



[www.fmshk.org](http://www.fmshk.org)

THE HONG KONG 香港醫訊  
*MEDICAL DIARY*

VOL.16 NO.7 JULY 2011

*Spasticity*



## Helping hands *around the world*



## Turning heads *with a* *wealth of publications*

**BOTOX**<sup>®</sup>: one of the world's most widely researched medicines<sup>1</sup>

### **BOTOX**<sup>®</sup>: a wealth of experience

- ▶ Treating serious and/or debilitating conditions for 20 years worldwide<sup>2</sup>
- ▶ Over 4000 clinical publications<sup>3</sup>
- ▶ More licensed indications than any other botulinum toxin:<sup>4-6</sup>
  - Strabismus and blepharospasm
  - Hemifacial spasm
  - Cervical dystonia
  - Dynamic equinus foot deformity due to spasticity in juvenile cerebral palsy
  - Wrist and hand disability due to upper limb spasticity associated with stroke
  - Severe axillary hyperhidrosis
  - Upper facial rhytides



#### References:

1. Allergan, data on file.
2. Meja N et al. Long-term botulinum toxin efficacy, safety, and immunogenicity. *Mov Disord* 2005;20(S1):592-597.
3. Search strategies are identical across all products. Searches are conducted on STN International<sup>®</sup> in the following databases: Medline<sup>®</sup>, Embase<sup>®</sup>, Embal<sup>®</sup>, Hcaplus<sup>®</sup>, Pascal<sup>®</sup>, Biosis<sup>®</sup>, Scisearch<sup>®</sup>. Data are reported separately from either peer-reviewed articles, letters, reports, or from congress abstracts, meeting posters. Results include clinical studies, case reports, reviews, and exclude trade magazines, news, book chapters.
4. BOTOX<sup>®</sup> Product Insert, Allergan HK, September 2009.
5. Dysport<sup>®</sup> Product Insert, Ipsen, June 2007.
6. Xeomin<sup>®</sup> Product Insert, Merz Pharma, January 2008.



## Contents

<b>Editorial</b>		<b>Radiology Quiz</b>	
■ Editorial	2	■ Radiology Quiz	27
<i>Dr. Dawson FONG</i>		<i>Dr. Vince LAU</i>	
<b>Medical Bulletin</b>		<b>Society News</b> 36	
■ Spasticity - Orthopaedic Perspectives	5	<b>Federation News</b> 32	
<i>Dr. Michael KT TO</i>		<b>Medical Diary of July</b> 34	
<i>Dr. W CHOW</i>		<b>Calendar of Events</b>	
■ MCHK CME Programme Self-assessment Questions	9	■ Course / Meeting 35	
■ The Medical Management of Cerebral Palsy- Paediatricians' Perspective	11		
<i>Dr. Mario Wai-kuong CHAK</i>			
■ Medical Treatment of Spasticity in Adults	16		
<i>Dr. Wai-man HUNG</i>			
■ Selective Dorsal Rhizotomy in Spastic Cerebral Palsy	21		
<i>Dr. Kwong-yui YAM</i>			
■ Physiotherapy in Spasticity Management for Children with Cerebral Palsy	24		
<i>Ms. Nerita NC CHAN</i>			
■ Computerised Gait Analysis	28		
<i>Ms. Miko LM LAO</i>			

## Disclaimer

All materials published in the Hong Kong Medical Diary represent the opinions of the authors responsible for the articles and do not reflect the official views or policy of the Federation of Medical Societies of Hong Kong, member societies or the publisher.

Publication of an advertisement in the Hong Kong Medical Diary does not constitute endorsement or approval of the product or service promoted or of any claims made by the advertisers with respect to such products or services.

The Federation of Medical Societies of Hong Kong and the Hong Kong Medical Diary assume no responsibility for any injury and/or damage to persons or property arising from any use of execution of any methods, treatments, therapy, operations, instructions, ideas contained in the printed articles. Because of rapid advances in medicine, independent verification of diagnoses, treatment method and drug dosage should be made.

## The Cover Shot



### Jamais Vu

The advent of the Mass Transit Railway (MTR) redefines commuting in Hong Kong since the late seventies. The first generation of subway carriage manufactured by Metrocammel - its full metallic, if not cold interior; clean and tidy if not monotonous ambience; its smoothness that one had to sit tight lest sliding from one end of the seat to the other - has become part of the collective memory for the generation at the time.

We all have packed in one of them but here it was void of person at all, exposing its pillars and posts, horizontal and vertical lining, its full uncompromising rigidity. It is a shot that I find little difference whether to take it in colour or B&W except for the higher ASA of the latter.

### Dr. Dawson FONG

MBBS(HK), FRCS(Edin), FCSHK, FHKAM(Surgery)  
Chief of Service and Consultant Neurosurgeon, Department of Neurosurgery, NT West Cluster





**Published by**  
The Federation of Medical Societies of Hong Kong

**EDITOR-IN-CHIEF**

Dr. MOK Chun-on  
莫鎮安醫生

**EDITORS**

Prof. CHAN Chi-fung, Godfrey  
陳志輝教授 (Paediatrics)  
Dr. CHAN Chun-hon, Edmond  
陳振漢醫生 (General Practice)  
Dr. KING Wing-keung, Walter  
金永強醫生 (Plastic Surgery)

**EDITORIAL BOARD**

Dr. CHAN Chi-kuen  
陳志權醫生 (Gastroenterology & Hepatology)  
Dr. CHAN Chi-wai, Angus  
陳志偉醫生 (General Surgery)  
Dr. CHAN Chun-kwong, Jane  
陳真光醫生 (Respiratory Medicine)  
Dr. CHAN Hau-ngai, Kingsley  
陳厚毅醫生 (Dermatology & Venereology)  
Dr. CHAN, Norman  
陳諾醫生 (Diabetes, Endocrinology & Metabolism)  
Dr. CHIANG Chung-seung  
蔣忠想醫生 (Cardiology)  
Prof. CHIM Chor-sang, James  
詹楚生教授 (Haematology)  
Dr. CHONG Lai-yin  
莊禮賢醫生 (Dermatology & Venereology)  
Dr. FAN Yiu-wah  
范耀華醫生 (Neurosurgery)  
Dr. FONG To-sang, Dawson  
方道生醫生 (Neurosurgery)  
Prof. HO Pak-leung  
何栢良教授 (Microbiology)  
Dr. KWOK Po-yin, Samuel  
郭寶賢醫生 (General Surgery)  
Dr. LAI Sik-to, Thomas  
黎錫滔醫生 (Gastroenterology & Hepatology)  
Dr. LAI Yuk-yau, Timothy  
賴旭佑醫生 (Ophthalmology)  
Dr. LAM Tat-chung, Paul  
林達聰醫生 (Psychiatry)  
Dr. LAM Wai-man, Wendy  
林慧文醫生 (Radiology)  
Dr. LEE Kin-man, Philip  
李健民醫生 (Oral & Maxillofacial Surgery)  
Dr. LEE Man-piu, Albert  
李文彪醫生 (Dentistry)  
Dr. LEUNG Kwok-yin  
梁國賢醫生 (Obstetrics & Gynaecology)  
Dr. LO See-kit, Raymond  
勞思傑醫生 (Geriatric Medicine)  
Dr. MAN Chi-wai  
文志衛醫生 (Urology)  
Dr. MOK, Mo-yin  
莫慕賢醫生 (Rheumatology)  
Dr. SIU Wing-tai  
蕭永泰醫生 (General Surgery)  
Dr. TSANG Wai-kay  
曾偉基醫生 (Nephrology)  
Prof. WEI I, William  
韋霖教授 (Otorhinolaryngology)  
Dr. WONG Bun-lap, Bernard  
黃品立醫生 (Cardiology)  
Dr. YU Chau-leung, Edwin  
余秋良醫生 (Paediatrics)

**Design and Production**

A-PRO MULTIMEDIA LTD www.apro.com.hk

# Editorial

## Dr. Dawson FONG

MBBS(HK), FRCS(Edin), FCSHK, FHKAM(Surgery)  
Chief of Service and Consultant Neurosurgeon,  
Department of Neurosurgery, NT West Cluster



Dr. Dawson FONG

### Editor

Medical knowledge and technology have grown so broad and deep that it is beyond any single person, no matter how outstanding to provide the best of treatment by oneself. In most instances, we need a team of collaborators, all experts in their own rights, working together.

Management of spasticity is a good example. Spasticity is a common sequela of neurological damage hampering daily activities and for children with cerebral palsy even moving around is a strenuous exercise let alone the social stigma that they have to live with all their lives. For adults recovering from trauma or strokes, spasticity is also a big problem they often encounter. If not well handled, spasticity might progress unchecked and eventually incarcerate its victims to the bed.

Physiotherapy is the mainstay of treatment but for some patients, medical or surgical intervention is needed during their rehabilitation. The selection of these patients calls for experience and expertise. Therefore a team looking after these patients requires specialists in the field of physiotherapy, gait laboratory study, neurology, developmental paediatrics, neurosurgery and orthopaedic surgery aligned to deliver a seamless care.

In this issue of the Medical Diary, there are six articles from various experts in the field – from the assessment, documentation of gait studies, formulation of treatment goals, selection for various treatment modalities and the treatment themselves. Readers will be revealed how these experts play their roles in a team and how their cooperation in a holistic approach would target spasticity and lead their patients to practicable goals along their course of neuro-rehabilitation.

I am thankful to all fellow authors for their contributions. The paper by Dr W Chow on 'Spasticity - Orthopaedic Perspectives' comes with questions for the purpose of CME.

Let me submit to you this July issue on Spasticity that I am sure you would find it delightful and informative reading.



NYCOMED

When life gets complicated...

Duodenal ulcer<sup>2,3</sup>, gastric ulcer<sup>2,3</sup>, moderate  
& severe forms of reflux esophagitis<sup>2,3</sup>,  
prevention of gastroduodenal ulcers  
induced by non-selective non-  
steroidal anti-inflammatory drugs  
(NSAIDS) in patients at risk  
with a need for continuous  
NSAID treatment<sup>1</sup>,  
Zollinger-Ellison  
Syndrome<sup>2,3</sup>.



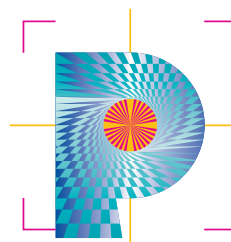
...there's one simple solution



Trusted in  
Gastroenterology

Nycomed (Hong Kong) Limited

Address : Units 3706-3708, 37/F AIA Tower,  
183 Electric Road, North Point, Hong Kong  
Telephone : 2133 9800 Fax : 2856 2728  
[www.nycomed.com](http://www.nycomed.com)



**PANTOLOC**<sup>®</sup>

pantoprazole  
40 mg / 20 mg tablets, 40 mg IV

For further information, please consult full prescribing information.  
References: 1. Package insert of PANTOLOC 20mg tablet.  
2. Package insert of PANTOLOC 40mg tablet.  
3. Package insert of PANTOLOC 40mg I.V.

HK/DM/PAN/02-04-2011

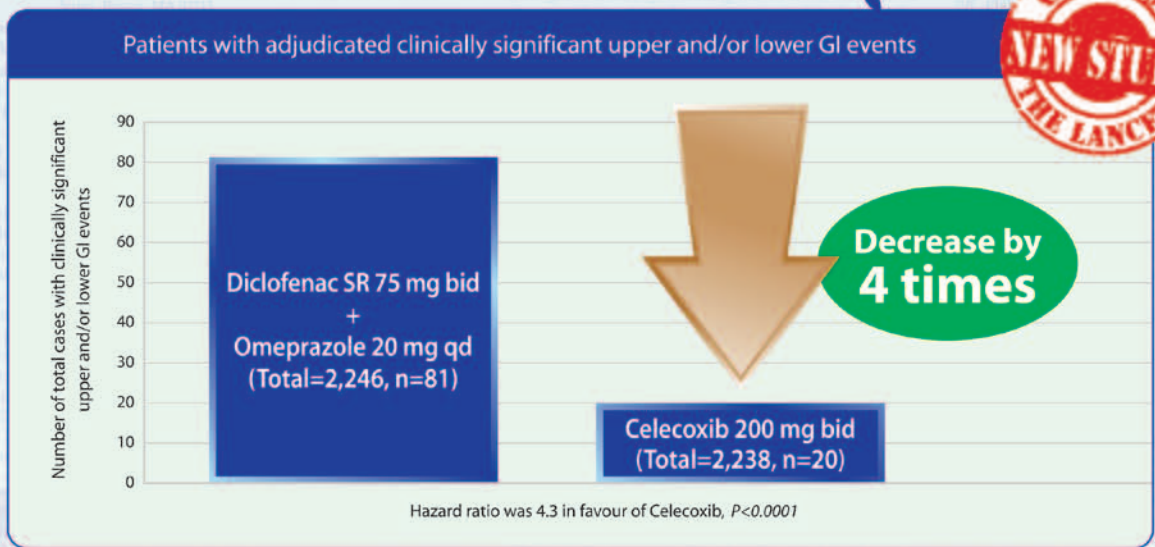
The Uncomplicated PPI

# CONDOR

Celecoxib versus Omeprazole and Diclofenac  
in patients with Osteoarthritis and Rheumatoid arthritis

## Celecoxib vs. Diclofenac + Omeprazole:

Patients treated with Celecoxib had a **LOWER RISK** of Clinically Significant Upper and/or Lower GI Events<sup>1</sup>



- Proven efficacy in various pain models<sup>2-5</sup>
- The Only FDA-approved COX-2 inhibitor<sup>6</sup>
- Option for millions of patients for over 10 straight years<sup>7</sup>



Strengthening confidence across Asia FOR OVER 10 YEARS

**CELEBREX**  
(CELECOXIB)

References: 1. Chan FKL, Lanas A, Scheiman J, Berger MF, Nguyen H, Goldstein JL. Celecoxib versus omeprazole and diclofenac in patients with osteoarthritis and rheumatoid arthritis (CONDOR) a randomized trial. *J Rheumatol*. 2010;37:167-174. 2. Graham DV, Chan FK. *Gastroenterology* 2008;134:1240-1257. 3. Nadarajah A, et al. *Singapore Med J* 2006; 47:534. 4. Petri M, et al. *J Rheumatol* 2004;31:1614-20. 5. Cheung R, et al. *Clin Ther* 2007;29 [Theme Issue]:2498-2510. 6. Food and Drugs Administration (FDA). <http://www.fda.gov>. Accessed February 10, 2011. 7. About Celebrex 2010. Available at URL <http://www.celebrex.com/about-celebrex.aspx>. Accessed Feb 10, 2011.

**CELEBREX ABBREVIATED PACKAGE INSERT** 1. TRADE NAME: Celebrex. 2. PRESENTATION: Capsules contain either 100 mg, 200 mg or 400 mg of celecoxib. 3. INDICATIONS: Adult: For relief of the signs and symptoms of osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, management of acute pain, treatment of primary dysmenorrhea. 4. DOSAGE: For more dosage information, please refer to the full package insert. 5. CONTRAINDICATIONS: Hypersensitivity to celecoxib, aspirin or other NSAIDs or demonstrated allergic-type reactions to sulfonamides or experienced asthma, urticaria, or allergic-type reactions after taking aspirin or other NSAIDs. Use as treatment for perioperative pain in the setting of CABG surgery. 6. WARNINGS & PRECAUTIONS: Increased risk of serious adverse cardiovascular thrombotic events: myocardial infarction, and stroke, which can be fatal; Patients with known CV disease or risk factors for CV disease may be at greater risk. Caution in patients with hypertension, fluid retention or heart failure; Can cause serious gastrointestinal events including bleeding, ulceration, and perforation of the stomach, small intestine or large intestine, which can be fatal. Extreme caution in patients with a prior history of ulcer disease or gastrointestinal bleeding; Special care should be taken as most spontaneous reports of fatal GI events are in elderly and debilitated patients; A patient with symptoms and/or signs suggesting liver dysfunction, or in whom an abnormal liver test has occurred, should be monitored carefully for evidence of the development of a more severe hepatic reaction. If clinical signs and symptoms consistent with liver disease develop, or if systemic manifestations occur, Celebrex should be discontinued. Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Renal toxicity has also been seen in patients in whom renal prostaglandins have a compensatory role in the maintenance of renal perfusion; not recommended in these patients with advanced renal disease. Anaphylactoid reactions have occurred in patients without known prior exposure to Celebrex. Should not be given to patients with the aspirin triad. This symptom complex typically occurs in asthmatic patients who experience rhinitis with or without nasal polyps, or who exhibit severe, potentially fatal bronchospasm after taking aspirin or other NSAIDs. Can cause serious skin adverse events such as exfoliative dermatitis, Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal. These serious events can occur without warning and in patients without prior known sulfa allergy. Patients should be informed about the signs and symptoms of serious skin manifestations and use of drug should be discontinued at the first appearance of skin rash or any other sign of hypersensitivity. Cannot be expected to substitute for corticosteroids or to treat corticosteroid insufficiency. Should not be administered to patients with aspirin-induced asthma and should be used with caution in patients with preexisting asthma. Has not been shown to reduce the risk of gastrointestinal cancer or the need for prophylactic coxibicy or other FAP-related surgeries in FAP. Therefore, the usual care of FAP patients should not be altered. The pharmacological activity in reducing inflammation, and possibly fever, may diminish the utility of these diagnostic signs in detecting infectious complications of presumed non-infectious, painful conditions. The concomitant use with any dose of a nonaspirin NSAID should be avoided due to the potential for increased risk of adverse reactions. 7. INTERACTIONS: ACE inhibitors and angiotensin II antagonists, aspirin, fucoxazole, furosemide, lithium, NSAIDs, warfarin and drugs that inhibit cytochrome P450 2C9. Potential interaction with drugs that are metabolized by P450 2C8. 8. PREGNANCY AND LACTATION: Pregnancy Category C; Pregnancy category D from 30 weeks of gestation onward because it may cause premature closure of the ductus arteriosus; Should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus, and should be avoided during the third trimester of pregnancy. Effects on labor and delivery in pregnant women are unknown. Caution should be exercised in nursing women. 9. SIDE EFFECTS: Abdominal pain, Dizziness, Dyspepsia, Flatulence, Nausea, Stool pain, Peripheral edema, Heavy menstrual, Disorientation, Headache, Insomnia, Pharyngitis, Rhinitis, Sinusitis, Upper respiratory infection, Rash. Reference: HK P (Jan 2005). Date of preparation: Nov 2010. FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.



Pfizer Corporation Hong Kong Limited

16/F, Stanhope House, 73B King's Road, North Point, Hong Kong

Tel: (852) 2811 9111

Fax: (852) 2579 0599

Website: [www.pfizer.com.hk](http://www.pfizer.com.hk)



## Spasticity - Orthopaedic Perspectives

**Dr. Michael KT TO**

FRCSEd (Ortho), FHKCOS, FHKAM (Orthopaedic Surgery)

**Dr. W CHOW**

FRCSEd (Ortho), FHKCOS, FHKAM (Orthopaedic Surgery)



Dr. Michael KT TO

Dr. W CHOW

*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 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 July 2011. The CME accreditation is in application. The number of CME credit is subject to the final decision of the organisations.*

### Background

Children who suffer from spastic cerebral palsy will have increased muscle tone due to hyperexcitability of the myotonic reflex arc. Together with the poor selective motor control, these primary abnormalities will drive the development of secondary deformities in the muscle (e.g. hamstring and gastrocnemius contracture) and in the skeleton (e.g. excessive femoral anteversion and external tibial torsion)<sup>1</sup>. It is not uncommon to see patients with spastic cerebral palsy developing scoliosis, hip adduction contracture, windswept deformity, knee flexion contracture, and rocker bottom foot deformity. The patients will have problems with mobility and posture as a result of these deformities.

