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Movement Disorder and Parkinson's Disease

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Disclaimer

The Cover Shot

A tai chi group function is held yearly to promote public awareness of Parkinson's disease. The performers are PD patients and their relatives.

The photo is taken by a young-onset PD patient.

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Editorial

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Specialist in Neurology

Editor

Dr. Kin-lun TSANG

Movement disorder is a branch of neurology studying diseases on movements of the whole body or body parts. The abnormality can either be reduced or excessive movements. According to the symptomatology, the "hypokinetic" side can be akinesia, apraxia or rigidity. The "hyper-" side includes tremor, dyskinesia, dystonia, chorea, myoclonus or tics. With much interest in this subject, I decided to have the first ever local medical journal issue to focus on the classical examples of movement disorders, Parkinson's disease. I am delighted to have experienced and eminent neurologists in the field to contribute on the articles. Drug treatment is often under the spotlight and readers must have come across Parkinson's disease drug treatment in other journals. I have always been fascinated by the way the clinical diagnosis of movement disorder being made. Unlike a heart condition which is diagnosed by auscultation or an abdominal problem which is revealed by palpation, the symptoms and signs of movement disorder are often overt and visible. The showmanship of eliciting the correct signs is very appealing and satisfying. Though a clinical diagnosis is often accurate in experienced hands, sophisticated investigations are indispensable in this era of medicine. One article will enlighten us on the state-of-art imaging study. Two other articles cover the gait and local epidemiology of Parkinson's disease respectively. To illustrate the multi-disciplinary work of this specialty, the paper from neurosurgeons shows their important roles. Finally, the lighter corner of this issue touches on tai chi.

The Hong Kong Movement Disorder Society has gathered a host of academics and clinicians. It has passed infancy and is making strides in promoting research, professional knowledge exchange and public awareness of movement disorder. I hope this issue can serve as a stepping stone.

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△ Vascular mortality: dabigatran 150mg BID (no. of events=274) vs. warfarin (no. of events=317).

‖ Life-threatening bleeding was a subcategory of major bleeding that consisted of fatal bleeding, symptomatic intracranial bleeding, bleeding with a decrease in the hemoglobin level of at least 50 g per liter, or bleeding requiring transfusion of at least 4 units of blood or inotropic agents or necessitating surgery.

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PRA-08-2012-14

Neuroimaging for Parkinson's Disease

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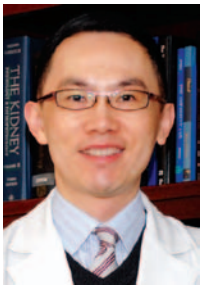

Dr. Anne YY CHAN Dr. Vincent MOK

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 October 2012.

Background

Idiopathic Parkinson's disease (PD) is the second most common neurodegenerative disease. In Hong Kong, the prevalence of PD is 0.186% and around 13000 people are suffering from PD. In 1817, James Parkinson described a phenomenon that we now believe is referring to parkinsonism in "An Essay on the Shaking Palsy". Nowadays, those patients who experience bradykinesia, resting tremor, rigidity and postural instability may often be labelled as having idiopathic PD. However, other parkinsonian-plus syndromes and secondary parkinsonian disorders may have overlapping clinical presentations with PD, making them difficult to be distinguished from PD. Neuroimaging can aid clinicians to make a more accurate pre-mortem diagnosis and thus correct and appropriate treatment could be provided.

MRI Brain

In the era before the molecular imaging with positron emission tomography (PET), clinicians depended on MRI brain to rule out structural lesions such as normal pressure hydrocephalus, which can be reversed by surgical drainage for patients who had parkinsonian features.

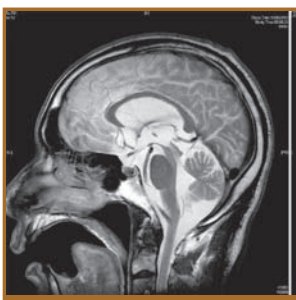

Figure 1. Midbrain atrophy with "Hummingbird" sign.

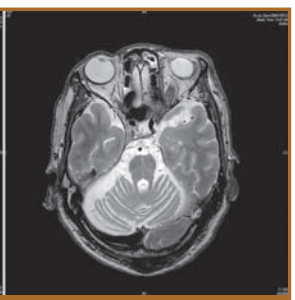

Figure 2. Pons atrophy with "HOT CROSS BUN" sign

Moreover, there are some patterns in MRI, which are specific to different parkinsonian-plus syndromes. Patients with progressive supranuclear palsy (PSP) will experience unsteady gait, frequent falls, impaired vertical gaze, bradykinesia and axial rigidity. The typical MRI brain findings in these patients appear to be a "hummingbird"[1,2] on the midsaggital cut of MRI scans due to midbrain atrophy (Figure 1). Patients with multiple system atrophy (MSA), however, will present with parkinsonism, autonomic dysfunction and ataxia. The typical MRI brain findings in these patients may include a 'hot cross bun' sign due to pons atrophy (Figure 2), cerebellar atrophy, putaminal atrophy and hypointensity, and/or slit-hyperintensity in the lateral margin of the putamen on T2-weighted sequences.[3]

Although these MRI features are specific, sensitivity of these features in the early stage of the disease is relatively low at around 50% only.[4]

PET Brain

Since the pathophysiology for neurodegenerative parkinsonism is related to dopaminergic neuronal loss, some radioligands, for example Fluorodopa PET, can help to assess the presynaptic nigrostriatal function. In Fluorodopa PET, the radioligand uptake will be reduced in the striatum especially pronounced in the posterior putamen for idiopathic PD. Note however that marked reduction of putaminal Fluorodopa uptake will also be found for both MSA and PSP as well. The next clinical question is "how can we differentiate idiopathic PD from MSA or PSP during the early stage of the disease especially when the signs and symptoms are subtle?" There are several ways to do so. For idiopathic PD, since there is up-regulation in the postsynaptic striatum in order to compensate for the presynaptic neuronal loss, there will be increased binding for putaminal region in 11C-Raclopride PET while there is reduced binding for MSA or PSP in this scan[5]. Besides molecular imaging for synaptic binding as mentioned above, glucose metabolism (FDG Brain PET) also helps to provide more information about PD, MSA, PSP and Dementia with Lewy Bodies (DLB). In fact, hypermetabolism noted over the dorsolateral putamen in FDG PET is highly suggestive of the diagnosis of idiopathic PD [5]. A recent study among the Chinese population in using FDG PET for differentiating idiopathic PD from parkinsonian-plus syndromes showed that the reduction of glucose metabolism on the bilateral putamen and cerebellum is indicative of MSA, while hypometabolism of the midbrain and mid frontal cortex are suggestive of PSP and hypometabolism in the bilateral occipital and parieto-occipital areas is characteristic of DLB.[6]

Clinical Application

How can we apply these neuroimaging techniques to our daily practices? The following case may help to illustrate the clinical utility of various PET techniques. A 58-year-old gentleman with a long-standing history of psychotic depression, requiring regular anti-depressants and anti-psychotics, presented with a one year history of progressive bradykinesia and cognitive decline, associated with visual hallucinations. The differential diagnoses were (1) sub-optimal control of his psychotic depression, which might require increasing dose of anti-psychotics; (2) drug induced parkinsonism, in which anti-psychotics might need to be tailed down; or (3) neurodegenerative parkinsonism (e.g. DLB), in which Ldopa drugs and/or acetylcholinesterase inhibitors might need to be considered. With this wide range of differential diagnoses, in which each had its different treatment approach, further neuroimagings might help to narrow down the possibilities. He subsequently underwent a PET scanning with both pre-synaptic (Fluorodopa), post-synaptic (11C-Raclopride) and also with glucose metabolism (FDG). The results showed that there were markedly decreased Fluorodopa uptake (Fig. A), moderate decreased 11C-Raclopride uptake (Fig. B) in bilateral basal ganglia which was indicative of impairment in the integrity of the striatonigral pathway; and there was decreased glucose metabolism over bilateral temporal-parietal and occipital regions in FDG PET (Fig. C). The overall clinical and radiological findings were compatible with DLB, requiring anti-psychotics for the visual hallucinations, low dose Ldopa for the parkinsonism and acetylcholinesterase inhibitors for the cognitive decline.

In summary, besides careful history taking and physical examination, neuroimaging can assist the clinicians to make an early and proper diagnosis for various parkinsonian syndromes and thus, correct treatment could be provided.

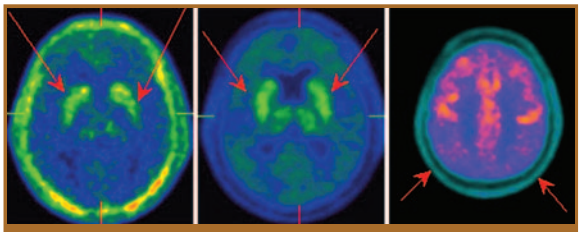

Fig. A : Markedly decreased DOPA uptake in Basal Ganglia

Fig. B : Decreased Raclopride uptake in Basal Ganglia

Fig. C : Decreased Glucose metabolism at bilateral occipital region at 18 FDG PET

References

1. Kato N, Arai K, Hattori T. Study of the rostral midbrain atrophy in progressive supranuclear palsy. J Neurol Sci. 2003;210:57–60.
2. Gröschel K, Kastrup A, Litvan I, Schulz JB. Penguins and hummingbirds: Midbrain atrophy in progressive supranuclear palsy. Neurology. 2006;66:949–50
3. Wenning GK, Colosimo C, Geser F, Poewe W. Multiple system atrophy. Lancet Neurol2004;3:93–103.
4. Brooks DJ, Seppi K; Neuroimaging Working Group on MSA. Proposed neuroimaging criteria for the diagnosis of multiple system atrophy. Mov Disord. 2009 May 15;24(7):949-64
5. Tatsch K. Positron emission tomography in diagnosis and differential diagnosis of Parkinson's disease. Neurodegener Dis. 2010;7(5):330-40.
6. Zhao P, Zhang B, Gao S. 18F-FDG PET study on the idiopathic Parkinson's disease from several parkinsonian-plus syndromes. Parkinsonism Relat Disord. 2012 Jan;18 Suppl 1:S60-2.

MCHK CME Programme Self-assessment Questions

Please read the article entitled "Neuroimaging for Parkinson's Disease" by Dr. Anne YY CHAN and Dr. Vincent MOK and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 October 2012. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. **Idiopathic Parkinson's disease is the commonest neurodegenerative disease.**
2. **MRI is diagnostic for Parkinson's disease.**
3. **Postural instability is a symptom of idiopathic Parkinson's disease.**
4. **A 'hot cross bun' sign on MRI is suggestive of multiple system atrophy.**
5. **MRI abnormalities are early in the course of parkinsonian-plus syndrome.**
6. **If PET is ordered for the diagnosis of parkinsonism, both dopa and glucose metabolism should be assessed.**
7. **Psychosis is a prominent feature in Dementia with Lewy Bodies disease (DLB).**
8. **11C-Raclopride PET is used to assess post-synaptic dopaminergic activity.**
9. **Parkinson's disease commonly runs in families.**
10. **Treatment response to Ldopa is equal in idiopathic Parkinson's disease and parkinsonism-plus syndrome.**

ANSWER SHEET FOR OCTOBER 2012

Please return the completed answer sheet to the Federation Secretariat on or before 31 October 2012 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Neuroimaging for Parkinson's Disease

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Answers to September 2012 Issue

Gilles de la Tourette Syndrome: Diagnosis and Medical Treatment

1. F **2.** T **3.** T **4.** F **5.** F **6.** T **7.** F **8.** T **9.** F **10.** T

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The Hong Kong College of Paediatricians (HKCPaed) and the Royal College of Paediatrics and Child Health (RCPCH) will hold a Joint Diploma in Child Health Examination in Hong Kong in 2013 awarding DCH (HK) and DCH (International) to successful candidates.

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Application: Candidates who wish to sit the examination in Hong Kong **MUST** apply through the Hong Kong College of Paediatricians (HKCPaed). For application details, please visit the HKCPaed website at www.paediatrician.org.hk/entcnews.htm or call the College Secretariat at 28718871.

Deadline for Application: Friday, 16 November 2012

Important Notice

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References:

1. Schlesinger N. Curr Rheumatol Rep 2010; 12(2):130-134. 2. Takano Y et al. Life Sci 2005; 76:1835-1847.
3. Becker MA et al. N Engl J Med 2005; 353(23):2450-2461.

