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Paediatric Neuroscience

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The Cover Shot

Mimi

Kitten Mimi is a typical British Blue – inquisitive to everything we do and yet too proud to let you hold her close; at one time she could be sessile watching you from high above or at another agile enough to catch a fly in mid-air! To capture a delightful split second of her takes some patience and fortuity.

It was taken with a Nikkor 105 mm Macro on a D700.

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Editorial

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Editor

Dr. Mario CHAK

It is my great pleasure to serve as the editor of this April issue of Hong Kong Medical Diary. The theme of this issue is "Paediatric Neuroscience". We would like to take this opportunity to discuss some common paediatric neurological disorders including ADHD, autism and epilepsy, as well as pharmacological, surgical and dietary treatment options. Current evidence, local experience and the current understanding of the underlying pathomechanisms of the disease are also discussed. Visual Art is explored as a way of improving psychological well-being. A multidisciplinary team approach to provide holistic care and empower patients to live with epilepsy in a positive way is also described.

Our contributing authors are all distinguished local experts in their respective field. Dr Venus Tam, an experienced child psychiatrist enlightens us about what is happening inside the brain of children with ADHD. Dr SM Lam, a local expert child psychiatrist discusses the pharmacological management of autistic spectrum disorder. Ms Mandy Fong, a specialist paediatric neurology nurse shares her experience in the provision of holistic care for children with refractory epilepsy. Dr Mario Chak, a paediatric neurologist of a multidisciplinary team and an Epilepsy Surgery Programme shares his experience in surgery, a ketogenic diet programme and immunotherapy in Landau Kleffner Syndrome. Finally, Professor Sophia Law, Associate Professor, Department of Visual Studies, Lingnan University describes how art can be used as a language for children in need. I would like to express my sincere thanks to all the authors and Dr Dawson Fong for his generous provision of a lovely cover photo.

I hope you will enjoy this issue and find these articles useful in your clinical practice.

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What's Happening Inside the Brain of Children with ADHD?

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Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is one of the most common psychiatric phenotypes to affect children and adolescents, with a worldwide prevalence estimated to be 5.29%. It is characterized by age-inappropriate inattentiveness, and/or hyperactivity-impulsivity that occur for at least 6 months in at least two domains of life[1]. Many affected children suffer serious impairment in cognitive, academic and social functioning, co-morbid with other behavioural and emotional disorders[2].

Early conceptualizations of ADHD focused on defective moral control of behaviour and deficits in behavioural inhibition. Later it was found that some biological causes such as birth trauma and brain injuries, particularly frontal lobe lesions, were related to ADHD symptoms. By the 1950s and 1960s this had evolved into the concept of 'minimal brain dysfunction' (MBD)[3]. In recent decades, psychopharmacological, genetic, neuropsychological, structural, and functional imaging data have provided strong evidence of neurobiological abnormalities that result in ADHD symptomatology.

Neuroimaging

Early studies of ADHD showed striking similarities in behavioural problems between children with ADHD and those who had suffered brain damage to the prefrontal region, the part of the brain responsible for executive functioning and self-regulation. Many different imaging methods are now available such as structural MRI, functional MRI (fMRI) and diffusion tensor imaging (DTI) to explore the structural and functional alterations in widespread brain regions and their connection with ADHD.

Structural imaging (MRI)

Early estimations showed an approximately 4–5% overall volume reduction of the cerebrum and cerebellum in children and adolescents with ADHD. Most replicated findings from the voxel-based and region of interest (ROI)-based structural MRI studies of patients with ADHD have suggested a significant decrease in the whole brain grey matter (GM) and white matter (WM) volume and significant regional underdevelopment in the prefrontal cortex (PFC), including the orbitofrontal cortex (OFC), dorsolateral prefrontal cortex (DLPFC), and superior frontal cortex (SFC), basal ganglia substructures (striatum and globus pallidus), and cerebellum[4]. Some studies also reported abnormal volume and cortical thickness of the temporal and parietal cortices.

