comprehend magnitude of the problem and its implications. In fact, this is also true for other specialties, where patients now have multiple comorbidities. Treating patients with seizures disorders who also have multiple medical comorbidities present a challenge as the seizures disorders can affect the management of the medical comorbidities and vice verse. Similarly, the anti-epileptic drugs used may have adverse effects through drugs interaction, cumulative side effects which can make optimising control of the disorders difficult, even leading to new medical problems. There is mounting evidence that many medical conditions co-exist with epilepsies, not just a mere coincidence. Conditions that have been shown to have direct causal relationship with seizures include stroke (not unexpected), brain tumours (not unexpected) and migraines and those with indirect causal relationships include hypertension, ischaemic heart disease, diabetes mellitus, obstructive sleep apnoea, and inflammatory bowel disease. Certain diseases share the same risk factors for epilepsy; cerebral palsy, Down syndrome, dementia and even chronic bronchitis. Other conditions that can result from seizure disorders or the treatments used include weight disorder, acne, eczema, cerebellar dysfunction, cardiac arrhythmias, conduction defects, peptic ulcer disease, calcium metabolism disorders, subfertility polycystic ovaries, pneumonia, headache and Parkinsonism. Other conditions reported to be increased in prevalence in patients with seizure disorders include; thyroid disease, rheumatoid arthritis, asthma, cataracts and chronic fatigue. Choice of medications need to take into account the presence or risk for these disorders, and doses need to be monitored and adjusted according to maximise seizure treatment response with minimal risk for adverse effects. For patient with seizures and migraine, valproate, topiramate and zonisamide may be the preferred treatment. IN overweight or obese patients, valproate should be avoided. Further weight gain can lead to a host of other medical problems (diabetes mellitus, metabolic syndrome and increase cardio-cerebrovascular risks and even cancers). The presence of other comorbid conditions reduces the quality of life and may even shorted life expectancy. Therefore, clinicians should be vigilant about medical comorbidities in patients with seizure disorders and not ascribe all new symptoms to epilepsy itself or to the medications. The third talk of the symposium was on ***'Surgery for Drug Resistance Epilepsy: Indications, Evaluation and Outcomes'*** delivered by *Yotin Chinvarun* (Thailand). With the improvement in the understanding of the aetiologies and pathogenesis of the various seizure disorders, surgical interventions have now become less favoured options. However, surgical interventions remain important in the armamentarium, particular for those categorised as drug resistance epilepsy. Surgical treatment for seizures include: resective and disconnection surgeries. In resective surgery, which by far the most common, the affected area is removed, in theory curing the patient. Temporal lobectomy is the most common resective surgery.

The second type of surgery, disconnection surgery such as corpus collosotomy interrupts the nerve pathways. Multiple subpial transections is another example of disconnective surgery and is usually done to preserve areas that are important for daily functioning. Disconnective surgeries are generally non-curative. Patients who are candidate for such interventions are those who failed adequate medical therapy (defined as failed two first line seizure medications, ones that are commonly effective in controlling the type of seizures). Patients should be referred for evaluation sooner if they are eligible. Presurgery workup include MRI with epilepsy protocol, interictal and ictal SPECT, PET, scalp and intracranial electroencephalograph (EEG) and cortical stimulation. Recent advances in functional imaging have improve presurgery workup. The long-term outcome of epilepsy surgery for temporal lobe epilepsy (TLE) is ~48% seizure free rate, sustained improvement in quality of life, and employment. For extra-temporal surgery (ETS) which is more heterogenous, the seizure free rate range between 27 and 46%, with most still benefiting from surgery. Major complications of surgery are infrequent and tend to be temporary or limited in their symptomology. The last talk for this symposium was on ***'Failed Initial Monotherapy; Substitutes or Add-on'*** by *Prof. Lim Shih Hui* (Singapore). Approximately half of patients with epilepsy will be seizure-free on their initial anti-epileptic therapy with the other half requiring either increase in dosing or addition or changing to another medication. To date, there is no definitive answer as to whether such patients would benefit from an alternative monotherapy or polytherapy. Therapeutic decision making should take into account factors that include; seizure type or syndrome, potential adverse effects of medications, mechanisms of actions of medications, comorbidities, potential interactions with other medications, age of patients, learning disabilities, teratogenicity, ability to adhere to treatment regime and tolerance of the previous medications. Prof. Lim opined that it is sensible to substitute rather than combine when the first anti-epileptic medication either produced an idiosyncratic reaction, is poorly tolerated at low or moderate dose, or produces no improvement in seizure control. Polytherapy may be preferred if patient tolerates their first or second medication well, but with a suboptimal response.