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Tai Chi and Parkinson's Disease

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Dr. Kin-lun TSANG

In February this year, an article from the New England Journal of Medicine reported a study of tai chi in Parkinson's disease.[1] The study was led by Fuzhong Li of the Oregon Research Institute in Eugene, Oregon, USA. Tai chi was tested in 195 people with mild-to-moderate Parkinson's disease. The participants attended twice-weekly hour-long group classes of either tai chi or two other kinds of exercise – stretching and resistance training, which included steps and lunges with ankle weights and a weighted vest. After six months of classes, the tai chi group did significantly better than the stretching group in tests of balance, control, walking and other measures. Compared with resistance training, the tai chi group did better in balance, control and stride, and about the same in other tests. Tai chi training was better than stretching in reducing falls, and as effective as resistance training. The improvements in the tai chi group continued during three months of follow-up after ceasing the classes. Li said the study showed tai chi was safe. It is easy to learn, and there is no special equipment. "People are looking for alternative programmes, and this could be one of them," he said. The strength of the study was that there were objective measures rather than relying on the patients' own reports. But a placebo effect cannot be totally discounted since the intervention could not be blinded.

I have no experience with tai chi and after a bit of searching, I want to share what I gathered about this exercise.

The beginning of tai chi was unclear and there was a lot of legend about its history. Everyone has heard about Chang Sanfeng (張三豐 ,1247--?). He first studied at Shao-Lin, the buddhist temple and the source of nearly all the Chinese kung fu, for about 10 years. Then he travelled all over China, learned from Taoism and then settled in the Wudang Mountain. He was a great master and recluse in Chinese history, as respected as the founder of all inner martial art, which is called Wudang Chuan. Wudang and Shao-Lin are the two major kung fu styles in China, one is called inner kung fu, another outer. He initiated tai chi in his late years (but it is said he lived for 130 years). After he created tai chi, it was not publicly taught, but as an esoteric technique. This was passed on for several generations, and there were talent people in every generation, known or unknown by the public.

Tai chi is a type of low-impact, weight-bearing, and aerobic -- yet relaxing -- exercise. Its energy expenditure is 3 to 6 METs. The principle involves movements of the arms and body together. Many people can understand the hand movements, but do not see the body movement. Tai Chi is a circle. Many people can make the arm circles but are not relaxed enough to make the body circles. All movements are from a circle. The arm circle follows the body circle. Do not go outside of the circle. The body movement is a circle. Do not turn the body to the maximum, always leave a little. The body should turn 90 degrees. Never overturn. The body needs to relax first, then make the movement. When you turn the body the weight changes sides. If the body turns to the right, the left leg takes the weight. One leg is relaxed and one is not relaxed. When the body turns, the hands follow. If the hands alone move (without the body) the movement is incorrect. By moving the body, the hands can move further. The body has three parts. The middle part, between the navel and the hips must be loose and flexible and able to rotate. If the upper part is too strong, the middle will not be able to relax and move. If the legs are too weak, the weight will be in the top of the body and there will be no root. Strong legs are important in tai chi. As you get older, the legs are the first things to grow weak. When the body is relaxed qi goes first to the dantian (丹田), then to the legs and the feet. When the legs are strong, the back will be strong. Tai chi breathing is not upper chest breathing. When you breathe using

natural breathing, you are stronger. Tai chi develops the use of natural breathing and with continued practice, natural breathing will be used all the time and not just when practising tai chi. More practice builds up the qi in the dantian. The dantian becomes like a football. More qi makes the body stronger.

Qigong

Qigong, from which tai chi originates, is a discipline that involves the mind, breath, and movement to create a calm, natural balance of energy that can be used in work, recreation or self-defence. Like yoga, where many varieties have evolved, there are more than 3,000 varieties of qigong and five major traditions: Taoist, Buddhist, Confucian, martial arts, and medical, and two major types: "soft" and "hard". Soft qigong is called inner qigong, of which tai chi is an example.

Types of Tai Chi

Yang, wu, and tai chi chih are three of the most popular styles of tai chi. The yang style, which includes 24 movements in its simple form (108 movements in the traditional form), is demanding because you must keep your stance wide and your knees bent most of the time; the wu style, which includes 24 to 36 movements in its shorter form (100 movements in the traditional), is gentler because it uses a narrow, higher stance where the knees are not bent as much as the yang style; and the tai chi chih style, which has 20 movements, also uses a higher stance, but with much less transfer of weight from one leg to the other than the other two. Because the wu style uses a high, narrow stance, it may be easier to do and ideal for improving balance. All of them are conducted slowly, deliberately, and gracefully, with each movement flowing seamlessly into the next without hesitation.

What are the benefits of tai chi?

In China, it is believed that tai chi can delay ageing and prolong life, increase flexibility and strengthen muscles and tendons, and aid in the treatment of heart disease, high blood pressure, arthritis, digestive disorders, skin diseases, depression, cancer, and many other illnesses. There has not been enough scientific evidence to support all of these claims. The followings are some of the documented benefits.

Balance and Falling

Most of the research on tai chi has been done in older individuals in the area of balance and fall prevention. This area of research is important because fall-related injuries are the leading cause of death from injury and disabilities among older adults. The most serious fall injury is hip fracture; one-half of all older adults hospitalised for hip fracture never regain their former level of function. Because tai chi movements are slow and deliberate with shifts of body weight from one leg to the other in coordination with upper body movements (sometimes with one leg in the air), it challenges balance and one could speculate that it would help improve balance and reduce fall frequency. This has been shown in some research.[2,3]

Strength and Endurance

A recent study of adults in their 60s and 70s who practised tai chi three times a week for 12 weeks (60-minute classes) were given a battery of physical-fitness tests to measure balance, muscular strength and endurance, and flexibility before and after the 12 weeks. Statistically significant improvements were observed in all balance, muscular strength and endurance, and flexibility measures after six weeks, and they increased further after 12 weeks. The authors of the study concluded that tai chi is a potent intervention that improved balance, upper- and lower-body muscular strength and endurance, and upper- and lower-body flexibility in older adults.[4]

Aerobic Capacity

Aerobic capacity diminishes as we age, but research on traditional forms of aerobic exercise shows that it can be improved with regular training. In another meta-analytic study, researchers looked at seven studies focusing on the effects of tai chi on aerobic capacity in adults (average age 55 years). The investigators found that individuals who practised tai chi for one year (classical yang style with 108 postures) had higher aerobic capacity than sedentary individuals around the same age. The authors stated that tai chi might be an additional form of aerobic exercise.[5]

Fibromyalgia

Fibromyalgia (FM) is one of the most common musculoskeletal disorders and is associated with high levels of impaired health and painful symptoms that frequently flair up without relief. The cause of FM is unknown, and there is no known cure. In a study of 39 subjects with FM who practised tai chi twice weekly for six weeks (one-hour classes), it was found that FM symptoms and health-related quality of life improved significantly after the study.[6] This should be good news for individuals who suffer from this disorder.

Stress

The demands of living are stressful for adults of all ages. Although one cannot point to studies showing a reduction in stress from practising tai chi (although in one study subjects who practise tai chi did report that mental control was one of the benefits), the breathing, movement, and mental concentration required of individuals who practise tai chi may be just the distraction you need from your hectic lifestyle. The mind-body connection is one that deserves attention, as it has been reported that breathing coordinated with body movement and eye-hand coordination promotes calmness.

Some more reasons to practise tai chi:

- Movements are low-impact and gentle and put minimal stress on your muscles and joints.
- The risk of injury is very low.
- It can be done anywhere, anytime.
- It requires very little space (no excuses apartment dwellers!) and no special clothing or equipment.
- Can do it at your own pace.
- It's noncompetitive.
- It can be done in groups or by oneself.
- There are lots of movements to keep oneself interested.

How much tai chi should one do?

There is not enough research to suggest what the optimal dose of tai chi is to accrue benefits. Studies have shown effects with as few as one hour of training per week, although for many individuals two to three times per week, at least in the beginning, is probably an effective dose since there is a fair amount to learn (like learning how to dance). It is the conventional wisdom in tai chi circles that a person needs at least one year of tai chi before one becomes proficient.

What precautions should be taken before practising tai chi?

Tai chi is gentle enough for almost everyone. However, precautions should be taken for people with arthritis, orthopaedic conditions (back pain, sprains, fractures, and severe osteoporosis), pregnancy, hernia, or medical condition that might be affected by exercise.

Tai chi for PD patients in Hong Kong

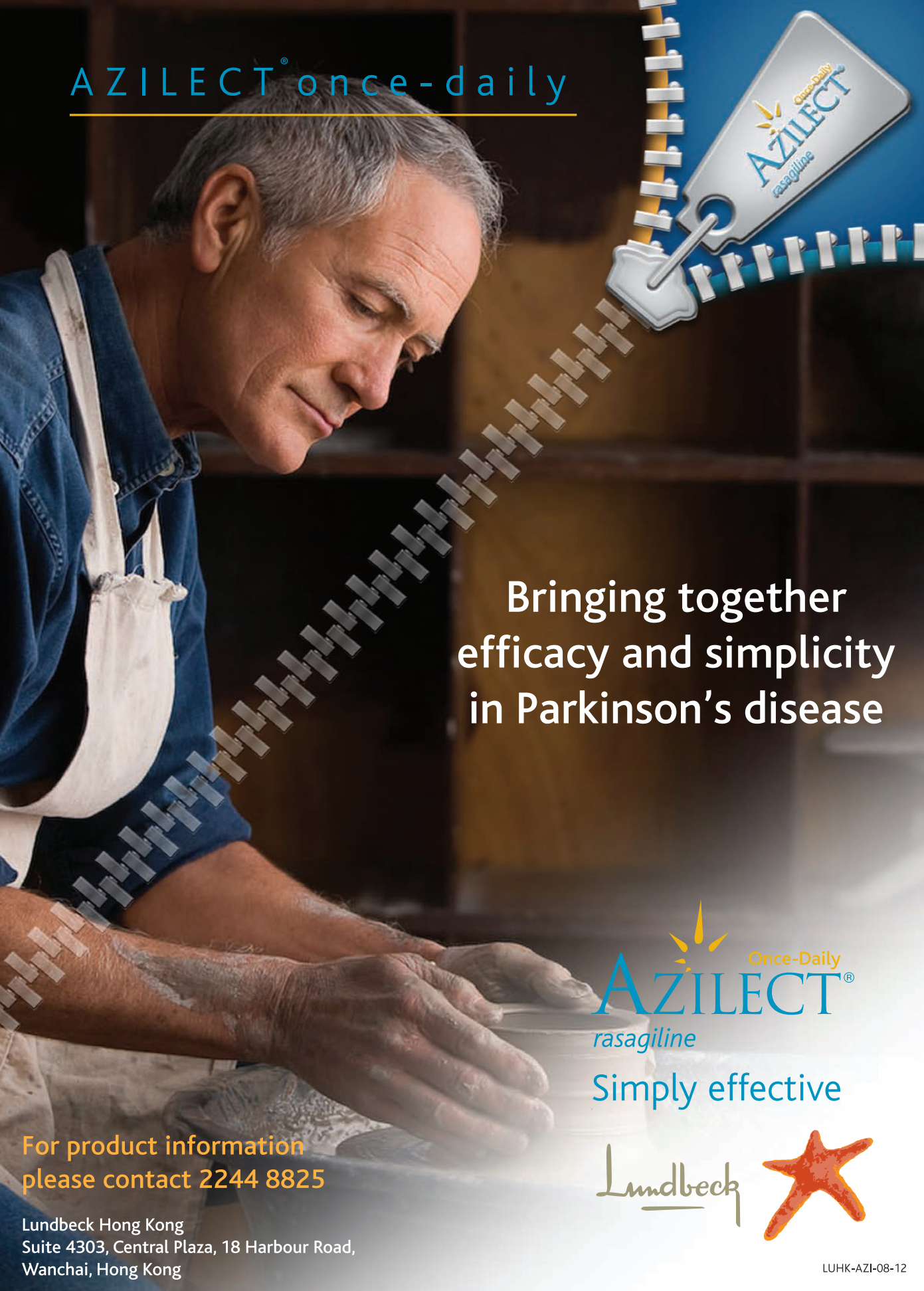
In Hong Kong, tai chi has been promoted as a regular exercise for Parkinson's disease patients. The Hong Kong Parkinson's disease Foundation and Hong Kong Tai Chi Association (香港太極總會) have jointly devised a Chang style tai chi involving 12 movements (鄭家太極十二式) tailored for PD patients. It is taught through a 10-session course by the Hong Kong Parkinson's Disease Association (a self-help group in affiliation with Rehabilitation Network) and is very welcomed by PD patients. Many patients continuously enroll into the course, not only to perfectionise their skills but to enjoy the moments with group practice. Every year there is a big event of group tai chi practice (愛心太極操) to promote the awareness of Parkinson's disease in Hong Kong. May I share the quotes from two patients who have benefited from regular tai chi exercises:

"In winter my joints used to be more stiff with weak legs. After I practised tai chi for 45-60 minutes everyday, my muscles are more supple, walking is easier and my legs can be lifted up more."

