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MEDICAL NEWS

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FOCUS: NEUROLOGY

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- Rapidly Progressive Dementia: Not Your Usual Memory Loss
- Deep Brain Stimulation for Parkinson's Disease

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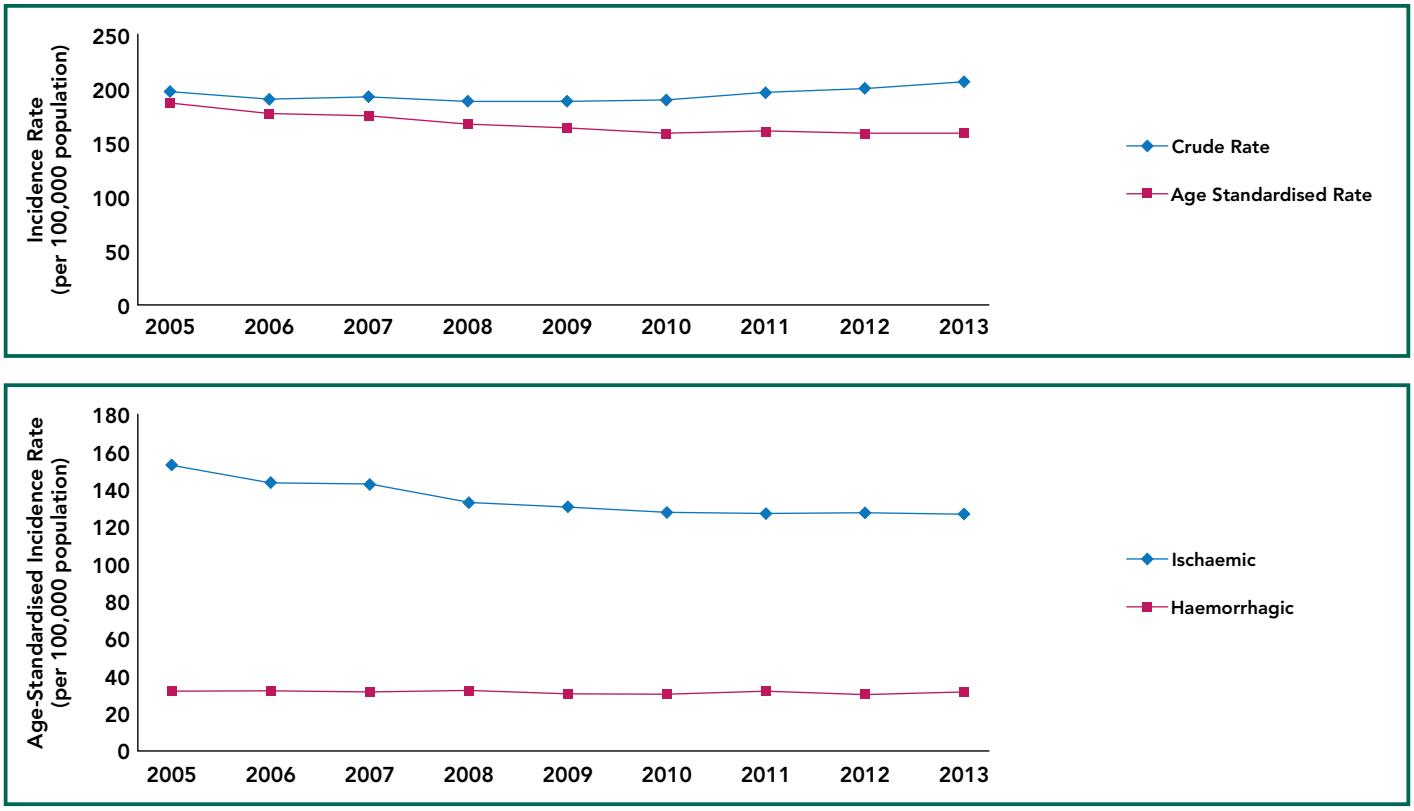
Hyper-Acute Stroke Treatment: Updates on a Rapidly Changing Field

Dr Wee Chee Keong, Consultant,
Department of Neurology, National Neuroscience Institute

Stroke is an enormous public health problem worldwide. The National Registry of Diseases Office reported that in 2014, there were about 7000 cases in Singapore, which approximates 20 cases per day. Most of those affected are aged over 60, and about 80% are ischaemic strokes. Stroke also ranks consistently amongst the top 5 causes of death in our country.

With better understanding of Pathophysiology and primary prevention, the age-specific incidence of stroke in Singapore has been declining. However, the absolute number of patients each year has been gradually increasing, likely due to demographic shifts. *(Refer to Figure 1)*



Subtype	2005	2006	2007	2008	2009	2010	2011	2012	2013
Ischaemic	4458	4373	4545	4455	4639	4749	4899	5137	5339
Haemorrhagic	951	994	1009	1097	1091	1125	1212	1202	1284
Unknown	47	33	24	31	29	16	30	25	19

Figure 1 Stroke Incidence Rates

WHAT HAPPENS AFTER A STROKE?

Results from the Oxfordshire Community Stroke Project showed[1] that, at 1 year after a stroke, approximately 23% of patients have demised. Amongst the survivors, about 35% are dependent in their activities of daily living (ADLs). In practice, the potential for permanent disability with a severely compromised quality of life is often the greatest fear patients have, sometimes even exceeding the fear of death itself.

The socio-economic burden is also significant, as stroke is a leading cause of adult disability in developed countries.

CAN WE PREVENT DISABILITY IN STROKE PATIENTS?

Animal studies in the 1980s by Astrup et al[2] showed that following acute vascular occlusion, not all brain tissue within an arterial territory infarcts immediately. Within the area of Oligaemia, there is a "penumbra", where the neuronal tissue, though non-functioning, remains viable. If perfusion could be restored quickly, these neurons could be salvaged and their function restored. It was theorised that stroke patients benefit similarly from penumbral salvage.

This hypothesis was proven in 1995, in the NINDS trial of tissue plasminogen activator for acute ischaemic stroke[3]. In this randomised controlled trial, 622 patients presenting within 3 hours of stroke onset were randomised to receive either Intravenous (IV) Alteplase at 0.9mg/kg or placebo.