The magnitude of spasticity changes as the patients mature. Usually, the spasticity is most severe at 4 years of age and gradually decreases up to the age of 12<sup>2</sup>. Therefore, it is very crucial to monitor the effects of the spasticity on the development of the affected children. Various treatments have been used to reduce the spasticity in patients with spastic cerebral palsy. Botulinum (Botox) and baclofen are commonly used medical therapies and selective dorsal rhizotomy is a very effective surgical method in reducing spasticity. Despite all these, orthopaedic surgical interventions are quite often needed for the management of gait abnormality, hip instability, joint contractures and ankle deformities<sup>3-5</sup>.

### Orthopaedic Surgery for Spastic Cerebral Palsy Patients – Multidisciplinary Approach

The role of orthopaedic surgeries in spastic cerebral palsy patients is to (1) reduce the effect of the spasticity of the muscles and (2) correct secondary bony deformities. The surgery should help to improve the walking ability for the potential walkers and sitting posture for the sitters. The indications of the surgeries have to be set clearly with the parents and caretakers before the surgeries in order to avoid any unrealistic expectations and misunderstanding. The keys to success in managing spastic cerebral palsy patients are (1) proper patient selection, (2) identification of

the underlying problems, (3) selection and execution of surgery, and (4) rehabilitation. Different bony and soft tissue surgeries have been described in managing spastic cerebral palsy. The choice of the surgery depends on the severity of the spasticity, selective motor control, age, pattern of the paralysis and types of deformities. Thorough and detailed assessment is very important since the patients' condition may fluctuate from time to time. In order to make the assessment more objective and comprehensive, a multidisciplinary approach, including the orthopaedic surgeons, physiotherapists and occupational therapists, is adopted in the pre-operative assessment. The assessment is often supplemented by modern gait analysis whenever possible. The team should be aware of the limitations of the patients and set reachable goals for them when deciding on the choice of treatment. The post-operative rehabilitation provided by nurses, physiotherapists, occupational therapists, and orthotists also play a very important role in helping these children to reach their full potential.

### Gross Motor Function Classification

The Gross Motor Function Classification System (GMFCS) is a commonly used classification to help classify the disabilities of children with cerebral palsy. It was first used for children from age 12 months to 12 years based on the observation of a child's self movements and need for assistive technology and/ or wheeled mobility<sup>6</sup>. The GMFCS was revised, expanded and further validated to include children up to the age of 18<sup>7</sup>. The GMFCS has five levels in which level I children can walk and run independently; whereas, level V children have very limited voluntary movements. GMFCS is a very useful treatment and prognostic guide for managing cerebral palsy children<sup>8-10</sup>. In general, orthopaedic surgery aims for gait modification for level I-III children and postural improvement for level IV and V children.

### Modern Gait Analysis and Surgery

For GMFCS I-III, the aim of the orthopaedic surgery is for gait improvement. Careful analysis of the abnormal gait pattern is very important. Apart from careful



clinical examinations, more detailed quantitative information can be acquired using computer-based gait analysis. Such motion analysis consists of 3-dimensional measurements of motion (kinematics), measurements of moments and power in the articulations of the lower limbs (kinetics), electromyography, dynamic foot pressure (pedobarography) and oxygen consumption measurement. It helps the clinicians to understand the interaction of the selective motor control, balance and spasticity of the patients during walking. More importantly, the effects of the secondary changes such as the lever-arm dysfunction of the lower limbs as well as the tertiary changes e.g. compensatory or coping mechanism for the primary and secondary abnormalities can be clearly delineated. The records can also be used for monitoring the progress of the disease as well as the outcome of the surgery.

## Timing of Surgery

In the past, CP children were commonly treated surgically every year until they reached skeletal maturity. Tendon lengthening was one of the most commonly performed surgeries on these children. Due to repeated hospitalisation and prolonged immobilisation, such "birthday surgeries" are no longer welcome. Today, single-event multilevel surgery (SEMLS), addressing all concomitant joint contractures in a single surgery, is advocated. Since the abnormality of one joint also affects the position of other joints, SEMLS corrects all the related deformities in one surgery and helps shorten the rehabilitation period. This also avoids repeated hospitalisation and overcorrection of the abnormalities. Since the skeleton continues to model as the child grows, it is advisable to defer any gait modification surgery until the child is relatively more skeletally mature e.g. after the age of 7. However, in situations like progressive hip subluxation<sup>11,12</sup> or severe joint contracture limiting the original walking potential in some children, early soft tissue release should be considered before the deformity progresses further.

## Gait Modification Surgery

The parents should be aware that gait modification surgery can change the gait pattern of their children but not to make it normal. Identifying common gait abnormalities, recognising their causes and understanding the interaction between the soft tissue and bony abnormalities are the keys to success in gait modification surgery. The biarticular muscles are more commonly affected in spastic cerebral palsy patients e.g. rectus femoris, hamstrings and gastrocnemius. Due to the biarticular involvement, they can result in very complex gait abnormalities<sup>13</sup>. There are four common gait abnormalities of the knees in cerebral palsy patients - jump knee, crouch knee, stiff knee, and recurvatum knee patterns<sup>14</sup>. Jump knee gait is quite frequently seen in spastic diplegic patients due to overactive hamstrings in the presence of tight or spastic gastrosoleus complex. Crouch knee gait can be related to the weakness or overlengthened triceps surae, external tibial torsion and or rocker bottom feet that disrupt the normal ankle plantarflexion knee extension couple. Stiff knee gait is caused by inappropriate phasic activity of the

rectus femoris resulting in excessive knee extension throughout the swing phase. Lastly, recurvatum knee gait is caused by spastic and contracted triceps surae with weakened hamstrings leading to hyperextension of the knee during the middle and late stance phase. Therefore, understanding the interaction of different muscles during walking is essential in the decision-making of gait improvement surgery.

## Lever-arm Dysfunction

Normal movement of a joint relies on the normal moment ( $M$ ) of a muscle joint complex, which is the product of muscle force ( $F$ ) multiplied by the lever arm ( $d$ ). Lever-arm dysfunction in cerebral palsy refers to the disruption of this moment generation because of abnormal development of the skeleton despite normal muscle force. In cerebral palsy, the skeleton develops differently because of the abnormal forces acting onto it and very often results in shortened lever arm. Since the moment ( $M$ ) =  $F \times d$ , the already weakened muscle force and shortened lever arm will produce ineffective moments. Depending on the site of the abnormal lever arm, the gait pattern will be affected accordingly e.g. coxa valga and excessive femoral anteversion will bring about ineffective lever arm at the hip leading to Trendelenburg gait. Five types of lever-arm dysfunction were described<sup>1</sup>: (1) short lever-arm (coxa valga), (2) flexible lever-arm (pes valgus), (3) malrotated lever-arm (external tibial torsion), (4) an abnormal pivot or action point (hip subluxation or dislocation), and/or (5) positional lever-arm dysfunction (crouch gait).

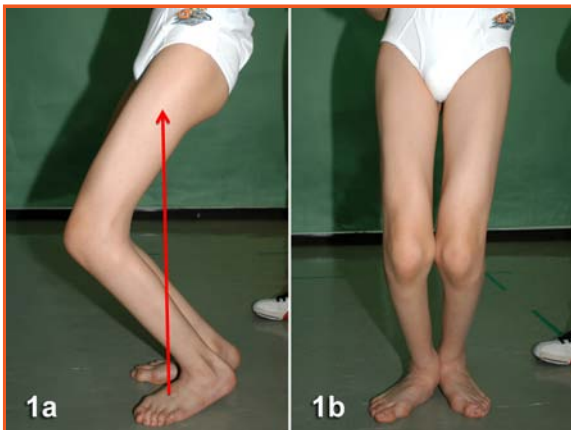
Abnormal moment of a joint can also affect the action of the neighbouring joints. This can be illustrated by the plantar flexion / knee extension (PF-KE) couple at the knee and ankle. With a competent soleus muscle to slow down the forward momentum of the tibia in the stance phase, the ground reaction force is maintained in front of the knee. This generates an extension moment at the knee without any additional action of the quadriceps. However, such PF-KE couple is disturbed in many spastic diplegic cerebral palsy patients because of the weakened triceps surae and lever-arm dysfunction. The malrotated lever arm of the foot (external tibial torsion) and flexible level arm (breakage of midfoot and planovalgus foot) will cause the ground reaction force (GRF) to shift more laterally and posteriorly to the normal position. The already weakened or overlengthened triceps surae fails to control the progression of the tibia over the planted foot during the stance phase leading to excessive ankle dorsiflexion. The resulting GRF therefore shifts to the posterior aspect of the knee and brings about flexion moment of the knee.

Proper bracing can compensate some of these deformities. But the severe lever-arm dysfunction will require bony surgeries to correct them. Some of the procedures include varus derotational osteotomy of the femur for coxa valga; derotation osteotomy of the tibia in patients with external tibial torsion; and foot stabilisation surgeries for severe planovalgus foot and lever-arm dysfunction to improve the function of the patients.



### Case Illustration

A 14 years old boy suffers from spastic diplegic cerebral palsy. He walked with significant crouch knee gait because of weakness in the triceps surae, bilateral knee flexion contracture, hamstring contracture and planovalgus foot deformity (Fig. 1). X-rays of the feet showed severe rocker bottom deformities with breakage in the midfoot (Fig. 2). The gait analysis showed persistent knee flexion and increased ankle dorsiflexion during stance phase (Fig. 3). He subsequently received posterior capsular release of both knees, bilateral hamstring lengthening and bilateral calcaneal lengthening to correct the lever arm dysfunction. Post-operatively, he could walk upright with significant improvement in the gait pattern (Fig. 4).



**Fig. 1a** Weakness of the triceps surae resulted in increase ankle dorsiflexion and shifted the ground reaction force posteriorly (red arrow). Both knees developed secondary fixed flexion contracture because of the loss of PF-KE couple and hamstring spasticity.

**Fig. 1b.** The patient had bilateral rocker bottom foot deformities, breakage in midfoot and forefoot abduction leading to an inefficient moment arm

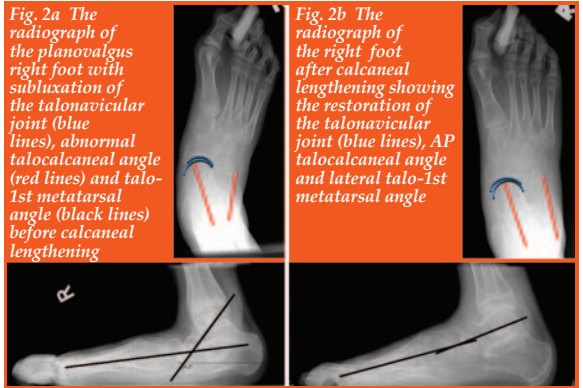


**Fig. 1c** Patient walked with significant crouching with persistent knee flexion and ankle dorsiflexion during stance phase. Note the severe rocker bottom foot deformity (red arrow)

**Fig. 1d** The lever-arm dysfunction of the feet resulted in excessive out-toeing on both sides

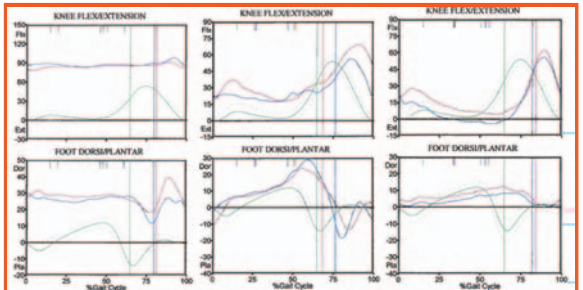
### Conclusion

Spasticity affects the normal action of the muscles and also the development of the skeleton in cerebral palsy patients. Orthopaedic surgery aims to correct these abnormalities e.g. muscle contracture and lever-arm dysfunction to improve the mobility and daily activities for these children. A multidisciplinary approach together with modern assessment tools such as gait analysis should be adopted in the decision-making when treating these patients. With a holistic approach, we aim to provide the best care to our patients and maximise their full potential.



**Fig. 2a** The radiograph of the planovalgus right foot with subluxation of the talonavicular joint (blue lines), abnormal talocalcaneal angle (red lines) and talo-1st metatarsal angle (black lines) before calcaneal lengthening

**Fig. 2b** The radiograph of the right foot after calcaneal lengthening showing the restoration of the talonavicular joint (blue lines), AP talocalcaneal angle and lateral talo-1st metatarsal angle



**Fig. 3a** Pre-operative gait analysis showed that the knees were held in flexion and the ankles in dorsiflexion throughout the stance phase (red line - left; blue line - right; green line - normal)

**Fig. 3b** Post-operative gait analysis without ground reaction orthosis showed significant improvement in the kinematics of the knees and ankles in the stance phase; but weakness in the triceps surae leading to residual ankle dorsiflexion and therefore weakening the PF-KE couple resulting in residual knee flexion during the stance phase

**Fig. 3c** Post-operative gait analysis with ground reaction orthosis to the ankles showed near normal kinematics in the knees



Fig. 4a The patient could walk upright with restoration of near normal PF-KE couple

Fig 4b. The planovalgus feet were corrected and the foot progression angles were relatively normal on both sides

### References

1. Novacheck TF, Gage JR. Orthopedic management of spasticity in cerebral palsy. Childs Nerv Syst. 2007 Sep;23(9):1015-31.
2. Hagglund G, Wagner P. Development of spasticity with age in a total population of children with cerebral palsy. BMC Musculoskelet Disord. 2008;9:150.
3. Nordmark E, Josenby AL, Lagergren J, Andersson G, Stromblad LG, Westbom L. Long-term outcomes five years after selective dorsal rhizotomy. BMC Pediatr. 2008;8:54.
4. Subramanian N, Vaughan CL, Peter JC, Arens LJ. Gait before and 10 years after rhizotomy in children with cerebral palsy spasticity. J Neurosurg. 1998 Jun;88(6):1014-9.
5. Carroll KL, Moore KR, Stevens PM. Orthopedic procedures after rhizotomy. J Pediatr Orthop. 1998 Jan-Feb;18(1):69-74.
6. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. Dev Med Child Neurol. 1997 Apr;39(4):214-23.
7. Palisano RJ, Rosenbaum P, Bartlett D, Livingston MH. Content validity of the expanded and revised Gross Motor Function Classification System. Dev Med Child Neurol. 2008 Oct;50(10):744-50.
8. Connelly A, Flett P, Graham HK, Oates J. Hip surveillance in Tasmanian children with cerebral palsy. J Paediatr Child Health. 2009 Jul-Aug;45(7-8):437-43.
9. Wood E, Rosenbaum P. The gross motor function classification system for cerebral palsy: a study of reliability and stability over time. Dev Med Child Neurol. 2000 May;42(5):292-6.
10. McDowell BC, Kerr C, Parkes J. Interobserver agreement of the Gross Motor Function Classification System in an ambulant population of children with cerebral palsy. Dev Med Child Neurol. 2007 Jul;49(7):528-33.
11. Bleck EE. The hip in cerebral palsy. Orthop Clin North Am. 1980 Jan;11(1):79-104.
12. Hoffer MM. Management of the hip in cerebral palsy. J Bone Joint Surg Am. 1986 Apr;68(4):629-31.
13. Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. Eur J Neurol. 2001 Nov;8 Suppl 5:98-108.
14. Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. Clin Orthop Relat Res. 1993 Mar(288):139-47.



THE UNIVERSITY OF HONG KONG LI KA SHING FACULTY OF MEDICINE  
DEPARTMENT OF PAEDIATRICS AND ADOLESCENT MEDICINE



## Postgraduate Diploma in Child and Adolescent Health

### 兒童及青少年健康深造文憑

\*Postgraduate Diploma in Child and Adolescent Health is a quotable qualification\*

2011 admission

This two year part-time course is designed to provide a systematic postgraduate programme in paediatric medicine and child/adolescent health to meet the educational needs of local medical practitioners.

On completion of the programme, students will be able to:

- ◆ attain competence in managing childhood diseases;
- ◆ tackle health problems in children and adolescents commonly encountered in daily clinical practice;
- ◆ recognize and provide expedient management for paediatric emergencies;
- ◆ make use of the most current clinical protocols for common childhood diseases ; and
- ◆ promote child and adolescent health in the community setting.

**Course structure:** The programme consists of lectures, clinical attachment and written assignment.

**Admission requirements:** Bachelor's degree in medicine from a recognized University or other qualification of equivalent standard; possess post registration experience of not less than one year

**Course fees:** HK\$ 52,000.00

For details, please visit <http://www.hku.hk/paed/>.

For enquiries, please contact Ms D Lai at 2817 0641 (Tel) / 2855 1523 (Fax) / [paedoff@hku.hk](mailto:paedoff@hku.hk).

**APPLICATION DEADLINE**  
**15 SEPT 2011**





## MCHK CME Programme Self-assessment Questions

Please read the article entitled "Spasticity - Orthopaedic Perspectives" by Dr. Michael KT TO and Dr. W CHOW 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 July 2011. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary. The CME accreditation is in application. The number of CME credit is subject to the final decision of the organisations.

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

1. In the Gross Motor Function Classification System (GMFCS), cerebral palsy patients are classified into five levels based on the patients' intellectual levels.
2. In general, orthopaedic surgeries aim for gait modification for GMFCS level I-III children and postural improvement for level IV and V children.
3. Modern gait analysis utilizes a computer-based system to study brain activity through the 3-dimensional measurement of motion (kinematics), measurements of moments and power in the articulations of the lower limbs (kinetics), electromyography, dynamic foot pressure (pedobarography) and oxygen consumption measurement.
4. In spastic cerebral palsy patients, hip subluxation is caused by spasticity of the lower limb muscle including the hip adductors and hip flexors.
5. In spastic cerebral palsy patients, muscles spasticity usually increases after the age of 4 and continues to increase until skeletally mature.
6. Gait modification surgery should be performed at older age when the patient is more skeletally mature e.g. after age 7.
7. According to Sutherland, the gait abnormalities of the knees in cerebral palsy patients can be classified into jump knee, crouch knee, stiff knee, and recurvatum knee patterns.
8. Gait analysis helps the clinicians to understand the interaction of the selective motor control, balance and spasticity of the cerebral palsy patients during walking.
9. When progressive hip subluxation is observed in a young spastic cerebral palsy patient despite adequate preventive measures e.g. hip abduction splints and physiotherapy, early surgical intervention should be considered despite he/she is skeletally immature.
10. Crouch knee gait can be caused by overlengthening of the triceps surae in spastic cerebral palsy patients.

## ANSWER SHEET FOR JULY 2011

Please return the completed answer sheet to the Federation Secretariat on or before 31 July 2011 for documentation. The CME accreditation is in application. The number of CME credit is subject to the final decision of the organisations.