"I am 81-year-old. Every time when I practise tai chi, I use my diaphragm rather than the chest for breathing. The diaphragmatic breathing exercises my tummy and gut as well and I can feel the gut moving. It helps my constipation."

References

1. Li F, Harmer P, Fitzgerald K, Eckstrom E, Stock R, Galver J, et al. Tai chi and postural stability in patients with Parkinson's disease. N Engl J Med. 2012 Feb 9;366(6):511-9.
2. Guan H, Koceja DM. Effects of long-term tai chi practice on balance and H-reflex characteristics. Am J Chin Med. 2011;39(2):251-60.
3. Liu B, Liu ZH, Zhu HE, Mo JC, Cheng DH. Effects of tai chi on lower-limb myodynamia in the elderly people: a meta-analysis. J Tradit Chin Med. 2011 Jun;31(2):141-6.
4. Xu DQ, Hong Y, Li JX. Tai Chi exercise and muscle strength and endurance in older people. Med Sport Sci. 2008;52:20-9.
5. Taylor-Piliae RE, Froelicher ES. Effectiveness of Tai Chi exercise in improving aerobic capacity: a meta-analysis. J Cardiovasc Nurs. 2004 Jan-Feb;19(1):48-57.
6. Wang C, Schmid CH, Rones R, Kalish R, Yinh J, Goldenberg DL, et al. A randomized trial of tai chi for fibromyalgia. N Engl J Med. Aug 19;363(8):743-54.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Deep Brain Stimulation for Movement Disorders

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Introduction

Since the report of the first deep brain stimulation (DBS) for Parkinson's disease came in 1987 by the French group AL Benebid et al, DBS has been established as the standard therapy for advanced Parkinson's disease[1]. It opened up a new, exciting horizon in functional neurosurgery. The treatment is still advancing and being explored in other areas than Parkinson's disease, such as movement disorders, essential tremors and dystonia. It has become a brand new surgical invention. However, its efficacy demands high-quality teamwork and a high degree of surgical precision. Other determining factors include patient selection, selection of targets, pre-operative imaging, stereotactic targeting, electrophysiological monitoring, fixation of electrode and post-operative adjustment. These consume time from experts in the field, who strive to achieve good results.

Parkinson's Disease

Parkinson's disease (PD) is a progressive disabling movement disorder and is characterised by three cardinal symptoms: resting tremor, rigidity and bradykinesia as described by James Parkinson two centuries ago[2]. The prevalence of PD in the general population is reported to be 0.2% to 0.3%. Although PD is a neurodegenerative disease of the elderly, 5% to 10% of the patients have symptoms before the age of 40.

The mainstay of medical management is the treatment with Ldopa and dopamine agonists. However, many of them (30%-50%) will end up into medical refractory state in 5 to 10 years of medication[3]. Patients will suffer from motor fluctuation, unpredictable Off-state or "freezing". Patients will also develop Ldopa induced dyskinesia (LID) when the daily Ldopa requirement increases. Patients at this stage should be considered for deep brain stimulation.

The target of subthalamic nucleus (STN) is a popular target of choice in many centres for its comprehensive relief in the cardinal symptoms. The symptom of Ldopa induced dyskinesia (LID) is also relieved when the Ldopa requirement is lowered after DBS. Despite its small size, the subthalamic nucleus can be accurately targeted with MRI stereotaxy. Together with microelectrode recording and macrostimulation, the surgical team can achieve a high degree of accuracy and precision. The overall result from STN DBS in PD was very good with improvement of the UPDRS part III score of over 50% at off-medication stage[4]. In additional, the improvement would be sustained over 5 years[5]. However, there is no evidence, so far, of neuro-protection.

There is a revival of interest for the target of globus pallidus interna (GPi). A randomised control trial, which compared subthalamic nucleus (STN) DBS and globus pallidus interna (GPi) DBS in Parkinson's disease[6,7], showed similar improvement in motor function. However, one component (visuomotor processing speed) in the neurocognitive function of the secondary outcome measures had a larger decline in STN group than the GPi group[7]. The overall score in depression rating also worsened in STN, as compared to GPi group[7]. Many previous reports have the same observation that cognitive and behavioural complications were observed more frequently in STN stimulation[5,6,8,9]. However, the cost-effectiveness issue was raised for GPi stimulation because of higher battery drain and higher Ldopa requirement. So both STN and GPi are still considered the target of choice for motor improvement in Parkinson's disease. It will be up to the patient's specific condition and expertise of the centre to decide on the target.

Essential Tremors

A thalamic nucleus, Vim, is the target of choice for essential tremors. All patients with essential tremors who opt for DBS treatment will have electrodes targeted to the Vim. A few patients with "tremor dominant" Parkinson's may also opt for DBS of Vim, even though the thalamic stimulation will not help with any of the other symptoms of PD. Vim stimulation is thought to provide the best possible tremor control among the DBS targets. Tremor control with DBS of the Vim occurs almost instantly- within a few seconds. Common side effects of DBS of Vim include a "pins and needles" sensation caused by stimulation of the sensory nucleus (VPL) which is just posterior to the Vim.

Dystonia

Of all forms of dystonia, primary generalised dystonia (also known as idiopathic torsion dystonia) has been most thoroughly studied. The results have generally been very good for DBS in the target of globus pallidus interna (GPi). The consensus among most experts in the field is that patients with primary generalised dystonia and symptoms do not respond to medical therapy are good candidates for DBS surgery[10].

Many forms of the primary dystonia are believed to be hereditary and may be caused by genetic abnormalities. Of these, one gene—the DYT1 gene—has been identified and is responsible for many patients with the early onset of the primary generalised dystonia. This is an autosomal dominant disorder with 30% to 40% penetrance. It is a common cause for early onset primary dystonia in children. Onset age is at about 12 years old with symptoms over the arms and legs. It progresses over a few years, spreading to the trunk and neck while sparing the face. Treatment of GPi DBS is very effective with almost 100% response rate[11].

Cervical dystonia (also known as spasmodic torticollis) is a focal dystonia, which is also approved for DBS therapy. Cervical dystonia can be present as part of primary generalised dystonia or segmental dystonia. The latter is a type of dystonia involving two connected body parts. Patients with cervical dystonia that do not respond to other medical therapy are also qualified candidates for DBS[12].

DBS appears to work in some patients with secondary dystonia, but the results are less dramatic than those with primary dystonia. One reason for the varied response of DBS for patients with secondary dystonia is that secondary dystonia has several different causes, including stroke, trauma, infections, metabolic abnormalities, and cerebral palsy. As a result, the brain areas involved in causing the dystonia are also likely to differ. Therefore, DBS in the GPi may not affect the structure(s) that cause the dystonic movements in patients with secondary dystonia. The one likely exception is tardive dystonia (dystonia that is caused by dopamine receptor antagonists, i.e. neuroleptics) There is growing evidence of significant improvement in dystonic symptoms from DBS that appears to be comparable to the results in primary generalised dystonia[13].

Complications of Deep Brain Stimulation

The requirement of high-degree precision and the complexity of surgery in DBS have made it a complication-prone operation. Complications of DBS are classified under these categories[14]

Operation-related complications

Haemorrhage

Intracerebral haemorrhage is the most devastating complication. There is an average of 7 to 8cm passage of electrode from the cortical surface to the targets (i.e. Globus pallidus interna or subthalamic nucleus STN). Several passes of the test electrode are required during microelectrode recording (MER) and macrostimulation so as to confirm the position of the target. Haemorrhage risk increases with the number of passes[15]. The risk for a single pass is about 0.2-0.5%. The overall risk of intracerebral haemorrhage in DBS is low (1-1.5% unilateral procedure). Meticulous surgical planning would minimise the surgical risk. A direct-MRI or composite-MRI targeting should be able to bring the electrode tip to within 2 mm from the target. This would minimise the number of passes.

Modern stereotactic surgery planning systems are all powered to plan a "safe-track" by avoiding cortical veins and major arteries. A microdriver can drive the electrode down the track steadily and slowly at an increment of 0.1mm.

Malposition of electrode

Most of the DBS targets are small targets (STN). The quadripolar stimulation electrode has a diameter of 1.2mm. An optimal target should have good stimulation response and wide side-effect free stimulation range. Malposition of the electrode implies surgical failure. Stimulation may show no beneficial effect or may cause side-effects. Microelectrode recording (MER) is a technique of electro-physiological recording of cellular discharge in the cells in the basal ganglia. A super-fine tip (<10um) microelectrode is capable of recording single cellular discharge along the track. A specific pattern and frequency of discharge would be recorded at the target (i.e. for STN irregular pattern of discharge at 20-50Hz) By MER, the risk of malposition of the electrode is minimised.

Hardware-related complications

A DBS system composes of an implantable pulse generator (IPG), an extension electrode and a stimulation electrode (lead). An IPG is usually implanted in the infraclavicular region subcutaneously. It is fixed to the fascia of the pectoris major muscle with sutures. The extension electrode connects the IPG to a lead subcutaneously. There is a connector between the lead and the extension electrode. The connector is usually placed at the retroauricular region and it has to be fixed with sutures. The lead is secured and anchored to a burr hole cap or an anchorage device to prevent migration. Hardware-related complications can arise in any part of the system which includes fracture of the electrode, lead migration, infection of or erosion of the implant. The reported hardware complications were from 5% to over 20%[14].

Stimulation-related complications

Sensorimotor conditions

Stimulation-related complications or side effects may occur when the lead is stimulating the target or its vicinity. They are the commonest problems encountered in patients and most of them are reversible and adjustable. Stimulation at the vicinity of STN may result in diplopia (anteromedial) and contralateral limbs numbness or tonic muscles pulling (lateral). Speech disturbance, dysphonia or dysarthria were also reported after bilateral DBS of the pallidus or STN.

Psychiatric conditions

The first reported stimulation-related (bilateral STN DBS) psychiatric condition was transient acute depression[16]. However, more case reports showed manic episodes[17]. The manic symptoms are believed to be stimulated by a lead surpassing the substantia nigra reticulate.

Life threatening conditions

A severe brain damage after a dental procedure was reported in a patient with bilateral STN DBS[18]. A radiofrequency diathermy was employed for a dental procedure in this patient and the patient resulted in vegetative state. For patients with DBS, MRI scanning of the body, radiofrequency therapy and the use of monopolar diathermy are contra-indicated.

The expected life span of a battery at a typical voltage for Parkinson's patients is about five years. At a very high voltage for dystonia, the battery may need to be replaced every two years. Replacing a battery is similar to the replacement of a pacemaker which can be done under local anaesthesia. A rechargeable device is now available and its expected life span is 9 years. Patients are required to "recharge" the device through telemetry over the chest wall every 2 to 3 days.

Conclusion

Deep brain stimulation has gone through a long way, from a brand new explorative surgical procedure to a widely accepted and applied treatment. The indications of DBS is stretching the field across from movement disorders (i.e. essential tremor, generalised dystonia) to psychiatric conditions (obsessive compulsive disorder and depression), to epilepsy and pain. Clinicians and scientists are fascinated by DBS, knowing its neuro-modulation functions. The use of DBS will continue to flourish across functional neurosurgery. However, this complication-prone procedure requires a high degree of accuracy and precision. The surgical team should observe for this requirement and navigate the steep learning curve through a team approach.