At 3 months post-stroke, significantly more patients in the Alteplase group (39%) were able to return to all their previous activities (mRS 0 - 1) as compared to the placebo group (26%). This was not without risk, as the risk of symptomatic intracranial haemorrhage was at 6.6% in the Alteplase group, versus 0.6% in the placebo group. However, overall, mortality was similar in both groups at 3 months post-stroke. *(Refer to Figure 2)*

The time window for IV Alteplase was later extended from 3 hours to 4.5 hours with the conclusion of the European ECASS III trial in 2008[4]; though predictably, the observed benefit was less, when compared to the original NINDS trial. The Number Needed to Treat (NNT) for a functional outcome of mRS 0 - 1 at 3 months post-stroke rose from 8 to 14.

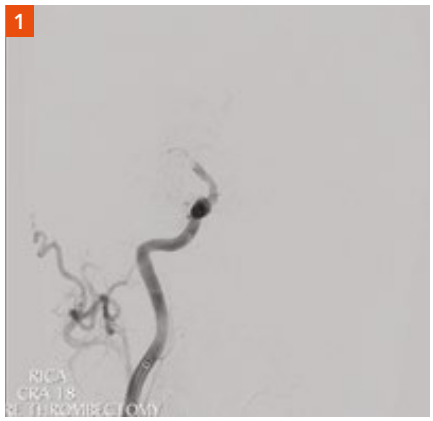
ENDOVASCULAR CLOT RETRIEVAL

The next significant breakthrough in hyper-acute stroke treatment came in 2015. Intravenous (IV) Thrombolysis was considered the standard of care at this stage. However, patients with occlusions of the large arteries in the anterior circulation still did poorly, as these tended not to recanalise with Alteplase alone; in fact, early recanalisation occurred in approximately 1/3 of patients with internal carotid artery occlusions.

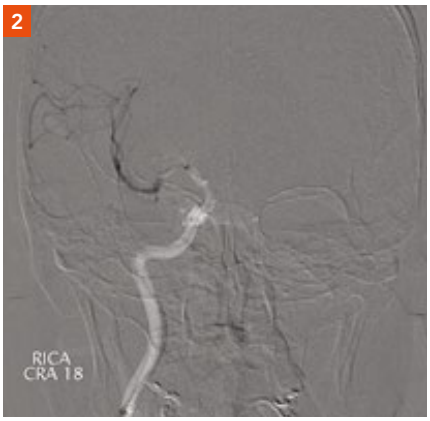
The Dutch investigators in the MR CLEAN trial[5] randomised 500 patients into 2 arms, one receiving endovascular treatment plus usual care (which may include IV Thrombolysis), and the other receiving only usual care (IV Alteplase within 4.5 hours of onset, if no contraindications were present). Patients presenting with a radiographically proven occlusion of the Internal Carotid Artery (ICA), or the first 2 segments of the Middle Cerebral Artery (MCA) or Anterior Cerebral Artery (ACA), and who were able to receive endovascular treatment within 6 hours of stroke onset, were recruited.

Modified Rankin Scale (mRS)	Description
0	No symptoms.
1	No significant disability. Able to carry out all usual activities, despite some symptoms.
2	Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities.
3	Moderate disability. Requires some help, but able to walk unassisted.
4	Moderately-severe disability. Unable to attend to own bodily needs without assistance, and unable to walk unassisted.
5	Severe disability. Requires constant nursing care and attention, bedridden, incontinent.
6	Death

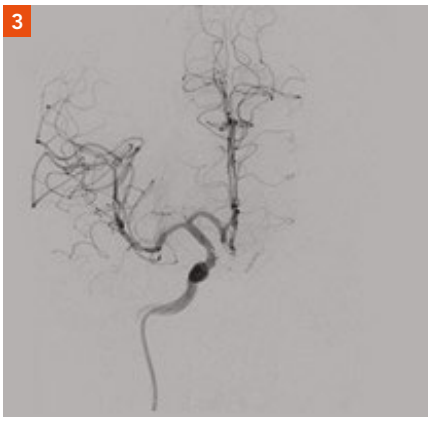
Figure 2 Modified Rankin Scale (mRS)



Right ICA Occlusion



Right ICA transversed by a Stent Retriever with Contrast in the Distal Branches



mTICI 3 Flow after Successful EVT

Figure 3 Series of Angiographic Images

At 3 months post-stroke, patients who received endovascular treatment were 2 times more likely to achieve an mRS of 0 - 2 (32.6% versus 19.1% or 2.16%). Following this, 5 other RCTs reporting positive results for endovascular treatment were published[6–10], albeit each with slightly different selection criteria and treatment protocols, but overall, displaying the powerful treatment effect of EVTs (Endovascular Thrombectomy) [11]. *(Refer to Figure 3)*

ENDOVASCULAR THROMBECTOMY (EVT) WITHOUT THROMOBOLYSIS

What was not very clear at the time was whether Endovascular Thrombectomy (EVT) alone would be effective for treating these patients, as there were only small numbers of them in the aforementioned RCTs.

I was on HMDP fellowship then, and the hospital I was in, was 1 of 2 tertiary centres providing neurointerventional services to the state of Western Australia. Many patients were arriving within the time frame for EVT, but were out of the 4.5-hour window period for IV Thrombolysis.

Yet others came within the 4.5-hour window period, but had clear contraindications to Alteplase, such as having undergone recent surgeries. With this in mind, we initiated a retrospective registry, based on the study of acute stroke cases who had received EVT[12].

Between the period of October 2013 to April 2016, there were 50 patients, with strokes secondary to a proximal occlusion in the anterior cerebral circulation, that received EVT. Contrary to the RCTs where only 15% of patients did not receive Alteplase, we found that in the real-world application, 58% of patients were ineligible for Alteplase.

Outcomes using the surrogate of the National Institute of Health Stroke Scale (NIHSS) showed an improvement of 8 points at 24 hours or a NIHSS of 0 – 2, which was similar between the 2 groups. These findings added to the body of literature that was being published concurrently, supporting the use of EVT in Alteplase-ineligible patients.

CURRENT MANAGEMENT OF STROKE PATIENTS: SGH AND NNI-PARTNER HOSPITALS

Keeping in mind that time is critical for the acute management of stroke patients, we engaged our partners in Civil Defence, who responded enthusiastically.

Ambulance paramedics are now trained in the use of the Cincinnati Prehospital Stroke Scale to make a preliminary diagnosis of an acute stroke. Emergency departments are pre-notified and patients are given priority access to neurologists and CT/MRI scans.