Longitudinal studies have shown that children with ADHD have a 3 to 5 year delay in the maturation of cortical thickness, with the greatest delays in the frontal and temporal brain regions.

A 2007 meta-analysis[5] of 21 studies published to date of the brain structure of individuals with ADHD (total of 565) compared with typical people of the same age (total of 583) revealed that at least five regions were significantly smaller in those with ADHD : a) the cerebellum, particularly the posterior inferior vermis, b) the front part of the corpus callosum (the splenium), a large bundle of nerve fibres that connects the right and left hemispheres, allowing cross-communication, c) the right side of the caudate nucleus (part of basal ganglia and the centre of the brain), d) the right hemisphere of the brain in general, e) the frontal regions of the brain.

Another meta-analysis by Dr. Tomohiro Nakao et al. in 2011[6] reviewed 14 separate studies measuring the volume of grey matter in the brain. Brain volume of children with ADHD was significantly smaller with the greatest reduction in the caudate region. These differences in brain volume improved with age and with the length of time children had been taking stimulant medication, implying that taking medication does not harm brain development and may facilitate maturation in brain size.

Functional MRI (fMRI)

Both task-based and resting-state fMRI have been frequently applied in children with ADHD, and have demonstrated atypical functional activations in the frontal, temporal, parietal lobes, and cerebellar regions[7].

Individuals with ADHD show increased connectivity in the affective network [anterior cingulate cortex (ACC), amygdala, nucleus accumbens, hypothalamus, anterior insula, hippocampus, and orbitofrontal cortex (OFC), with reciprocal connections to autonomic, visceromotor, and endocrine systems], that are attributed to emotional control deficits.

Individuals with ADHD also had consistently hypo-activated ventral attention network regions [temporoparietal junction (TPJ) and ventral frontal cortex (VFC)] and bilateral dorsal attention network [intraparietal sulcus (IPS) and the frontal eye field (FEF)], that affected their ability to reorient attention to relevant stimuli. Meta-analysis has shown that the activity of the

bilateral dorsolateral prefrontal cortex (DLPFC) [part of the cognitive control network for inhibition and goal-directed decision making] is reduced in individuals with ADHD, and has been implicated in planning, working memory and attentional processes.

Spontaneous slow-frequency functional activities have been reported in multiple brain regions including the precuneus/posterior cingulate cortex, medial prefrontal cortex, and dorsal ACC, which form the default mode network (DMN), during wakeful resting-state fMRI acquisition. Individuals with ADHD displayed excess levels of DMN activity, in the form of attenuated up-regulation of the DMN, during goal-directed tasks that was associated with attentional disturbances and reduced performance[8].

Some research has focused on the effect of medication on brain development. A meta-regression analysis revealed that the percentage of patients prescribed stimulant medication was correlated with increasing (ie. more normal) grey matter volume in the right caudate, over and above the effects of increasing age. fMRI studies also consistently showed that acute doses of methylphenidate enhance and normalize activation in the basal ganglia as well as their functional connectivity with the fronto-cortical and cerebellar regions[9].

Diffusion tensor imaging (DTI)

Diffusion tensor imaging (DTI) is an imaging technique that can be used to assess fractional anisotropy (FA) of white matter tracts in the brain, with lower white matter (WM) FA values indicating alterations in WM fiber orientation and integrity that reflect a widespread dysmaturation of myelination and thus structural brain connectivity. The findings of white matter tract deficits suggest that structural deficits in ADHD are not just confined to specific regions but affect the structural interconnectivity between regions and hence entire neural networks[10].