References

1. Benabid AL, Pollak P, Louveau A, et al. Combined thalamotomy and stimulation sterotactic surgery of the VIM thalamic nucleus for bilateral Parkinson's disease. Appl Neurophysiol 1987;50:344-6.
2. Parkinson J. London Sherwood,Nealy and Jones. Essay on the shaking palsy 1817:16
3. Danny TM Chan, Vincent CT Mok, Wai S Poon, KN Hung, XL Zhu. Surgical management of Parkinson's disease : a critical review. Hong Kong Medical Journal 2001;7:34-9
4. Limousin P, Pollak P, Benazzouz A, et al. Effect on parkinsonian signs and symptoms of bilateral subthalamic nucleus stimulation. Lancet 1995;345:91-5.
5. Paul Krack, Alim-Louis Benabid (2003) Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson's disease. N Engl J Med 2003;349:1925
6. The Deep-Brain Stimulation for Parkinson's Disease Study Group (2001). Deep-brain stimulation of the subthalamic nucleus or the pars interna of the globus pallidus in Parkinson's disease. N Engl J Med. 2001;345:956
7. Kenneth A Follet, Frances M Weaver, Matthew Stern et al. Pallidal versus subthalamic deep-brain stimulation for Parkinson's disease. N Engl J Med 2010;362:2077-91
8. Valerie C. Anderson, Kim J Burchiel, Penelope Hogarth et al. Pallidal vs subthalamic nucleus deep brain stimulation in Parkinson disease. Arch Neurol. 2005;62:554-60
9. German Parkinson Study Group, Neurostimulation Section. A randomized trial of deep brain stimulation for Parkinson's disease. N Engl J Med 2006;355:896-908
10. Deep-brain stimulation for Dystonia Study group. Pallidal deep brain stimulation in primary generalized or segmental dystonia. N Engl J Med 2006;355:1978-90
11. Markun LC, Starr PA, Air EL, Marks WJ Jr et al. Shorter disease duration correlates with improved long-term deep brain stimulation outcomes in young-onset DYT1 dystonia. Neurosurgery 2012;71(2):325-30
12. Zelma HT Kiss, Kristina Doig-Beyaert, Michael Eliasziw et al. Functional and Stereotactic Section of the Canadian Neurosugrical Soceity and the Canadian Movement Disorders Group.The Canadian multicentre study of deep brain stimulation for cervical dystonia. Brain 2007;130:2879-2886
13. Caroline Andrews, Iciar Aviles-Olmos, Marwan Hariz, Thomas Foltynie. Which patients with dystonia benefit from deep brain stimulation? A metaregression of individual patient outcomes. J neurol Neurosurg psychiatry 2010;81:1383-89
14. Danny TM Chan, XL Zhu, Jonas Yeung et al.Complications of deep brain stimulation: A collective review. Asian Journal of Surgery 2009;30(4):258-63
15. Marwan I. Hariz (2002) Complications of deep brain stimulation surgery. Movement disorder 17(3):S162-S166.
16. Bejjani BP (1999) Transient acute depression induced by high-frequency deep brain stimulation. The New England Journal of Medicine 340(19):1476-1480
17. Kuliseveky J, Berthier ML, Gironell A, Pascual-Sedano B, Molet J, Pares P (2002) Mania following deep brain stimulation for Parkinson's disease. Neurology 59:1421-1424
18. Nutt JG, Anderson VC, Peacock JH, Hammerstad JP, Burchiel KJ (2001) DBS and diathermy interaction induces severe CNS damage. Neurology 56:1384-1386

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References:
1. Garcia-Alamino JM et al. Cochrane Database of Systematic Reviews, Issue 4, 2010.

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20 Nov	Changing mother baby friendly practice	Ms. Christine LAM Nurse Consultant Department of Obstetrics & Gynaecology Queen Elizabeth Hospital
27 Nov	Common Perinatal Psychiatric Disorders and the impact on Child Development	Dr. May Yin-king MIAO Private specialist in Psychiatry
4 Dec	Management of preterm labour - a continuing challenge	Dr. Wing-cheong LEUNG Chief of Service Department of Obstetrics & Gynaecology Kwong Wah Hospital
11 Dec	Comprehensive Child Development Service	Ms. Hoi-ying LAU Registered Nurse Midwife Department of Obstetrics & Gynaecology Princess Margaret Hospital
18 Dec	Genetics and common diseases in obstetric practice	Dr. Carman Wing-sze LAI Resident Specialist Department of Obstetrics & Gynaecology Queen Mary Hospital
	Prenatal genetic counselling	Dr. Anita Sik-yau KAN Associate Consultant Department of Obstetrics & Gynaecology Queen Mary Hospital

Date : 13 November 2012 – 18 December 2012 (Every Tuesday)
Time : 7:00 p.m. – 8:30 p.m.
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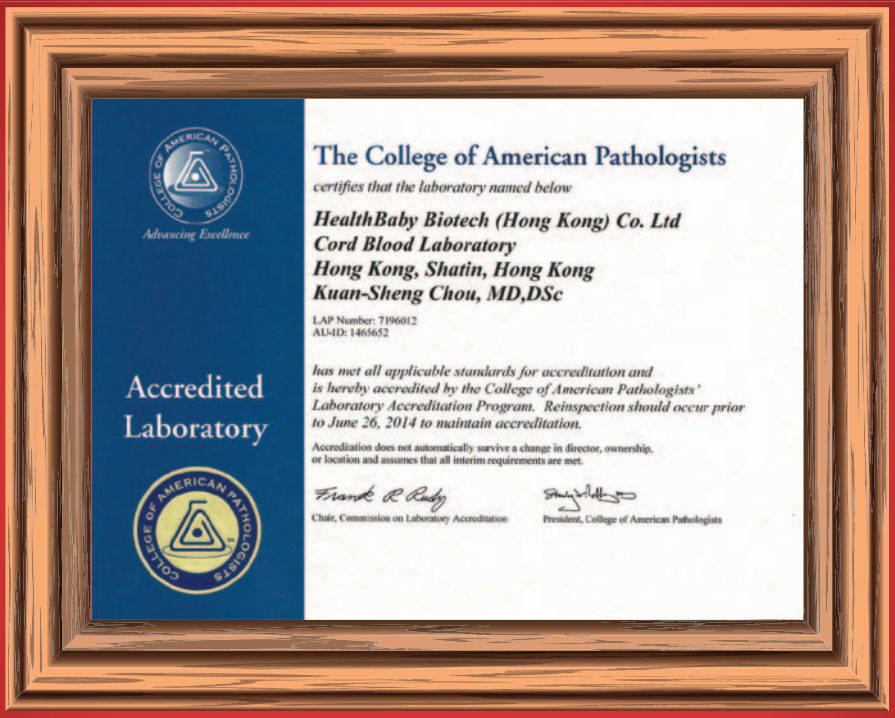

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10 Nov	Brain tumours	Dr. Gilberto Ka-kit LEUNG Assistant Professor, The University of Hong Kong Dr. Danny Tat-ming CHAN Associate Consultant, Prince of Wales Hospital
17 Nov	Cerebrovascular diseases	Dr. Kar-ming LEUNG Consultant, Kwong Wah Hospital Dr. Shing-chau YUEN Consultant, Tuen Mun Hospital
24 Nov	Common spine problems	Dr. Wai-man HUNG Associate Consultant, Tuen Mun Hospital Dr. David Tin-fung SUN Associate Consultant, Prince of Wales Hospital
8 Dec	Common paediatric neurosurgical problems	Dr. Xian-lun ZHU Consultant, Prince of Wales Hospital Dr. Kwong-yui YAM Consultant, Tuen Mun Hospital
15 Dec	Functional neurosurgery	Dr. Michael Wing-yan LEE Associate Consultant, Pamela Youde Nethersole Eastern Hospital Dr. Kwan-ngai HUNG Consultant, Queen Mary Hospital

Date : 3 November 2012 – 15 December 2012 (Skip 1 December, Every Saturday)
Time : 2:30 p.m. – 4:00 p.m.
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References:

1. Braun M, Cawello W, Poole K, Horstmann R. Steady-state pharmacokinetics of rotigotine in patients with early-stage Parkinson's disease. Poster presented at: 9th Congress of the European Federation of Neurological Societies; September 17–20, 2005; Athens, Greece.
2. Watts RL, Jankovic J, Waters C, Raiput A, Boroojerdi B, Rao J. Randomized, blind, controlled trial of transdermal rotigotine in early Parkinson disease. *Neurology*. 2007;68:272–276.
3. Giladi N, Mair KJ, Badenhorst F, et al, for the SP 826 Study Group. Effects of rotigotine transdermal patch on early morning motor function, sleep quality, and daytime sleepiness in patients with idiopathic Parkinson's disease. Results of a multi-center, multinational trial. Poster presented at: 10th Congress of the European Federation of Neurological Societies; September 2–5, 2006; Glasgow, UK.
4. LeWitt PA, Lyons KE, Pahwa R, for the SP 650 Study Group. Advanced Parkinson disease treated with rotigotine transdermal system: PREFER study. *Neurology*. 2007;68:1262–1267.

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Progression and Survival of Parkinson's Disease

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Parkinson's disease (PD) is a primary neurodegenerative disease that runs a relentlessly progressive course. Although it is amendable to symptomatic treatment, effective treatments that can alter the disease course are still lacking. There are ample data and evidences on the progression of various aspects on PD in Western studies. Local Chinese data are, however, sparse. Thorough understanding of its clinical course will not only help to better understand the disease, but have huge implications on treatment strategies. Motor and non-motor progressions as well as survival of PD patients, with stresses on their implications to future treatment strategies are discussed.

Motor Progression in PD

Various assessments have been developed to rate the severity of PD by measuring the motor manifestation and to assess the ability to perform daily functional activities.

Hoehn and Yahr (H&Y) staging is a scale assessing the combined features of motor impairment and disability. This staging is a well known and easily performed test, and its rating is based upon examination of the patient.

0 - no signs of disease
1 - unilateral disease (on one side)
1.5 - unilateral disease plus axial involvement
2 - bilateral disease, without impairment of balance
2.5 - bilateral disease, with recovery on the pull test
3 - mild to moderate bilateral disease; needs assistance to prevent falling on pull test; - physically independent
4 - severe disability, but still able to walk or stand unassisted
5 - wheelchair-bound or bedridden unless aided

H & Y stage less than 3 is usually classified as mild in severity, 3 is mild to moderate whereas stage 4 and 5 are those with advanced disease. Reviewing 171 newly diagnosed PD patients who were prospectively followed in our PD clinic[1], at baseline 165 patients were in H&Y stage 2.5 or less and no patient was in stage 4 or 5. After around 10 years' disease, only 67 patients remained at stage 2.5 or less. In other words, 104 (61%) patients were in stage 3 or above. Comparing with a Japanese study, they too found that after 10 years of Ldopa treatment 61% patients were in stage 3 or above.[2]

Previous studies showed that median latencies to stage 3 ranged from 3.5 to 11 years[3-5], while that to stage 5 ranged from 6 to 15 years. The discrepancies between studies are likely due to different study methodologies and heterogeneity of different PD cohorts studied.

Another widely used rating scale is the Unifying Parkinson's Disease Rating Scale (UPDRS). It scores mental state, activity of daily living, motor impairment and complications of therapy. Previous evidences showed that in early PD, there was a 8 to 14 points annual decline in total UPDRS scores(range 0-164)[6-8], while in more advanced PD, a 3 points[9] annual was noted. It seems that early PD has a faster progression. This is in line with studies using imaging ligands which showed an annualised rate of reduction in striatal markers of about 4% to 13% in patients with PD.[10-15]

A significant proportion of PD patients showed deterioration in motor impairment, which is more rapid in the initial phase of disease. Therefore in order to effectively alter the disease course, intervention should preferably be initiated at the earliest phase of the disease.

Motor Complications

Levo-dopa (Ldopa) remains the most potent and effective treatment for PD. But after the first years of honey moon period when Ldopa treatment are effective and without troublesome side effects, motor complications in the form of motor fluctuation and dyskinesia would appear. These not only cause disability but also limit the full use of Ldopa in control of PD motor symptoms. In our cohort, Ldopa exposure was noted in 169 (99%) patients, of whom 63.9% had motor dyskinesia and 75.1% suffered from motor fluctuation at around 10 years of treatment. Indeed approximately half the patients developed motor dyskinesia after 5 years of treatment studies[16-17]. Researches are ongoing for other treatment modalities with comparable potency, but better side effect profiles to Ldopa.

Non-motor Features

PD has been considered as a motor disease for a long time, and hence motor symptoms are primarily assessed in clinical studies. However, there is increasing awareness of the non-motor aspects of the disease, including cognitive decline, behavioural changes, sleep and autonomic dysfunctions.

After following a cohort of PD patients for 15 years, Hely[18] found that 48% of the 52 surviving patients had dementia, half of the patients had depression, and half had hallucinations. Thirty-five percent had orthostatic hypotension. Our Chinese cohort also revealed a similar picture[1]: after 11 years of longitudinal interval assessments, 83 of the 171 PD (49%) developed dementia, 81(47%) had psychosis and 103 (60%) had sensory complaints. Postural hypotension was found in 58 (34%) patients.

Some of the non-motor symptoms e.g. REM sleep behavioural disorder[19-21], hyposmia[22], constipation[23] frequently antedate the development of the classical motor symptoms in Parkinson's disease. Better understanding of the premotor evolution in PD is important in guiding the search for both risk and protective factors in PD.