Once a diagnosis of an acute ischaemic stroke is established, eligible patients are given IV Alteplase within 4.5 hours, with 10% of the dosage as a bolus and the remainder as an infusion over the course of an hour. For patients with a suspected occlusion of the proximal anterior circulation within 6 hours of onset, a CT/MR angiography is typical.

If a clot is found, the neurointerventionalist is informed and a decision is made regarding EVT. This is performed on-site at the Singapore General Hospital and the Tan Tock Seng Hospital. A "drip and ship" model is employed in other hospitals covered by NNI neurologists, where Intravenous Alteplase is initiated and the infusion is given en route to the patient, whilst being transferred to an end-vascular facility.

"TIME IS BRAIN"

Regardless of whichever hospital the stroke patient initially arrived in, the key is timely intervention. It was estimated that for strokes due to a large vessel occlusion, 1.9 million neurons are lost each minute.

Many patients are still unaware of the necessary urgency of stroke treatment and seek emergency assistance for acute treatment beyond the required time frame. Some patients even arrive days after the onset of stroke. Despite the many treatment advances in recent years, little is available to help them.

WHAT CAN GENERAL PRACTITIONERS DO?

As primary care partners, we hope that you can help to educate patients on the symptoms of stroke and the need for emergency care. In particular, this includes elderly patients and those with cardiovascular risk factors.

Should a patient visit the clinic with an acute stroke, it is probably better to send them for treatment via the emergency ambulance (in view of the priority access given), rather than through other forms of transport.

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Dr Wee Chee Keong is a Neurologist at the National Neuroscience Institute. He has a keen interest in ischaemic strokes and reperfusion therapies. He recently returned from a year-long fellowship with Professor Graeme J. Hankey at Sir Charles Gairdner Hospital in Perth, with a focus on hyper-acute stroke treatment.

Back in Singapore, he contributes actively to the acute stroke team, members of whom are rostered every day of the year to help provide treatment to the stroke patients at the hospital.

GPs can call for appointments through the GP Appointment Hotline at **6357 7095** or scan the **QR code** for more information.

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Rapidly Progressive Dementia: Not Your Usual Memory Loss

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CASE 1

A 70-year-old Chinese man with no past medical history of note presented with a 2-week history of cognitive impairment.

He had problems with short-term recall, being unable to remember a list of groceries to buy, misplacing items and forgetting to turn off the tap. He also had difficulty turning on his computer. In terms of language, he was noted to have word-finding difficulty and a poverty of speech.

On clinical examination, he was oriented to the time, place and person, but not the day nor date. He was unable to perform the 3-item recall, and was noted to have perseveration of speech and dysphasia. Limb examination was remarkable for bilateral lower limb rigidity.

Blood investigations were unremarkable for any metabolic, autoimmune or infective causes. An MRI of the brain showed cortical diffusion restriction, involving both the temporal and parietal lobes bilaterally, as well as the cingulate gyri and left frontal lobe.
[Refer to Image 1]

An electroencephalogram (EEG) showed periodic sharp wave complexes in the left hemisphere, and cerebrospinal fluid (CSF) analysis was normal. A diagnosis of rapidly progressive dementia, secondary to sporadic Creutzfeldt-Jakob disease (CJD), was made.

The patient's symptoms progressed rapidly. He developed myoclonic jerks and was treated symptomatically with clonazepam and levetiracetam. A month after the onset of symptoms, he had become fully dependent and was in an akinetic mute state. He was subsequently transferred to the inpatient hospice care.

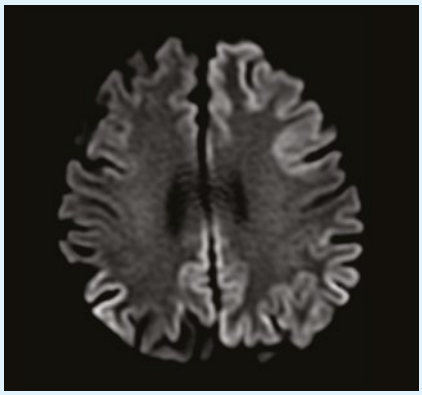

Image 1 MRI showing Cortical Restricted Diffusion

CASE 2

A 67-year-old Malay housewife, who was previously well, was admitted to the hospital for a 5-minute episode of transient unresponsiveness during a religious class. Extensive investigations with an MRI of the brain, EEG and telemetry monitoring for cardiac arrhythmias were negative. She was discharged with an outpatient Neurology referral.

Over the next 3 months, the patient complained of short-term memory loss. For example, she had no memory of having gone to her grandson's primary school, despite being there several times during that period. Her husband noted that she became progressively more anxious, irritable and socially withdrawn. In addition, she also complained of intermittent, brief episodes of chills associated with goose bumps and abnormal epigastric sensation, up to 20 times a day.

An outpatient cognitive testing revealed the evidence of reduced attention and executive dysfunction. A repeat EEG showed epileptiform activity in the left frontal and temporal lobes, suggesting that her episodes of "chills" were due to focal seizures.

A repeat MRI scan of the brain was normal, and a CSF analysis was unremarkable. The initial blood investigations were also normal.

A presumptive diagnosis of possible autoimmune limbic encephalitis was made on the basis of her cognitive decline, personality change and focal seizures. The testing of serum for autoimmune encephalitis antibodies subsequently came back positive for the anti-LGI1 receptor antibody, thus confirming the diagnosis.

She was treated with high dosage intravenous methylprednisolone, followed by a tapering dose of oral corticosteroids. Rapid improvement in her cognitive function and seizure control ensued.

Dementia is defined as a decline in cognition associated with functional impairment. In Rapidly Progressive Dementia (RPD), deterioration from the symptom onset to a diagnosis of dementia progresses at a rate faster than that expected for typical dementia. Though a time frame of 2 years is sometimes used, most cases of RPD develop sub-acutely, from weeks to months [1].

Table 1 **Causes of Rapidly Progressive Dementia**

Causes of Rapidly Progressive Dementia
Prion disease e.g. Creutzfeldt-Jakob disease (CJD)
Non-prion Neurodegenerative disease e.g. Frontotemporal Dementia, Corticobasal Syndrome, Alzheimer disease
Immune-mediated/Paraneoplastic e.g. Limbic Encephalitis
Vascular e.g. Ischaemic/Haemorrhagic Stroke, Subdural Haemorrhage, Vascular Dementia, CNS Vasculitis
Neoplasm e.g. Metastases, CNS Lymphoma
Infection e.g. HSV, HIV, Progressive Multifocal Leukoencephalopathy (PML), Syphilis, Whipple disease
Toxic-metabolic e.g. Electrolyte and Endocrine abnormalities, Illicit drug use

Most patients with RPD present in the 6th or 7th decade of life, though patients as young as 12-years-old have been reported. This is because RPD is essentially a clinical syndrome, which encompasses many varied etiologies [Refer to Table 1].