A recent meta-analysis reviewed the ROI-based studies that assessed the WM integrity, and provided evidence of several disturbed WM regions in children with ADHD, including the inferior and superior longitudinal fasciculus, anterior corona radiate, cortico-spinal tract, cingulum, CC, internal capsule, caudate nucleus, and cerebellum. Review of the voxel-based analysis (VBA) studies also confirmed WM changes in these regions, and found extensive differences across the four brain lobes, as well as areas within the basal ganglia, uncinated fasciculus, and forceps minor. The neural pathways that are associated with the areas of abnormal WM reviewed above are the pathways that connect the cortical regions, cortical-striatum and cortical-cerebellum.

Genetic studies

Research into the genetics of ADHD started with family studies, adoption studies and twin studies. They demonstrated that ADHD clusters in families and is thus considered one of the most heritable disorders with an estimated mean heritability of 75%[11]. With the recent advances in molecular genetic research, there has been an increasing number of studies looking for various candidate genes.

Genetic linkage studies

Since 2002, there have been 15 linkage studies performed for ADHD to screen the genetic loci involved in this disease. Regions 16p13 and 17p11 are the most promising loci since they have twice been reported as significant linked loci with ADHD in one GWL study and one candidate region linkage study respectively[12].

Candidate-gene association studies

Although the degree of association varies and results are very inconsistent for the same locus, there is strong evidence for the association of genes from several important pathways or systems with ADHD. Recent studies focused on three neurotransmission systems, i.e. dopaminergic, serotoninergic and noradrenergic systems. In addition, the evaluation of common variants in 16 genes involved in the regulation of neurotransmitter release and 10 genes encoding neurotrophic factors and their receptors in ADHD showed the contribution of the SNARE system and neurodevelopment system to ADHD.

The most comprehensive meta-analysis was performed by Gizer et al.[13], and investigated 38 markers within 18 genes and identified significant associations between several candidate genes including DAT1, DRD4, DRD5, 5HTT, HTR1B, and SNAP25 and childhood ADHD.

Genes involved in the metabolism of tyrosine, tryptophan and other neurotransmitters were highlighted in the "hot genes" list as well, including COMT, DBH, MAOA and MAOB, TPH1 and TPH2, TH, and DDC. Genes related to neurodevelopment and neuroplasticity, such as BDNF, have also been widely investigated.

Genome-wide association studies

To improve the power of GWASs, a meta-analysis was conducted in a total sample of 2064 trios, 896 cases and 2455 controls[14], although no significant associations were determined. Considering the complex heterogeneity of ADHD, a larger sample might be useful to identify a significant association signal. Through a comparison with data on other disorders, such as schizophrenia, bipolar disorder, autism and depression, within a Psychiatric GWAS Consortium (PGC), it is suggested that genome-wide significance can only be achieved with a sample size of 12,000 individuals including both cases and controls for psychiatric diseases. To extend the scope of ADHD GWAS, an international collaboration should be encouraged.

Environmental factors

Environmental factors can significantly modulate genetically programmed brain development during foetal life. One harmful factor is maternal smoking[15].

Children with ADHD who were exposed prenatally to smoking had the smallest cerebellum volume compared with normal children. Smoking exposure during pregnancy was also associated with decreased fractional anisotropy in the region of the corpus callosum containing fibres to the premotor cortex. Decreased fractional anisotropy in these areas was related to more sensation seeking in those individuals exposed to maternal smoking during pregnancy.

Other environmental factors such as the amount of alcohol consumed and lead exposure during pregnancy is also associated directly with the degree of inattention and hyperactivity in children.

Conclusion

ADHD is a highly heritable disorder with onset during childhood. The pathophysiology is more complex than previously believed. It reflects the mystery of interaction between gene and environment.

With advances in technology, there is an increasing amount of research on neurobiological causes of ADHD. Studies consistently show that children with ADHD have a reduced volume of the whole brain and different brain regions involved in attention and impulse control. fMRI and DTI studies have shown that functional and structural abnormalities in different networks are associated with ADHD.

To date more than 300 candidate genes have been proposed to be related to the susceptibility or aetiology of ADHD, but over 70% of these genes have been reported only once in one single study. Nonetheless some "hot genes" such as DAT1 and DRD4 are identified to have strong association with ADHD.