### Spasticity - Orthopaedic Perspectives

**Dr. Michael KT TO**

FRCSEd (Ortho), FHKCOS, FHKAM (Orthopaedic Surgery)

**Dr. W CHOW**

FRCSEd (Ortho), FHKCOS, FHKAM (Orthopaedic Surgery)

1  2  3  4  5  6  7  8  9  10

Name (block letters): \_\_\_\_\_ HKMA No.: \_\_\_\_\_ CDSHK No.: \_\_\_\_\_

HKID No.: \_\_\_ - \_\_\_ X X (X) HKDU No.: \_\_\_\_\_ HKAM No.: \_\_\_\_\_

Contact Tel No.: \_\_\_\_\_

### Answers to June 2011 Issue

Pathology and Medical Therapy of Benign Prostatic Hyperplasia

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

# LEXUS

Ultimate Comfort

Ignite your Passion  
The New Lexus Luxury Sedan is Coming



Click on [www.newlexus.com.hk](http://www.newlexus.com.hk)  
for the passion on the new Lexus.

**Kowloon/N.T.:** G/F Mita Centre, 552-566 Castle Peak Road, Kwai Chung.

**Hong Kong:** Shop A, G/F Harcourt House, 39 Gloucester Road, Wanchai.

**Macau:** No.207-215, Av.do Dr. Rodrigo Rodrigues, G/F, Block "C", Nam Kwong Building, Macau. Tel: (853) 2851-3388 (Free Parking)

Tel: 2880-4151 (Free Valet Parking)

Tel: 2511-9430 (Free Valet Parking)

Tel: (853) 2851-3388 (Free Parking)

[www.lexus.com.hk](http://www.lexus.com.hk)





# The Medical Management of Cerebral Palsy- Paediatricians' Perspective

**Dr. Mario Wai-kwong CHAK**

MBBS, MRCP, FHKCPaed, FHKAM(Paed)

Associate Consultant, Department of Paediatrics and Adolescent Medicine, New Territories West Cluster



Dr. Mario Wai-kwong CHAK

## What is Cerebral Palsy?

Cerebral palsy is a broad term describing a group of permanent disorders of the development of movement and posture causing activity limitations that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbance of sensation, perception, cognition, communication, and /or behaviour, and/or seizure disorder.<sup>1</sup>

## Magnitude of the Problem in Hong Kong

According to a local cross-sectional survey of children aged 6 to 12 years performed between September 2003 and June 2004, among 435,572 mainstream primary schools and special needs school children, 578 were identified with cerebral palsy. The overall prevalence was 1.3 per 1000 children. The age-specific prevalence rate varies from 1.04 to 1.50 per 1000 children.<sup>2</sup> The figures are compatible with other studies that range from 1.2 to 2.5 per 1000 live births.

Approximately 38% of children with cerebral palsy attended mainstream schools. For those studying in special schools, 96% attended institutions for the physically handicapped or the severely mentally handicapped. Among the 219 children with cerebral palsy studying in mainstream schools, 26% received educational support, and 61% received outpatient therapy. Only 12% received both supporting services. No educational or therapy support was received by 26% of children.<sup>2</sup>

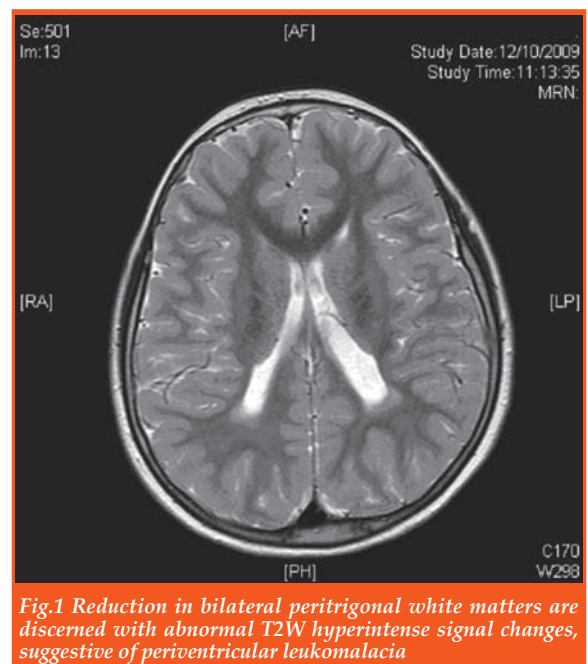
## Risk Factors

There are prenatal, perinatal and post-natal risk factors for development of cerebral palsy. Certain risk factors could help us make an early diagnosis. The prenatal risk factors include intrauterine infections, malformations of cortical development, premature delivery. The earlier the maturity, the higher percentage of survivors would likely have cerebral palsy. The perinatal risk factors include: amnionitis, antepartum haemorrhage, neonatal meningitis, perinatal stroke and birth asphyxia. The post-natal risk factors include: CNS infection, ischaemic and haemorrhagic strokes, traumatic brain injury, hypoxic-ischaemic events etc. It is increasingly apparent that cerebral palsy can result from the interaction of

multiple risk factors, and in many cases no identifiable cause can be found.<sup>3</sup>

## How to Make a Clinical Diagnosis?

Very often the diagnosis is delayed until the obvious clinical sign or deformity is detected. In order to make an early diagnosis, the recognition of early signs is important. Early signs include: developmental delay especially gross motor aspect (for example: poor head control, delayed sitting, standing and walking), toe walking, abnormal muscle tones either hypotonia or hypertonia, hyperreflexia, persistent primitive reflex, unusual posturing, early hand preference, spine deformity, failure to thrive, visual, hearing and speech problems etc.



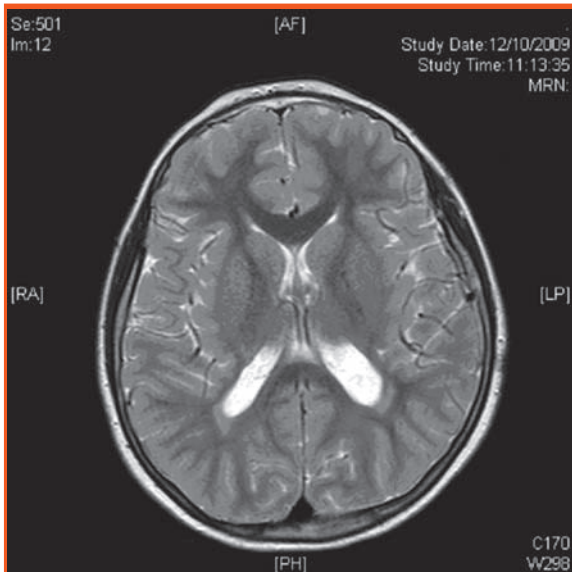
*Fig.1 Reduction in bilateral peritrigonal white matters are discerned with abnormal T2W hyperintense signal changes, suggestive of periventricular leukomalacia*

## What Other Types of Investigation Help to Make a Diagnosis?

Until recently, correlation between radiographic findings and clinical presentation in cerebral palsy was weak. Advances both in imaging technology and in quantitative motor assessments are now changing this



picture. In the clinical context of cerebral palsy, MRI brain could reveal the following radiological features: 1) signal or cystic changes over periventricular white matter e.g. periventricular leukomalacia (fig.1&2) in ex-preemie diplegic patients; 2) signal changes over thalamus and basal ganglia in dystonic term babies with perinatal hypoxic-ischaemic injury; 3) features suggestive of cortical infarctions over watershed area, Porencephaly and Schizencephaly in hemiplegia; 4) involvement of the internal capsule and cerebral cortex in spastic tetraplegia; 5) features of malformation of cortical development.



*Fig. 2 The same ex-premature patient has thinning of the posterior body of corpus callosum and mild dilatation of both trigones, suggestive of periventricular leukomalacia*

## When to Consider Other Differential Diagnoses Apart from Cerebral Palsy?

When a patient has spasticity or dystonia in the absence of any risk factors for cerebral palsy, one should consider other differentials. For example hereditary spastic paraplegia in a diplegic patient with family history of lower limb spasticity; adrenal leucodystrophies in a clumsy hemiparetic child with preceding normal motor function, cerebellar tumour or Pelizaeus-Mezbacher Syndrome in an ataxic child with nystagmus. Dopa responsive dystonia, tyrosine hydroxylase deficiency or other neuro transmitter deficiency should also be considered in a dystonic child without any perinatal risk factors.<sup>4</sup> Many rare neuro-metabolic and neuro-genetic diseases could mimic cerebral palsy. These diagnoses require a high index of clinical suspicion, sometimes with the collaboration of chemical pathology and clinical genetics for laboratory confirmation.

## Classification of Cerebral Palsy

The definition of cerebral palsy covers a wide range of clinical presentations and variable activity limitations; it is therefore useful to further categorise individuals into classes or groups. The traditional classification schemes

have been focusing primarily on the topography of the affected limbs, with an added modifier describing the predominant type of tone or movement abnormality.<sup>1</sup> Based on dominant movement disorder and limb involvement, cerebral palsy can be classified into the following main groups: spastic hemiplegia, spastic diplegia, spastic tetraplegia, dyskinetic and ataxic cerebral palsy.<sup>3</sup>

## Accompanying Impairments

Apart from motor impairment, there are many non-motor neurodevelopment or sensory problems associated with cerebral palsy that include: Intellectual Impairment, learning disabilities, epilepsy/abnormal EEG, speech and language disorders, psychological impairments, hearing impairments, sensory impairments, ophthalmologic defects, strabismus, dysfunctional voiding patterns, gastro-oesophageal reflux disorder, constipation, malnutrition, oral motor dysfunction, oral health problems, bone and mineral density disorders and sleep disturbances etc.

## UMN Syndrome and Spasticity

Features of the UMN syndrome in cerebral palsy include both "positive signs" and "negative signs". "Positive signs" include spasticity, spasms, clonus and hyperreflexia. "Negative signs" include weakness, loss of voluntary motor control, dexterity or fatigability. Botulinum toxin is useful in targeting the positive signs of UMN syndrome, while the negative signs are more difficult to manage. Spasticity has been most commonly defined as a velocity-dependent increase in passive muscle tone, associated with features of the UMN syndrome. It is seen in over 70% of those with cerebral palsy.<sup>5</sup>

## Why should We Make an Early Diagnosis?

The increase of the muscle tone will make muscle stiffer, causing muscle contracture. The latter will alter normal biomechanics and affect normal bony lever-arm system. Mal-alignment and skeletal deformity form a vicious cycle that can be broken with early effective intervention of the spasticity.

## What is Dystonia?

Dystonia is a movement disorder in which involuntary muscle contractions (sustained or intermittent) result in twisting and repetitive movements. Examples include choreo-athetosis, tongue protrusion, facial grimacing and /or abnormal postures such as back arching. Dystonia often co-exists with spasticity and when present needs to be taken into consideration in planning spasticity treatments, as the outcome may be less predictable.<sup>5</sup>

## What is Botulinum Toxin-A?



Botulinum toxin-A is a potent neurotoxin derived from the bacterium *Clostridium Botulinum*. In 1949, it was discovered that botulinum toxin could temporarily block neuromuscular transmission by stopping the release of acetylcholine from the motor neuron end plate in the muscle. This leads to a specific weakening of the muscle and a reduction in tone. Botulinum toxin-A was first used to treat strabismus in humans in the 1970s, and spasticity in 1989. Since then the spectrum of indications has expanded tremendously - focal dystonia, hemifacial spasm, torticollis and spasticity in children with cerebral palsy, just to name a few.<sup>6</sup>

## How to Use Botulinum Toxin-A Injection?

The toxin is available in fixed dose ampoules and will normally require reconstitution in normal saline. The reconstituted solution is then injected directly into the spastic muscle. The toxin will spread within the muscle and cause local relaxation over two to three days. This relaxation effect usually lasts around 2-3 months.<sup>6</sup>

## Who may Benefit from Botulinum Toxin-A?

Botulinum toxin-A would benefit patients whose spasticity interferes with function or daily cares or causing pain, and in whom the muscles to be injected have some dynamic ranges as determined using the Tardieu Scale. The dynamic contracture is the difference between the R1 (first catch as the muscle is moved quickly through its range of motion) and the R2 (end of passive range of motion). The greater the dynamic range, the greater the potentials to have a favourable response to Botulinum toxin-A.<sup>5</sup>

## Clinical Indications of Botulinum Toxin-A Injection for Spasticity in Cerebral Palsy

### *For functional use and ease of care in lower limbs*

Calf muscle spasticity causing equinus during walking or difficulty with splint use; hip adductor muscle spasticity causing scissoring during walking or difficulty with perineal hygiene; Hamstring muscle spasticity causing crouching gait or difficulty with positioning or sitting; tibialis posterior muscle causing equinovarus foot deformity,<sup>5</sup> iliopsoas muscle spasticity causing hip flexion contracture.

### *For functional use and ease of care in upper limb*

Elbow flexor spasticity limiting arm extension, for example for reaching, and affecting cosmetic appearance when walking; Thumb in palm causing difficulty in grasping objects; fisting hand causing difficulty in hand opening or leading to excoriated skin flexor creases; pronator spasticity limiting supination; shoulder spasticity limiting arm abduction or causing pain.<sup>5</sup>

## Possible Side-effects of Botulinum Toxin-A

A small number of patients may develop flu-like symptoms. Excessive weakness of the injected muscle can occur, especially when there is underlying weakness masked by the spasticity. This may be a problem in small muscles, such as forearm, where the Botulinum toxin – A may spread to the adjacent muscles. The weakness will reverse completely over time as the body naturally removes the toxin and spasticity returns. Most episodes of excessive local weakness last less than six weeks.

Rarely a child may experience generalised weakness due to a more systemic effect. This is rare and transient. Incontinence with loss of bowel and bladder control can occur after injection into the upper leg muscles. This may manifest as frequency and soiling with onset about a week after injection. The other side effects include dry mouth and pain on injection.<sup>5</sup>

## Current Evidence and Recommendation

The assessment of children with cerebral palsy and evaluation of outcomes following injection of Botulinum toxin-A are complex. The involvement of a multidisciplinary team is recommended. The recent international consensus concludes that injection of botulinum toxin-A in children with cerebral palsy is generally safe although systemic adverse events may occur, especially in children with more physical limitation (GMFCS V). The recommended dosage is intermediate between previous consensus statements. The committee further concludes that injection of Botulinum toxin-A is effective in the management of lower limb spasticity in children with cerebral palsy, and when combined with physiotherapy and the use of orthoses, these intervention may improve gait and goal attainment.<sup>7</sup> It is a grade A evidence for the use of Botulinum toxin to reach individualised therapeutic goals for paediatric upper limb hypertonia, grade B (probably effective) for tone reduction following Botulinum toxin injections and grade U evidence (inconclusive) for improvement in upper limb activity and function. Botulinum injections are generally found to be safe and well tolerated with the most common side effect identified as a transient decrease in grip strength.<sup>8</sup>

## How Often should We Inject?

About 5-10% of patients on repeated botulinum toxin injections develop clinical resistance. This is likely due to the development of antibodies to the toxin. Our policy is to give injection at least three months between injections to minimise this possibility.

## Muscle Localisation for Botulinum Toxin-A Injection

It is demonstrated that palpation is inadequate for muscle identification, especially in the forearm or deep lower limb muscles.<sup>9,10</sup> Topical analgesia (EMLA cream) with sedation (e.g. Midazolam) is a method to decrease the pain of the procedure.

Electromyography (EMG) was used to control injections

in the upper limb in children with cerebral palsy. However, children with cerebral palsy have a limited ability to co-operate and perform selective movements.<sup>11</sup> The acoustic signal is reduced in sedated children and the painful procedure usually requires several attempts until correct positioning of the needle is achieved. EMG can be useful in children with dystonia to identify muscles that are responsible for dystonic posture or movement.

Electrical stimulation is independent of selective voluntary motor function, and the patient's ability to co-operate<sup>12</sup> is unaffected by sedation. Therefore it offers better accuracy and practicality in children with cerebral palsy than EMG. The procedure can be time-consuming and painful and is therefore usually performed in combination with sedation.

Ultrasound allows quick visual identification of the target muscle, differentiation from adjoining structures, exact localisation of the needle tip in the desired position of the muscle and real time adjustment of injected volume and number of injection sites according to individual muscle anatomy.<sup>13-15</sup>

## Conclusions

Cerebral palsy is a broad term describing a group of heterogeneous disorders of movement and posture. Although it is an old terminology, it encompasses a group of patients with similar clinical problems, prognosis and treatment. It allows different medical professionals to communicate. Although the brain insult is static, the pattern and severity of the movement disorder may evolve during childhood. The role of paediatricians is to make an early diagnosis and intervene early. Current evidence has demonstrated that Botulinum toxin-A is an effective way in managing lower limb spasticity. Since patients with cerebral palsy have multi-dimensional problems, an interdisciplinary team has been set up in Tuen Mun Hospital to provide them with a comprehensive and holistic care.

## References

1. Bax M, Goldstein M, Rosenbaum P.; Executive committee for the definition of cerebral palsy. Proposed definition and classification of cerebral palsy, April 2005. *Dev. Med Child Neurol.* 2005 Aug;47(8):571-6.
2. WKL Yam, HSS Chan. Prevalence study of cerebral palsy in Hong Kong children *Hong Kong Med J* 2006; 12:180-4
3. Forsyth R, Newton R. Paediatric Neurology Oxford Specialist Handbooks in Paediatrics
4. WL Yeung, V Wong. Expanding phenotype and clinical analysis of tyrosine hydrolase deficiency. *J Child Neurology* 26(2):179-187.
5. Clerk K, Lannin N, O'Flaherty S. Treating children with botulinum toxin the children's hospital at Westmead.
6. Barnes MP, Ward AB. Oxford handbook of rehabilitation medicine.
7. Love SC, Novak I, Kentish M. Botulinum toxin assessment, intervention and after-care for lower limb spasticity in children with cerebral palsy: international consensus statement. *Eur J Neurol.* 2010 Aug; 17 Suppl 2:9-37.
8. Fehling D, Novak I, Berweck S, Hoare B. Botulinum toxin assessment ,intervention and follow-up for paediatric upper limb hypertonicity: international consensus statement *Eur J Neurol.* 2010 Aug;17 Suppl 2:38-56.
9. MollyFM, Shill HA, Kaelin-LangA, Karp BI. Accuracy of muscle localization without EMG; Implications for treatment of limb dystonia. *Neurology*2002;58:805-807.

10. Chin TY, Natrass GR, Selber P, Graham HK. Accuracy of intramuscular injection of botulinum toxin A in juvenile cerebral palsy: a comparison between manual needle placement and placement guided by electrical stimulation. *J Pediatr Orthop* 2005;25:286-291.
11. Janovic J. Needle EMG guidance for injection of botulinum toxin. Needle EMG guidance is rarely required. *Muscle Nerve* 2001;24:1568-1570.
12. Barbano RL. Needle EMG guidance for injection of botulinum toxin. Needle EMG guidance is useful. *Muscle Nerve* 2001; 24:156-1568.
13. Berweck S, Feldkamp A, Francke A, Nehles J, Schwerin A, Heinen F. Sonography-guided injection of botulinum toxin A in children with cerebral palsy. *Neuropediatrics* 2002;33:221-223.
14. Willenborg MJ, Shilt JS, Smith BP, Estrada RL, Castle JA, Koman LA. Technique for iliopsoas ultrasound guided active electromyography-directed botulinum a toxin injection in cerebral palsy. *J Pediatr Orthop* 2002; 22:165-168.
15. Fietzek U, Berweck S, Wissel J, Heinen F. Sono's Anatomy, 1st edn. Munich: Child and Brain, 2008.