In the adult population, most are due to irreversible neurodegenerative diseases – usually rapidly progressive forms of more common dementias, such as frontotemporal dementia, corticobasal syndrome, vascular dementia and rarely, Alzheimer disease and dementia with Lewy bodies [2,3].

The other important group of neurodegenerative diseases are those due to abnormal prion protein deposition, such as Creutzfeldt-Jakob disease (CJD). A typical patient is highlighted within **Case 1**. Patients with CJD present with global cognitive decline and other neurological features, including cerebellar ataxia, visual disturbances, parkinsonism and myoclonus.

Diagnosis is made by a combination of clinical, radiological and EEG criteria. A brain biopsy is rarely necessary. The treatment is palliative, and the disease is invariably fatal with a median survival of 6 - 11 months.

The incidence of CJD is one per million of the population per year. At the National Neuroscience Institute at the Tan Tock Seng Hospital Campus, we see an average of 3 cases per year, with 29 cases diagnosed from 2006 to 2016 [4].

More importantly, however, between 17% to 23% of RPD patients have potentially treatable etiologies [1], which include immune-mediated, paraneoplastic, neoplastic, infective and toxic-metabolic conditions [Refer to Table 1].

In these patients, the reversibility of dementia improves with early diagnosis and treatment. Hence, it is incumbent on the evaluating physician or neurologist to perform a comprehensive evaluation to identify a treatable cause of RPD.

SYMPTOMS AND INITIAL EVALUATION

Perhaps the most crucial part of the initial evaluation of a patient with RPD is to obtain the clinical history from a reliable historian and, wherever possible, from multiple corroborative sources [1].

The first step is to exclude the presence of delirium, often due to a reversible systemic cause, which can cause the rapid decline of pre-existing cognitive impairment or dementia [2].

Secondly, some patients who present with so-called RPD may, in actual fact, already have long-standing undiagnosed dementia. Subtle cognitive decline may have progressed insidiously for a few years, but patients present only when the memory loss is significant enough to come to their family members' attention - thus mimicking an RPD. This is especially so for non-Alzheimer dementias, that tend to cause executive dysfunction and personality changes, with the relative sparing of short-term memory. Hence, a corroborative history from multiple sources is important.

The next step is to establish the progression of symptoms. An abrupt onset or stepwise progression is the hallmark of a vascular etiology, while an acute presentation over days may indicate an infective or toxic-metabolic cause.

In RPD, the initial presenting symptoms and signs are also useful for neurolocalisation and diagnosis. This requires a detailed evaluation, not only of the classical symptoms of dementia (amnesia, agnosia, aphasia, apraxia, agnosia, executive dysfunction), but also other features that localise to various affected regions of the brain. Therefore, a detailed neurological examination is compulsory to identify signs of cerebellar, extrapyramidal or brainstem dysfunction [1].

In **Case 2**, our patient presented with short-term memory loss, focal seizures with epigastric aura and a personality change, all of which localise to the limbic system. This led to the clinical suspicion and eventual diagnosis of autoimmune limbic encephalitis, a treatable cause of RPD [5].

The presence of systemic symptoms and the patient's medical and drug history also help to narrow down the differential diagnoses. Common drugs that may affect cognition, particularly in elderly patients, include anticholinergics and benzodiazepines. In a patient with risk factors for sexually transmitted diseases, HIV and syphilis need to be considered; while a patient with anorexia and cachexia may be harbouring a malignancy.

WHAT IS THE ROLE OF THE GENERAL PRACTITIONER (GP)?

A GP who encounters a patient who is suspected of RPD should not hesitate to refer him or her to a specialist Neurology clinic for an early evaluation. Screening of the renal and liver function, electrolytes (especially of sodium and calcium), thyroid function, vitamin B12 and folate levels – and where relevant, HIV testing – can also be performed in the primary care setting.

Abrupt or rapid progression of symptoms over days should prompt a referral to the Emergency department, not only for the exclusion of a life-threatening cause, but also for prompt evaluation and treatment.

WHAT ARE THE LIKELY TREATMENT OPTIONS BY THE SPECIALIST?

In the Neurology clinic, a full evaluation of the patient's cognition and an MRI of the brain are obligatory. Further ancillary investigations, such as an EEG, CSF analysis and more specialised blood investigations, are often necessary. Depending on the clinical context, systemic evaluation with CT or PET imaging for occult imaging may also be performed.

Treatment options depend on the etiology of the RPD, ranging from symptomatic (in the context of neurodegenerative diseases) to potentially reversible (in the setting of infections, metabolic disorders, autoimmune conditions and neoplasms).

WHEN WILL THE PATIENT BE REFERRED BACK TO THE GP?

Patients with treatable conditions (for example, hypothyroidism) may be referred back to their GP when specialist titration of treatment is no longer required.

Some patients with neurodegenerative dementias, such as Alzheimer or vascular dementia, whose symptoms are well-controlled with medications such as cognitive enhancers, antidepressants and/or antipsychotics, can also be followed up by their GP. Patients with limited life expectancy may either be transferred to inpatient hospice care or discharged to the care of a primary care physician in the home care setting.

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Deep Brain Stimulation for Parkinson's Disease

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PARKINSON'S DISEASE (PD) is a chronic and degenerative brain disorder that results in motor impairment. After 4 to 6 years of treatment, medication-induced complications can develop. As the disease advances, symptoms become more severe and other problems related to the patient's gait, cognition and musculoskeletal system develop.

In Singapore, PD is the second most common neurological disorder. Local studies have shown that the disease occurs in three out of every 1,000 people aged 50 and above. It is estimated that there are between 4,000 to 5,000 PD patients in Singapore [1].

Oral medication is the standard of care in early PD; however, as the disease progresses and medication-induced complications develop, patients may become refractory to this treatment option.

Deep Brain Stimulation (DBS) is the most common surgical treatment performed on people with PD, when medications alone are no longer able to adequately control their motor symptoms. It is also required when medication-related problems, particularly levodopa-induced dyskinesia (involuntary movements) and motor fluctuations, significantly affect the quality of life of the patient.