In order to gain a greater understanding of the psychopathology of ADHD, it is necessary to combine the efforts of different researchers. An understanding of the underlying neurobiology might change the future treatment options and clinical practice.

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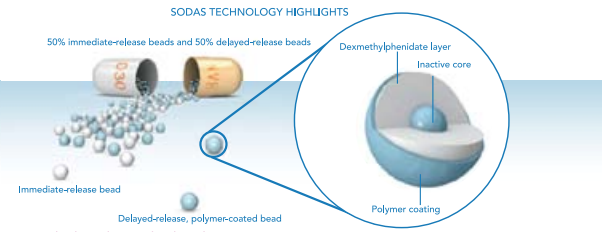

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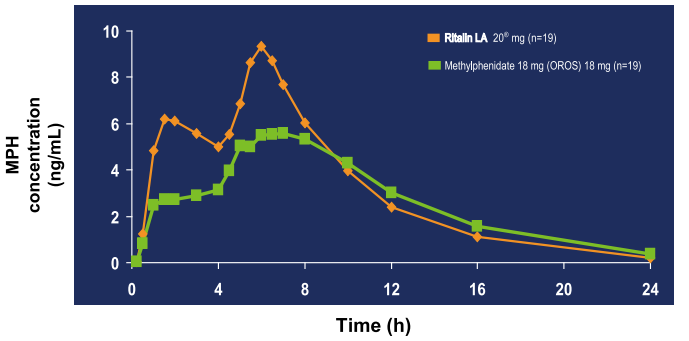

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References:

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A Brief Overview of the Pharmacological Management of Autism Spectrum Disorder

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Dr Siu-man LAM

1) Introduction

Autism spectrum disorder (ASD) manifests in early childhood and is characterized by qualitative abnormalities in social interactions, markedly aberrant communication skills and restricted repetitive behaviours, interests and activities. The definition of ASD in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)[1] encompasses the previous manual's autistic disorder (autism), Asperger's Disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified. ASD is recognized as a complex neurodevelopmental disorder, often becoming clinically apparent in the second to third years of life. Accurate diagnosis is usually made by a combination of a detailed history from reliable informant(s) and direct observation of behaviour. Nonetheless the diagnostic process can be complicated by considerable heterogeneity in the manifestation of core deficits, by variation in ability level and by developmental changes.

Once thought to be a rare disorder with a prevalence of 3 to 5 per 10,000 children, the prevalence of ASD is now commonly quoted as around 1% [2, 3]. Reported rates of ASD have been rising in many countries over the last two decades and the lay public has referred to this as an 'autism epidemic'. It remains unclear how much of these data represent an actual increase and how much reflect changes in diagnostic definition and practices, as well as increasing awareness among the general public and within the medical profession. More people are now first diagnosed with autism as adolescents and adults, possibly owing to increased public and professional recognition[4]. A male excess of between 3:1 and 4:1 is generally observed[5]. Up to two-thirds of affected individuals will have intellectual disability (mental retardation). Autism is thus a relatively common, chronic and substantially disabling disorder, with significant costs to both the affected individual and family members.

Although the etiology of autism is unknown, hypotheses include genetic abnormalities, obstetric complications, exposure to toxic agents and prenatal infections. Maternal rubella is associated with a significantly higher rate of autism in the offspring. Additionally, tuberous sclerosis is associated with autism as a comorbid disorder. Approximately 10% of children with ASD exhibit a known medical condition. The claims that ASD may be linked to vaccination, in particular the combined measles, mumps and rubella (MMR) vaccine, have not been supported by several large scale population studies[6].