Symposium 8: Dementia Epidemiology in Asia

The first talk in this symposium was delivered by *Paulus Anam Ong* (Indonesia) who talked on the situation of dementia in Indonesia, '***Prevalenc eof dementia in Indonesia***'. Indonesia is the most populace nation in the Southeast Asian region with ~250 million, of which 7.59% are elderly. With the ageing population, it is estimated that the total number of people with dementia will increase. Importantly, public awareness of dementia, symptoms, manifestations and the associated complications remain low. To date, there is no data on the prevalence of dementia in Indonesia. However, it has been estimated that ~1 million of the overall population has dementia (from another resource). *Dr Nagaendran Kandiah* (Singapore) spoke on ***'Alzheimer's disease among Asians: contribution of vascular factors to cognitive and clinical characteristics.'*** Alzheimer's disease (AD) is the most common cause of dementia worldwide with a global prevalence estimated at 3.9% in people older than 60 years. AD co-exist with other neurodegenerative and vascular pathologies. Clinical studies have estimated that 10-20% of patients with AD have with other vascular disease, while autopsy studies reported 8 to 35%. There is growing evidence than vascular pathology (cerebrovascular disease) may be crucial factor in determining progression of AD. The third talk by *Dr Vorapun Senanarong* (Thailand) '***Epidemiology of Dementia in Thailand'*** reported that the prevalence of late onset dementia in Thailand is estimated to range between 2 to 10%. Currently, dementia is the 4_{th} top disability adjusted life years (DALYs) among elderly women in Thailand. A published study by the speaker reported that 48.2% of elderly subjects (aged 60 and above) had some memory complaints. Young onset dementia, defined as dementia onset before the age of 65 years is rare. The last talk was delivered by *Dr Joseree-AnnS. Catindig* (Philippines) on the ***'Prevalence of Dementia in the Philippines.'*** Similar to many

Southeast Asian nations, there is currently no data on the prevalence of dementia in the Philippines. Similar to problems faced by neighbouring nations, public awareness of this increasing condition is poor and most are not even aware that they are living with someone who have signs and symptoms of dementia, a condition that is largely attributed to as part of ageing.