# FOR LEASE

## OFFICES – suitable for clinics

### Manning House

48 Queen's Road Central

No Agency Fee is required

Very attractive rental package

- \* 832 - 952 sq.ft.
- \* High concentration of clinics
- \* Next to Central MTR exits
- \* Professional property management

LEASING HOTLINE :  
Ms Pang - 2844 3259  
Ms Lee - 2844 3257





## DCH (Diploma in Child Health Examination) Written Examination (MRCPCH Part IA) 2011

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 2011 awarding DCH (HK) and DCH (International) to successful candidates.

The Examination is divided into two parts, Written (MRCPCH Pt IA) and Clinical. The DCH Written Examination is a common paper shared with the MRCPCH Part IA. The MRCPCH Part IA Examination is held three times a year in Hong Kong. The next MRCPCH Part IA Examination will be held on **Tuesday, 11 October 2011**. The examination fee is **HK\$4,250** for Part IA. Candidates who wish to enter the examination must hold a recognized medical qualification in Hong Kong.

**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](http://www.paediatrician.org.hk/entcnews.htm) or call the College Secretariat at 28718871.

**Deadline for Application: Tuesday, 18 July 2011**

### Important Notice

#### New Clinical Examination for DCH from April 2011

A new format of the DCH clinical examination has been adopted since April 2011. Details of the new format and other relevant information can be viewed on the RCPCH website at: [www.rcpch.ac.uk](http://www.rcpch.ac.uk)



The Hong Kong College of Paediatricians (HKCPaed) and the Royal College of Paediatrics and Child Health (RCPCH) will be holding a Joint Diploma in Child Health Clinical Examination in Hong Kong in October 2011, awarding DCH (HK) and DCH (International) to successful candidates.

The DCH Clinical Examination will be held on **25 October 2011** and will be run in a new format. Please visit the HKCPaed Website for information on the format of the new DCH Clinical Examination: <http://www.paediatrician.org.hk/councilnews.htm#dch>

The DCH Clinical Examination is open to registered medical practitioners in Hong Kong. Candidates who have already successfully passed the Written Paper 1A since January 2004 are eligible to apply. In addition, candidates who passed the Part IA examination in May 2005 or thereafter should have at least 6 months of Paediatric practice (resident medical officer or intern within 5 years prior to the date of the DCH Clinical Examination) in a recognized institution with acute hospital admissions. There are no exemptions from the Paper 1A examination.

A new DCH Syllabus has been introduced since November 2009. It will serve as the basis for assessments for the DCH Clinical Examination to be held in Hong Kong in October 2011. The new Syllabus is available for viewing at the following link on the RCPCH Website: <http://www.rcpch.ac.uk/training-examinations-professional-development/examinations/diploma-child-health/dch-clinical-struct>

#### Application:

Candidates who wish to sit the DCH Clinical Examination in Hong Kong **MUST** apply through the Hong Kong College of Paediatricians. Application form, details of application and the new format can be found on the HKCPaed website at [www.paediatrician.org.hk/entcnews.htm](http://www.paediatrician.org.hk/entcnews.htm). Examination Fee is HK\$ 8,100. Available places are limited and will be allocated on a 'first come first served' basis.

**Opening date: 20 June 2011**

**Closing date: 18 July 2011**

# Medical Treatment of Spasticity in Adults

**Dr. Wai-man HUNG**

*Department of Neurosurgery, Tuen Mun Hospital, Tuen Mun, Hong Kong*

Spasticity is a common clinical problem among neurological patients especially in the field of neuro-rehabilitation. It reduces functional mobility and is often a major obstacle for both carers and therapists to deliver quality care and training. In severe cases, it can progress into limb contracture that poses great impact on patients' quality of life.

Spasticity in adults usually occurs after a major stroke, severe brain as well as spinal cord injuries. Patients suffering from these conditions may have neurological deficits of upper motor neuron lesion type and limb spasticity will set in early in rehabilitation. Prevention and clinical vigilance is most important for patients at risk and once confirmed, management of adult spasticity can be divided into physical, medical and surgical components. Medical treatment is our focus in this article.

Medical treatment for adult spasticity can be divided into 3 categories:

- A. Oral medication
- B. Focal treatment
- C. Intrathecal medication

## Oral Medication

### *Baclofen*

Baclofen is the commonest oral anti-spastic drug used in our daily practice. It is a GABA  $\beta$  receptor agonist that works by a pre-synaptic inhibitory effect on the release of excitatory neurotransmitters such as glutamate, aspartate, and substance P.<sup>1</sup> Although baclofen is widely prescribed in both supra-spinal and spinal neurological conditions, its higher efficacy on spasticity of spinal cord origin has been demonstrated in many double-blind studies.<sup>2</sup> The drug is believed to act at the spinal cord level by inhibition of polysynaptic spinal reflexes.

Oral doses are typically 5-20 mg given 2-4 times/day. The maximum daily dosage is 80 mg. Careful upward titration from the minimum dose until an adequate relief is recommended.

Baclofen shares with other anti-spastic medications the side effects of drowsiness, fatigue, and muscle weakness which are often dose-dependent. The effect of each dose lasts for 3 to 5 hours. As a general rule, the dose of baclofen can usually be increased until the desired benefit is achieved or side effects become intolerable.

Discontinuation of baclofen is associated with

withdrawal symptoms which are more likely after prolonged use for more than a couple of months, irrespective of the actual dosage. The severity of symptoms depends on the rate at which baclofen is discontinued. It should therefore be tapered down slowly and in case of acute symptoms, baclofen can be recommenced.

### *Dantrolene sodium*

Dantrolene is less commonly used in Hong Kong. The mode of action is peripheral with a direct effect on skeletal muscles so that all muscles including non-spastic ones become weaker. It suppresses the release of calcium ions from the sarcoplasmic reticulum thus inhibiting excitation, contraction and coupling<sup>3</sup>, regardless of the origin of the spasticity - spinal or cerebral.<sup>4</sup>

Dantrolene should be started at 25 mg daily and can be increased over several weeks to a maximum of 400mg daily in divided doses.

Drowsiness, dizziness, weakness, fatigue and diarrhoea are frequent side effects. Because of its peripheral action, sedative side effects are much milder than that of baclofen. Since there were reports of hepatotoxicity in long-term high dosage treatment regimens, dantrolene should be avoided in those with impaired liver function.

### *Tizanidine*

Tizanidine is available in some countries for some time but only in Hong Kong recently. It is an imidazoline derivative and mainly affects spinal polysynaptic reflexes. Its agonistic action at noradrenergic alpha 2 receptors results in both direct impairment of excitatory amino acid release from spinal interneurons and concomitant inhibition of facilitatory coeruleo spinal pathways.<sup>5</sup> Tizanidine has similar anti-spastic efficacy as baclofen but induces less clinical weakness and is better tolerated. Side effects are mild and include dry mouth, fatigue, and dizziness with very occasional elevation in liver enzymes.

The recommended initial dosage of tizanidine is 4 mg orally once a day. It can be repeated as needed at 6 to 8 hour intervals to a maximum of 3 times a day. The dose may be gradually increased (every 4 to 7 days) in increments of 1 to 2 mg until the desired response is attained. The manufacturer recommends that the total daily dose should not exceed 36 mg nor should a single dose be greater than 12 mg.

### *Diazepam*



This was the first anti-spastic agent ever used. The effect of diazepam in spasticity is probably mediated by its ability to enhance the action of the inhibiting neurotransmitter GABA. There are few double-blind controlled studies of adequate size but these do confirm the effectiveness of diazepam. Its effect lasts for 6 to 8 hours but with unacceptable drowsiness and weakness. Indeed it has a limited usefulness in most situations.

### Other anti-spastic agents

There are still other choices such as gabapentin, clonidine, glycine, L-dopa, etc. Recently, Cannabis has also been studied for the management of spasticity but the clinical efficacy and safety need to be evaluated.

## Focal Treatment

Most spasticity is focal and affects one or a few muscle groups. Focal treatment targets just those affected muscle and does not exert systemic side effects like oral medications do occasionally.

### Botulinum toxin

Botulinum toxin has revolutionised the treatment of focal spasticity. Botulinum toxin is produced by the bacterium *Clostridium botulinum*. There are seven serotypes A to G. It blocks the pre-synaptic release of acetylcholine from nicotinic and muscarinic nerves. Therapeutic injections of botulinum toxin have been used since the early 1980s. It was first used by an ophthalmologist in San Francisco, Alan Scott, to correct strabismus. The toxin was chosen for this purpose because of its ability to produce a focal, dose-related temporary weakness of muscles that lasts about 3 months.<sup>6</sup>

The first report of the use of botulinum toxin in spasticity was published in 1989 by Das and Park. Others randomised clinical trials (RCTs) involving patients with spasticity resulting from a variety of diseases (mainly stroke and multiple sclerosis) have clearly shown that botulinum toxin type A (Dysport<sup>®</sup> and Botox<sup>®</sup>) temporarily reduces spasticity in the elbow, wrist and finger flexors of the upper limb, and hip adductors and ankle plantar flexors in the lower limb. The clinical benefits from reduction of spasticity are best shown in the upper limb, with less disability of passive function and reduce caregiver burden. In the lower limb, there is improved perineal hygiene from hip adductor injections. The benefits of reducing ankle plantar flexor tone are less well established. Pain is also reduced, possibly by mechanisms other than muscle weakness. Improved active function has not yet been clearly demonstrated in RCTs, only in open-label trials. The safety of botulinum toxin-A is impressive, with minimal (mainly local) adverse effects.

Therapeutic botulinum toxin is available in two serotypes, type A (botulinum toxin-A) and type B (botulinum toxin-B). There are two formulations of botulinum toxin-A, Botox<sup>®</sup>, which is available worldwide, and Dysport<sup>®</sup> outside North America. It is important to point out that the two formulations of botulinum toxin-A are not dose equivalent.

### Phenol or alcohol nerve blocks

In case of budget constraint, an alternative is to use phenol or alcohol to abort spasticity by nerve block, motor point block or intramuscular neurolysis. Any peripheral nerve that is readily accessible can be blocked. Examples include:

1. Obturator nerve for adductor spasticity
2. Posterior tibial nerve for calf spasticity
3. Sciatic nerve for hamstring spasticity
4. Median, ulnar, or musculocutaneous nerves for flexor arm spasticity

Comparing to botulinum toxin, the injection technique of phenol is more demanding. Side effects such as pain or inflammation are common. If a mixed motor/sensory nerve is injected, painful dysaesthesiae may result and can be permanent. Its effect is immediate and lasts for as long as eight months and in some cases permanent.

### Selection of patients for Injection

Involved therapists and physicians come together for the selection. Based on a careful assessment and documentation of the range of motion of joints, muscle power, muscle tone according to the Modified Ashworth Scale (MAS) (Figure 1) and the presence of contracture, patients are selected for botulinum toxin therapy. Treatment protocols are set not only to relieve spasticity alone but according to the 3 domains - body function and structures, activities and participation stipulated under International Classification of Function by the WHO. A measurable outcome is stated in the record to gauge effectiveness of the treatment.

Grade	description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when the affected part(s) is moved in flexion or extension.
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.
2	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.
3	Considerable increase in muscle tone, passive movement difficult.
4	Affected part(s) rigid in flexion or extension.

Figure 1. Modified Ashworth Scale

### Selection of muscle groups and Injection technique of Botulinum Toxin

By studying the spasticity pattern over affected joints, muscle groups are selected for injection. In case of co-contraction, both flexor and extensor muscle groups have to be injected for better control. For example, a larger dose should be given to hamstring than to rectus femoris of a spastic lower limb in order to maintain a reasonable lower limb tone for maintaining the upright posture.

If Botox<sup>®</sup> is used, there are several useful guidelines in dose planning:



- Each vial of Botox® containing 100 units is diluted with 2 ml of normal saline to a solution of 0.02 ml/ unit
- Maximum body dose per visit in an average adult should be less than 600 units
- For big muscle groups such as pectoralis major or gastrocnemius, biceps, 100 to 120 unit per muscle per visit can be given
- For smaller muscle groups such as flexor carpi ulnaris or flexor hallucis longus, 30 to 50 units per muscle per visit suffice

Detailed dosage recommendation is available from pharmaceutical formulary.

Identification of anatomical landmarks and palpation are the basics for accurate injection. However precision can be further enhanced by technology.

- Electromyogram (EMG) guidance:** By attaching the cannulised, Teflon-coated monopolar hypodermic needle to an EMG machine, voluntary contraction of the target muscle could be picked up as a crisp staccato. Injection is then done precisely to the muscle as desired.
- Electrical stimulation:** Low-amperage electrical stimulation directly through the bare tip of the insulated hypodermic needle may be used to produce visible contraction in the target muscle. The needle is repositioned until contractions may be reproduced by the lowest stimulation intensities. In our own experience, it is the most effective way to confirm the correct placement of the injection needle. (Figure 2)

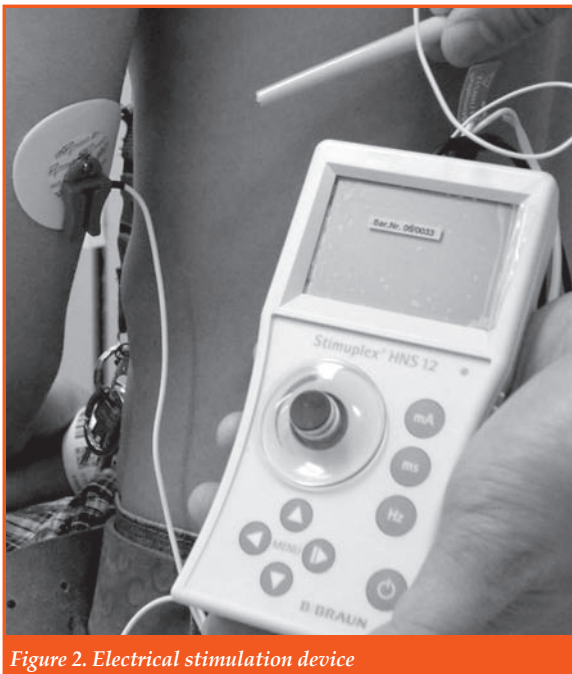


Figure 2. Electrical stimulation device

- Ultrasonography:** It has an advantage over other modes of localisation tools in that a clear anatomical structure is visualised in real time during the injection. It is particular useful in cases of severe muscle atrophy due to long standing spasticity, where usual anatomical palpation and skin landmarks cannot be easily applied. However, there is definitely a learning curve for this application.

### Repeat Injections

The toxin gives a window period for the patient to have rigorous training as well as castings for the limbs to alleviate spasticity and prevent contracture. Generally speaking, we would consider repeating injection for patients after 3 to 6 months if the symptoms recur. In our experience, the patients' condition usually improves after a few cycles of injections, such as a better and steadier gait pattern. Further botulinum toxin injection might be needed for an evolving goal as a patient pursues a higher level of independence.

### Intrathecal Medication

#### Intrathecal baclofen (baclofen pump)

Another way of administration of medication is to apply the anti-spastic agent directly on to the spinal cord. The use of intrathecal baclofen was first described in 1984. A programmable drug pump (Figure 3) is implanted subcutaneously to deliver baclofen intrathecally via a silastic catheter. Baclofen is administered either in regular boluses or by continuous infusion. The daily dose has to be adjusted according to the clinical effect and usually ranges from 50 to 1000 microgram per day. Azouvi et al (1996) administered intrathecal baclofen by an implanted pump to 80 patients with severe and disabling spinal spasticity with an average follow-up period of 37 months.<sup>7</sup> All individuals showed a significant decrease in tone and spasms. The efficacy remained stable except in cases of mechanical problems of the pump or catheter. An interesting study by Dressandt and Conrad (1996) demonstrated lasting reduction of spasticity after ending intrathecal treatment.<sup>8</sup> The spasticity remained absent or markedly reduced after stopping treatment in 7 out of 27 individuals. The reason for this continuing reduction in spasticity, however, is not clear.

To confirm that a patient is suitable for implantation, a trial dose is necessary. 50 microgram of baclofen in 1 ml volume is injected via lumbar puncture or a spinal catheter in one minute. Clinical response is expected in the next 4 to 8 hours. Further test doses can be given with 75 microgram on Day 2 or 100 microgram on Day 3. If the patient does not show any positive response up to 100 microgram, he will not be considered a suitable candidate.



Figure 3 Baclofen pump by Medtronic

The pump is fully programmable and allows a constant infusion of small doses of baclofen. The technique is particularly useful in severe and resistant spasticity. However, there are disadvantages:

- A surgical procedure is required
- The set up and the hardware is expensive,



3. Repeated refilling of drug is required that poses financial burden to the patient or the treatment providers
4. There are risks of catheter migration or dislodgement and prompt action has to be taken. There is significant stress on manpower and a dedicated team for emergency maintenance is needed.
5. There is a risk of pump failure leading to termination or over-dosage of baclofen.

## Spasticity Management for Adults in NT West Cluster\*

Our service for spasticity in adults is an extrapolation from the experience in managing children with cerebral palsy for the past decade. The only difference is perhaps the acute onset of the former. In the same manner, we need to gather a committed team of physiotherapists, occupational therapists, social workers and physicians with training in rehabilitation who will be screening patients from the acute phase onward, as part of neuro-rehabilitation – an integral part of a holistic service for our neurosurgical patients.

Rehabilitation should commence soon after the initial stabilisation of the acute clinical condition. Specialised nurses proactively assess patients and will watch out for the development of spasticity since their early hospitalisation. Limb stretching exercise is started for all patients with neurological deficits. Spasticity may occur as early as the first few weeks of the insult. We advocate early monitoring of patients' body and limbs' tone with MAS. Botulinum toxin can be given to patients even before discharge to the rehabilitation ward if initial physiotherapy treatment cannot control the worsening spasticity. Rigorous physical therapy and appropriate splintage follow. Prevention is better than cure and early action makes a difference!

Botox<sup>®</sup> clinic by the team continues after hospitalisation and a rehabilitation programme is tailor-made for the specific goals set for patients utilising physical therapy and medication discussed above.

## Conclusion

Spasticity management requires a multi-disciplinary team approach. Early detection and an appropriate physical treatment protocol would maximise the effects of medication for spasticity. Apart from various oral medications, botulinum toxin is a preferred choice for focal spasticity for its safety and reliability. Although the cost involved is substantial, it is still worth the ensuing better quality of life of these patients.