2) Medication management in autism

The mainstay of interventions for ASD is non-pharmacological, with educational and psychosocial interventions aiming at improving language acquisition and maximizing communication and social skills. Nonetheless pharmacological management, particularly of associated maladaptive problem behaviour and comorbid psychiatric symptoms, is common and increasing. Such symptom clusters are common in individuals with ASD and are strongly associated with caregiver stress as they interfere with socialization and education progress. They also severely affect the quality of life of the affected individuals and their families, and are often the reason that families seek medical treatment[7]. A UK study revealed up to 75% of adults with autism and intellectual disabilities were prescribed at least one psychotropic medication[8]. Factors associated with increasing medication use include older age, poorer functioning and higher level of maladaptive, problematic behaviour[9].

(A) Pharmacotherapy for the core symptoms

The core symptoms refer to the social and communicative deficits and repetitive pattern of behaviour. As in any chronic condition with unknown cause and lack of specific cure, treatment of core symptoms has been subject to many 'treatment fads' with many unproven therapeutic options being heavily promoted. A classic example is the use of secretin, a gastrointestinal peptide, in the early 1990s that claimed to have prosocial effects in autism and was once promoted as a potentially 'miraculous cure'[10]. However large placebo-controlled trials failed to replicate these improvements. A wide variety of pharmacological agents, including antidepressants, antipsychotics, glutamatergic drugs, has also been subject to clinical trials but to date, no pharmacological agent is regarded as effective in the treatment of such core symptoms. Additionally, the adverse effects of many of these agents have to be taken into consideration. Consequently, recent clinical guidelines from the National Institute for Health and Care Excellence & Social Care Institute for Excellence do not recommend any medication as being specifically effective for core symptoms.

(B) Comorbid psychiatric symptoms

ASD sufferers have a high prevalence of psychiatric comorbidities[11]. At a theoretical level, it is not always clear whether such symptoms constitute true psychiatric 'comorbidity' in that they are caused by the same underlying pathophysiological process as autism, or whether they co-occur with autism because of

an interaction between the disorder and concurrent stressors. The latter may include increased social demands, inadequate support at school, bullying or family conflict. Nonetheless irrespective of their relationship with the primary condition, it is important to understand and be alert for these symptoms as they often cause as much functional impairment as the core disorder, and are often a key target for intervention. Common comorbid symptoms include aggressive outbursts and self-injury, hyperkinesis and inattention, and mood and anxiety disorders.

(i) Aggression and self-injuries

Aggression can severely affect the functioning of people with autism. Second generation antipsychotics (SGAs) are the most frequently used psychotropics for these conditions. The US Food and Drug Administration has approved risperidone and aripiprazole for the symptomatic treatment of irritability (including aggression and self-injury). The adverse effects of SGAs include increased appetite and weight gain, dyslipidaemia and insulin resistance, somnolence, extrapyramidal symptoms and prolactin elevation. Evidence for other SGAs is still very preliminary. The use of olanzapine, a very commonly used SGA, is supported by a small randomized controlled trial[12] that found it to be effective in 50% of children but it is strongly associated with weight gain and other physical morbidity. Open label trials of quetiapine have indicated that the response rate and tolerability are poor[13]. Ziprasidone is used off-label to treat serious behaviour disorders associated with autism but the risk of QTc prolongation limits its use. The opiate antagonist, naltrexone, once used as treatment of the social deficits with largely negative results, has demonstrated beneficial effects in reducing self-injury in individuals with intellectual disabilities, including those with ASD[14]. Evidence regarding the use of mood stabilisers, including lithium and valproate, is mixed and their use is not generally supported[15].

