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  - Severe axillary hyperhidrosis
  - Upper facial rhytides

References:
1. Allergan, data on file.
3. Search strategies are identical across all products. Searches are conducted on 3rd International on the following databases: Medline®, Embase®, Cochrane Library®, PM囿, Biosis®, Scopus®. Data are reported separately from other peer-reviewed articles, letters, reports, or from congress abstracts, meeting posters. Results include clinical studies, case reports, reviews, and excluded trade magazines, news, book chapters.
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### 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
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.
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References: 1. Package insert of PANTOLOC 20mg tablet. 2. Package insert of PANTOLOC 40mg tablet. 3. Package insert of PANTOLOC 40mg IV.

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Spasticity - Orthopaedic Perspectives

Dr. Michael KT TO
FRCSEd (Ortho), FHKCOS, FHKAM (Orthopaedic Surgery)

Dr. W CHOW
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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 anteverision and external tibial torsion). 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. 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.

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. The GMFCS was revised, expanded and further validated to include children up to the age of 18. 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. 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 mechanisms 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 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, hamstring and gastrocnemius. Due to the biarticular involvement, they can result in very complex gait abnormalities. There are four common gait abnormalities of the knees in cerebral palsy patients - jump knee, crouch knee, stiff knee, and recurvatum knee patterns. Jump knee gait is quite frequently seen in spastic diplegic patients due to overactive hamstrings in the presence of tight or spastic gastrocnemius 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 x 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 antversion will bring about ineffective lever arm at the hip leading to Trendelenburg gait. Five types of lever-arm dysfunction were described: (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 will 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).

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. 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
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.

Spasticity - Orthopaedic Perspectives

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The Medical Management of Cerebral Palsy-Paediatricians’ Perspective

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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.

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. 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.

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.

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.

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 leucomalacia (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, Peronecephaly and Schizencephaly in hemiplegia; 4) involvement of the internal capsule and cerebral cortex in spastic tetraplegia; 5) features of malformation of cortical development.

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 hypereflexia. “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.

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 viscous 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.

What is Botulinum Toxin-A?

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. 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.
Botulinum toxin-A is a potent neurotoxin derived from the bacterium Clostridium Botulinum. In 1949, it was discovered that botulinum toxin could temporarily blocks 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 on 1989. Since then the spectrum of indications has expended tremendously - focal dystonia, hemifacial spasm, torticollis and spasticity in children with cerebral palsy, just to name a few.

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.

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.

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; 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.

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.

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. 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.

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. 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. 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 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.

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

DCH (Diploma in Child Health Examination) Written Examination (MRCPCH Part 1A) 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 1A Examination is held three times a year in Hong Kong. The next MRCPCH Part 1A Examination will be held on Tuesday, 11 October 2011. The examination fee is HK$4,250 for Part 1A. 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 or call the College Secretariat at 28718871.

Deadline for Application: Tuesday, 18 July 2011

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**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

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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 1A 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. 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 β receptor agonist that works by a pre-synaptic inhibitory effect on the release of excitatory neurotransmitters such as glutamate, aspartate, and substance P. 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. 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, beclofen 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, regardless of the origin of the spasticity - spinal or cerebral.

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 beclofen. 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. 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 occasionally 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.

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® and Botox®) 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®, which is available worldwide, and Dysport® 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.

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® 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.

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 administrated 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. 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. 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.

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:

1. A surgical procedure is required
2. 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® 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

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

(Effective from October 2009)

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Non-Peak Hour: 9:30am - 5:30pm
Peak Hour: 5:30pm - 10:30pm

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### Postgraduate Diploma in Community Geriatrics

**Jointly organized with Hong Kong Geriatrics Society**

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<td>Session</td>
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### Postgraduate Diploma in Community Psychological Medicine

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

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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**
Selective Dorsal Rhizotomy in Spastic Cerebral Palsy

Dr. Kwong-yui YAM

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

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. 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.

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

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, urodynamic 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). 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.
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 aceterminophen. 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 \(^8\). 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\(^9\).

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

Physiotherapy in Spasticity Management for Children with Cerebral Palsy

Ms. Nerita NC CHAN

MSc
Physiotherapy Department, Tuen Mun Hospital

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. 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. 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), 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 tonicity. 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.

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 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 iliaccus 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 iliaccus 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. The use of outcomes measure is similar in each study. In both domains of activity and participation, they used Gross Motor Function Measures (GMFM) to measure changes in motor function. Three dimensional (3-D) gait analysis or the Physician Rating Scale (PRS) 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), 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.

**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.

A typical deviated gait pattern is the flexion of the knee during midstance, either crouch pattern or jump knee pattern. 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. After thorough examination and confirmation with gait analysis, muscles targetted 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. The change in...
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; not too fat in body build;
- **Slim** - intellectually good enough for carrying out training;
- **Smart** - best result for one with supportive family and carer for postoperative intensive training;
- **Support** - intellectually good enough for carrying out training;

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 activities27 - and Paediatric Evaluation of Disability Inventory (PEDI) are used. Our result is promising and has been published21.