Survival

Most western studies showed reduced mortality in PD patients in the post-Ldopa era than those patients in the pre-Ldopa era.[5] .Reported 10-years standard mortality ratio (SMR) in PD ranged from 0.9 – 1.8[24-29] Our Chinese cohort showed a 10 years SMR of 1.1, not significantly different from that of the general population. Since PD is a degenerative disease, it is believed that SMR would increase with disease duration. Indeed, other cohort studies have noted increasing SMRs over time, suggesting that advanced PD has a more negative effect on survival than early PD.[18,25,30]

Conclusion

Forty years after the introduction of Ldopa, it remains to be the most potent and effective treatment in PD patients. Ldopa has proven to be able to reduce disability in the first few years of disease, and improves survival. Indeed studies have shown that survival of the first 10 years in PD patients in the post-Ldopa era is approaching that of the general population, yet more than half of these patients progress into moderate to severe disease, facing various non-motor and motor features that are non-dopa responsive. In addition, more than half of the patients suffered from motor complications while on long-term Ldopa therapy.

Various lines of evidence showed that PD is not just a disease with motor impairment. Some of the non-motor symptoms appear before motor onset, making pre-motor diagnosis and identification of at risk patients possible. Since PD progression is fast in the initial phase, when prevention of further irreversible damage is possible, researches are now directed to identification of both the high risk groups in developing PD, and the finding of disease modifying agents.

References

1. M Auyeung, T H Tsoi, V Mok et al. 10 years survival and outcome in a prospective cohort of new onset Chinese Parkinson's disease patients. J Neurol Neurosurg Psychiatry 2012;83:607-611.
2. Sato K, Hatano T, Yamashiro K, et al. Prognosis of Parkinson's disease: time to stage III, IV, V, and to motor fluctuations. Mov Disord 2006; 21: 1384–95.
3. Hely MA, Morris J, Reid W, et al. The Sydney multicentre study of Parkinson's disease: progression and mortality at 10 years. J Neurol Neurosurg Psychiatry 1999;67:300-7.
4. J. Muller, G.K. Wenning, K. Jellinger Progression of Hoehn and Yahr stages in parkinsonism: a clinicopathologic study NEUROLOGY 2000;55:888–891
5. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. Neurology 1967;17:427e42
6. Parkinson Study Group. A controlled, randomised, delayed-start study of rasagiline in early Parkinson disease. Arch Neurol 2004;61: 561–66.
7. Parkinson Study Group. Effects of tocopherol and deprenyl on the progression of disability in early Parkinson's disease. The Parkinson Study Group. N Engl J Med 1993; 328: 176–83.
8. Parkinson Study Group. Effect of lazabemide on the progression of disability in early Parkinson's disease. The Parkinson Study Group. Ann Neurol 1996; 40: 99–107.
9. Guido Alves, Tore Wentzel-Larsen, Dag Aarsland et al. Progression of motor impairment and disability in Parkinson disease A population-based study. NEUROLOGY 2005;65:1436–1441
10. Morrish PK, Rakshi JS, Bailey DL, Sawle GV, Brooks DJ. Measuring the rate of progression and estimating the preclinical period of Parkinson's disease with
11. [18F]dopa PET. J Neurol Neurosurg Psychiatry. 1998;64:314-319.
12. Sawle GV, Colebatch JG, Shah A, et al. Striatal function in normal aging: implications for Parkinson's disease. Ann Neurol. 1990;28:799-804.
13. Parkinson Study Group. Dopamine transporter brain imaging to assess the effects of pramipexole vs levodopa on Parkinson disease progression. JAMA. 2002; 287:1653-1661.
14. Marek K, Innis R, vanDyck C, et al. [123I]_-CIT/SPECT imaging assessment of the rate of Parkinson's disease progression. Neurology. 2001;57:2089-2094.
15. Jankovic J, Kapadia AS. Functional decline in Parkinson's disease. Arch Neurol. 2001;58:1611-1615.
16. Holloway RG, Shoulson I, Fahn S, et al. Pramipexole vs levodopa as initial treatment for Parkinson disease: a 4-year randomized controlled trial. Arch Neurol 2004; 61:1044 –1053.
17. Rascol O, Brooks DJ, Korczyn AD, De Deyn PP, Clarke CE, Lang AE. A five year study of the incidence of dyskinesia in patients with early Parkinson's disease who were treated with ropinirole or levodopa. N Engl J Med 2000; 342:1484 –1491.
18. Hely M, Morris JGL, Reid WGJ, et al. Sydney multicenter study of Parkinson's disease: non L-dopa-responsive problems dominate at 15 years. Mov Disord 2005;20:190-9.
19. Wing YK. Li SX. Mok V. Lam SP. Tsoh J. Chan A. Yu MW. Lau CY. Zhang J. Ho CK.Prospective outcome of rapid eye movement sleep behaviour disorder: psychiatric disorders as a potential early marker of Parkinson's disease. J Neurol Neurosurg Psychiatry. 83(4):470-2, 2012 Apr
20. Schenck CH, Bundlie SR, Mahowald MW. Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep behaviour disorder. Neurology 1996; 46:388 –393.
21. Postuma RB, Lang AE, Massicotte-Marquez J, Montplaisir J. Potential early markers of Parkinson disease in idiopathic REM sleep behavior disorder. Neurology 2006;6:845– 851.
22. Henderson JM, Lu Y, Wang S, Cartwright H, Halliday GM. Olfactory deficits and sleep disturbances in Parkinson's disease: a case-control survey. J Neurol Neurosurg Psychiatry 2003 Jul;74(7):956–8.
23. Abbott RD, Petrovitch H, White LR, Masaki KH, Tanner CM, Curb JD, et al. Frequency of bowel movements and the future risk of Parkinson's disease. Neurology 2001 Aug 14;57(3):456–62.
24. Chen H, Zhang SM, Schwarzschild MA, et al. Survival of Parkinson's disease patients in a large prospective cohort of male health professionals. Mov Disord 2006;21:1002-7.
25. Hely MA, Morris J, Reid W, et al. The Sydney multicentre study of Parkinson's disease: progression and mortality at 10 years. J Neurol Neurosurg Psychiatry 1999;67:300-7.
26. Hughes TA, Ross HF, Mindham RHS, et al. Mortality in Parkinson's disease and its association with dementia and depression. Acta Neurol Scand 2004;110:118e23.
27. Herlofson K, Lie SA, Arsland D, et al. Mortality and Parkinson disease. A community based study. Neurology 2004;62:937e42.
28. Elbaz A, Bower JH, Peterson BJ, et al. Survival study of Parkinson disease in Olmsted County, Minnesota. Arch Neurol 2003;60:91e6.
29. Fall PA, Saleh A, Fredrickson M, et al. Survival time, mortality, and cause of death in elderly patients with Parkinson's disease: a 9-year follow-up. Mov Disord 2003;18:1312e16.
30. Diem-Zangerl A, Seppi K, Wenning GK, et al. Mortality in Parkinson's disease: a 20-year follow-up study. Mov Disord 2009;24:819e25.-increasiing SMR

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2:00pm-2:05pm	Opening Address	Prof. Shu-Leong Ho Co-Chair : Dr. Tsoi Tak Hong Dr. Leonard Li Sheung Wai
2:05pm-2:25pm	Current Understanding of Parkinson's Disease and Diagnosis	Prof. Shu-Leong Ho
2:25pm-2:45pm	Management of Early Parkinson's Disease	Dr. Jonas Yeung
2:45pm-3:05pm	Management of Late Parkinson's Disease and Long Term Outcome	Dr. Man Au Yeung
3:05pm-3:20pm	Q & A Section & Present Souvenir	
3:20pm-3:50pm	Tea Break	
3:50pm-4:10pm	Physical Therapy for Parkinson's Disease	Dr. Margaret Mak
4:10pm-4:30pm	Occupational Therapy for Parkinson's Disease	Ms. Connie Lee
4:30pm-4:50pm	Speech and Swallowing Therapy for Parkinson's Disease	Ms. Lorinda Kwan
4:50pm-5:05pm	Q & A Section & Present Souvenir	
5:05pm-5:10pm	Closing Remarks	Dr. Tsoi Tak Hong Dr. Leonard Li Sheung Wai

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Freezing of Gait

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Introduction

Freezing of gait (FoG) is a common disabling phenomenon in patients with advanced Parkinson's disease (PD). For example, in the Sydney Multicentre Study of Parkinson's disease (Hely *et al.*, 2008), 81% of the 20-year survivors suffered from freezing. Similarly, in a local 10-year prospective cohort of PD (Auyeung *et al.*, 2012), 87% had FoG and the median time for its development was 6 years. The importance of FoG is underscored by its association with falls and fractures (Bloem *et al.*, 2004). Moreover, FoG has a significant impact on the quality of life measured by the PDQ-39 (Moore *et al.*, 2007, Rahman *et al.*, 2008). In this article, we will briefly examine the phenomenology, pathophysiology and treatment options of FoG.

Phenomenology

FoG can be defined as "an episodic inability to generate effective stepping in the absence of any known cause other than parkinsonism or high-level gait disorders (Giladi and Nieuwboer, 2008)." It can happen in a number of situations. Patients can experience freezing when they initiate walking (*start hesitation*) and on turning (*turning hesitation*). In these cases, the patients are unable to initiate or maintain smooth locomotion, and their feet take short shuffling steps or even get "glued" to the floor. Besides, FoG is also commonly encountered when patients are about to reach a destination (*destination hesitation or destination freezing*). They might stop too early when they reach a chair or a wheelchair, in which they intend to sit down. FoG is more usual in certain stressful situations which limit time and space, e.g. walking through a crowded space (*tight quarter hesitation*), crossing a busy street at the green man signal, answering a doorbell, or entering an elevator when the door is about to close (Okuma, 2006). Usually more than one subtype of FoG co-exist in the same patient. Apart from walking, freezing phenomena can also affect speech and handwriting. One possible reason for clinicians to overlook the problem of FoG is that it appears more frequently at home than in the clinic office setting when the patients are being observed.

FoG is more commonly observed in the advanced stage of PD. In a study of FoG on 100 consecutive PD patients, significant association was found between the duration of disease and the presence of FoG (Lamberti *et al.*, 1997). FoG can develop before or after the commencement of Ldopa therapy. Moreover, the positive association between FoG and the duration of Ldopa therapy is not consistent (Giladi *et al.*, 1992, Lamberti *et al.*, 1997). FoG is more linked to the akinetic-rigid form of PD than the tremor predominant form. In those PD patients who suffer from motor fluctuations, FoG can be observed in both "off" and "on" states (Schaafsma *et al.*, 2003). In their study, most of the FoG episodes occurred during the "off" state, and the duration of the freezing episodes in the "on" state was significantly shorter than those in the "off" state.

Apart from PD, FoG has been reported in other neurological conditions (Factor, 2008). These include pure akinesia, progressive supranuclear palsy, multiple system atrophy, corticobasal ganglionic degeneration, dementia with Lewy bodies, vascular parkinsonism, post-encephalitic parkinsonism and normal pressure hydrocephalus. When severe FoG develops in the early stage of the disease, diagnoses other than PD should be excluded (such as progressive supranuclear palsy).

Pathophysiology

The human gait is regulated by cortico-basal ganglion-brainstem circuits which modulate central pattern generators (CPGs) in the spinal cord. CPGs are networks of neurons which are dispersed over several spinal segments and generate rhythmical movements of the legs. Supraspinal control then modifies this stereotyped locomotor pattern which is necessary for gait initiation, turning, termination etc. Neuroimaging techniques like positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have helped us to map out the abnormal circuitry involved in FoG. Imaging findings in PD patients with and without FoG suggest that FoG may emerge when altered cortical control of gait (under-activation of supplementary motor cortices, SMA) combines with a limited ability of the brainstem (mesencephalic locomotor region, MLR) to react to this alternation (Nutt et al., 2011).

Treatment

Treatment options of FoG include medications, rehabilitation strategies and deep brain stimulation.

Medications

Dopaminergic Agents
The classical belief is that, like many dopamine-resistant symptoms of PD with the involvement of pathways other than the nigrostriatal system, FoG does not

respond to conventional dopaminergic therapy. For example, as observed by Lamberti et al., 90% of their patients reported no changes of FoG after Ldopa therapy (Lamberti *et al.*, 1997). In fact, dopaminergic therapy may even worsen FoG. Espay et al. has demonstrated a worsening of "on" state FoG after Ldopa in a dose-dependent fashion from the "on" to the "supra-on" state (Espay *et al.*, 2012). Another study, however, has shown significant improvement of "off" state FoG in terms of duration and frequency with Ldopa treatment (Schaafsma *et al.*, 2003). They hypothesised that the pathophysiology of "off" state FoG is different from that of the "on" state. In the Earlier versus Later Ldopa Therapy in Parkinson Disease (ELLDOPA) study, Ldopa has delayed the development of FoG (Fahn, 2005). Based on the above findings, if FoG occurs only or predominantly in the "off" state, then we should treat it as a wearing off phenomenon by increasing the dose of dopaminergic agents. The treatment of "on" state FoG is less certain. Some patients respond to increased Ldopa dosages even if the optimal control of other parkinsonian signs has been achieved. However, others may respond to dose reduction of dopaminergic agents.