DBS therapy is an adjustable and reversible surgical treatment and more than 140,000 patients worldwide have been treated with DBS. In Singapore, more than 100 patients have benefited from DBS therapy. DBS has been approved for the following indications:

- Essential tremor
- Parkinson's disease
- Primary dystonia
- Obsessive-compulsive disorder
- Epilepsy

SYMPTOMS OF PARKINSON'S DISEASE

PD is clinically defined by the presence of bradykinesia (slowness in movement), with at least one cardinal motor feature (rigidity or rest tremor).

In addition to the cardinal motor features, patients with PD also have non-motor symptoms involving a multitude of functions, such as disorders of the sleep–wake cycle regulation, cognitive impairment, disorders of mood and affect, autonomic dysfunction (mainly orthostatic hypotension, urogenital dysfunction, constipation and hyperhidrosis), as well as sensory symptoms (most prominently hyposmia) and pain [2].

Common motor features of PD

Figure 1

BENEFITS OF DBS THERAPY

DBS therapy can give PD patients an additional 5 hours of good movement control per day, compared to the best medical therapy (using only oral medication). This can help the patient to regain the ability to perform normal daily activities and to improve their quality of life.

WHO SHOULD AND WHEN TO GO FOR DEEP BRAIN STIMULATION?

In February 2013, the EARLYSTIM study published in The New England Journal of Medicine provided Class I evidence for the use of DBS therapy in the earlier stages of Parkinson's disease.

The trial recruited DBS patients in the early stages of PD (disease duration of ≥4 years and disease severity below 3 on the Hoehn and Yahr scale) and showed that DBS provided superior benefits for patients with early motor complications from PD, as compared to those who received only the best medical therapy [3].

These findings led to an FDA approval in 2016, for DBS in patients with at least 4 years of disease duration and 4 months of motor complications, as an adjunct therapy for patients who are not adequately controlled with medications.

Deep brain stimulation for PD has a window of opportunity, where it may be most effective for PD patients. The window opens when a patient has the disease for at least 4 years' duration that is not adequately controlled by medication, including motor complications for at least 4 months or of longer-standing duration.

Typically, the patient may present with one or more of the following symptoms observed:

- "On" time characterised by disabling dyskinesias (or other non-motor side effects)
- "Off" time characterised by disabling tremor, rigidity, or akinesia/bradykinesia
- Unpredictable motor fluctuations
- Medication-resistant tremor

The window of opportunity for DBS closes when:

- Symptoms no longer respond to dopaminergic medication
- The patient is severely disabled, even in the best "on" state
- Medical conditions prevent surgery
- The patient has dementia

(Refer to Figure 2)

Benefits of DBS Therapy

Figure 2

DBS is not a cure for PD and it may not be suitable for everyone. Hence, people with PD will need to undergo a set of comprehensive tests and evaluations to assess their suitability before proceeding to a surgery.

Refer to *Figure 3* for an overview of the disease progression and the therapeutic window.

Careful patient selection is performed by a multidisciplinary approach (comprising of neurologists, specialist nurses, neurosurgeons, anaesthetists, speech therapists, occupational therapists, physiotherapists, psychiatrists, psychologists and social workers). After surgery, once the device is turned on, our patients experience an immediate improvement in their movement. This leads to a significant benefit in their overall function and quality of life.

DBS patient support group sessions in the hospitals are held regularly to provide a platform for the PD community to share their experiences, and to provide insights and support.

HOW DOES DBS THERAPY WORK?

The exact mechanisms of how focal stimulation by DBS exerts local and systemic effects across the brain networks remain unclear. Initial views were based on the classic "rate model", where the motor symptoms of PD were attributed to altered neuronal firing in the basal ganglia, wherein DBS creates a reversible "functional" lesion or a physiological block.

More recent models suggest that DBS acts through multifactorial mechanisms, including immediate neuromodulatory effects, synaptic plasticity and long-term neuronal reorganisation [4,5].

WHAT IS IN A DBS THERAPY SYSTEM?

A small, pacemaker-like device is surgically placed under the skin, just beneath the collarbone. The device sends electrical signals through lead wires to a precise location in the brain, in either the subthalamic nucleus (STN) or *globus pallidus interna* (GPi) that controls movement. These signals block some of the brain's messages that cause the annoying and disabling motor symptoms associated with PD. *(Refer to Figure 4)*