\*NT West Cluster consists of Tuen Mun, Pok Oi and Castle Peak Hospitals under the Hospital Authority

## References

1. Richard J. Greenwood, Micheal P. Barnes, et al., "Handbook of Neurological Rehabilitation" 2nd edition, Psychology Press
2. Duncan, G.W., Shahani, B.T., & Young, R.R. (1976). An evaluation of baclofen treatment for certain symptoms in patients with spinal cord lesions. *Neurology*, 26, 441-446.
3. Young, R.R., & Delwaide, P.J. (1981). Spasticity. *New England Journal of Medicine*, 304, 28-33, 96-99.
4. Michael P. Barnes, Anthony B. Ward, "Oxford Handbook of Rehabilitation Medicine", Oxford University Press
5. Coward, D. Coward, D.M. (1994). Tizanidine: neuropharmacology and mechanism of action. *Neurology*, 44 (suppl. 9), S6-11.
6. Geoffrey Sheean, University of California, San Diego, USA. Botulinum toxin treatment of adult spasticity. *Drug Safety* 2006; 29 (1): 31-48.
7. Azouvi, P., Mane, M., Thiebaut, J.B., et al. (1996). Intrathecal baclofen administration for control of severe spinal spasticity: functional improvement and long term follow-up. *Arch. Pys. Med. Rehab.*, 77, 35-39.
8. Dressandt, J., & Conrad, B. (1996). Lasting reduction of severe spasticity after ending chronic treatment with intrathecal baclofen. *J. Neuro., Neurosurgery. & Psychiatry*, 60, 168-173.



## Rental Fees of Meeting Room and Facilities at The Federation of Medical Societies of Hong Kong

(Effective from October 2009)

Venue or Meeting Facilities	Member Society (Hourly Rate HK\$)			Non-Member Society (Hourly Rate HK\$)		
	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays
Multifunction Room I (Max 15 persons)	150.00	105.00	225.00	250.00	175.00	375.00
Council Chamber (Max 20 persons)	240.00	168.00	360.00	400.00	280.00	600.00
Lecture Hall (Max 100 persons)	300.00	210.00	450.00	500.00	350.00	750.00
<b>Non-Peak Hour: 9:30am - 5:30pm</b> <b>Peak Hour: 5:30pm - 10:30pm</b>						
LCD Projector	500.00 per session					
Microphone System	50.00 per hour, minimum 2 hours					



# 2011 / 2012

Part-time Postgraduate Courses

[www.fmpg.hku.hk](http://www.fmpg.hku.hk)



Department of Family Medicine and Primary Care  
The University of Hong Kong

## Postgraduate Diploma in Community Geriatrics 社區老年醫學深造文憑

Jointly organized with Hong Kong Geriatrics Society

	Interactive Workshops	Clinical Attachment
Study period	September 2011 - June 2012	
Time	Saturday afternoons (2:30 - 5:15pm)	Weekday afternoons (2:00 - 4:00pm) or evenings (6:30 - 8:30pm)
Session	5 sessions	25 sessions
Venue	HKU Sassoon Road campus	Public hospitals
Assessment	Written assignments, written and clinical examination	
Tuition fee	\$42,000	

Quotable Qualifications by the  
Medical Council of Hong Kong

## Postgraduate Diploma in Community Psychological Medicine 社區精神醫學深造文憑

Jointly organized with Department of Psychiatry, The University of Hong Kong

	Interactive Workshops	Clinical Attachment
Study period	September 2011 - June 2012	
Time	Saturday afternoons (2:00 - 4:00pm)	Weekday afternoons (2:00 - 4:00pm)
Session	20 sessions	20 sessions
Venue	A centrally located venue	Public hospitals / clinics
Assessment	Case report, written and clinical examination	
Tuition fee	\$42,000	

Application can be made through HKU online system at  
[www.asa.hku.hk/admissions/tpg/](http://www.asa.hku.hk/admissions/tpg/)  
or you can contact Ms Tang for further details.

**Extended closing date for application: 15 July 2011**

Enquiry Tel: 2518 5681 Fax: 2814 7475 Email: [fmpg@hku.hk](mailto:fmpg@hku.hk)  
Address: 3/F, Ap Lei Chau Clinic, 161 Main Street, Ap Lei Chau, Hong Kong





# Selective Dorsal Rhizotomy in Spastic Cerebral Palsy

**Dr. Kwong-yui YAM**

*Consultant Neurosurgeon, Department of Neurosurgery, Tuen Mun hospital, Hong Kong*



Dr. Kwong-yui YAM

## Introduction

Cerebral palsy (CP) is a neurological disorder encompassing a group of motor conditions that cause physical disability in a developing child. It is caused by a non-progressive damage to the motor control centres of the developing brain and can occur during pregnancy, during labour or after birth up to the age of three.

The incidence of CP is around 3 per 1,000 live births. In a local study, the overall prevalence was 1.3 per 1000 children<sup>1</sup>. Spastic CP is classified according to the region of the body affected. Spastic diplegia is by far the commonest type of cerebral palsy, accounting for 70% to 80% of all cases. It involves the lower extremities only. In spastic quadriplegia, all four limbs are involved.

Spasticity is defined as a movement disorder characterised by a velocity-dependent increase in tonic stretch reflexes. Children with spastic CP have a neuromuscular mobility impairment stemming from an upper motor neuron lesion in the brain over the areas in the motor cortex or the corticospinal tract, which descends in the spinal cord, in the lateral columns and carries signals for voluntary movement of skeletal muscles. The damage impairs the ability of nerve receptors in the spine to properly receive gamma amino butyric acid, leading to hypertonia in the muscles innervated by these damaged tracts.

In spastic CP, patients also have other signs and symptoms of upper motor neuron syndrome like muscle weakness, paucity of active movement, inability to perform fine movement, brisk tendon jerk reflexes and positive Babinski sign. Consequently, their muscles have reduced dexterity, weakness and fatigability, which will translate into deficient daily activities. Deformities of the bones and joints of the limbs are common after prolonged muscle imbalance and spasticity.

Current treatments for spasticity include oral muscle relaxant medications, physiotherapy, orthotic devices, and repeated intramuscular injections of botulinum toxin. Orthopaedic procedures include tenotomy, tendon lengthening, tendon transfer and osteotomy. Neurosurgical procedures are peripheral neurotomy, selective dorsal rhizotomy and continuous intrathecal baclofen infusion. The spectrum of therapeutic interventions ranges from diffuse to focal, temporary to permanent. A multidisciplinary team approach is needed because these patients often require different treatment modalities at different ages and stages of the disease. In general, stretching exercise, splints and

physiotherapy are prescribed for patients at age of 2-3. Botulinum toxin injections, casting and orthosis would be added at age of 3 to 5. A permanent tone reduction procedure like SDR would be performed when these patients show static gross motor dysfunction. SDR is usually performed at the age of 6-7. Soft tissue surgery aiming at fine-tuning joint range and alignment will be done one year after SDR. Bone surgery will be reserved until skeletal maturity is reached.

## Selective Dorsal Rhizotomy Clinic

Selective dorsal rhizotomy is a neurosurgical procedure performed in patients suffering from spastic cerebral palsy. The selective de-afferentation of sensory nerve rootlets from L1 to S2 results in a reduction of contracting stimuli to muscles, decreasing spasticity, and thereby improves motor function. A number of case series suggested that selective dorsal rhizotomy reduces spasticity substantially, improves ambulatory function, and involves no unacceptable short-term risk when performed by experienced multidisciplinary teams<sup>2-4</sup>.

In 1997, the Department of Neurosurgery established the Selective Dorsal Rhizotomy (SDR) Clinic in Tuen Mun Hospital. The team members include physiotherapists from the hospital, special schools and Child Assessment Service of the Department of Health, paediatric neurologists and developmental paediatricians, orthopaedic surgeons, neurosurgeons and urologists. The clinic provides a platform for multidisciplinary approach to patients with spastic CP and their relatives. The clinic provides screening and assessment to select potential surgical candidates for SDR. Setting a treatment plan with realistic therapeutic goal for each individual patient follows. We also provide them with pre-operative training programme and peri-operative rehabilitation protocol. The families have to understand and be committed to the important preoperative physiotherapy training which will last for 2-3 months before a decision on SDR is made. The clinic will follow up patients to monitor their progress after surgery and formulate subsequent treatment plan.

## Patient Selection

The selection criteria for Spastic diplegic patients for SDR include:

1. Spastic - Spasticity of the lower limbs interfering normal functions, disturbing fluidity of gait or movement.

2. Strong - Fair to good lower limb muscle strength and control
3. Straight - Fair to good trunk control with no fixed orthopaedic deformity
4. Slim - Not too heavy or obese
5. Smart - Normal to near normal intelligence
6. Social support - A supporting and motivated family

The selection criteria for Spastic quadriplegic patients for SDR include:

1. Significant lower limb spasticity interfering with positioning and care
2. No severe dystonia
3. No fixed contracture at multiple joints

The decision-making on patient selection has to be individualised, as most of the SDR candidates will not meet all criteria. All team members play a part in the decision making process. Unsuitable candidates will be discharged with a rehabilitation plan. They will be reassessed in nine to twelve months time before coming to a final verdict.

SDR is a functional neurosurgical procedure with the purpose of improving lower limb function. In order to monitor and document the clinical progress, we have adopted a series of tools and examinations. Main outcome measures include Modified Ashworth Scale, passive range of joint movement, the Gross Motor Function Measure, the Paediatric Evaluation of Disability Inventory, the Canadian Occupational Performance Measure, urodynamics study, oxygen consumption and three-dimensional gait analysis. We schedule the test immediately before SDR, six months, twelve months and up to six years after SDR.

## The Procedure

We performed the first SDR procedure in December 1996. We modified the procedure and used multiple level laminoplasty (L2-S1) for dura exposure after Dr. Peacock's surgical demonstration during the neurosurgical commissioned training in 1997 (figure 1). In October 2006, we used a less invasive single level laminectomy at the level of conus medullaris for SDR (figure 2)<sup>5</sup>. After laminectomy, the surgeon will open the dura and arachnoid at the midline. The dorsal roots will be separated from the anterior motor roots by using patties. The dorsal root will be further subdivided into 4-5 rootlets and their clinical response to electrical stimulation tested. Abnormal rootlets will be cut. The procedure will be performed on L1 to S1 dorsal roots on both sides.

The term "selective" is used because we rely on the Intra-operative Trigger Electromyography (EMG) response to find the rootlets causing abnormal muscle spasms before cutting them. We insert paired needle electrodes into the deltoids, hip adductors, vastus medialis, hamstrings, gastrocnemius and the external anal sphincter for EMG monitoring during SDR. A pair of active electrodes is used to stimulate individual rootlet during surgery.

The EMG criteria for selecting an abnormal nerve rootlet include:

1. Low stimulation threshold
2. A tetanic or polyphasic to a tetanic stimulation of a 50Hz chain lasting for one second
3. A spread of EMG response to the contralateral side (figure 3)

Besides EMG, we also use the on-table clinical response during dorsal rootlet stimulation as selection criteria. It has been observed that according to the segmental innervation of the lower limb, the higher the muscle tone, the more extensive the rhizotomy would be. In general, 40-60% of the rootlets would be cut during SDR. In the first two postoperative days, the patient is nursed on lateral position and turned every 2 hours. A Foley's catheter is removed on day 3. The patient is mobilised on day 4 and both patient and parents will participate in intensive physiotherapy programme later on as outpatients in the following two months.

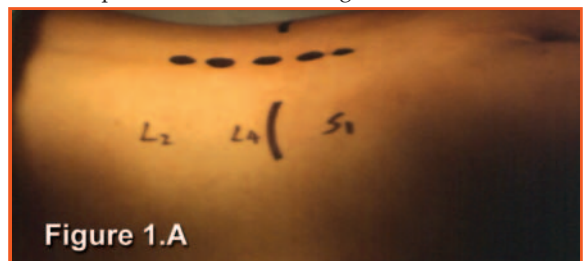


Figure 1.A

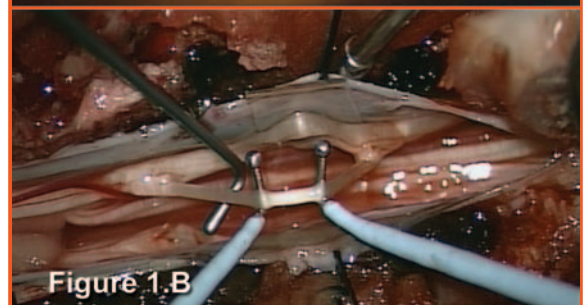


Figure 1.B

Figure 1. A. Skin incision for multiple level laminoplasty. B. Dorsal rootlets at the S1 level were exposed after multiple level laminoplasty. The paired electrodes were used to apply electrical stimulation during surgery.

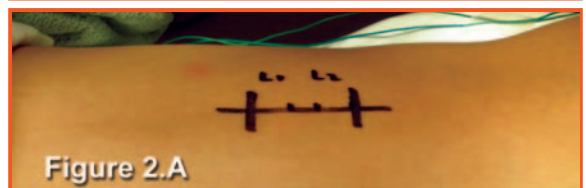


Figure 2.A

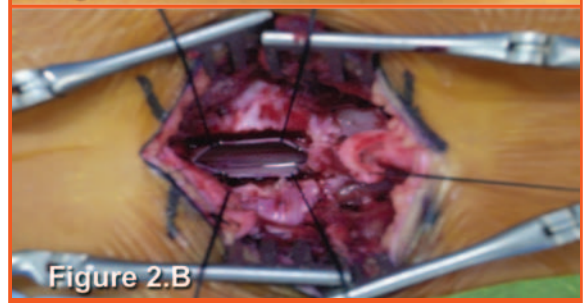


Figure 2.B

Figure 2. A. Skin incision for single level approach targeting at the conus medullaris. B. The conus medullaris was exposed and SDR was performed in this 2 cm dura opening under operating microscope.

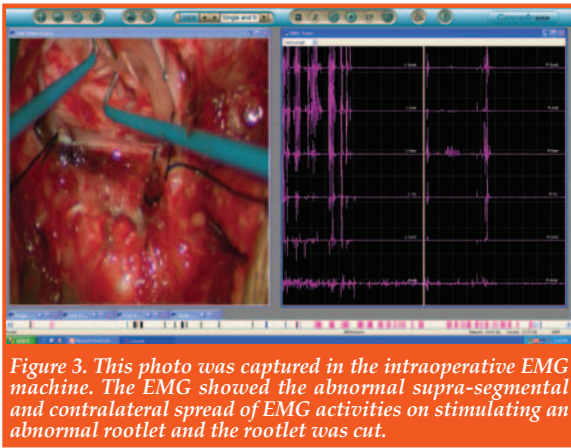


Figure 3. This photo was captured in the intraoperative EMG machine. The EMG showed the abnormal supra-segmental and contralateral spread of EMG activities on stimulating an abnormal rootlet and the rootlet was cut.

## Clinical Outcomes

We have performed around eighty SDR procedures since 1996. 80% of the surgical candidates belong to the spastic diplegic group and 10% belong to the spastic quadriplegic group. 75% of them had surgery done before the age of nine. There was no major surgical complication. Wound pain and postoperative low-grade fever were fairly common, usually lasting a few days and improved after acetaminophen. There was no wound infection, cerebral spinal fluid leak or pseudomeningocele after surgery. A small proportion of them developed dysuria after surgery but no urinary tract infection was found. Around 20% of the SDR patients complained of some degree of lower limb numbness that usually resolved after a few days to weeks.

There was no long-term complication such as spinal deformity. The progression of hip joint pathology was in accordance with their initial status according to the Gross Motor Function Classification System (GMFCS) and was not related to surgery itself. There was no deterioration of sphincter function seen. We observed that the reduction of lower limb muscle tone was long lasting. The patients have no recurrence of lower limb spasticity after surgery for a mean follow up of 6 years. We found that these patients experienced substantial reduction in spasticity after SDR, as documented by a marked reduction in Modified Ashworth Score of the lower limbs when the baseline Modified Ashworth Score was compared with findings 12 months after SDR. There was significant improvement in combined hip abduction range with the knee in extended position (R2) and in selective control scoring. The patients exhibited significant improvement in Gross Motor Function Measure total score and in dimensional scores in crawling, kneeling, walking, running, and jumping after selective dorsal rhizotomy plus physiotherapy. Improvements in walking were also reflected by significant improvements in Observational Gait Scores. Changes and improvement in instrumental gait analysis and oxygen consumption were also observed<sup>6,7</sup>. Patients usually developed transient lower limb weakness after SDR. The weakness might last for 4-8 weeks and the GMFM also dropped during this period of time. In all patients, the weakness recovered after physiotherapy and training. Most of them showed improvement in

GMFM after two to three months of intensive physical therapy.

Our orthopaedic colleagues will look into the musculoskeletal issues of spastic CP patients. For post SDR patients, the orthopaedic surgeons would decide on the appropriate intervention such as iliopsoas release, heel cord release, derotational osteotomy, foot/ankle stabilisation procedures, and muscle transfer procedures at around a year after SDR. We believe musculoskeletal surgery and SDR are complementary procedures with additional benefits over ambulatory function and will enhance the quality of life of our patients. It has been shown that SDR performed in patients at a young age – 2 to 4 years old – can prevent subsequent soft tissues and joint deformity and reduces the need of future orthopaedic intervention<sup>8</sup>.

Around 50% of our surgical candidates have abnormal urodynamic study such as detrusor instability and hyper-reflexic bladder before SDR. None reported any deteriorating urinary symptoms and signs after SDR. Fifty percent of patients with detrusor instability, as confirmed by a pre-SDR urodynamic study, demonstrated improvements in their urinary symptoms and signs after SDR. This finding has been confirmed in some patients with repeated urodynamic studies.

## Conclusion

We conclude that selective dorsal rhizotomy, together with intensive postoperative physiotherapy improve the lower limb muscle tone, range of motion across joints and leads to subsequent improvements of lower limb ambulatory function. SDR is a safe procedure and operation-related complications are uncommon. A multidisciplinary team capable of providing the whole spectrum of spasticity treatments is mandatory for quality management and care of patients suffering from spastic cerebral palsy.

## References

1. Winnie KL Yam, Sophelia HS Chan. Prevalence study of cerebral palsy in Hong Kong children. *Hong Kong Med J* 12:180-4
2. Peacock WJ, Staudt LA. Functional outcomes following selective posterior rhizotomy in children with cerebral palsy. *J Neurosurg* 1991;74:380-85.
3. McLaughlin JF, Bjornson KF, Astley SJ, et al. The role of selective dorsal rhizotomy in cerebral palsy: Critical evaluation of a prospective clinical series. *Dev Med Child Neurol* 1994;36:755-69.
4. Steinbok P. Outcomes after selective dorsal rhizotomy for spastic cerebral palsy. *Childs Nerv Syst* 2001;17:1-18.
5. Park TS, Johnston JM: Surgical techniques of selective dorsal rhizotomy for spastic cerebral palsy. *Neurosurgical Focus* 21: Issue 2, August 2006
6. K Y Yam, Dawson Fong. Selective Posterior rhizotomy: results of five pilot cases *Hong Kong Medical Journal* (1999; 5:287-290).
7. Sophelia H S Chan, K Y Yam. Selective Dorsal Rhizotomy in Hong Kong: Multidimensional Outcome Measures. *Paediatric Neurology* (2008;39:22-32).
8. Donncha F. O'Brien, T S Park. A review of orthopedic surgeries after selective dorsal rhizotomy. *Neurosurg Focus* 21 (2):E2, 2006



# Physiotherapy in Spasticity Management for Children with Cerebral Palsy

Ms. Nerita NC CHAN

MSc

Physiotherapy Department, Tuen Mun Hospital



Ms. Nerita NC CHAN

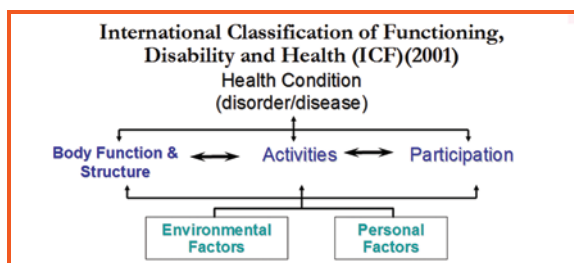
## Introduction

Spasticity is a major challenge for patients with neurological problems. Children with cerebral palsy (CP) always encounter different degrees of movement disorder in the presence of spasticity. However, spasticity is not all evil for those with neurological deficits<sup>1</sup>. It could act as a brace to support the individual's weight for transferring or walking. Therefore, a detailed assessment and motion analysis is essential in generating a specific goal for spasticity management that should be started as early as possible to prevent irreversible changes in musculo-skeletal system which will further distort the biomechanics of movement.