**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

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**References**

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)
Computerised Gait Analysis

Ms. Miko LM LAO

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

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. 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. 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. This information is then correlated with the CGA data to help determine the potential causes of the gait deviations.

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.
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)

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)

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)

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). Maximum COP displacements in anterior-posterior and mediolateral direction can be used to quantify postural instability.

Figures:
- Figure 3: Retro-reflective markers are attached to patient.
- Figure 4: Joint kinematics represented in graphic form which is more easily to be understood.
- Figure 5: Ground reaction force acts on the patient.
- Figure 6: Joint kinetics represented in graphic form which is more easily to be understood.
- Figure 7: Wireless sensors are attached to the patient to record muscle activities.
- Figure 8: Prolonged muscle activities are shown.
- 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.

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.

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

Certificate Course on
Respiratory Medicine 2011

Jointly organised by
The Federation of Medical Societies of Hong Kong
Hong Kong Thoracic Society

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

14 Sep 2011
Topic: Polysomnography – hook-up, acquisition and interpretation
Speaker: Dr. Jamie Chung-mei LAM

21 Sep 2011
Topic: Collaborative care for COPD patients in community
Speaker: Dr. Kahlin CHOO

28 Sep 2011
Topic: Occupational lung diseases in Hong Kong
Speaker: Dr. Henry Kai-him KWOK

12 Oct 2011
Topic: Radiological imaging in respiratory medicine
Speaker: Dr. Thomas Yun-wing MOK

19 Oct 2011
Topic: Mechanical and non-invasive ventilation
Speaker: Dr. Wai-ning CHAN

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

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.
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- 2011 Paediatric Update No. 2 – Childhood Allergy
- HKMA Dragon Boat Team Practice Session
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- FMSHK Officers’ Meeting
- HKMA Council Meeting
- HKMA MPS CME – Mastering Your Risk Workshop
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<td><strong>2011 Paediatric Update No. 2 – Childhood Allergy</strong>&lt;br&gt;Organiser: Hong Kong College of Paediatricians, Chairman: Dr. Sik-nin WONG, Speakers: Various, Venue: Hospital Authority Head Office M Floor, Lecture Theatre</td>
<td>Ms. Vanessa WONG&lt;br&gt;Tel: 2527 8773  Fax: 2785 1830 3 CME Points</td>
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<td>3 7:30 pm – 8:30 pm</td>
<td><strong>HKMA Dragon Boat Team Practice Session</strong>&lt;br&gt;Organiser: The Hong Kong Medical Association, Venue: Sai Kung</td>
<td>Miss Alice TANG &amp; Miss Sharon HUNG&lt;br&gt;Tel: 2527 8285</td>
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<td>4 7:30 pm – 8:30 pm</td>
<td><strong>An Unknown Complication for A Known Procedure</strong>&lt;br&gt;Organiser: Hong Kong Urological Association, Chairman: Dr. Wai-ki MA &amp; Dr. Kim-chung TO, Speaker: Dr. Ka-tung CHIU, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon</td>
<td>Dr. Hong-hoi HUNG / Ms. Tammy HUNG&lt;br&gt;Tel: 2958 6006 / 9609 6064 Fax: 2958 6076 / 8344 5115</td>
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<td>5 1:45 pm</td>
<td><strong>HKMA Tai Po Community Network – Acne Vulgaris – An Update</strong>&lt;br&gt;Organiser: HKMA Tai Po Community Network, Speaker: Dr. William Yum-ming TANG, Venue: Tai Po</td>
<td>Miss Candice TONG&lt;br&gt;Tel: 2527 8285 1 CME Point</td>
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<td>5 2:00 pm – 5:00 pm</td>
<td><strong>FMSHK Officers’ Meeting</strong>&lt;br&gt;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</td>
<td>Ms. Sonia CHEUNG&lt;br&gt;Tel: 2527 8898 Fax: 2865 0345 Ms. Christine WONG&lt;br&gt;Tel: 2527 8285</td>
</tr>
<tr>
<td>5 9:00 pm</td>
<td><strong>HKMA Council Meeting</strong>&lt;br&gt;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</td>
<td></td>
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<tr>
<td>6 6:30 pm</td>