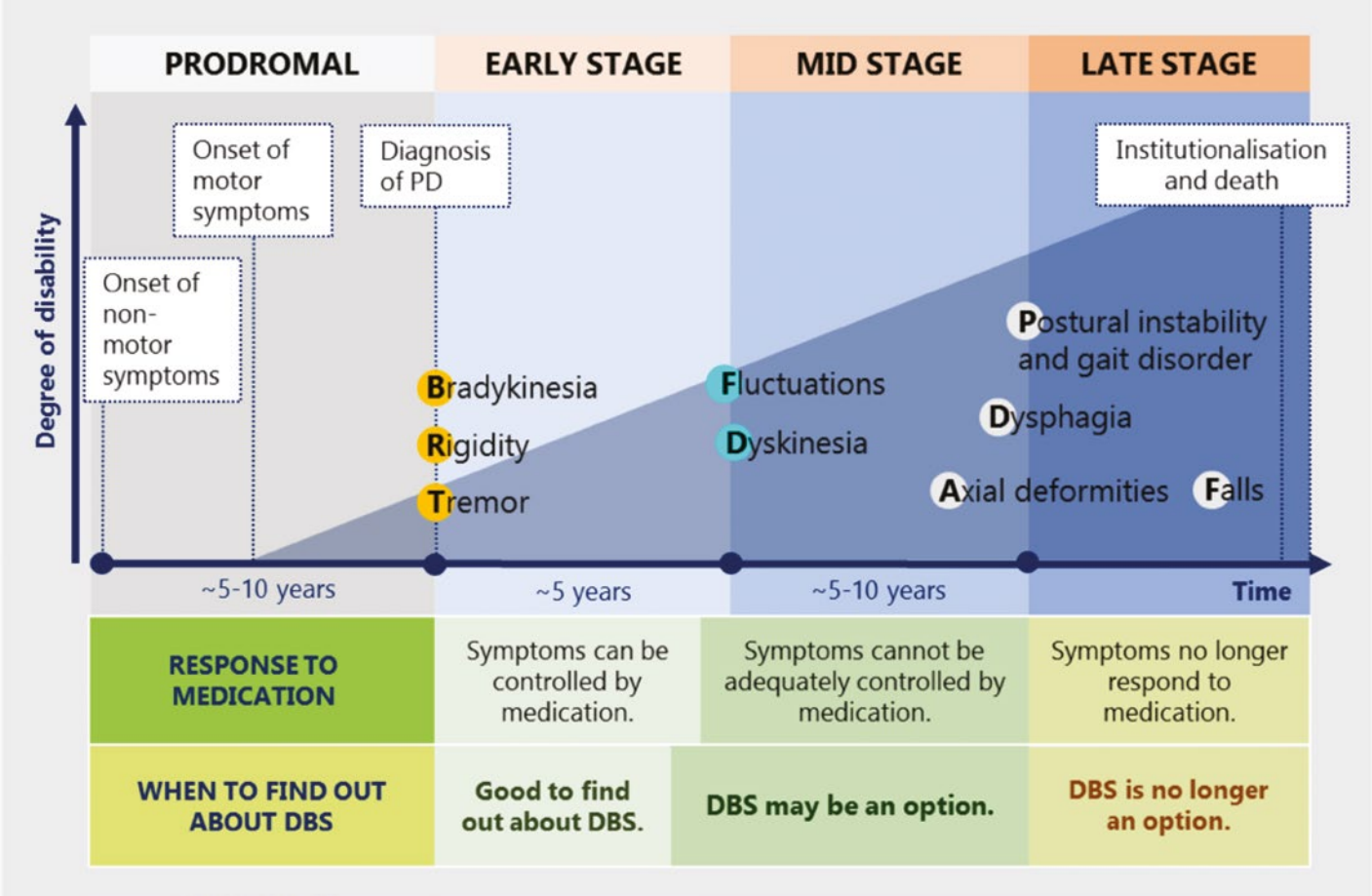

Figure 3 Motor Symptoms associated with PD Progression, Response to Medication and the Therapeutic Window for DBS

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HOW IS DBS SURGERY PERFORMED?

DBS surgery is a minimally invasive procedure. Before the surgery, the patient is required to go for a brain MRI where the MRI images will be used on the day of the operation.

The surgery consists of 2 stages. In the first stage, a head frame is attached to the patient's head and the purpose of the frame is to ensure that the head is immobile, so that the neurosurgeon can accurately and precisely place the DBS lead wires into the target location within the brain. The patient will go for a CT Scan with the head frame in place. The neurosurgeon then uses the pre-operative MRI and CT image to plan the best trajectory for the entry point of the lead electrodes.

Before the leads are placed permanently, several steps are taken to ensure that the permanent electrode is placed in the optimal position. A test electrode is used to locate the best spot in the brain. The location of the test electrode is checked by listening to the brain through microelectrode recording (MER; *Refer to Figure 5*). Besides MER, the patient under sedation will be woken up and asked to perform simple motor tests, such as moving their limbs or talking.

In the second stage, the patient will undergo the insertion of the pulse generator (or battery). Typically, the length of the hospital stay for a DBS surgery is 5 to 7 days.

The DBS battery will be turned on approximately 1 month after the surgery. Over the next 3 to 6 months, the patient will be followed up closely by the neurologist or APN for programming to find the best stimulation settings for the patient. In most cases, after programming, the patient's PD medications will be reduced.