The role of physiotherapists in assessing function, defining disability, undertaking biomechanical assessment and providing mobility aids/casting/orthoses and motor training/stretching exercises is critical for the success of medical and surgical interventions for spasticity management.

## Spasticity and Functions

More than 80% of children with cerebral palsy in Hong Kong belong to the spastic type<sup>2</sup>. Spasticity prevents or limits the development of motor function. Management of spasticity for children with CP aims at improving the biomechanical alignment of the body and in turn improves functions. According to the International Classification of Functioning, Disabilities and Health (ICF)<sup>3</sup>, a treatment goal should be set at improving the Activity and Participation level of patients. Each party may have different goals - able to move by whatever means such as rolling, crawling or creeping and to reach out for toys may be the first goal for a child; to be able to walk and start schooling is the most important for parents while for health care professionals, it is most imperative to prevent deformity and delay surgical intervention. All these goals should be aligned to generate a holistic and comprehensive treatment regime for the children with CP.



## Role of Physiotherapy in Spasticity Management

There are 2 components of hypertonia - neurogenic and biomechanical. The neurogenic component refers to the overactive muscle contraction and biomechanical component the stiffening and shortening of the muscle and soft tissue. If these are left untreated, a vicious cycle is triggered off by the unopposed contraction of spastic and dystonic muscle groups leading to an abnormal limb posture, and in turn worsen soft tissue shortening and biomechanical changes in the contracted muscles. This further prevents muscle lengthening and perpetuates tonic<sup>4</sup>. There is indeed no time to lose. At our first assessment, we should confirm that spasticity is hindering functions. Secondly, the pattern of spasticity is analysed to see if it is generalised, focal or multi-focal.

Massage, myofascial release and acupressure are manual techniques that can reduce tone immediately. Passive stretching programmes, splintage and positioning are all essential in prevent deterioration of body alignment. Facilitation of active control of the limbs and strengthening exercises to the trunk and limb muscles are essential in promoting functional movement of children with CP.

If physical treatment alone is not sufficient to overcome the increased muscular tone or its mechanical consequences, particularly in moderate to severe spasticity, medical treatment and other interventions should be considered early. Intramuscular botulinum toxin injection is a treatment of choice for focal spasticity. Its effect lasts 2 to 3 months during which there is better motor control and allows intensive therapy. Functional gain can be observed even after the effect wears off<sup>5</sup>.

Physiotherapists also have a major role in selecting candidates for selective dorsal rhizotomy (SDR) and to lead these children throughout the long course of intensive physiotherapy that follows.

## Early Intervention with Botulinum Toxin

How early should we intervene? How do we know if spasticity is interfering or facilitating functions? Most health care professional or carers target at standing and walking while ignoring the important role of crawling for kids with which they explore the environment and



learn from it. Crawling exercise builds up both core and girdle muscles<sup>6</sup> in preparing for a better development of gross motor function. Patients need good power and control in the shoulder girdle so that they can move in and out between 4 point kneeling and sitting position, to rotate body on sitting posture with one arm support and to reach for toys. The core muscles and the pelvic control are important in preparing for standing and walking. Therefore, treatment to improve the upper limbs weight bearing and hip dissociation may be the first goal that needs to be addressed. Spastic muscles over the upper limbs such as the biceps, brachialis, flexor carpi ulnaris, pronator teres and quadratus may be examined and considered for focal injection with botulinum toxin to improve the elbow and wrist extension for better weight bearing during crawling. To promote the lower limbs movement during crawling, hip adductors and iliacus may also be considered for injection.



**Sitting** is also an area that we need to emphasise for those with spastic diplegia. Ability to sit stably and play with toys facilitates learning. Mothers would be happy to see a baby sit properly for feeding and a good alignment in sitting prevents spinal deformity. Spasticity of lower limb muscles - hamstrings, hip adductors and iliopsoas - hampers sitting posture. These kids either sit with a round back (photo 1) because of the pull from the hamstrings muscle or they compensate with "W" sitting posture (kneel sit) so as to alleviate the pull from the spastic muscles. Botulinum toxin to proximal muscles of the lower limbs such as hamstrings, hip adductors or iliacus will help to adjust the spinal alignment while sitting and with proper sitting posture, trunk muscles could be effectively trained.

Most of the previous studies concentrated on the result of spasticity management to lower limb muscles and its relation to walking<sup>7,8,9,10</sup>. The use of outcomes measure is similar in each study. In both domains of activity and participation, they used Gross Motor Function Measures (GMFM)<sup>11</sup> to measure changes in motor function. Three dimensional (3- D) gait analysis<sup>12</sup> or the Physician Rating Scale (PRS)<sup>13</sup> by the use of video recording were also common tools in measuring changes in gait pattern. However, seldom would studies mention the early intervention of botulinum toxin to prevent the deformity of the foot arches. Children with spastic diplegia always have increased muscle tone over both calf muscles which cause equinus gait pattern with

collapse of medial foot arches. Early botulinum toxin injection of the gastro-soleus muscle followed with casting can effectively prevent and delay the deformity of the feet. This should be done as early as weight bear and before independent walking when they are aged around one to two.

When a patient is able to walk, with or without aids, at level I, II and III of Gross Motor Function Classification System for Cerebral Palsy (GMFCS I, II and III)<sup>14</sup>, 3-D gait analysis is useful in documenting the fine changes in lower limbs kinematics and kinetics. It is also helpful in analysing the movement pattern and deciding which muscle groups need botulinum toxin injection, at a single or multiple levels. A prospective study at Tuen Mun Hospital concluded that botulinum toxin is useful in improving the gait pattern and GMFM, most effectively within 3 months after injection<sup>15</sup>.



Photo 1

## Multilevel Botulinum Toxin Injection

Walking involves a sequence of muscle contraction and relaxation and in children with CP, weakness, spasticity, poor control, incoordination and impaired proprioception come into play. A detailed examination gives physiotherapists a general picture of limb function so that different muscle groups that require botulinum toxin injection could be mapped out and an appropriate treatment plan formulated. Recent study showed that multilevel botulinum toxin is effective in managing the problem in children with lower limb spasticity<sup>8,16</sup>.

A typical deviated gait pattern is the flexion of the knee during midstance<sup>17</sup>, either crouch pattern or jump knee pattern<sup>18</sup>. This is often caused by muscle imbalance resulting from a combination of spasticity of flexor muscle (iliopsoas, hamstrings) and weakness of extensor (glutei, quadriceps, gastro-soleus) leading to fixed muscle contractures during development and further deterioration in mobility<sup>19</sup>. After thorough examination and confirmation with gait analysis, muscles targeted for injection are usually the iliopsoas, hamstrings and gastrocnemius. Soleus is skipped if true crouch pattern (dorsiflexion of ankle joint in midstance) is noted to avoid further weakening the muscle.

After injection, intensive physiotherapy, serial casting followed by orthoses are indicated for optimal effects on the muscle strength and length<sup>20</sup>. The change in



gait pattern is expected with better knee extension in midstance.

## Selective Dorsal Rhizotomy

After a series of tone management with intensive physiotherapy punctuated with botulinum toxin injections, the child would probably be around 4 to 5 years old and selective dorsal rhizotomy (SDR) can be considered. A suitable candidate for SDR is typified by 6 S's –

- Spastic** - spasticity is still a problem;
- Strong** - good strength of lower limbs and trunk muscles;
- Straight** - able to stand straight with good alignment;
- Slim** - not too fat in body build;
- Smart** - intellectually good enough for carrying out training;
- Support** - best result for one with supportive family and carer for postoperative intensive training<sup>21</sup>.

Those with severe dystonia, dyskinesia or significant fixed orthopaedic deformity are not suitable candidates.

The team at NTWC\* has, to date, done the largest number of SDR in Hong Kong. Separate teams of physiotherapists are involved with the assessment and the actual training of patients to avoid bias in our prospective study. Under the domain of body structure and function according to ICF, we documented the changes of muscle tone, range of motion, strength and the selective control of the lower limbs. We perform gait analysis, oxygen consumption test, GMFM to measure the change in activity level. On participation level, Canadian Occupational Performance Measure (COPM) - a family-centred tool that guides participants to identify difficulties in their self-care abilities, school participation, and leisure activities<sup>22</sup> - and Paediatric Evaluation of Disability Inventory (PEDI) are used. Our result is promising and has been published<sup>21</sup>.

## Summary

Spasticity management for children should be started as early as possible. Expertise in movement analysis and multidisciplinary collaboration in defining a practicable goal based on the ICF framework is essential in achieving good outcome. Timely intensive physiotherapy training is a key to success.

\*NTWC – *New Territories West Cluster consists of Tuen Mun Hospital, Pok Oi Hospital and Castle Peak Hospital under the Hospital Authority*

## References

1. Boyd, R. N. and Graham, H. K. (1999), Objective measurement of clinical findings in the use of botulinum toxin type A for the management of children with cerebral palsy. *European Journal of Neurology*, 6: s23–s35.
2. Yam, KL and Chan, HS (2006), Prevalence study in Cerebral Palsy in HK Children. *Hong Kong Medical Journal*, 12:3:180-184
3. Dahl, TH (2002), International Classification of Functioning, Disability and Health: An introduction and Discussion of its Potential Impact on Rehabilitation services and research. *Journal of Rehabilitation Medicine*: 34: 201-204
4. Royal College of Physicians, British Society of Rehabilitation Medicine, Chartered Society of Physiotherapy Association of Chartered Physiotherapists Interested in Neurology, Spasticity in adults: management using botulinum toxin . National guidelines. London: RCP, 2009
5. Chan NC, Chan SH, Kwong K L, Chak WK. A Seven Years Review on the Effect of Botulinum toxin A injection for children with Cerebral Palsy. *Hong Kong Hospital Authority Convention 2007*. (Poster Presentation)
6. Queiroz Bc, Caqlari MF, Amorim CF, Sacco IC (2010), Muscle activation during four Pilates core stability exercises in quadruped position. *Archives of Physical Medicine and Rehabilitation*, Jan: 91(1): 86-92
7. Ryll U, Bastiaenen C, De Bie R, Staal B (2011) Effect of leg muscle botulinum toxin A injection on walking in children with spasticity-related cerebral palsy: a systematic review. *Developmental Medicine & Child Neurology*, Mar 53(3):210-6
8. Scholtes VA, Dallmeijer AJ, Knol DI et al (2006). The combined effect of lower-limb multilevel botulinum toxin type A and comprehensive rehabilitation on mobility in children with cerebral palsy: a randomized clinical trial. *Archives of Physical Medicine and Rehabilitation*, 87:1551-8
9. Reddihough DS, King JA, Coleman GJ et al (2002). Functional outcome of botulinum toxin A injections to the lower limbs in cerebral palsy. *Developmental Medicine & Child Neurology*, 44:820-7
10. Steenbeck D, Meester-Delver A, Becher JG, Lankhorst GJ (2005). The effect of botulinum toxin type A treatment of the lower extremity on the level of functional abilities in children with cerebral palsy: evaluation with goal attainment scaling. *Clinical Rehabilitation*; 19:274-82
11. Flett PJ, Stern LM, Waddy H, Connell TM, Seeger JD, Gibson SK (1999). Botulinum toxin A versus fixed cast stretching for dynamic calf tightness in cerebral palsy. *Journal of Paediatric Child Health*; 35: 71-7
12. Corry IS, Cosgrove AP, Duffy CM, Mcneil S, Taylor TC, Graham HK (1998). Botulinum toxin A compared with stretching casts in the treatment of spastic equinus: a randomized prospective trial. *Journal of Pediatric Orthopedic*; 18: 304-11.
13. El-Etribi MA, Salem ME, El-Shakankiry HM, El-Kabky AM, El-Mahboub SM (2004). The effect of botulinum toxin A injection on spasticity, range of motion and gait patterns in children with spastic diplegic cerebral palsy: an Egyptian study. *International Journal of Rehabilitation Research*; 27:275-81
14. Wood, E. and Rosenbaum, P. (2000), The Gross Motor Function Classification System for Cerebral Palsy: a study of reliability and stability over time. *Developmental Medicine & Child Neurology*, 42: 292-296
15. Chan NC (2003). Botulinum Toxin A injection to children with cerebral palsy: A Local experience. *Brainchild*. Hong Kong Society of Child Neurology and Developmental Paediatrics; 4 (1): 5-8
16. Scholtes VA, Dalimeijer AJ, Knol DL et al (2007). Effect of multi-level botulinum toxin A and comprehensive rehabilitation on gait in cerebral palsy. *Pediatric Neurology*; 36:30-9
17. Wren TA, Rethlefsen S, Kay RM (2005). Prevalence of specific gait abnormalities in children with cerebral palsy: influence of cerebral palsy subtype, age, and previous surgery. *Journal of Pediatric Orthopedic*; 25:79-83
18. Gage JR, editor.(2004). *The treatment of gait problems in cerebral palsy*. London: Mac Keith Press
19. Johnson DC, Damiano DL, Abel MF (1997). The evolution of gait in childhood and adolescent cerebral palsy. *Journal of Pediatric Orthopedic*; 17:392-6
20. Leach J (1997). Children undergoing treatment with botulinum toxin: the role of the p6: 194-207 physical therapist. *Muscle Nerve Suppl*;
21. Chan HS, Yam KY, Chan NC, Lau PH, Poon YC, Cheung HM, Chak WK (2008). Selective Dorsal Rhizotomy in Hong Kong: Multidimensional Outcome Measure. *Paediatric Neurology*, Vol 39, issue 1, p22-32, 2008
22. Law M, Baptiste S, Carswell A, McColl MA, Polatajko H, Pollock N (1994). *Canadian Occupational Performance Measure 2nd edition*. Toronto: CAOT publication: 1-58.

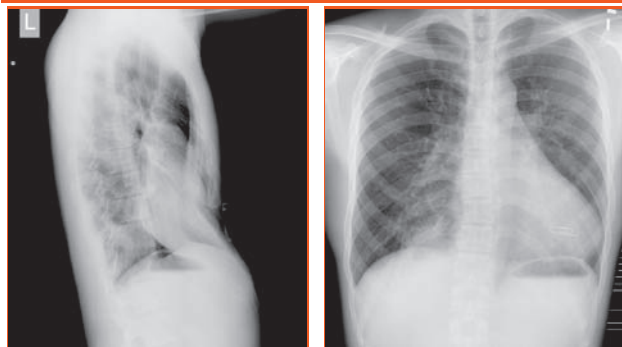




## Radiology Quiz

Dr. Vince LAU

Department of Radiology, Queen Mary Hospital




### Questions:

1. What are the findings?
2. What is the diagnosis?
3. What is the treatment?

(See P.37 for answers)

Enhancing the practice of primary care physicians as our goal to serve the medical profession and the Society



THE UNIVERSITY OF HONG KONG  
LI KA SHING FACULTY OF MEDICINE

香港大學李嘉誠醫學院

## Postgraduate Diploma in Diagnosis and Therapeutics in Internal Medicine (PDipIntMed&Therapeutics)

醫學內科診斷及治療深造文憑

*Approved by  
Medical Council as  
quotable qualification*

**PROGRAM FEES**  
Composition fee for the 2 year program is HK\$23,000 (subject to approval)

**ADMISSION REQUIREMENTS**  
Holder of a primary medical degree with post registration experience of not less than 12 months

**DEADLINE OF APPLICATION**  
31 August 2011

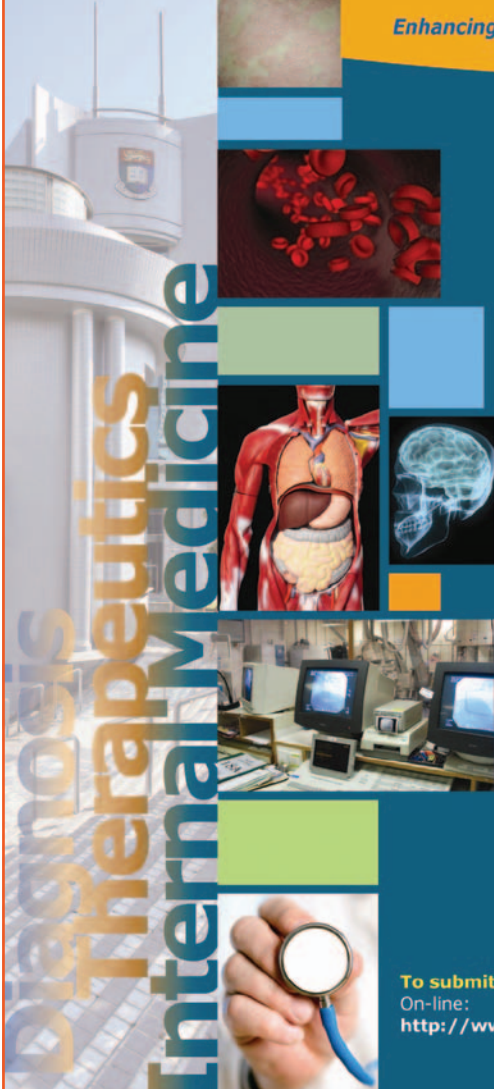
**VENUE**  
William MW Mong Block  
Faculty of Medicine Building  
21 Sassoon Road  
Pok Fu Lam, Hong Kong

**ORGANIZER**  
Department of Medicine  
The University of Hong Kong  
Queen Mary Hospital  
Hong Kong

**To submit an application:**  
On-line:  
<http://www.hku.hk/medicine/postdip.htm>

Call for Application  
for Admission  
in September 2011

Diagnosis  
Therapeutics  
Internal Medicine



# Computerised Gait Analysis

**Ms. Miko LM LAO**

M.Phil (Bioengineering), Pg.D (Epidemiology & Biostatistics)  
Manager, Centre of Gait and Motion Analysis, NTWC



Ms. Miko LM LAO

## Introduction

Computerised gait analysis (CGA) is a systematic evaluation of locomotion by which gait characteristics are measured, abnormalities identified, causes suggested and treatments formulated. It is not intended to replace clinical examinations, but as an adjunct to understand the impairment better. The treatment decision should be made in full consideration of the patient's condition, physical examination and medical history.