Symposium 9: Headache and Neuralgia

The first talk of the symposium was delivered by *Dr Shahul Hameed Nainar* (Brunei Darussalam); ***'Burden of Headache'***. Headache is the most prevalence neurological symptoms and reason for consultation and referral to a neurologist. In most instances, headaches are benign and self limiting. However, some may have more sinister causes. The most common headaches is the Primary Headache Disorder such as migraine and tension type headache (TTH). The prevalence peaks during the most productive years between the ages of 25-55 years. Therefore, apart from the individual burden, chronic headache is also associated with societal and economic burden that is considerable; important cause of work time, absenteeism and reduced productivity. There are more data on headaches (prevalence to impact on daily lives and treatment) from the West compared to developing countries. In developing countries, greater emphasis have always been on more life threatening conditions such as stroke, epilepsy and infections. However, just like the West and more developed nations, chronic headache represent a substantial burden to neurologists in developing countries. The next speaker *Dr Siwaporn Chankrachang* (Thailand) spoken on ***'Medication Overuse Headache (MOH'***, an interesting entity, previously referred to as rebound headache, many non-neurologists are not familiar with. MOH is a secondary cause of chronic daily headache due to overuse of acute headache medications. In fact, all treatments use to treat acute headache can lead to MOH. MOH is defined as headaches that are experienced 15 or more days in a month for at least three months and have developed or markedly worsened during medication overuse. Overuse of medication is often motivated by a patient's desire to treat his headache or fear of future headache. Medication overuse can make headache refractory to preventive medications. MOH can be difficult to treat. Patient should reduce reliance on acute medication and be started on preventive medications. However, patients should be made that preventive medications may not be fully effectiveness and medication overuse has to be stopped. The third talk by *Dr Regina Macalintal-Canlas* was on the link between Migraine and Stroke ***'Migraine and Stroke… the link'***. Both stroke and migraine are among the most common neurological problems seen by neurologist. They often present with combination of headaches and neurological deficits secondary to alteration of cerebral blood flow. However, they occur in different age groups. So where is the link? The association between migraine and stroke is complex and multi-dimensional and the link can be; migraine-induced stroke, migraine-related stroke, co-existing migraine and stroke. Women aged under 45 with migraine have been found to have three folds increase for stroke in those without aura; six folds in those with aura. Patients with migraine, in particular those with aura have more cerebrovascular risk compared to controls. History of migraine is also frequently found in patients with stroke (14.9% vs. 9.1%) in case control study. With regard to the underlying pathophysiology, there is no evidence of migraine directly causing stroke, However, migraine physiology has agents which may contribute to risk of stroke such as the cortical spreading depression and vasoconstrictive agents. Other similarities include; 677TT and ACE D/DI genes, cardiovascular risk factors such as patent foramen of Ovale (PFO) and hypercoagulability. However, more research is needed to evaluate the link between these two common neurological conditions. The last talk of this symposium was on ***'Chronic Migraine Treatment'*** by *Dr Julia Shahnaz Merican* (Malaysia). While there is no cure for migraine, there are available treatment that can effectively control the symptoms. Apart from avoidance of precipitating factors (i.e. physical exertion, diet, hormonal changes, stress and anxiety, sleep deprivation or excess and environmental factors), treatment of

chronic migraine fall into two groups: acute and preventative. Acute medications are available over the counter or can be prescribed and are taken on a as required basis. The aim of the treatment is to terminate or shorten the duration of attack. Examples of medications for acute attack are: non specific;- NSAIDs, combination analgesic (usually acetaminophen related), opioids, neuroleptics/anti-emetics, and specific;- ergotamine or triptans. The preventive (prophylactic) medications are used for patients with frequent attacks and aim to reduce the frequency and severity of attacks. Preventive medications need to be taken on regular basis. The most commonly used medication for prevention are the beta-blockers (propranolol). To date, only five medications have been approved by the FDA for treating chronic migraines; propranolol, timolo, divalproex, topiramate and onabotulinum Toxin A. Anti-hypertensive such as the ACE inhibitors have also been shown to be effective as preventive medications.