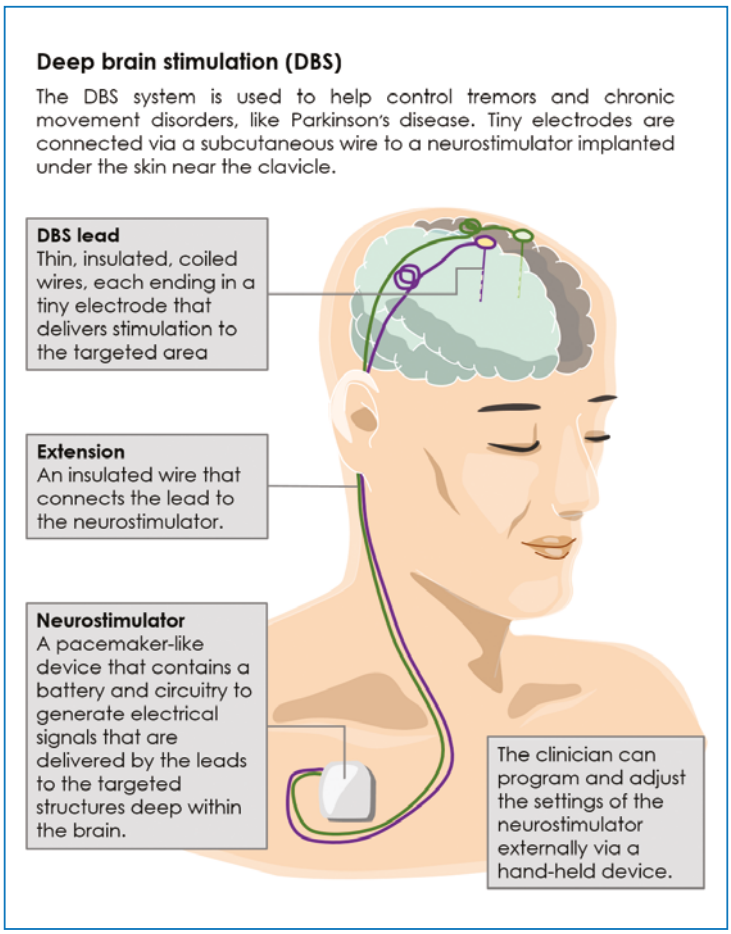


Figure 4 Components of the Deep Brain Stimulation system

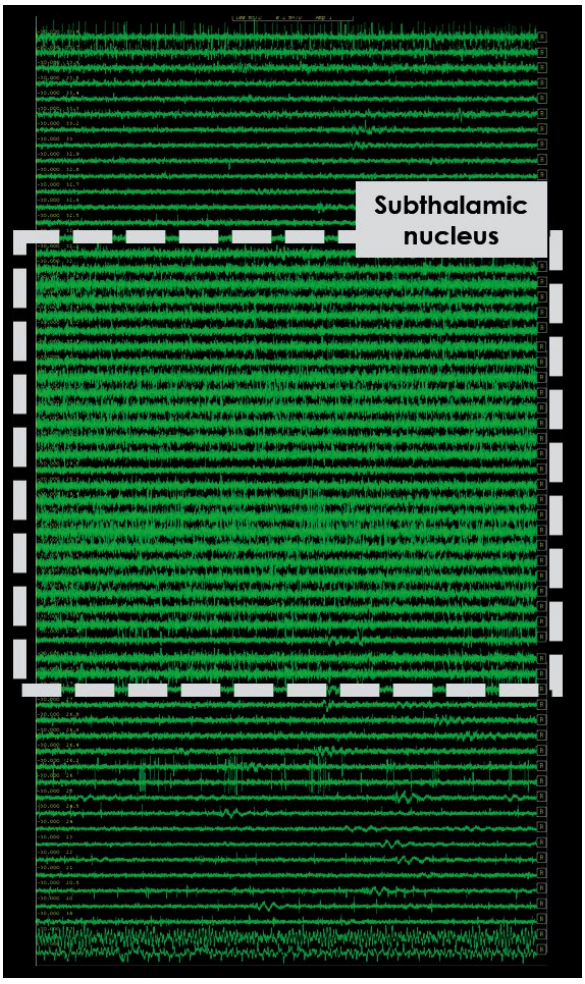
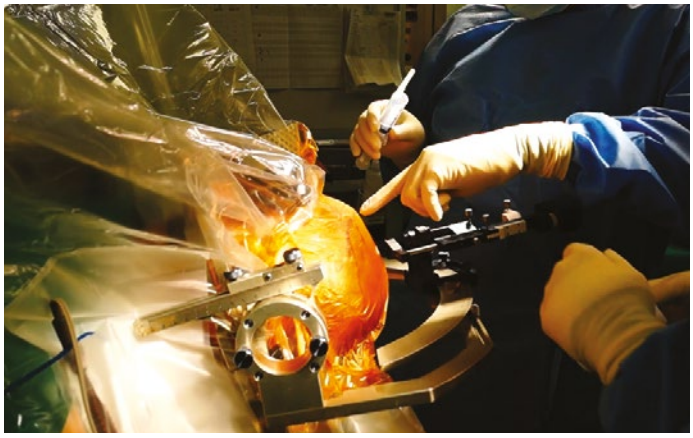


Figure 5 **Diagram of Microelectrode Recording (MER)**
The subthalamic nucleus (STN) has a characteristic firing pattern, which can be detected with MER

SUMMARY

As PD progresses at different rates for everyone, it is important to consider all the treatment options at the early stages of the disease.

By considering and starting DBS therapy early and at the appropriate time, patients with PD can experience greater therapeutic benefits and a better quality of life. Delaying DBS therapy might result in missing that window of opportunity altogether.



Deep Brain Stimulation Surgery

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Assistant Professor Nicolas Kon Kam King is a Consultant at the Department of Neurosurgery at the National Neuroscience Institute (NNI), where he subspecialises in Functional Neurosurgery, Deep Brain Stimulation and Neuro-Oncology. He practises at both the Singapore General Hospital and the Tan Tock Seng Hospital campuses of the NNI. He is also the Co-Director of the NNI Deep Brain Stimulation programme.

GPs can call for appointments through the GP Appointment Hotline at **6357 7095** or scan the **QR code** for more information.

New Care Model for Pregnant Women with Gestational Diabetes

In Singapore, up to one in five women are at risk of Gestational Diabetes Mellitus (GDM), a condition that puts them at an increased risk of certain conditions during pregnancy, including the development of high blood pressure, incidence of preterm labour, maternal complications and the development of Type 2 Diabetes Mellitus (T2DM) in their lifetime.

Despite the risks, about 90% to 95% of women with GDM do not undergo regular check-ups after delivery to ascertain if their condition has resolved, in a bid to monitor their diabetes condition. They also do not maintain a routine screening for T2DM, of at least once every three years.

In September 2017, the KK Women's and Children's Hospital, as well as the Temasek Foundation Cares, introduced the **Temasek Foundation Cares GDM Care programme** to improve the detection, care and support for this vulnerable group.

It aims to pilot a novel model of care to encourage all pregnant women who receive antenatal care in the KK Women's and Children's Hospital to undergo a GDM screening. It also encourages them to receive appropriate antenatal and postnatal care, as well as to engage in follow-up sessions to track and manage their diabetes condition.

The $1.09 million programme aims to benefit about 5,400 women with GDM and their families over a three-year period.

"The incidence of GDM is rising globally due to an increasing average age for child-bearing, as well as an increased prevalence of obesity in the population, exposing both the mother and the baby to increased health and mortality risks," explains the programme lead, Professor Tan Kok Hian, who is also the Head and Senior Consultant at the Perinatal Audit and Epidemiology Unit, Department of Maternal Fetal Medicine, KK Women's and Children's Hospital.

"Children born from pregnancies affected by GDM tend to be big babies weighing more than four kilogrammes at birth, putting them at a higher risk of suffering birth trauma and a lack of glucose in the bloodstream, which can lead to long-term negative health effects. They also have higher risks of developing obesity and T2DM later in life," Prof Tan adds.

Diabetes Care Navigator and Nurse Clinician, Asmira Bte Mohamed Rahim, guides a pregnant patient on the self-administered finger-prick test to monitor her blood sugar levels.

THE PROGRAMME AND ITS BENEFICIARIES

Under the pilot programme, all expectant mothers in the KK Women's and Children's Hospital are offered the routine GDM screening between 24 and 28 weeks of gestation.

Expectant mothers with GDM are also guided by a team of Diabetes Care Navigators in observing a care plan for optimal management of the condition, to achieve the best outcomes for both the mother and child.

Educational support will be provided to the patients and their families during pregnancy and after delivery to encourage healthier lifestyles and to minimise the health risks arising from a pregnancy affected by GDM.

If the condition persists after delivery, they can be referred to a network of care partners for further evaluation and for the follow-up management of their condition.

Subsidies will also be provided to women who require further financial assistance to optimise care for themselves and their families.

"The Temasek Foundation Cares GDM Care programme is truly a welcome boost to our ongoing efforts to enhance care and to improve the health for women and children.

With the early detection, timely intervention and close follow-up care afforded by this structured care and education programme, we are closer to optimising the prevention and management of diabetes and its associated health risks for our future generations," says Prof Tan.