## Clinical use of computerised gait analysis

To date, CGA has its greatest clinical value as a test for individuals with central nervous disorders associated with spasticity, especially children with cerebral palsy (CP). To increase mobility and prevent deformity, various medications, non-surgical therapy regimens, bracing, assistive devices, and/or orthopaedic and neurosurgical procedures are prescribed for these children. In the past, many orthopaedic procedures were performed separately during a child's growing years. But it is far more desirable to perform multiple orthopaedic procedures at a single surgical session. This approach avoids the psychological impact of multiple separate procedures, optimises functional improvement by using a single operation, and reduces the medical costs. CGA offers objective measurements not provided by clinical examinations and helps clinicians to select the most appropriate procedure.

Many prospective and retrospective studies were published regarding the utilisation of CGA in clinical decisions for children and adolescents with cerebral palsy<sup>1, 3-4, 6-8, 10-12</sup>. These studies demonstrated that CGA plays a role both in confirming or refuting indications for surgery or in delaying it.

Wren et al compared the clinical course of a group of 313 children who had undergone CGA with another non-CGA group of 149 children<sup>14</sup>. After adjusting for differences in age and severity of functional problems, it was found that the CGA group had more distinct procedures during the initial surgery than the non-CGA group. Only 11% of the CGA group children needed additional surgery in contrast to 32% of the non-CGA group.

## Role of CGA in NT West Cluster\*

Centre of Gait and Motion Analysis at Tuen Mun

Hospital provides a full spectrum of gait and motion analysis services including physical examination, video records, temporal-distance data, three dimensional joint kinematics and kinetics results, electromyography data, balance ability and metabolic energy expenditure, to patients suffering from a variety of diseases.

### Physical Examination

Measurements are made of the patient at rest. They include passive joint range of motion, joint contracture, muscle strength and tone, bony deformity and neurological assessment. (Fig 1) This information is then correlated with the CGA data to help determine the potential causes of the gait deviations.



Figure 1. Physiotherapist measures the passive range of motion

### Video Records

Specialised computer-interfaced video cameras measure patient motion. An initial video clip provides qualitative documentation of how a patient walks. Close-up views of a specific motion and recording in slow motion allow the observer to evaluate the walking pattern. For example, close-up views of the feet provide a means to evaluate hind foot position and motion. (Fig 2)



Figure 2. Close-up view shows the foot position and motion.





**Temporal-distance Data**

We look at velocity, cadence, stride length, step length and percentage of stance/swing. These measurements of functional level allow comparison with subsequent progress.

**3D Joint Kinematics**

Markers with retro-reflective surface are placed on the patient's skin, aligning with specific bony landmarks and joints. (Fig 3) As the patient walks along a straight pathway in the laboratory, infrared beams reflected from these markers are tracked by eight high-speed cameras, all interfaced with a central computer to generate 3D motions trajectories of these markers. Computation of the marker position data gives us the angular orientation of particular body segments as well as the angles between segments (joint angles). (Fig 4)

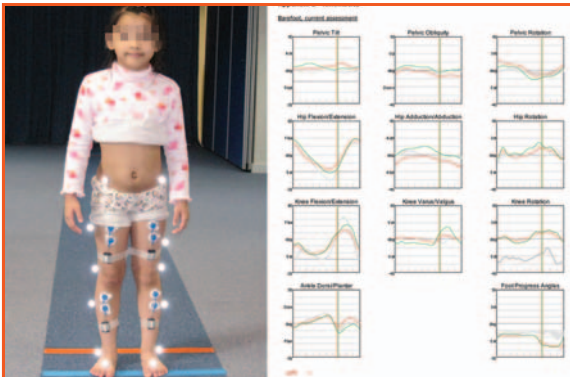


Figure 3. Retro-reflective markers are attached to patient.

Figure 4. joint kinematics represented in graphic form which is more easily to be understood.

**3D Joint Kinetics**

Multicomponent force platforms imbedded in the walkway provide measurement of reaction between the foot and the ground as the patient walks over them. (Fig 5) The mechanics of walking can thus be further analysed. The data can be assessed directly or used to calculate the load in and across the joints. We can then tell how much load each joint is producing while the patient walks (joint moments and joint powers). (Fig 6)

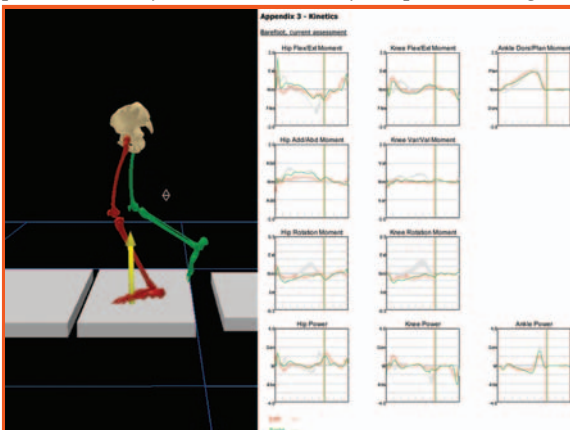


Figure 5. Ground reaction force acts on the patient

Figure 6. joint kinetics represented in graphic form which is more easily to be understood.

**Electromyography**

Wireless electrodes placed on the surface of specific muscles give us dynamic electromyography (EMG). (Fig 7) This technique measures the electrical potential generated by the muscle when it is activated. It discerns whether the muscles are contracting in a concerted manner to produce the required forces, if the timing of these contractions is appropriate or do the muscles need to be strengthened or are some pulling too hard because of spasticity. This information can be used with joint kinematics and kinetics results to understand better the subject's neuromuscular abnormalities. (Fig 8)

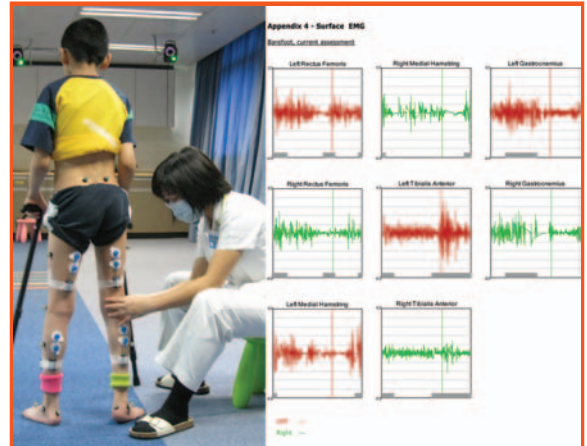


Figure 7. Wireless sensors are attached to the patient to record muscle activities.

Figure 8. Prolonged muscle activities are shown.

**Balance Ability**

Loss of coordination or balance is often observed in patients with neurological disease. The severity of this deficit at presentation and after treatment is often subjective. Advanced biomechanical technology equipped in our Centre provides quantitative data to complement standard clinical assessment. The force platform detects changes of postural sway by assessing the ground-reaction forces. (Fig 9) These ground-reaction forces are used to calculate the centre of pressure (COP)<sup>5</sup>. Maximum COP displacements in anterior-posterior and mediolateral direction can be used to quantify postural instability<sup>2,9</sup>.



Figure 9. Patient stands on the force platform. Her COP can be calculated. Exaggerated COP displacement indicates poor balance instability.



**Energy Expenditure**

Energy consumption during walking provides an estimate of the overall ambulatory status of the patient. (Fig 10) Patients with movement disorders spend more energy in walking. Oxygen consumption is therefore a good measurement of the severity of such disability and can be used to gauge subsequent improvement after treatment.



*Figure 10. A small breathing mask is worn to measure the oxygen consumption.*

An interdisciplinary team composed of neurosurgeons, orthopaedic surgeons, neurologists, physiotherapists and bioengineers will then review all these data and formulate a treatment recommendation (Fig. 11) consisting of a combination of physiotherapy, medications, bracing and surgery.

<b>Gait Analysis Report</b>	
Name:	
HKID:	
Date of Birth:	
Date of Analysis:	
<b>G. Discussion and Recommendations</b>	
<i>Observational gait analysis</i>	
Patient usually walks with knee walker in school. She walks with trunk forward lean, the hip extension is limited with slightly knee crouch. Both feet are in equinus position throughout the gait cycle. Dragging of feet is severe especially on left side.	
With the use of quadripods, patient walks with more asymmetrical pattern especially in trunk and pelvic rotation, crouch pattern is getting worse. Moreover, the speed is much slower than that with knee walker.	
<i>Instrumented gait analysis</i>	
In kinematic, anterior pelvic tilt is noted. Hip extension is limited over both sides. Crouch knee pattern is detected in stance phase and stiff knee pattern is slightly improved as compared with 1 year before. Dragging of both feet persist with more severe on left side. In transverse plane, the pelvic rotation is more obvious as compared to 1 year ago.	
In EMG, the firing of rectus is more prominent in swing phase with more severe on left side.	
<i>Energy consumption</i>	
The O2 cost is 10 times of age matched normal. There is 27.3% increase as compared with last year assessment.	
<i>Recommendations</i>	
Orthopedic intervention:	
1. Hip - bilateral psoas release over pelvic trim, bilateral adductor longus release, ? rectus release proximal L (based on the abnormal firing during swing phase)	
2. Knee - bilateral semi-T to adductor magnus transfer, gracilis release +/- semiM (need to repeat testing of biceps femoris power)	
3. Ankle - R side: gastroc slide and tibialis posterior intramuscular slide. L side: gastroc slide + split tibialis posterior transfer (? through IOH)	
Dr. K.Y. Yam Consultant Department of Neurosurgery	Ms. Nanta Chan Physiotherapist I Department of Physiotherapy

*Figure 11. Recommendation is made by an interdisciplinary team.*

In summary, computerised gait analysis is vital for the evaluation of pathological gait in a more objective fashion. Its beauty lies in the integration of many different analyses to arrive at a comprehensive assessment of gait pattern.

*\*NT West Cluster consists of Tuen Mun Hospital, Pok Oi Hospital and Castle Peak Hospital under the Hospital Authority*

**References**

1. Chang FM, Seidl AJ, Muthusamy K, Meiningner AK, Carollo JJ. Effectiveness of instrumented gait analysis in children with cerebral palsy--comparison of outcomes. J Pediatr Orthop. 2006 Sep-Oct;26(5):612-6.
2. Corriveau H, Hebert R, Prince F, Raiche M. Postural control in the elderly: an analysis of test-retest and interrater reliability of the COP-COM variable. Arch Phys Med Rehabil. 2001 82(1):80-5.
3. DeLuca PA, Davis RB 3rd, Öunpuu S, Rose S, Sirkin R. Alterations in surgical decision making in patients with cerebral palsy based on three-dimensional gait analysis. J Pediatr Orthop. 1997 Sep-Oct;17(5):608-14.
4. Filho MC, Yoshida R, Carvalho Wda S, Stein HE, Novo NF. Are the recommendations from three-dimensional gait analysis associated with better postoperative outcomes in patients with cerebral palsy? Gait Posture. 2008 Aug;28(2):316-22.
5. Goldie PA, Bach TM, Evans OM: Force platform measures for evaluating postural control: reliability and validity. Arch Phys Med Rehabil. 1989 70:510-517.
6. Gough M, Shortland AP. Can clinical gait analysis guide the management of ambulant children with bilateral spastic cerebral palsy? J Pediatr Orthop. 2008 Dec;28(8):879-83.
7. Kawamura CM, de Moraes Filho MC, Barreto MM, de Paula Asa SK, Juliano Y, Novo NF. Comparison between visual and three-dimensional gait analysis in patients with spastic diplegic cerebral palsy. Gait Posture. 2007 Jan;25(1):18-24.
8. Kay RM, Dennis S, Rethlefsen S, Reynolds RA, Skaggs DL, Tolo VT. The effect of preoperative gait analysis on orthopaedic decision making. Clin Orthop. 2000 Mar;(372):217-22.
9. Lafond D, Corriveau H, Hebert R, Prince F: Intrasession reliability of center of pressure measures of postural steadiness in healthy elderly people. Arch Phys Med Rehabil. 2004 85(6):896-901.
10. Lofterød B, Terjesen T, Skaaret I, Huse AB, Jahnsen R. Preoperative gait analysis has a substantial effect on orthopedic decision making in children with cerebral palsy: comparison between clinical evaluation and gait analysis in 60 patients. Acta Orthop. 2007 Feb;78(1):74-80.
11. Lofterød B, Terjesen T. Results of treatment when orthopaedic surgeons follow gait-analysis recommendations in children with CP. Dev Med Child Neurol. 2008 Jul;50(7):503-9.
12. Molenaers G, Desloovere K, Fabry G, De Cock P. The effects of quantitative gait assessment and botulinum toxin A on musculoskeletal surgery in children with cerebral palsy. J Bone Joint Surg Am. 2006 Jan;88(1):161-70.
13. Onambele' GL, Maganaris CN, Mian OS, Tam E, Rejc E, McEwan IM, Narici MV. Neuromuscular and balance responses to flywheel inertial versus weight training in older persons. Journal of Biomechanics. 2008 41:3133-3138.
14. Wren TA, Woolf K, Kay RM. How closely do surgeons follow gait analysis recommendations and why? J Pediatr Orthop B. 2005 May;14(3):202-5.



# Certificate Course on Respiratory Medicine 2011

Jointly organised by



The Federation of Medical  
Societies of Hong Kong  
香港醫學組織聯合會



Hong Kong  
Thoracic Society  
香港胸肺學會



AMERICAN COLLEGE OF  
CHEST  
PHYSICIANS  
HONG KONG AND MACAU CHAPTER LIMITED  
美國胸肺學院  
(港澳分會)

## Objectives:

To enhance knowledge of the participants on the interpretation of common respiratory investigations, and to update the practical management of common respiratory problems.

**7 Sep 2011**

**Topic :** A comprehensive review of lung function testings

**Speaker :** Dr. Johnny Wai-man CHAN  
陳偉文醫生

**14 Sep 2011**

**Topic :** Polysomnography – hook-up, acquisition and interpretation

**Speaker :** Dr. Jamie Chung-mei LAM  
林頌眉醫生

**21 Sep 2011**

**Topic :** Collaborative care for COPD patients in community

**Speaker :** Dr. Kahlin CHOO  
俞佳琳醫生

**28 Sep 2011**

**Topic :** Occupational lung diseases in Hong Kong

**Speaker :** Dr. Henry Kai-him KWOK  
郭啟謙醫生

**12 Oct 2011**

**Topic :** Radiological imaging in respiratory medicine

**Speaker :** Dr. Thomas Yun-wing MOK  
莫恩榮醫生

**19 Oct 2011**

**Topic :** Mechanical and non-invasive ventilation

**Speaker :** Dr. Wai-ming CHAN  
陳惠明醫生

**Dates :** 7 September – 19 October 2011 (Every Wednesday)

**Time :** 7:00 p.m. – 8:30 p.m.

**Venue :** Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong

**Language Media :** Cantonese (Supplemented with English)

**Course Fee :** HK\$750 (6 sessions)

**Certificate :** Awarded to participants with a minimum attendance of 70%

**Enquiry :** The Secretariat of The Federation of Medical Societies of Hong Kong

**Tel.:** 2527 8898

**Fax:** 2865 0345

**Email:** [info@fmshk.org](mailto:info@fmshk.org)

## CME / CPD Accreditation in application

A total of 9 CNE points for the whole course and the points will be awarded according to the number of hours attended. Application form can be downloaded from website: <http://www.fmshk.org>



## The Federation Presidents' and Editors' Dinner 2011

The Federation Presidents' and Editors' Dinner 2011 was successfully held on 13th June, 2011 at the Hong Kong Club. It was a great occasion to meet the presidents of the member societies and the editors of the Hong Kong Medical Diary for reunion and fraternity.

Our special thanks to the Meetings and Exhibitions Hong Kong of the Hong Kong Tourism Board and Swire Travel to be the supporting organisation of the Dinner. The evening was made most memorable with the delightful violin performance by a group of lovely young students from the Takako Nishizaki Violin Studio.

During the Dinner, the Federation's Executive Committee updated our work and future plans of activities. Our EXCO was also much encouraged by the positive comments and suggestions received from our member societies. Souvenirs were presented to the editors of the Medical Diary in expressing our heartfelt thanks for their great support and contributions.









Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
<ul style="list-style-type: none"> <li>* 2011 Paediatric Update No. 2 – Childhood Allergy</li> <li>* HKMA Dragon Boat Team Practice Session</li> </ul> <p><b>3</b></p>	<ul style="list-style-type: none"> <li>* An Unknown Complication for A Known Procedure</li> </ul> <p><b>4</b></p>	<ul style="list-style-type: none"> <li>* HKMA Tai Po Community Network – Acne Vulgaris – An Update</li> <li>* FMSHK Officers' Meeting</li> <li>* HKMA Council Meeting</li> </ul> <p><b>5</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Your Risk Workshop</li> </ul> <p><b>6</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Adverse Outcomes Workshop</li> </ul> <p><b>7</b></p>	<p><b>1</b></p>	<p><b>2</b></p>
<ul style="list-style-type: none"> <li>* HKMA Dragon Boat Team Practice Session</li> <li>* HKMA Certificate Course on Family Medicine 2011</li> </ul> <p><b>10</b></p>	<ul style="list-style-type: none"> <li>* HKMA Hong Kong East Community Network – Treatment beyond LDL in Real-life Practice</li> <li>* HKMA Annual General Meeting</li> </ul> <p><b>12</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Adverse Outcomes Workshop</li> <li>* Hong Kong Neurosurgical Society Monthly Academic Meeting – Normal Pressure Hydrocephalus</li> <li>* HKMA Central, Western &amp; Southern Community Network – Certificate Course on Psychiatry (Session 4)</li> </ul> <p><b>13</b></p>	<ul style="list-style-type: none"> <li>* HKMA Kowloon City Community Network – In Realty: What is the Implication in Reality</li> <li>* HKMA NT West Community Network – Lecture Series on BPH &amp; Urology – Practical Tips on Management for Primary Care Physicians</li> <li>* HKMA Structured CME Programme with Hong Kong Samaritan &amp; Hospital Year 2011 – Lymphadenopath</li> </ul> <p><b>14</b></p>	<ul style="list-style-type: none"> <li>* HKMA Kowloon City Community Network – UPPITER: What is the Implication in Reality</li> <li>* HKMA NT West Community Network – Lecture Series on BPH &amp; Urology – Practical Tips on Management for Primary Care Physicians</li> <li>* HKMA Structured CME Programme with Hong Kong Samaritan &amp; Hospital Year 2011 – Lymphadenopath</li> </ul> <p><b>15</b></p>	<ul style="list-style-type: none"> <li>* HKMA – KLN East Community Network; HA – UCH; HKCFP - CME Course for Health Personnel 2011</li> <li>* Hong Kong Surgical Forum – Summer 2011</li> </ul> <p><b>16</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Your Risk Workshop</li> </ul> <p><b>23</b></p>
<ul style="list-style-type: none"> <li>* HKMA Dragon Boat Team Practice Session</li> </ul> <p><b>17</b></p>	<ul style="list-style-type: none"> <li>* FMSHK Executive Committee Meeting</li> </ul> <p><b>19</b></p>	<ul style="list-style-type: none"> <li>* HKMA Central, Western &amp; Southern Community Network – Certificate Course on Psychiatry (Session 5)</li> </ul> <p><b>20</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Your Risk Workshop</li> </ul> <p><b>21</b></p>	<ul style="list-style-type: none"> <li>* HKMA Yau Tsim Mong Community Network – Latest Advances in Gastric Cancer Management</li> </ul> <p><b>22</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Your Risk Workshop</li> </ul> <p><b>29</b></p>	<ul style="list-style-type: none"> <li>* HKMA MPS CME – Mastering Your Risk Workshop</li> </ul> <p><b>30</b></p>
<ul style="list-style-type: none"> <li>* HKMA Summer Vigor Mini Dragon Boat Race</li> </ul> <p><b>24</b></p>	<p><b>25</b></p>	<p><b>26</b></p>	<ul style="list-style-type: none"> <li>* (1) Military Lung Lesion – What Else Could that be if not TB?</li> <li>(2) A Rapidly Growing Lung Mass</li> <li>* HKFMS Foundation Meeting</li> </ul> <p><b>28</b></p>	<ul style="list-style-type: none"> <li>* (1) Military Lung Lesion – What Else Could that be if not TB?</li> <li>(2) A Rapidly Growing Lung Mass</li> <li>* HKFMS Foundation Meeting</li> </ul> <p><b>28</b></p>	<p><b>29</b></p>	<p><b>31</b></p>