Symposium 10: Neuro-ophthalmology and Neuro Otology

This symposium discussed on several topics common to neurology and the fields of ophthalmology and otology. The first talk was by *Dr Elliot Eng* (Singapore) on assessment of peripheral cause of dizziness; ***'When Dizziness Becomes Peripheral...'***. Dizziness or vertigo is a common presentation to the general and specialist clinics. The symptom is non specific and can be a manifestation of many conditions. Establishing the cause of dizziness can be difficult but can be achieved through thorough and stepwise approach. However, clinicians must be knowledge of the neurological pathways that cause manifestation of dizziness. The causes of dizziness can be broadly categorised into central and peripheral causes. In this talk, the speaker concentrated on the peripheral causes: problem localised to the ear or the neurological connections. Causes of dizziness or vertigo include benign paroxysmal positional vertigo (BPPV), inflammation of the inner ear, Meniere's disease, vestibular migraine, labyrinthitis and more sinister causes such as acoustic neuroma. Risk factors include age, medications (i.e. anti-hypertensive, anti-epileptics and tranquilizers) and previous history of dizziness. The second talk was on ***'Diplopia beyond III, IV & VI– Clinical approach'*** by *Dr Mohan Ramalingam* (Brunei Darussalam). In this talk, the speaker covered the basic physiological mechanisms of diplopia from ocular to cortical levels. Causes of binocular diplopia other than pathologies affecting the cranial nerves III, IV and VI and also the neuromuscular junction lesion were discussed. Mechanical causes of diplopia was also covered. In the third talk, *Dr Clement Tan* (Singapore) discussed in detail the ***'The Afferent Visual System'***. The afferent visual system consist of the eye (retina, ganglion cells) the neurological connections (optic nerves, optic chiasm, optic tract, lateral geniculate nucleus, superior colliculi, pretectal nuclei, suprachiasmatic nucleus, optic radiations), and the cortex (Calcarine cortex—primary visual cortex) of the brain. Examinations of the afferent visual system include; visual acuity testing, contrast sensitivity testing, light stress test, colour vision testing, examinations of the pupil, light brightness comparisons and visual field testing. In the last talk of this symposium, *Dr Alvin Seah* (Singapore) talked about ***'Vision and Sleep: Melanopsin-Containing Retinal Ganglion Cells'***. In this talk, the speaker shared with the audience (more so for the non-neurologist) recent understanding of the visual pathways and the link between vision and sleep. Before the discovery of photosensitive retinal ganglion cells (pRGC), it was thought that photoreceptors cells were only rods (pigment; rhodopsin) and cones (photopsin I, II and III). Each opsin reacts best with photons of a different colour (Spectral sensitivity) and the reactions between the opsins and photons result in hyperpolarisation of the photoreceptor cells, so called phototransduction. This in turn releases glutamate which affects bipolar cells, and the signals are filtered through bipolar and amacrine cells and are ultimately transmitted to the retinal ganglion cells (RGCs). RGCs are the cell that send axons that collectively make up the optic nerve. With the discovery of pRGCs, which themselves contain photopigments and can respond to light even in the absence of rods and cones, led to the understanding the link

between visual pathway and sleep. The pigments in the pRGCs, melanopsin have different spectral sensitivity are involved with control of pupil size, connection to the suprachiasmatic nucleus of the hypothalamus and the ovilary pretectal nucleus of the midbrain; involvement in the day-night (circadian) rhythm.