Selegiline

Selegiline is a monoamine oxidase type B inhibitor and frequently used in the management of PD. In the Deprenyl and Tocopherol Antioxidative Therapy of Parkinsonism (DATATOP) trial, 800 patients with early PD were randomised to placebo, selegiline alone, vitamin E alone, or a combination of selegiline and vitamin E. Selegiline was strongly associated with a reduced risk of FoG (Giladi et al., 2001). In the BLIND-DATE study, 368 subjects from the DATATOP cohort who were on both selegiline and Ldopa were re-randomised to either selegiline/Ldopa or placebo/Ldopa. After 2 years, the subjects on selegiline had less FoG (Shoulson et al., 2002). It is not known whether these benefits were due to symptomatic effects or some neuroprotective effects. Anyway, we should consider treating patients who are likely to develop FoG in the future (e.g. absence of tremor) with selegiline. Further studies are needed to examine the usefulness of selegiline in patients with established FoG.

Methylphenidate

The use of a non-dopaminergic approach in the treatment of FoG has been ongoing. One of these is methylphenidate (MPD). MPD is an amphetamine-like psychomotor stimulant that modulates both the dopaminergic and norepinephrine systems. Its benefits are so far controversial. On one hand, positive effects on walking speed have been demonstrated with low and high doses of MPD (Auriel et al., 2006, Devos *et al.*, 2007). On the other hand, a randomised, double blinded, placebo controlled study has failed to demonstrate gait improvement by MPD (Espay *et al.*, 2011). Therefore, we have to wait for more trials on the safety and efficacy of this drug before we can use it regularly in patients with FoG.

Rehabilitation Strategies

Both visual and auditory cues can alleviate FoG. A striking example of external cue is a remarkable preservation of ability to ride a bicycle in a PD patient with severe FoG (kinesia paradoxica) (Snijders and Bloem, 2010). Applying strips on the floor and inverted cane are common visual cues. Donovan et al. tested the utility of laserlight visual cues and found modest efficacy in overcoming FoG and reducing falls in PD patients (Donovan *et al.*, 2011). Alternatively, auditory cueing devices also provide improvement in walking speed, stride length and freezing. These devices can be easily incorporated into portable media players with minimal disruption of the daily routine of patients (Ledger *et al.*, 2008).

Deep Brain Simulation

Deep brain stimulation (DBS) to either the subthalamic nucleus (STN) or globus pallidus internus (GPi) can improve FoG. In some patients, the use of both Ldopa and STN stimulation can lead to additive effects. "On" state FoG can be alleviated indirectly by reduction of Ldopa dosage, which is usually achievable after DBS to STN (Ferraye *et al.*, 2008).

Certain strategies are now available to us to fine tune the stimulation parameters of DBS to achieve better results. Bilateral uncoordinated gait and marked gait asymmetry are associated with FoG (Plotnik *et al.*, 2005). By manipulation of the stimulation voltage to either STN, DBS can improve frequency and duration of FoG through normalisation of gait symmetry and coordination (Fasano *et al.*, 2011). Lower frequency stimulation (60 Hz, instead of the commonly used frequency of 130 Hz) to STN has been shown to alleviate FoG (Moreau *et al.*, 2008, Xie *et al.*, 2012).

In recent years, a new therapeutic target known as the pedunculopontine nucleus (PPN) has emerged for experiment. This nucleus is localised within the brainstem and is part of the mesencephalic locomotor region (MLR) which is involved in locomotion in both animals and humans. Debates are still ongoing concerning the efficacy, patient selection, target localisation and stimulation parameters (Ferraye *et al.*, 2010).

Conclusion

Freezing of gait is a major source of disability for PD patients and remains difficult to be treated. We now understand more about the pathophysiology underlying this condition. Future researches should focus on the specific targeted therapies and preventive strategies.

References

AURIEL, E., HAUSDORFF, J. M., HERMAN, T., SIMON, E. S. & GILADI, N. 2006. Effects of methylphenidate on cognitive function and gait in patients with Parkinson's disease: a pilot study. Clin Neuropharmacol, 29, 15-7.

AUYEUNG, M., TSOI, T. H., MOK, V., CHEUNG, C. M., LEE, C. N., LI, R. & YEUNG, E. 2012. Ten year survival and outcomes in a prospective cohort of new onset Chinese Parkinson's disease patients. J Neurol Neurosurg Psychiatry, 83, 607-11.

BLOEM, B. R., HAUSDORFF, J. M., VISSER, J. E. & GILADI, N. 2004. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Mov Disord, 19, 871-84.

DEVOS, D., KRYSTKOWIAK, P., CLEMENT, F., DUJARDIN, K., COTTENCIN, O., WAUCQUIER, N., AJEBBAR, K., THIELEMANS, B., KROUMOVA, M., DUHAMEL, A., DESTEE, A., BORDET, R. & DEFEBVRE, L. 2007. Improvement of gait by chronic, high doses of methylphenidate in patients with advanced Parkinson's disease. J Neurol Neurosurg Psychiatry, 78, 470-5.

DONOVAN, S., LIM, C., DIAZ, N., BROWNER, N., ROSE, P., SUDARSKY, L. R., TARSY, D., FAHN, S. & SIMON, D. K. 2011. Laserlight cues for gait freezing in Parkinson's disease: an open-label study. Parkinsonism Relat Disord, 17, 240-5.

ESPAY, A. J., DWIVEDI, A. K., PAYNE, M., GAINES, L., VAUGHAN, J. E., MADDUX, B. N., SLEVIN, J. T., GARTNER, M., SAHAY, A., REVILLA, F. J., DUKER, A. P. & SHUKLA, R. 2011. Methylphenidate for gait impairment in Parkinson disease: a randomized clinical trial. Neurology, 76, 1256-62.

ESPAY, A. J., FASANO, A., VAN NUENEN, B. F., PAYNE, M. M., SNIJDERS, A. H. & BLOEM, B. R. 2012. "On" state freezing of gait in Parkinson disease: a paradoxical Ldopa-induced complication. Neurology, 78, 454-7.

FACTOR, S. A. 2008. The clinical spectrum of freezing of gait in atypical parkinsonism. Mov Disord, 23 Suppl 2, S431-8.

FAHN, S. 2005. Does Ldopa slow or hasten the rate of progression of Parkinson's disease? J Neurol, 252 Suppl 4, IV37-IV42.

FASANO, A., HERZOG, J., SEIFERT, E., STOLZE, H., FALK, D., REESE, R., VOLKMANN, J. & DEUSCHL, G. 2011. Modulation of gait coordination by subthalamic stimulation improves freezing of gait. Mov Disord, 26, 844-51.

FERRAYE, M. U., DEBU, B., FRAIX, V., GOETZ, L., ARDOUIN, C., YELNIK, J., HENRY-LAGRANGE, C., SEIGNEURET, E., PIALLAT, B., KRACK, P., LE BAS, J. F., BENABID, A. L., CHABARDES, S. & POLLAK, P. 2010. Effects of pedunculopontine nucleus area stimulation on gait disorders in Parkinson's disease. Brain, 133, 205-14.

FERRAYE, M. U., DEBU, B. & POLLAK, P. 2008. Deep brain stimulation effect on freezing of gait. Mov Disord, 23 Suppl 2, S489-94.

GILADI, N., MCDERMOTT, M. P., FAHN, S., PRZEDBORSKI, S., JANKOVIC, J., STERN, M. & TANNER, C. 2001. Freezing of gait in PD: prospective assessment in the DATATOP cohort. Neurology, 56, 1712-21.

GILADI, N., MCMAHON, D., PRZEDBORSKI, S., FLASTER, E., GUILLORY, S., KOSTIC, V. & FAHN, S. 1992. Motor blocks in Parkinson's disease. Neurology, 42, 333-9.

GILADI, N. & NIEUWBOER, A. 2008. Understanding and treating freezing of gait in parkinsonism, proposed working definition, and setting the stage. Mov Disord, 23 Suppl 2, S423-5.

HELY, M. A., REID, W. G., ADENA, M. A., HALLIDAY, G. M. & MORRIS, J. G. 2008. The Sydney multicenter study of Parkinson's disease: the inevitability of dementia at 20 years. Mov Disord, 23, 837-44.

LAMBERTI, P., ARMENISE, S., CASTALDO, V., DE MARI, M., ILICETO, G., TRONCI, P. & SERLENGA, L. 1997. Freezing gait in Parkinson's disease. Eur Neurol, 38, 297-301.

LEDGER, S., GALVIN, R., LYNCH, D. & STOKES, E. K. 2008. A randomised controlled trial evaluating the effect of an individual auditory cueing device on freezing and gait speed in people with Parkinson's disease. BMC Neurol, 8, 46.

MOORE, O., PERETZ, C. & GILADI, N. 2007. Freezing of gait affects quality of life of peoples with Parkinson's disease beyond its relationships with mobility and gait. Mov Disord, 22, 2192-5.

MOREAU, C., DEFEBVRE, L., DESTEE, A., BLEUSE, S., CLEMENT, F., BLATT, J. L., KRYSTKOWIAK, P. & DEVOS, D. 2008. STN-DBS frequency effects on freezing of gait in advanced Parkinson disease. Neurology, 71, 80-4.

NUTT, J. G., HORAK, F. B. & BLOEM, B. R. 2011. Milestones in gait, balance, and falling. Mov Disord, 26, 1166-74.

OKUMA, Y. 2006. Freezing of gait in Parkinson's disease. J Neurol, 253 Suppl 7, VII27-32.

PLOTNIK, M., GILADI, N., BALASH, Y., PERETZ, C. & HAUSDORFF, J. M. 2005. Is freezing of gait in Parkinson's disease related to asymmetric motor function? Ann Neurol, 57, 656-63.

RAHMAN, S., GRIFFIN, H. J., QUINN, N. P. & JAHANSHAHI, M. 2008. Quality of life in Parkinson's disease: the relative importance of the symptoms. Mov Disord, 23, 1428-34.

SCHAAFSMA, J. D., BALASH, Y., GUREVICH, T., BARTELS, A. L., HAUSDORFF, J. M. & GILADI, N. 2003. Characterization of freezing of gait subtypes and the response of each to Ldopa in Parkinson's disease. Eur J Neurol, 10, 391-8.

SHOULSON, I., OAKES, D., FAHN, S., LANG, A., LANGSTON, J. W., LEWITT, P., OLANOW, C. W., PENNEY, J. B., TANNER, C., KIEBURTZ, K. & RUDOLPH, A. 2002. Impact of sustained deprenyl (selegiline) in Ldopa-treated Parkinson's disease: a randomized placebo-controlled extension of the deprenyl and tocopherol antioxidative therapy of parkinsonism trial. Ann Neurol, 51, 604-12.

SNIJDERS, A. H. & BLOEM, B. R. 2010. Images in clinical medicine. Cycling for freezing of gait. N Engl J Med, 362, e46.

XIE, T., KANG, U. J. & WARNKE, P. 2012. Effect of stimulation frequency on immediate freezing of gait in newly activated STN DBS in Parkinson's disease. J Neurol Neurosurg Psychiatry, 83, 1015-7.

Dermatological Quiz

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Private Dermatologist

Dr. Lai-yin CHONG

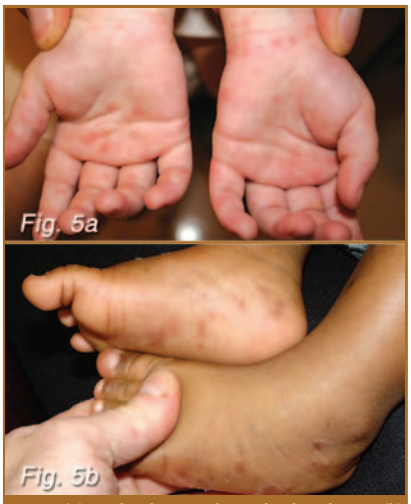

Fig. 5: (a) Multiple pustules at both palms & (b) Similar lesions and desquamation at both soles

This 1-year-old boy presented with recurrent multiple pustules over both palms and soles for 2 months (Fig. 5a & 5b). The lesions came in bouts with individual lesion lasting around 1-2 weeks. He was otherwise well and there was no significant family history. Pus swabs for bacterial and viral culture were negative. Complete blood picture showed eosinophilia. He was treated as Scabies but without any significant improvement. Parents were anxious about the pustulation and its recurrent nature.