Date / Time	Function	Enquiry / Remarks
<b>3</b> 2:00 pm – 5:00 pm <b>SUN</b> 3:00 pm (10, 17)	<b>2011 Paediatric Update No. 2 – Childhood Allergy</b> Organiser: Hong Kong College of Paediatricians, Chairman: Dr. Sik-nin WONG, Speakers: Various, Venue: Hospital Authority Head Office M Floor, Lecture Theatre <b>HKMA Dragon Boat Team Practice Session</b> Organiser: The Hong Kong Medical Association, Venue: Sai Kung	Ms. Vanessa WONG Tel: 2871 8773 Fax: 2785 1850 3 CME Points Miss Alice TANG & Miss Sharon HUNG Tel: 2527 8285
<b>4</b> 7:30 pm – 8:30 pm <b>MON</b>	<b>An Unknown Complication for A Known Procedure</b> Organiser: Hong Kong Urological Association, Chairman: Dr. Wai-kit MA & Dr. Kim-chung TO, Speaker: Dr. Ka-fung CHIU, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Hing-hoi HUNG / Ms. Tammy HUNG Tel: 2958 6006 / 9609 6064 Fax: 2958 6076 / 8344 5115
<b>5</b> 1:45 pm <b>TUE</b> 8:00 pm – 10:00 pm 9:00 pm	<b>HKMA Tai Po Community Network – Acne Vulgaris – An Update</b> Organiser: HKMA Tai Po Community Network, Speaker: Dr. William Yuk-ming TANG, Venue: Tai Po <b>FMSHK Officers' Meeting</b> 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 <b>HKMA Council Meeting</b> Organiser: The Hong Kong Medical Association, Chairman: Dr. Kin CHOI, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Miss Candice TONG Tel: 2527 8285 1 CME Point Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345 Ms. Christine WONG Tel: 2527 8285
<b>6</b> 6:30 pm <b>WED</b> (23)	<b>HKMA MPS CME – Mastering Your Risk Workshop</b> Organiser: The Hong Kong Medical Association, Speaker: Dr. Ka-lam HAU, Venue: The Hong Kong Medical Association Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Department Tel: 2527 8452 2.5 CME Points
<b>7</b> 6:30 pm <b>THU</b> (13)	<b>HKMA MPS CME – Mastering Adverse Outcomes Workshop</b> Organiser: The Hong Kong Medical Association, Speakers: Dr. Justin Ngai-sing CHENG & Dr. Emily Chi-wan HUNG, Venue: The Hong Kong Medical Association Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Department Tel: 2527 8452 2.5 CME Points
<b>10</b> 2:00 pm <b>SUN</b>	<b>HKMA Certificate Course on Family Medicine 2011</b> Organiser: The Hong Kong Medical Association, Dr. Kit-kuen IP & Dr. Hak-hon HUNG, Venue: QEH	HKMA CME Department Tel: 2527 8452 3 CME Points
<b>12</b> 1:00 pm <b>TUE</b> 9:00 pm	<b>HKMA Hong Kong East Community Network – Treatment beyond LDL in Real-life Practice</b> Organiser: HKMA Hong Kong East Community Network, Chairman: Dr. Chi-lap AU, Speaker: Dr. Bernard Bun-lap WONG, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong <b>HKMA Annual General Meeting</b> Organiser: The Hong Kong Medical Association, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Miss Candice TONG Tel: 2527 8285 Ms. Christine WONG Tel: 2527 8285
<b>13</b> 7:30 am <b>WED</b> 1:00 pm (27)	<b>Hong Kong Neurosurgical Society Monthly Academic Meeting – Normal Pressure Hydrocephalus</b> Organiser: Hong Kong Neurosurgical Society, Chairman: Dr. Fung-ching CHEUNG, Speaker: Dr. Chi-hung YU, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon <b>HKMA Central, Western &amp; Southern Community Network – Certificate Course on Psychiatry (Session 4 &amp; 5)</b> Organiser: HKMA Central, Western & Southern Community Network, Chairmen: Dr. Ming-yuen LAM & Dr. Yim-kwai LAW, Speakers: Dr. Cindy Pui-yu CHIU & Prof. Dominic Tak-shing LEE, Venue: The Hong Kong Medical Association Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Dr. Gilberto LEUNG Tel: 2255 3368 Fax: 2818 4350 1.5 CME Points Mr. Alan LAW Tel: 2527 8285 1 CME Points
<b>14</b> 1:00 pm <b>THU</b> 1:00 pm 2:00 pm	<b>HKMA Kowloon City Community Network – JUPITER: What is the Implication in Reality</b> Organiser: HKMA Kowloon City Community Network, Chairman: Dr. Chu-wah CHIN, Speaker: Dr. Wing-bun CHAN, Venue: Spotlight Recreation Club, 4/F, Screen World, Site 8, Whampoa Garden, Hunghom, Kowloon <b>HKMA NT West Community Network – Lecture Series on BPH &amp; Urology Practical Tips on Urology for Primary Care Physicians</b> Organiser: HKMA NT West Community Network, Chairman: Dr. Aaron Fook-kay LEE, Speaker: Dr. Man-chiu CHEUNG, Venue: Plentiful Delight Banquet, Yuen Long, N.T. <b>HKMA Structured CME Programme with Hong Kong Sanatorium &amp; Hospital Year 2011 – Lymphadenopathy</b> Organiser: The Hong Kong Medical Association, Chairman: Bun-lap WONG, Speaker: Dr. Raymond H.S. LIANG, Venue: The Hong Kong Medical Association Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Candice TONG Tel: 2527 8285 Mr. Alan LAW Tel: 2527 8285 1 CME Point HKMA CME Department Tel: 2527 8452 1 CME Point
<b>16</b> 1:30 pm <b>SAT</b>	<b>HKMA – KLN East Community Network; HA – UCH; HKCFP - CME Course for Health Personnel 2011</b> Organiser: HKMA – KLN East Community Network, Chairman: Dr. Danny Ping-kwan MA, Speaker: Dr. Emily Wai-ho TANG, Venue: UCH <b>Hong Kong Surgical Forum – Summer 2011</b> Organiser: Department of Surgery, The University of Hong Kong; Queen Mary Hospital & Hong Kong Chapter of American College of Surgeons, Venue: Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong	Mr. Alan LAW Tel: 2527 8285 1.5 CME Points Forum Secretary, Hong Kong Surgical Forum Tel: 2255 4882 / 2255 4886 Fax: 2819 3416 Email: hksf@hku.hk Website: <a href="http://www3.hku.hk/surgery/forum.php">http://www3.hku.hk/surgery/forum.php</a>
<b>19</b> 8:00 pm – 10:00 pm <b>TUE</b>	<b>FMSHK Executive Committee Meeting</b> Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chambers, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345





Date / Time	Function	Enquiry / Remarks
<b>21 THU</b> 1:00 pm	<b>HKMA Yau Tsim Mong Community Network – Latest Advances in Gastric Cancer Management</b> Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr. Tai-yam LEE, Venue: Eaton Smart, Hong Kong	Miss Candice TONG Tel: 2527 8285
<b>24 SUN</b> 8:00 am	<b>HKMA Summer Vigor Mini Dragon Boat Race</b> Organiser: The Hong Kong Medical Association, Venue: Sai Kung	Miss Alice TANG & Miss Sharon HUNG Tel: 2527 8285
<b>28 THU</b> 6:30 pm – 8:00 pm	<b>(1) Miliary Lung Lesion – What Else Could that be if not TB?</b> <b>(2) A Rapidly Growing Lung Mass</b> Organiser: Hong Kong Thoracic Society / American College of Chest Physicians (HK & Macau), Chairpersons: Prof. David Shu-cheong HUI & Mr. Hok-sum CHAN, Speakers: Dr. So-shan NG & Dr. Dorothy Chung-ngar LIU, Venue: LG1, Lecture Room, Ruttonjee Hospital, Hong Kong	Dr. Fanny wai-san KO / Dr. Arthur chun-wing LAU Tel: 2632 2785
8:30 pm – 10:00 pm	<b>HKFMS Foundation Meeting</b> Organiser: The Federation of Medical Societies of Hong Kong and HKFMS Foundation Limited, Venue: Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345

## Upcoming Certificate Courses of the Federation of Medical Societies of Hong Kong

Date	Course No	Course Name	Target Participants	CME/CNE
7/7/2011 - 18/8/2011	C184	Certificate Course on Paediatric Nutrition	Medical, Health and Child-Care Professionals	9 CNE Points; CME/CPD Accreditation in application
12/7/2011 - 16/8/2011	C178	Certificate Course on Occupational Hygiene Practice	Healthcare Workers	9 CNE Points; CME/CPD Accreditation in application
5/8/2011 - 9/9/2011	C183	Certificate Course on Communication and Swallowing Problem in the Elderly Population	Paramedical Professional	9 CNE Points; CME/CPD Accreditation in application
7/9/2011 - 19/10/2011	C177	Certificate Course on Respiratory Medicine 2011	Nurses and Allied Health Professionals	9 CNE Points; CME/CPD Accreditation in application
8/9/2011 - 13/10/2011	C181	Certificate Course on Renal Medicine 2011	Medical and Health Professionals	9 CNE Points; CME/CPD Accreditation in application



## Society News

### News from Member Societies

Name of member societies	President	Hon. Secretary	Hon. Treasurer
American College of Chest Physicians (HK & Macau Chapter)	Dr. Johnny Wai-man CHAN	Dr. Chun-wing LAU	Dr. Chun-wing LAU
British Medical Association (HK Branch)	Dr. Raymond See-kit LO	Dr. Terry HUNG	Dr. Clarence LEUNG
College of Nursing, Hong Kong	Ms. June Wing-mui LUI	Ms. Ellen Wai-yin KU	Ms. Gloria Tao-ying HUNG
Hong Kong Association for Integration of Chinese-Western Medicine	Dr. Vivian Chi-woon WONG TAAM	CMP Ping-shun CHAN	CMP Hung-pun TAM
Hong Kong College of Cardiology	Dr. Chung-seung CHIANG	Dr. Kam-tim CHAN	Dr. Shu-kin LI
Hong Kong College of Chinese Medicinal Nursing	Mr. David Tai-wai LEUNG	Ms. Shuk-ling CHAN	Ms. Po-fung LAI
Hong Kong College of Emergency Medicine	Dr. Chor-chiu LAU	Dr. NG Fu	Dr. Yau-tak WONG
Hong Kong College of Radiologists	Dr. Chun-key LAW	Dr. Stephen C.W. CHEUNG	Dr. Cheuk-man TONG
Hong Kong Dental Association	Dr. Sigmund Sai-man LEUNG	Dr. Raymond Kin-man LEE	Dr. Vincent Fun-shing LEUNG
Hong Kong Paediatric Nephrology Society	Dr. Wai-ming LAI	Dr. Winnie Kwai-yu CHAN	Dr. Kwok-wai LEE
Hong Kong Physiotherapy Association Ltd.	Ms. Yee-hung POON	Ms. Kit-yin LAM	Mr. Chi-ming CHAN
Hong Kong Psychogeriatric Association	Dr. Siu-wah LI	Dr. Paulina Po-ling CHOW	Ms. Yuk-mui LAU
Hong Kong Society for Microbiology & Infection	Dr. Dominic N.C. TSANG	Dr. Vincent Chi-chung CHENG	Dr. Rebecca Kit-yee LAM
Hong Kong Society for Molecular Diagnostic Sciences Ltd.	Dr. Daniel Chuen-chu TAM	Mr. Wai-ting HUI	Dr. Wing-cheong YAM
Hong Kong Society for Nursing Education	Dr. Sharron Shuk-kam LEUNG	Mr. Edmond Tak-fai TONG	Dr. Vico Chung-lim CHIANG
Hong Kong Society of Certified Prosthetist - Orthotists	Dr. Aaron Kam-lun LEUNG	Mr. Wilson Yau-keung CHAN	Dr. Man-sang WONG
Hong Kong Society of Clinical Chemistry	Dr. Lap-kay LAW	Ms. Judy Po-shan LAI	Ms. Yan-ping IU
Hong Kong Society of Critical Care Medicine Ltd.	Dr. Wing-wa YAN	Dr. Alfred Yan-fat CHAN	Dr. Pik-kei CHAN
Hong Kong Society of Endocrinology, Metabolism and Reproduction	Dr. Peter Chun-yip TONG	Dr. Cheung-hei CHOI	Dr. Wing-yee SO
Hong Kong Society of Medical Genetics	Dr. Fai-man LO	Dr. Priscilla Miu-kuen POON	Mr. Wing-kwong CHAN

The FMSHK would like to send its congratulations to the new office-bearers and look forward to working together with the societies.



## Answer to Radiology Quiz

### Diagnosis:

Pectus excavatum.

### Findings:

-Indistinct right heart border.  
-Leftward displacement of heart with decreased heart density.

### Discussion:

Pectus excavatum (funnel chest) is a congenital chest wall deformity characterised by concave depression of the sternum. Compression of the heart causes characteristic findings on frontal CXR of an indistinct right heart border, decreased heart density and displacement of the heart to the left. The anterior ribs have an accentuated downward slope, giving rise to a "reverse 7" appearance with the horizontal posterior ribs.

The indistinct right heart border can mimic right middle lobe pathology but a lateral CXR confirms the sternal deformity. Pectus excavatum is usually an isolated anomaly but can be associated with Marfan's syndrome, Noonan's syndrome, foetal alcohol syndrome and homocystinuria.

Surgical repair is performed in severe cases, in which CT scan would be useful for detailed pre-op assessment.

**Dr. Vince LAU**

*Department of Radiology, Queen Mary Hospital*

**The Federation of Medical Societies of Hong Kong**  
4/F Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, HK  
Tel: 2527 8898 Fax: 2865 0345

<b>Patron</b>	The Honourable Donald TSANG, GBM	曾蔭權先生
<b>President</b>	Dr. LO See-kit, Raymond	勞思傑醫生
<b>1st Vice-President</b>	Prof. CHAN Chi-fung, Godfrey	陳志峰教授
<b>2nd Vice-President</b>	Dr. LO Sze-ching, Susanna	盧時楨醫生
<b>Hon. Treasurer</b>	Mr. LEE Cheung-mei, Benjamin	李祥美先生
<b>Hon. Secretary</b>	Dr. CHAN Sai-kwing	陳世炯醫生
<b>Executive Committee Members</b>	Dr. CHAN Chi-wing, Timmy Dr. CHAN Chun-kwong, Jane Dr. CHAN Hau-ngai, Kingsley Prof. CHIM Chor-sang, James Dr. HUNG Che-wai, Terry Ms. KU Wai-yin, Ellen Dr. LEUNG Ka-kit, Gilberto Dr. MAN Chi-wai Dr. MOK Chun-on Dr. NG Yin-ikwok Dr. WONG Mo-lin, Maureen Ms. YAP Woan-tyng, Tina Dr. YU Chau-leung, Edwin Dr. YUEN Shi-yin, Nancy	陳智榮醫生 陳真光醫生 陳厚毅醫生 詹楚生教授 洪致偉醫生 顧慧賢女士 梁嘉傑醫生 文志倫醫生 吳發強醫生 黃慕蓮醫生 葉婉婷女士 余秋良醫生 袁淑賢醫生

### Founder Members

**British Medical Association (Hong Kong Branch)**  
英國醫學會 (香港分會)

<b>President</b>	Dr. LO See-kit, Raymond	勞思傑醫生
<b>Vice-President</b>	Dr. WU, Adrian	鄒揚源醫生
<b>Hon. Secretary</b>	Dr. HUNG Che-wai, Terry	洪致偉醫生
<b>Hon. Treasurer</b>	Dr. LEUNG, Clarence	梁顯信醫生
<b>Council Representatives</b>	Dr. LO See-kit, Raymond Dr. CHEUNG Tse-ming	勞思傑醫生 張子明醫生

**The Hong Kong Medical Association**  
香港醫學會

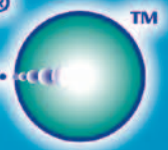
<b>President</b>	Dr. CHOI Kin	蔡堅醫生
<b>Vice- Presidents</b>	Dr. CHAN Yee-shing, Alvin Dr. CHOW Pak-chin	陳以誠醫生 周伯展醫生
<b>Hon. Secretary</b>	Dr. LEE Fook-kay	李福基醫生
<b>Hon. Treasurer</b>	Dr. LEUNG Chi-chiu	梁子超醫生
<b>Council Representatives</b>	Dr. CHAN Yee-shing Dr. CHOW Pak-chin	陳以誠醫生 周伯展醫生
<b>Chief Executive</b>	Mrs. LEUNG, Yvonne Tel: 2527 8285 (General Office) 2527 8324 / 2536 9388 (Club House in Wanchai / Central) Fax: 2865 0943 (Wanchai), 2536 9398 (Central) Email: hkma@hkma.org Website: http://www.hkma.org	梁周月美女士

### The HKFMS Foundation Limited 香港醫學組織聯會基金

<b>Board of Directors</b>		
<b>President</b>	Dr. LO See-kit, Raymond	勞思傑醫生
<b>1st Vice-President</b>	Prof. CHAN Chi-fung, Godfrey	陳志峰教授
<b>2nd Vice-President</b>	Dr. LO Sze-ching, Susanna	盧時楨醫生
<b>Hon. Treasurer</b>	Mr. LEE Cheung-mei, Benjamin	李祥美先生
<b>Hon. Secretary</b>	Dr. CHAN Sai-kwing	陳世炯醫生
<b>Directors</b>	Mr. CHAN Yan-chi, Samuel Prof. CHIM Chor-sang, James Ms. KU Wai-yin, Ellen Dr. WONG Mo-lin, Maureen Dr. YU Chak-man, Aaron	陳恩賜先生 詹楚生教授 顧慧賢女士 黃慕蓮醫生 余則文醫生



**Temodal**<sup>®</sup>  
temozolomide  
capsules



# *Long-term Survival with Glioma Lies in your Hands*

Before prescribing, please consult the full prescribing information.



**Merck Sharp & Dohme (Asia) Ltd.**

27/F., Caroline Centre, Lee Gardens Two, 28 Yun Ping Road, Causeway Bay, Hong Kong  
Tel: (852) 2574 4241 Fax: (852) 2834 0756

Copyright © 2010 Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Whitehouse Station, N.J., USA. All rights reserved.