(ii) Symptoms of attention deficit/hyperactivity disorder (ADHD)

Symptoms of ADHD (inattention, hyperactivity and impulsivity) are very common in ASD, particularly in children and adolescents[16], and may severely impair an individual's functioning and warrant treatment. A RCT of methylphenidate has demonstrated improvement in ASD cases[17]. Nonetheless trials suggest that the response rate in people with autism is lower than in neurotypical individuals, and side effects such as irritability and poor appetite are more common. Psychostimulants may have a positive effect on some aspects of social communication, such as joint attention initiations, in children with ASD and hyperactivity. Atomoxetine, a selective noradrenaline reuptake inhibitor, is another commonly used non-stimulant for the treatment of ADHD, a small controlled trial[18] suggested a beneficial effect on hyperactivity, impulsivity and oppositionality, e.g. clonidine and guanfacine, may also be helpful for hyperactivity[19] common side effects included sedation and hypotension. SGAs including risperidone and aripiprazole have been found helpful in reducing hyperactivity[20], but their use must be balanced against their potentially serious metabolic side effects.

(iii) Mood and anxiety disorders

Patients with ASD are reported to be at increased risk of depression and anxiety[21,22]. These problems may be easily overlooked in ASD patients especially when their presentation may be atypical as a result of core deficits. For example, anxiety is often particularly intense and can interact with core symptomatology to mimic thought disorder or abnormal beliefs. Mood and anxiety disorder tend to emerge in later childhood and adolescence and is often associated with the child's increased self-awareness and increasing social demands. As in other childhood emotional disorders, psychosocial management is the first line treatment. For persistent and severe problems, open label trials of selective serotonin reuptake inhibitors (SSRIs) including fluoxetine, fluvoxamine, sertraline, citalopram and escitalopram have shown improvement.

3) Conclusion

Pharmacological research in the context of ASD is still in its infancy and the current evidence base for a pharmacological approach is small. Clinicians will need to carefully interpret the clinical evidence and make appropriate judgments when applying it to the specific, real world situation of their patients. Most trials have been short term. There remains a lack of important information on the long term safety and efficacy of drugs, given the lifelong disabilities associated with ASD. The standard of evidence to date does not allow for a definitive treatment protocol for various symptom clusters. More research is required to determine whether a combination of drug and behavioural treatment will result in additional benefits for the long term development of our patients. It should also be noted that many drugs are not licensed for children because of a corresponding lack of clinical trial data. Nevertheless they are used quite widely by specialists with the view that parents should be made aware of this off-label use and consent to it.

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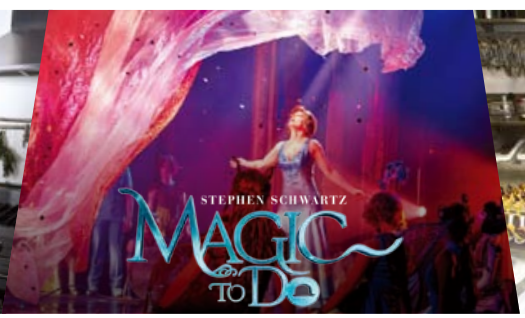

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Holistic Care in Paediatric Refractory Epilepsy: The Role of the Paediatric Neurology Nurse Specialist

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Introduction

Refractory epilepsy refers to the continued occurrence of seizures after a trial of two or more antiepileptic drugs. It is a group of disorders characterized by different seizure types, etiologies and prognosis[1]. Management of refractory epilepsy thus poses a challenge that requires early medical attention and a multi-disciplinary team approach. Thorough investigation of each patient is required in order to determine the underlying aetiology and select appropriate and effective intervention. The present choice of treatment modalities include resective epilepsy surgery, vagal nerve stimulator implantation and a ketogenic diet. All these options should be considered in the patient with refractory epilepsy.[2]

A multi-disciplinary team for paediatric refractory epilepsy was established at Tuen Mun Hospital, Hong Kong in 2005. It comprises paediatric epileptologists, neurosurgeons, radiologists, nuclear medicine physicians, EEG technicians, neuro-psychologist/ clinical psychologist, child psychiatrist, developmental behavioural paediatrician, pathologist, dietitian, occupational therapist, physiotherapist, speech therapist and neurology nurse specialist.[3]

The Role of the Paediatric Neurology Nurse Specialist: Listening, Discussion, Counseling and Provision of Empathic Care