For more information on the **Temasek Foundation Cares GDM Care programme**, please contact the **KKH Obstetrics Day Assessment Centre at 6394 2097.**

Singapore's First Donor Human Milk Bank Opens at the KK Women's and Children's Hospital

In August 2017, the KK Women's and Children's Hospital launched Singapore's first **Donor Human Milk Bank Programme** to provide a donated ready supply of safe, pasteurised human breast milk for vulnerable premature and sick neonates, whose mothers are not able to provide enough breast milk to support their babies' needs.

Funded by the Temasek Foundation Cares, the $1.37 million three-year pilot programme aims to recruit approximately 375 healthy and eligible mothers, who are willing to donate their excess breast milk, to benefit 900 babies receiving neonatal care in the KK Women's and Children's Hospital, the Singapore General Hospital and the National University Hospital.

"Breast milk is the best form of nutrition for babies, containing white blood cells and antibodies that protect the baby against infections and improve their chances of survival. The fat globules in breast milk enable better brain and vision development.

This makes breast milk especially beneficial for premature and sick newborns, who have immature and weak digestive systems that make them prone to feeding intolerance and predisposes them to necrotising enterocolitis, which is potentially lethal.

Guest of Honour, Madam Halimah Yacob (centre) at the launch of Singapore's first Donor Human Milk Bank, with (from left) Assoc Prof Ng Kee Chong, Chairman, Medical Board, KKH; Prof Alex Sia, CEO, KKH; Mr Richard Magnus, Chairman, Temasek Foundation Cares; Prof Ivy Ng, Group CEO, SingHealth; Ms Woon Saet Nyoon, Chief Executive, Temasek Foundation Cares; and Dr Chua Mei Chien, Head and Senior Consultant, Department of Neonatology, KKH.

Providing safe, pasteurised donated breast milk to these vulnerable babies allows them to benefit from this ideal source of nutrition, while also significantly improving their chances of development and recovery," says Dr Chua Mei Chien, Director, Temasek Foundation Cares - Donor Human Milk Bank Programme, as well as Head and Senior Consultant, Department of Neonatology, KK Women's and Children's Hospital.

The first of its kind in Singapore, the KK Human Milk Bank will collect, screen, process and store breast milk received from donors, following strict international guidelines for laboratory testing, processing and storage of the pasteurised milk, before it is dispensed for use.

Eligible donors will be required to undergo a stringent donor screening process and blood tests, as well as to receive education on the handling and storage of the breast milk, prior to donation.

Every year, about 350 very low birth-weight infants receive neonatal intensive care in Singapore's public hospitals.

Despite the best efforts to support breastfeeding, up to 80% of sick neonates in the Neonatal Intensive Care Unit and Special Care Nursery receive formula milk meant for premature babies, either totally or partially, during their hospital stay. This is due to an inadequate supply of breast milk from their own mothers.

"As a Baby-Friendly Hospital Initiative-certified hospital, we are committed to improving neonatal and infant health, and supporting mothers in their endeavours to breastfeed by providing the necessary education before birth, imparting breastfeeding skills during their hospital stay and supporting them even after they are discharged.

This precious supply of ready, safe, pasteurised donor breast milk will be greatly beneficial in helping us to reduce the risk of potential complications in the babies, while optimising their immunity, development and overall health," adds Dr Chua.

DONOR ELIGIBILITY

Mothers whose baby is less than one-year-old, and who meet the following criteria, are eligible to donate their breast milk to the KK Human Milk Bank:

- Do not smoke
- Do not use illegal drugs or other prohibited substances
- Do not routinely consume more than two standard alcoholic drinks per day
- Do not routinely consume three or more cups of coffee, tea, or other caffeine-stimulant drinks per day, including cola and stimulant soft drinks
- Have not lived in or travelled to the UK between 1980 and 1996 for a total or cumulative period of six months
- Have not been tested positive for HIV, Hepatitis B and C or Syphilis

More information about the Temasek Foundation Cares - Donor Human Milk Bank Programme is available via the website: **https://www.kkh.com.sg/MilkBank/Pages/Home.aspx.**

Potential donors are encouraged to contact the KK Human Milk Bank at:
Tel: **6394 1986**
Email: **milkbank@kkh.com.sg**

Gender Does Make A Difference

Treating male patients with dyslipidaemia could be more challenging

Across the globe, women have shown consistently more favourable lipid profiles as compared to men. Locally, as well, the National Health Surveys have revealed the same since the 1990s.

While our lifestyle habits have been linked to this gender difference in several overseas population-based studies, a more recent study conducted by the Department of Research of SingHealth Polyclinics has brought to light some of these lifestyle behaviours in the local context. The study was published in the journal, *Proceedings of Singapore Healthcare*.

For instance, fewer Singaporean men were found to have attained their LDL-Cholesterol (LDL-C) Treatment Goals as compared to women. This appeared to have been associated with a greater reluctance to embark on dietary changes, unlike women, who tended to be more weight and health-conscious.

It was also found that men dined out more regularly, which could make it harder for them to make the necessary changes. In the study, a higher proportion of women were homemakers, which could place them in a better position with regard to their food choices.

Selecting more healthy options when dining out is the way to go, says Dr Tan Ngiap Chuan, the principal investigator of the study.

"But we do not suggest an abstinence from the less healthy food options. They can, instead, moderate their intake in terms of the quantity and its frequency."

Among the men, those of Chinese ethnicity and with lower educational levels were found to have better LDL-C control, as were non-smokers and non-drinkers. Both the activities of smoking and alcohol intake are known factors that lead to a rise in LDL-C.

"Therefore, primary care physicians should actively advise their patients to stop smoking, and to moderate their alcohol intake to no more than one unit per week, regardless of the type of alcohol consumed," said Dr Tan.

Interestingly, among women, those who perceived the lipid-lowering therapy to be expensive were less likely to achieve their LDL-C Treatment Goal. Physicians can help to overcome this barrier, by proactively raising the issue during their consultations, and by helping the affected patients obtain financial assistance.

"It is important that physicians recognise that it will be more challenging to manage their male patients with dyslipidaemia. A multi-faceted team-based approach may be one option, focusing on medication adherence and lifestyle changes," advised Dr Tan.

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Courses

Public Forum
National Neuroscience Institute
Parkinson's Disease

Date : 25 November 2017, Saturday
Time : 9.00 am – 11.00 am
Venue: Bishan Community Club, No. 51 Bishan Street 13, Singapore 579799

CME Points Accreditation: Nil | **Fees:** Free

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- The Importance of Exercise for People with Parkinson's Disease
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- Moving Together Towards a Brighter Tomorrow

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