Symposium 11: Neuromuscular Diseases

This symposium covered diseases of the neuromuscular system, a big topic in the field of neurology. *Dr Rawiphan Witoonpanich* (Thailand), discussed about the ***'Electrophysiological diagnosis of neuromuscular junction disorders'***. The clinical presentations of neuromuscular junction (NMJ) disorders often consist of familiar symptoms and signs which frequently pose a diagnostic challenge to the clinicians. NMJ disorders can be presynaptic or post synaptic. Electrophysiological studies play important role in establishing the diagnoses. A classical example of NMJ disorders is myasthenia gravis (MG). Other NMJ disorders include Commonly used studies include the repetitive nerve stimulation (RNS) and single fibre electromyography (SFEMG). Different NMJ disorders give different patterns and awareness and recognitions of the various pattern is central to use of electrophysiological studies in diagnosing NMJ disorders. In the second talk, *Dr Ludwig Damian* (Philippines) talked on ***'Updates in the treatment of Myasthenia'***. After diagnosing, myasthenia gravis (MG), it is important to search for underlying association, specifically a thymoma. The current management of MG is largely through immune modulation and this can be done through several options. Apart from the use of traditional immunosuppressant such as steroid and steroid sparing immune modulators, use of monoclonal antibody such as CD20 Rituximab has now been shown to useful. However, this has been based on small studies. Rituximab which depletes the B cells can lead to long duration of immune suppression. However, it is also associated with risk of serious infections. Use of monoclonal antibody for MG is a promising strategy though more evidence is needed to better understand the precise roles in MG. In the third talk, *Dr Umapathi Thirugnanum* (Singapore) provided an ***'Update on immune mediated neurpathies'***. In this talk, the speaker used a series of illustrative cases to discuss on common immune mediated neuropathies such as Guillain Barré syndrome, chronic inflammatory demyelinating polyneuropathy, multifocal motor neuropathy, sensory neuronopathy and vasculitic neuropathy. IN the next talk, *Prof Goh Khean Jin* (Malaysia) concentrated specifically on the ***'Limb-Girdle Muscular Dystrophy in Malaysians'***. Muscular dystrophies are a heterogeneous group of disorders where progressive muscle weakness and dystrophic changes are the central features leading to increase debilitations of the patients. Limb-girdle muscular dystrophies (LGMD) specifically refer to a disease group that has an autosomal inheritance (dominant or recessive) with late onset and predominantly affecting the proximal muscles. This differentiates it from the more common X-linked Duchenne/Becker and fascio -scapulohumeral muscular dystrophies as well as the congenital muscular dystrophies. There are currently 14 different autosomal recessive LGMD (Type 2) and eight autosomal dominant (Type 1) described. In their experience with patients with LGMD (N=76), 11.8% were found to have LGMD2A based on mutations in the CAPN3 gene. Based on immunohistochemistry study, 10 patients had reduced dysferin staining, seven had confirmed mutations of the dysferin gene, confirming the diagnosis of LGMD2B. Six patient has reduced or absent staining for sarcoglycan; of whom two (2.6%) had mutations for LGMD2D and three (3.9%) for LGMD2E.

Symposium 12: Neurology of Sleep Disorders

The first talk of this symposium discussed on the link between sleep disorder and stroke by *Dr Yotin Chinvarun* (Thailand): ***'Sleep-Disorder Breathing and Acute Ischaemic Stroke'***. Large scale studies have established that patients with moderate to severe untreated obstructive sleep apnoea (OSA) are at increased risk for stroke and other cardiovascular events; both fatal and non-fatal events. With the growing prevalence of obesity, this is a major concern. Changes associated with OSA such as hypoxemia, reduction of cerebral blood flow,