Providing information to a patient with epilepsy is not a one-way process. Both the patient and his/her family need to have opportunities to ask questions and voice any concerns they may have[4]. The specialist nurse listens to the patient as well as their immediate and extended family members while trying to empathise with their concerns and frustrations. The specialist nurse will spend time with patients in hospital and at home. Patient and family members are encouraged to discuss any concerns related to epilepsy such as disease prognosis, how epilepsy affects their life, and the issue of Sudden Unexplained Death in Epilepsy (SUDEP)[5]. Continuous discussion with the patient and family members enables the specialist nurse to gain a deeper understanding of how the disease impacts their lives. Patients and families also gain insight into their epilepsy and this is essential for self-management and patient empowerment.

Provide Education on Epilepsy

A paediatric neurology nurse can educate patients and their family about the pathophysiology of seizures, different treatment options, and the potential side effects of medication. Fear related to seizure may be relieved by teaching about seizure trigger factors and how to avoid or minimize them, as well as acute seizure management and use of emergency rescue medication such as rectal diazepam. Information can be tailored to the individual patient's needs and level of understanding about their illness.

Facilitate Patient Empowerment: Living with Epilepsy in a Positive Way

Empowerment is a reciprocal social process wherein the patient is helped to participate in their community with competence and to take control over the factors that affect their life[6]. Families are empowered when they have participatory competency in the care of their children and when they are allowed to participate in decision making.

Building a relationship, facilitating participatory experiences and helping families by education and information-giving are all roles that are fulfilled by a neurology nurse. Using a systematic framework to assess, plan, implement and evaluate management strategies will aid in the development of family competencies in order to achieve empowerment.

As a Key Member to Co-ordinate Multi-disciplinary Team Care

In the hospital setting, the neurology nurse specialist facilitates close liaison with other professionals and coordinates the multi-disciplinary service need for children with epilepsy. The proactive and complementary role of the neurology nurse will minimize fragmented care and bring an integrated approach to the complex management of epilepsy patients.

As a Main Contact Person of Care: Liaising between Professionals

The neurology nurse acts as the consistent point of contact for affected children, adolescents and family, as well as other related health care professionals. The

neurology nurse also coordinates outpatient and inpatient assessments including electroencephalogram (EEG), magnetic resonance imaging (MRI), long term video EEG monitoring (L-T VEEG), Ictal single-photon emission computed tomography (Ictal SPECT) and positron emission scan (PET) as well as other investigations including blood tests[7]. Furthermore, the neurology nurse coordinates with partners in the multi-disciplinary team such as the physiotherapist, occupational therapist and social worker. The neurology nurse can arrange in-patient admission if a patient's epilepsy control deteriorates and serves as a central source of information for ward nurses and related health care professionals. This facilitates continuity of effective care.

The neurology nurse will accompany the patient during some investigations, for example during a SPECT scan to facilitate the identification of seizure and provide acute seizure care to the patient.

Provide Relevant Information during Pre-surgical Evaluation for Epilepsy Surgery Programme

The neurology nurse serves as the point of contact for information about investigations required including details about procedures and preparation, as well as potential complications. Various treatment options for refractory epilepsy can also be discussed such as epilepsy surgery, vagal nerve stimulator implantation and adopting a ketogenic diet.

Co-ordinate Ketogenic Diet Programme

When anti-epileptic pharmacotherapy is ineffective, other treatment modalities may be used. The ketogenic diet (KD) is high in fat with adequate protein and low carbohydrate. Fat is converted in the liver to fatty acids and ketone bodies and creates ketosis that is known to reduce the occurrence of seizure[8]. A ketogenic diet needs to be formulated and monitored by a dietitian with the patient in hospital.