Questions:

1. What is your provisional clinical diagnosis?
2. What are the differential diagnoses?
3. How do you manage this patient?

(See P.32 for answers)

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
	* HKMA Dragon Boat Team – CPA Cup – National Day Celebration Dragon Boat Invitational Race 2012 1	2	3	4	* HKMA Shatin Doctors Network - Latest Advances in Osteoporosis Management – What Doctor Should Know 5	* HKMA Dragon Boat Team Celebration Dinner cum CME Lecture 6
* HKMA Tennis Tournament 2012 7	* Breakthrough conjugate technology in the prevention of invasive pneumococcal disease in adults * Bilateral Ureteric Obstruction 8	* HKMA Kowloon West Community Network - Pneumococcal Disease and its Prevention in Adults * HKMA Yau Tsim Mong Community Network - Updates in NSAID-related Gastroduodenal Injury * New Clinical Evidence in Treatment of Heavy Menstrual Bleeding * BodyTalk Fundamentals Seminar Introductory Talk * FMSHK Officers' Meeting * HKMA Council Meeting 9	* Hong Kong Neurosurgical Society Monthly Academic Meeting- The Challenge of Neurological Rehabilitation for Neurosurgical Patients * HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 2) 10	* HKMA NTW Community Network - Certificate Course on Eye Diseases (Session 1) "New Cataract Extraction Technique with Femtosecond Laser" * HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2012 – The applications of health psychology in health care 11	12	* Infectious Diseases Seminar on "Travel and Infectious Diseases" * HKCS Annual Scientific Meeting - Advanced Management on Nocturia * Refresher Course for Health Care Providers 2012/2013 13
* Swimming Gala * HKMA Tennis Tournament 2012 14	15	16	* HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 3) 17	* FMSHK Executive Committee Meeting 18	* HKMA Kowloon City Community Network - Mitigating the Risk Associated with Diabetes Treatment 19	* Hong Kong Bench Press Championships 2012 * OSHK A to Z Symposia Series: "E"- symposium * HKMA CME – Health Personnel 2012 20
* HKMA Tennis Tournament 2012 21	22	23	* HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 4) 24	* HKMA NTW Community Network - Certificate Course on Eye Diseases (Session 2) "Glaucoma - Advances in Diagnosis and Management" * HKMA Hong Kong East Community Network – Update on Quadrivalent HPV Vaccine - Moving to HPV Gender Neutral Vaccination * FMSHK Foundation Committee Meeting 25	26	27
* HKMA Tennis Tournament 2012 28	29	30	* HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 5) 31			

Date / Time	Function	Enquiry / Remarks
1 MON 8:00 am	**HKMA Dragon Boat Team – CPA Cup – National Day Celebration Dragon Boat Invitational Race 2012** Organiser: Hong Kong Institute of Certified Public Accountants, Venue: Typhoon Shelter, Shaukeiwan	Miss Phoebe WONG Tel: 2527 8285
5 FRI 1:00 pm	**HKMA Shatin Doctors Network - Latest Advances in Osteoporosis Management – What Doctor Should Know** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Dr. WONG Chun Wa, Venue: Jasmine Room, Level 2, Royal Park Hotel	Ms. Evan LAW Tel: 8199 8970 2 CME points
6 SAT 7:00 pm	**HKMA Dragon Boat Team Celebration Dinner cum CME Lecture** Organiser: The Hong Kong Medical Association, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Miss Phoebe WONG Tel: 2527 8285
7 SUN 8:00 pm	**HKMA Tennis Tournament 2012** Organiser: The Hong Kong Medical Association, Venue: Kowloon Tong Club	Miss Phoebe WONG Tel: 2527 8285
8 MON 6:30 pm	**Breakthrough conjugate technology in the prevention of invasive pneumococcal disease in adults** Organiser: Hong Kong Medical Association & HK Society for Infectious Diseases, Speaker: Prof. Heinz Josef Schmitt, Venue: Regency Ballroom, Hyatt Regency Hotel, Tsim Sha Tsui	Ms. Sylvia HO Tel: 2963 5536 1.5 CME points
7:30 pm	**Bilateral Ureteric Obstruction** Organiser: Hong Kong Urological Association, Chairman: Dr. SC KWOK, Speaker: Dr. Y CHIU Yi, Venue: Multi-disciplinary Simulation and Skills Centre, 4/F, Block F, QEH	Ms. Tammy HUNG Tel: 9609 6064 1 CME point
9 TUE 1:00 pm	**HKMA Kowloon West Community Network - Pneumococcal Disease and its Prevention in Adults** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. WONG Wai Hong, Speaker: Dr. WONG Chun Kwan, Bonnie, Venue: Cyrstal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Candice TONG Tel: 2527 8285
1:00 pm	**HKMA Yau Tsim Mong Community Network - Updates in NSAID-related Gastroduodenal Injury** Organiser: HKMA Yau Tsim Mong Community Network, Chairman: Dr. LAM Tzit Yuen, David, Speaker: Dr. LAI Hin, Larry, Venue: Pearl Ballroom, Level 2, Eaton Smart, Hong Kong, 380 Nathan Road, Kowloon	Miss Candice TONG Tel: 2527 8285 1 CME point
1:45 pm	**New Clinical Evidence in Treatment of Heavy Menstrual Bleeding** Organiser: HKMA-Tai Po Community Network, Speaker: Dr. YEO Lee Kung, Evelyn, Venue: CHIU Chow Garden Restaurant, Shop 01-03, 1/F, Uptown Plaza, No. 9 Nam Wan Road, Tai Po	Mr. Manson CHUG Tel: 8200 2132 1 CME point
6:30-7:30 pm	**BodyTalk Fundamentals Seminar Introductory Talk** Certification Course for BodyTalk at Holistic Central, 13Fl, Asia Standard Tower, 59-65 Queen's Road, Central (www. Bodytalksystem.com.hk)	Ms. Angie TOURANI Tel: 6683 2755
8:00 pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. TSE Hung Hing, Venue: HKMA Head Office (5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
10 WED 7:00 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting- The Challenge of Neurological Rehabilitation for Neurosurgical Patients** Organiser: Thong Kong Neurosurgical Society, Chairman: Dr. Gilberto LEUNG, Speaker: Ms. Nerita CHAN, Venue: Seminar Room, Ground Floor, Block A, Queen Elizabeth Hospital	Dr. Gilberto LEUNG Tel: 2255 3368 1.5 CME points
1:30 pm	**HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 2)** Organiser: The Hong Kong Medical Association, Speaker: Dr. Raymond HF CHAN, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Ms. Viviane LAM Tel: 2527 8452 2 CME points
11 THU 1:00 pm	**HKMA NTW Community Network - Certificate Course on Eye Diseases (Session 1) "New Cataract Extraction Technique with Femtosecond Laser"** Organiser: HKMA NTW Community Network, Chairman: Dr. NGAI Pak Wai, Philip, Speaker: Dr. YIH Lai Bong, Jean Paul, Venue: Maxim's Palace Chinese Restaurant, Tuen Mun	Mr. Alan LAW Tel: 2527 8285
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2012 – The applications of health psychology in health care** Organiser: The Hong Kong Medical Association, Speaker: Dr. FUNG Shuk Man, Amy, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Ms. Viviane LAM Tel: 2527 8452 1 CME point
13 SAT 2:00 pm	**Infectious Diseases Seminar on "Travel and Infectious Diseases"** Organiser: The Hong Kong Medical Association, Chairman: Dr. Tse Hung Hing; Dr. So Man Kit, Thomas; Dr. TSANG Tak Yin, Owen, Speakers: Dr. FAN Pang Yung, Dr. Bonnie WONG, Dr. John Simon WINGATE & Dr. Sarah BORWEIN, Venue: Lecture theatre, 7/F, Block H, Princess Margaret Hospital, 2-10, Princess Margaret Hospital Road, Lai Chi Kok, Kowloon	Ms. Viviane LAM Tel: 2527 8452 2.5 CME points
2:00 pm	**HKCS Annual Scientific Meeting - Advanced Management on Nocturia** Organiser: Hong Kong Continence Society, Speakers: Dr. LEUNG Man Fuk & Dr. TONG Bing Chung, Venue: G/F, M Block, Queen Elizabeth Hospital	Mr. Calvin CHAN Tel: 2377 9801
2:30 pm	**Refresher Course for Health Care Providers 2012/2013** Organiser: The Hong Kong Medical Association, Speaker: Ms. Joey CHENG, Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME points
14 SUN 1:00 pm	**Swimming Gala** Organiser: The Hong Kong Medical Association, Venue: Michael Clinton Swimming Pool, Hong Kong Polytechnic University	Miss Phoebe WONG Tel: 2527 8285
8:00 pm	**HKMA Tennis Tournament 2012** Organiser: The Hong Kong Medical Association, Venue: Kowloon Tong Club	Miss Phoebe WONG Tel: 2527 8285

Date / Time	Function	Enquiry / Remarks
17 WED 1:30 pm	**HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 3)** Organiser: The Hong Kong Medical Association, Speaker: Ms. HO Ester, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Ms. Viviane LAM Tel: 2527 8452 2 CME points
18 THU 8:00 pm	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
19 FRI 1:15 pm	**HKMA Kowloon City Community Network - Mitigating the Risk Associated with Diabetes Treatment** Organiser: HKMA Kowloon City Community Network, Chairman: Dr. CHIN Chu Wah, Speaker: Prof. Alan SINCLAIR, Venue: Ballroom, Hyatt Regency Hotel, Tsim Sha Tsui	Ms. Candice TONG Tel: 2527 8285
20 SAT 11:30 am	**Hong Kong Bench Press Championships 2012** Organiser: Hong Kong Weightlifting and Power-lifting Association, Venue: Morse Park Sports Center, Kowloon	Miss Phoebe WONG Tel: 2527 8285
1:00 pm	**OSHK A to Z Symposia Series: "E"- symposium** Organiser: Osteoporosis Society of Hong Kong, Chairmen: Dr. Eddie CHOW & Dr. Anita KAN, Speakers: Dr. Andrew YY HO, Dr. William TSANG & Dr. TP IP, Venue: Star Room, Level 42, Langham Place Hotel, Mongkok	Ms Teresa MAN Tel: 2577 1922
1:30 pm	**HKMA CME – Health Personnel 2012** Organiser: The Hong Kong Medical Association, Chairman: Dr. Kwan YU, Speaker: Dr. Cindy CHAN Mei Yun, Venue: Lecture Theatre, G/F, Block F, UCH	Ms. Gary WONG Tel: 3513 4821 1.5 CME points
21 SUN 8:00 pm	**HKMA Tennis Tournament 2012** Organiser: The Hong Kong Medical Association, Venue: Kowloon Tong Club	Miss Phoebe WONG Tel: 2527 8285
24 WED 1:30 pm	**HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 4)** Organiser: The Hong Kong Medical Association, Speaker: Ms. CHENG Joey, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Ms. Viviane LAM Tel: 2527 8452 2 CME points
25 THU 1:00 pm	**HKMA NTW Community Network - Certificate Course on Eye Diseases (Session 2) "Glaucoma - Advances in Diagnosis and Management"** Organiser: HKMA NTW Community Network, Chairman: Dr. LEE Fook Kay, Aaron, Speaker: Dr. YUEN Shi Yin, Nancy, Venue: Maxim's Palace Chinese Restaurant, Tuen Mun	Mr. Alan LAW Tel: 2527 8285
1:00 pm	**HKMA Hong Kong East Community Network – Update on Quadrivalent HPV Vaccine - Moving to HPV Gender Neutral Vaccination** Organiser: HKMA Hong Kong East Community Network, Speaker: Prof. CHAN Kar Loen, Karen, Venue: HKMA Wanchai Premises,5/F, Duke of Windsor Social Services Building, 15 Hennessy Road, Wanchai	Miss Candice TONG Tel: 2527 8285
8:00 pm	**FMSHK Foundation Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
28 SUN 8:00 pm	**HKMA Tennis Tournament 2012** Organiser: The Hong Kong Medical Association, Venue: Kowloon Tong Club	Miss Phoebe WONG Tel: 2527 8285
31 WED 1:30 pm	**HKMA CME – The Hong Kong Medical Association Community Network Exercise Prescription Courses (Session 5)** Organiser: The Hong Kong Medical Association, Speaker: Prof. Stanley HUI/ Mr. Sam WONG, Venue: HKMA Central Premises, 2/F. Chinese Club Building, 21-22 Connaught Road Central, HK	Ms. Viviane LAM Tel: 2527 8452 2 CME points