decreased cardiac output, cardiac arrhythmias, blood pressure swings, increased sympathetic activity, baroreceptors dysfunction, endothelial dysfunction, inflammatory changes, decreased fibrinolytic activity and increase platelet dependent modifiable aggregrability may be responsible for onset or rapid progression of stroke during sleep in patients with sleep disordered breathing (SDB). SBD is associated with increased long term mortality. Cheyne-Stokes breathing, a pathological breathing pattern is commonly found in half of patients with heart failure, but can also be found in 20.6% of patients suffering from acute lacunar strokes. Therefore, stroke itself can predispose to the development of Cheyne-Stokes breathing. Treatment of SDB in stroke patients include prevention and early treatment of secondary complications such as aspiration, respiratory infections and pain. There should be cautious use of any sedative medications and patients advised to avoid any alcohol. Body position is also important as it can predispose to obstructive events especially in patient at risk or have OSA. Use of CPAP has been reported to be beneficial in up to 70% of selected stroke patients. Bilevel positive pressure therapy may be tried in patients who failed CPAP. Use of adaptive servo ventilation has been shown to be useful in patients with Cheyne-Stokes breathing. In the second talk; *Prof. Lim Shih -Hui* (Singapore) spoke on ***'Sleep disturbances in Epilepsy'***, an area that is probably under appreciated in clinical practice. In fact, sleep disturbance is consistently ranked among the top three adverse events in patients with epilepsy. Subjective sleep complaints in the previous six months have been reported by 15 to 40% of patients with epilepsy compared to controls. Sleep disturbance, like other conditions, can lead to reduced quality of life, independent to the negative impacts of the underlying conditions. Lack of good sleep can significantly impact neurocognitive and psychological functions which may already been impaired as results of anti-epileptic medications. In adult, presence and frequency of seizure activities influence quality of sleep and contribute to alteration of sleep architecture. This leads to a vicious cycle of frequent arousal, inadequate sleep resulting in seizure activities, control, psychological impact and disturbed sleep. In children, factors that contribute to sleep disturbance include younger age, developmental delay, increased frequency of seizures, symptomatic seizures and co-existing sleep disorder. The medications used to treat the underlying disease also cause sleep disturbance; especially the older generations anti-epileptic drugs. The dose and duration of treatment also affect sleep cycle. The last talk of this symposium focused on sleep disturbance as result of movement disorders and dementia, in particular Parkinson's Disease; ***'Movement Disorders and Dementia'*** delivered by *Dr Lim Li Ling* (Singapore). Sleep disturbances are common and increase with advancing age. However, it is more in patients with movement disorders and dementia and not surprisingly, patients with Parkinson's Disease have been shown to suffer from various sleep disturbances, contributing to the negative impact of the condition; impaired quality of life. In fact, sleep disturbance are often severe and typically overlooked and inadequately treated in patients with Parkinson's Disease. Sleep disturbance in Parkinson's Disease have been attributed to combination of factors; effects of medications, comorbid conditions (i.e. depression), abnormal movements leading to physical discomforts, neurodegenerative effects of the sleep-wake mechanism. Abnormalities in sleep architecture have been shown to correlate with duration of disease. Common sleep disorders are insomnia (53%), daytime somnolence (32%) and unfreshing sleep. Snoring have been reported by 72% of patients with Parkinson's Disease. Unlike the motor component, there is much that is not known regarding sleep disturbance sin Parkinson's Disease. Parkinson's Disease have also been associated with several sleep disorders; REM behavioural disorder (REMBD), Restless Legs Syndrome (RLS) with Periodic Limb Movement of sleep, nightmares and Obstructive Sleep Apnoea (OSA). Various abnormalities of sleep patterns have been described including sleep fragmentation, increase arousals, decreased sleep efficiency and abnormal muscle tone during REM sleep. Sleep disturbance is also very common among Singaporean patients with Parkinson's Disease, but generally overlooked due

to lack of awareness among the public and also clinicians.

Teaching Course (Saturday 19th October 2013)

The teaching course was part of the programme targeting non-neurologist or aspiring juniors doctors are interested in neurology. This teaching course consistent of nine sessions covering various aspects of neurology: ***'Neurological History and Examination'*** by *Prof. Lim Shih Hui* (Singapore), ***'Use of Neurodiagnostic Studies'*** - *Dr N V Ramani* (Singapore), ***'Headaches and Facial Pa***in' - *Dr Dk Hjh Nur ashikin Pg Hj Tengah* (Brunei Darussalam), ***'Approach to Dizziness'*** - *Dr Umapathi Thirugnanam* (Singapore), ***'Episodic Loss of Consciousness'*** - *Dr See Siew Ju* (Singapore), ***'Stupor and Coma'*** - *Dr Jessie T Colacion* (Brunei Darussalam), ***'Sensory Disorders'*** - *Dr Hjh Anas Naomi DP Haji Harun* (Brunei Darussalam), ***'Overview of Afferent Visual Pathway Disorders for Non-Neurologist'*** - *Dr Mohan Ramalingam* (Brunei Darussalam) and ***'Weakness and Gait Disorder'*** - *Dr Norazeida Yassin* (Brunei Darussalam).

NOTE: Some materials not covered in the lecture presentations have been added into the summary for benefits of the general readers.