<td><strong>HKMA MPS CME – Mastering Your Risk Workshop</strong>&lt;br&gt;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</td>
<td>HKMA CME Department&lt;br&gt;Tel: 2527 8452 2.5 CME Points</td>
</tr>
<tr>
<td>7 6:30 pm</td>
<td><strong>HKMA MPS CME – Mastering Adverse Outcomes Workshop</strong>&lt;br&gt;Organiser: The Hong Kong Medical Association, Speakers: Dr. Justin Ngai-sing CHENG &amp; 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</td>
<td>HKMA CME Department&lt;br&gt;Tel: 2527 8452 2.5 CME Points</td>
</tr>
<tr>
<td>10 2:00 pm</td>
<td><strong>HKMA Certificate Course on Family Medicine 2011</strong>&lt;br&gt;Organiser: The Hong Kong Medical Association, Speaker: Dr. Kit-kuen IP &amp; Dr. Hak-hoon HUNG, Venue: QEH</td>
<td>HKMA CME Department&lt;br&gt;Tel: 2527 8452 3 CME Points</td>
</tr>
<tr>
<td>12 1:00 pm</td>
<td><strong>HKMA Hong Kong East Community Network – Treatment beyond LDL in Real-life Practice</strong>&lt;br&gt;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, 13 Hennessy Road, Hong Kong</td>
<td>Miss Candice TONG&lt;br&gt;Tel: 2527 8285</td>
</tr>
<tr>
<td>13 7:30 am</td>
<td><strong>Hong Kong Neurosurgical Society Monthly Academic Meeting – Normal Pressure Hydrocephalus</strong>&lt;br&gt;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</td>
<td>Dr. Gilberto LEUNG&lt;br&gt;Tel: 2255 3366 Fax: 2818 4350 1.5 CME Points</td>
</tr>
<tr>
<td>13 1:00 pm</td>
<td><strong>HKMA Central, Western &amp; Southern Community Network – Certificate Course on Psychiatry (Session 4 &amp; 5)</strong>&lt;br&gt;Organiser: HKMA Central, Western &amp; Southern Community Network, Chairman: Dr. Ming-yiu LAM &amp; Dr. Yin-kwai LAW, Speakers: Dr. C.-t. Dr.-yu CHIU &amp; 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</td>
<td>Mr. Alan LAW&lt;br&gt;Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>14 1:00 pm</td>
<td><strong>HKMA Kowloon City Community Network – JUPITER: What is the Implication in Reality</strong>&lt;br&gt;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, Hung hom, Kowloon</td>
<td>Miss Candice TONG&lt;br&gt;Tel: 2527 8285</td>
</tr>
<tr>
<td>14 1:00 pm</td>
<td><strong>HKMA NT West Community Network – Lecture Series on BPH &amp; Urology Practical Tips on Urology for Primary Care Physicians</strong>&lt;br&gt;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.</td>
<td>Mr. Alan LAW&lt;br&gt;Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>14 2:00 pm</td>
<td><strong>HKMA Structured CME Programme with Hong Kong Sanatorium &amp; Hospital Year 2011 – Lymphadenopathy</strong>&lt;br&gt;Organiser: The Hong Kong Medical Association, Chairman: 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</td>
<td>HKMA CME Department&lt;br&gt;Tel: 2527 8452 1 CME Point</td>
</tr>
<tr>
<td>16 1:30 pm</td>
<td><strong>HKMA – KLN East Community Network; HA – UCH; HKCFP - CME Course for Health Personnel 2011</strong>&lt;br&gt;Organiser: HKMA – KLN East Community Network, Chairman: Dr. Danny Ping-kwan MA, Speaker: Dr. Emily Wai-ho TANG, Venue: UCH</td>
<td>Mr. Alan LAW&lt;br&gt;Tel: 2527 8285 1.5 CME Points</td>
</tr>
<tr>
<td>19 8:00 pm – 10:00 pm</td>
<td><strong>FMSHK Executive Committee Meeting</strong>&lt;br&gt;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</td>
<td>Ms. Sonia CHEUNG&lt;br&gt;Tel: 2527 8896 Fax: 2865 0345</td>
</tr>
</tbody>
</table>

**Calendar of Events**

**VOL.16 NO.7 JULY 2011**

**THE HONG KONG MEDICAL DIARY**
Calendar of Events

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

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<thead>
<tr>
<th>Date</th>
<th>Course No</th>
<th>Course Name</th>
<th>Target Participants</th>
<th>CME/CNE</th>
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<tr>
<td>7/9/2011 - 19/10/2011</td>
<td>C177</td>
<td>Certificate Course on Respiratory Medicine 2011</td>
<td>Nurses and Allied Health Professionals</td>
<td>9 CNE Points; CME/CPD Accreditation in application</td>
</tr>
<tr>
<td>8/9/2011 - 13/10/2011</td>
<td>C181</td>
<td>Certificate Course on Renal Medicine 2011</td>
<td>Medical and Health Professionals</td>
<td>9 CNE Points; CME/CPD Accreditation in application</td>
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Society News

News from Member Societies

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

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
Long-term Survival with Glioma Lies in your Hands

Before prescribing, please consult the full prescribing information.

Merck Sharp & Dohme (Asia) Ltd.
22/F, Caroline Centre, Lee Gardens Two, 28 Yung Ping Road, Causeway Bay, Hong Kong
Tel: (852) 2574 4741 Fax: (852) 2864 0756

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