Upcoming Meeting

1-4/11/2012	**BodyTalk Fundamentals Seminar** Certification Course for BodyTalk at White Lotus Center, 20/F, Car Po Commercial Bldg, 18-20 Lyndhurst Terrace, Central, Hong Kong (www.bodytalksystem.com.hk)	Mrs Angie TOURANI Tel: 6683 5855
2/12/2012	**2012 Paediatric Update No.3 – Paediatric Neurology** Organiser: Hong Kong College of Paediatricians, Chairmen: Dr. Sik Nin WONG & Dr. Shun Ping WU, Speakers; Dr. Shun Ping WU, Dr. Sheila WONG, Dr. Louis CK MA & Dr. Sophelia CHAN, Venue: Pao Yue Kong Auditorium, G/F, HK Academy of Medicine Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Hong Kong College of Paediatricians Tel: 2871 8773 3 CME points (Category A)

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the Family Medicine Division, Hong Kong Sanatorium and Hospital

2012

18/11

Cardiology

Updates on Management of Acute Coronary Syndrome
Dr. KWOK On Hing, Vincent
Director, Cardiac Catheterization and Intervention Centre
Director, Cardiology Centre
Hong Kong Sanatorium & Hospital

Diagnosis and Management of Common Cardiovascular Diseases in General Practice
Dr. HO Hung Kwong, Duncan
Honorary Consultant in Cardiology, Hong Kong Sanatorium & Hospital

Orthopaedics

Latest Development in the Treatment of Osteoarthritis of the Knee
Dr. WU Wing Cheung, Stephen
Head, Department of Orthopaedics & Traumatology
Director, Orthopaedic & Sports Medicine Centre
Hong Kong Sanatorium & Hospital

16/12

Urology

Updates on Management of Urolithiasis
Dr. WONG Wai Sang
Director, Urology Centre
Hong Kong Sanatorium & Hospital

Nephrology

Diagnosis and Management of Common Kidney Diseases
Dr. LAI Kar Neng
Director, Department of Health Assessment
Director, Nephrology Centre
Hong Kong Sanatorium & Hospital

2013

20/1

Endocrinology

Updates on Management of Type 2 Diabetes Mellitus and starting Insulin in Primary Care
Dr. TSUI Yee Ling, Elaine
Assistant Director, Department of Health Assessment
Co-Director, Endocrine & Diabetes Centre
Hong Kong Sanatorium & Hospital

Gastroenterology

Updates on Management of Irritable Bowel Syndrome
Dr. CHAN On On, Annie
Director, Gastroenterology & Hepatology Centre
Hong Kong Sanatorium & Hospital

17/2

Hematology

Updates on Management of Anemia
Dr. LIANG Hin Suen, Raymond
Head, Department of Medicine
Director, Comprehensive Oncology Centre
Director, Haematology Centre
Hong Kong Sanatorium & Hospital

Oncology

Updates on Management of Common Oncology Emergency
Dr. LEUNG Wai Tong, Thomas
Associate Director, Comprehensive Oncology Centre
Hong Kong Sanatorium & Hospital

17/3

Infectious Disease

Updates on Management of Tuberculosis
Dr. TANG Siu Fai, Bone
Honorary Consultant in Microbiology, Hong Kong Sanatorium & Hospital

Paediatrics

Updates on Management of Recurrent Urinary Tract Infections

Updates on Management of Febrile Convulsion
Dr. LOW Chung Kai, Louis
Honorary Consultant in Paediatrics, Hong Kong Sanatorium & Hospital

2013

21/4

Surgery

Updates on Management of Colonic Polyps
Dr. CHAN Chi Wai, Angus
Director, Endoscopy Centre
Associate Director, Surgery Centre
Hong Kong Sanatorium & Hospital

Plastic Surgery

Updates on Management of Burns, Wounds, Facial Lacerations and Scars
Dr. KING Wing Keung, Walter
Director, Plastic & Reconstructive Surgery Centre
Hong Kong Sanatorium & Hospital

19/5

Radiology

Recent Advances in Imaging for Ischemic Heart Diseases
Dr. CHAN Ka Fat, John
Honorary Consultant in Radiology, Hong Kong Sanatorium & Hospital

Recent Advances in Interventional Radiology
Dr. Al Ho Gong, Victor
Honorary Consultant in Radiology, Hong Kong Sanatorium & Hospital

Recent Advances in Imaging applicable to General Practice
Dr. LO GOH, Gladys
Radiologist-in-Charge, Department of Diagnostic & Interventional Radiology
Hong Kong Sanatorium & Hospital

Nuclear Medicine

Application of PET scan in Dementia
Dr. LEUNG Yim Lung, Eric
Deputy Director, Department of Nuclear Medicine & Positron Emission Tomography
Hong Kong Sanatorium & Hospital

Recent Advances in Nuclear Medicine Applicable to General Practice
Dr. WONG Ka Nin
Honorary Consultant in Nuclear Medicine, Hong Kong Sanatorium & Hospital

23/6

Allergy

A selection of Allergy cases
Dr. LEE Tak Hong
Director, Allergy Centre
Hong Kong Sanatorium & Hospital

Food Allergies and Applications of Skin Testing
Ms. June CHAN
Senior Dietitian, Allergy Centre
Hong Kong Sanatorium & Hospital

Dermatology

Dermatology Pictures Quiz
Dr. FUNG Ho Chuen, Alec
Honorary Consultant in Dermatology, Hong Kong Sanatorium & Hospital

Skin Manifestation of Systemic Diseases
Dr. WONG Hok Wai, Wendy
Honorary Consultant in Dermatology, Hong Kong Sanatorium & Hospital

21/7

Ophthalmology

Dry eyes & Eye Emergencies
Dr. LEUNG Yu Lung, Dexter
Honorary Consultant in Ophthalmology, Hong Kong Sanatorium & Hospital

ENT

Neck Mass - Look, Feel & then What?
Dr. WEI, William Ignace
Head, Department of Surgery
Director, Li Shu Pui ENT Head & Neck Surgery Centre
Hong Kong Sanatorium & Hospital

18/8

Respiratory

Updates on Management of Obstructive Lung Disease
Dr. LAM Bing
Director, Respiratory Medicine Centre
Hong Kong Sanatorium & Hospital

O&G

What alerts you that your Patient is Suffering from a Gynaecological Cancer?
Dr. TAM Kar Fai
Honorary Consultant in Obstetrics & Gynaecology, Hong Kong Sanatorium & Hospital

Venue	:	Auditorium, 4/F, 2 Village Road, Happy Valley, HK, Hong Kong Sanatorium and Hospital
Time	:	1:00 - 2:00pm (Lunch), 2:00 - 4:40pm(Lecture)
Fee	:	HK$300 for the whole course, HK$50 per lecture
Enquiries	:	2835 8800 (Tel), 2835 8008 (Fax), hospadm@hksh.com (Email)
Registration Form	:	Download from HKSH website - http://www.hksh.com/CME.pdf

*Rev. 6/8/2012

Answer to Dermatological Quiz

1. Infantile acropustulosis. This is a recurrent vesiculopustular eruption of the palms and the soles, typically begins between the first 2-12 months of life. Individual bouts of lesions last 1 to 2 weeks and recur in 2 to 4-week intervals. Apart from the skin, there is no visceral involvement. The aetiology is still unknown.

2. Scabies must be ruled out first before diagnosing infantile acropustulosis. In infants or young children, scabies can present in atypical forms with vesiculo-pustular and nodular lesions at the palms, soles and body folds. Other differential diagnoses include pompholyx (dyshidrotic eczema), palmoplantar pustular psoriasis, hand-foot-and-mouth disease, pustular impetigo and neonatal transient pustular melanosis.

3. Parents should be reassured of the self-limited and non-contagious nature. Almost all cases will resolve spontaneously before 3 years of age. Treatment is often unnecessary as most patients are asymptomatic. Topical steroids can be tried in more severe cases and oral antihistamines may be useful.

Dr. Lai-yin CHONG
MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)
Private Dermatologist

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- helps support **calcium absorption** [4-6] and **fat absorption** [7,8]
- together with Oligofructose (soluble dietary fibre) help support **GI Health** [9-11]

In a clinical trial, infants fed ILLUMA Stage 1 had **improved stool consistency** as compared to infants fed control formula, which was closer to that of infants fed with human milk [12]

The information is for Healthcare Professionals reference only. Further information is available upon request.

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Breastfeeding statement
Human milk is the best for babies. Infant formula is intended to replace human milk when mothers do not breastfeed. Good maternal nutrition is important for preparation and maintenance of breastfeeding. Introducing partial bottle feeding could negatively affect breastfeeding, and reversing a decision not to breastfeed is difficult. Professional advice should be followed on infant feeding. Infant formula should be prepared and used as directed. Unnecessary or improper use of infant formula may present a health hazard. Social and financial implications should be considered when selecting a method of infant feeding.

ILLUMA Stage 2 is a nutritious follow-on formula for babies six months to one year of age. ILLUMA Stage 2 is not a breast milk substitute. ILLUMA Stage 2 has been specially formulated for use as a nutritional supplement for the transition to the semi-solid and solid food portion of the older infant's diet.

Reference
1. Data on file. Wyeth Nutrition (Master Formulation #IRSP1P003). 2. Data on file. Wyeth Nutrition (Master Formulation #IRSP2P002). 3. Data on file. Wyeth Nutrition (Master Formulation #IRSP3P002). 4. Peacock M. Am J Clin Nutr. 1991;54:261S-265S. 5. Greer FR, Krebs NF, American Academy of Pediatrics Committee on Nutrition. Pediatrics. 2006;117(2):578-585. 6. Holick MF. J Nutr. 1996;126(4 Suppl):1159S-1164. 7. Kennedy K, et al. Am J Clin Nutr. 1999;70(5):920-927. 8. Lopez- Lopez A, et al. Early Human Development. 2001;65 Suppl:S83-S94. 9. Schneeman BO. J Nutr. 1999;129:1424S-1427S. 10. Data on file, Pfizer (Wyeth) Nutrition. Stool composition and stool characteristics in healthy term infants fed human milk or infant formulas (protocol 9055A1-3001-PH), CSR-79151, 01 Nov 2010. 11. Niness KR. J Nutr. 1999;129:1402S-1406S. 12. Yao M, et al. J Pediatr Gastroenterol Nutr. 2010 abs;50(Suppl2):E207-E208.

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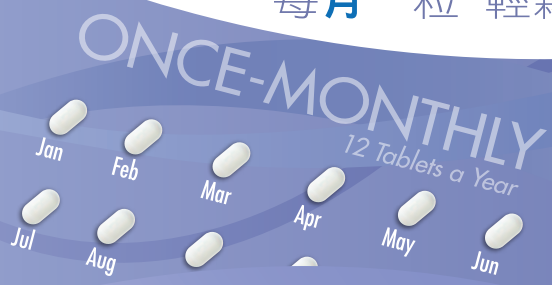
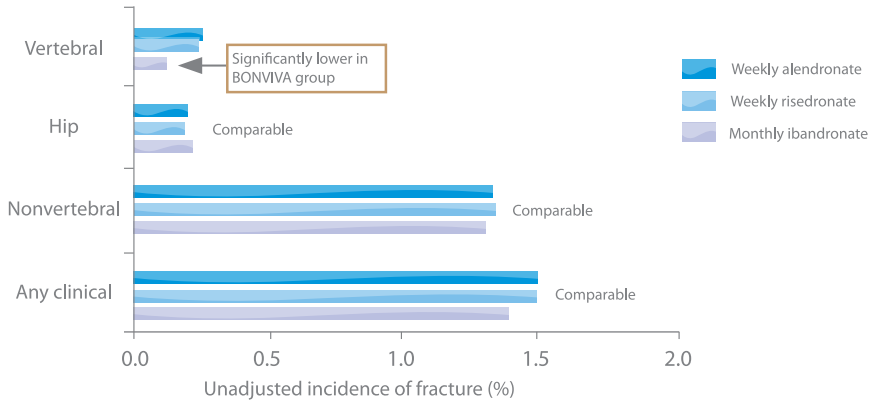


The VIBE* Study

Proven efficacy: Once-monthly Bonviva vs. weekly bisphosphonates (BP)

One year retrospective observational fracture study[1]



Bonviva-treated patients had statistically lower incidence of vertebral fractures.[1]

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significant lower risk vs. Alendronate (p=0.004)

BONVIVA
61%
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Ref:1. Bone. 2009;44:758–765.

Full prescribing information available upon request

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