The KD programme is multi-disciplinary. The neurology nurse works with the dietitian and neurologist. The nurse will explain the concept of ketogenic diet to the patient and their family and, following their agreement, commence the pre-diet work up. Blood and urine tests will be performed to exclude any undiagnosed metabolic disease in which a ketogenic diet would be contraindicated, for example pyruvate carboxylase deficiency, fatty acid oxidation problems, organic acidurias or high cholesterol and high triglyceride level[9,10]. Baseline EEG, ECG and ultrasound of the kidney should also be performed and the patient's health history, seizure semiology, body weight and past dietary pattern assessed and recorded. A pharmacist will review the patient's current medication to determine carbohydrate content that may affect the ketogenic effect of dietary treatment and to minimize the carbohydrate and sugar content as much as possible. A dietician will review the patient's current diet to determine carbohydrate content and dietary menu.

Provided baseline workup does not identify any contraindications, the child will be admitted to hospital for a few days to start the KD. The patient will be closely monitored to allow early identification of any acute side effects, for example dehydration, hypoglycaemia or hyperketosis, and to monitor seizure pattern as well as to compliance with diet. Prior to discharge the neurology nurse must ensure that the child and parent understand the treatment plan; how to use ketostix; when and how to seek medical advice if the patient gets sick. Thereafter the patient is followed up in the outpatient clinic.

Co-ordinate Vagal Nerve Stimulator Implantation (VNS) Programme

VNS device implantation can reduce the frequency or duration of seizures in refractory epilepsy patients. The neurology nurse will explain the device to the patients and their family and facilitate the pre-operative work-up. A multi-disciplinary meeting will be held pre-operatively.

The VNS system consists of an implanted device with an electrode; the device contains a lithium battery and microprocessor sealed inside a titanium case. The device is placed under the skin on the chest wall by a neurosurgeon and a wire runs from the device to the vagus nerve in the neck. The neurologist programmes the strength and timing of impulses to the vagus nerve. The patient should be unaware of the intermittent stimulation. A special magnet is provided for the patient. If the patient experiences warning signs of a seizure, sweeping this magnet over the VNS will provide additional electrical stimulation and abort the siezure seizure[11]. The neurology nurse will offer psychological support and follow-up care following implantation of the VNS device.

The neurology nurse will offer psychological support and follow-up care after implantation of the VNS device.

Support Ongoing Epilepsy Patient Care

Ongoing specialist nursing care will improve patient and family satisfaction, quality of care related to patient education, communication, and self-care and reduce the need for hospital readmission[12]. In the Refractory Epilepsy Programme, the neurology nurse ensures continuity of care by providing medical information and emotional support for the patient and arranging referral and phone consultations when appropriate. The specialist nurse also liaises with school teachers or other agencies such as a family social worker if necessary. Follow up out-patient appointments are arranged if required.

Strengthen Nursing Care in Refractory Epilepsy

The neurology nurse leads the nursing team. Treatment of patients with refractory epilepsy is continuously reviewed and updated to ensure optimum standards of care. The specialist nurse also ensures that ongoing professional education is provided to nursing and other health care professionals to maintain up to date knowledge about epilepsy.

Conclusion

The neurology specialist nurse serves as an educator, counselor and patient advocate. She is instrumental in helping patients and their family gain insight into their epilepsy, especially those with intellectual disability[13]. In management of the refractory epilepsy patient, the paediatric neurology nurse plays an essential role in both acute and long term care. By developing a good rapport with the patient and their family, we aim to empower them to live with refractory epilepsy in a positive way and optimize their quality of life.

Table 1: Summary of the Role of a paediatric neurology specialist Nurse in a paediatric refractory epilepsy programme

- Listen, Discuss, Counsel and Provide Empathic Care to Patients/Families
- Provide Education on Epilepsy
- Empower Patients/Families
- Coordinate Multi-disciplinary Team Care
- Act as Main Contact Person for Patient Care
- Liaise between Professionals
- Provide Relevant Information in Pre-surgical Evaluation for Epilepsy Surgery Programme
- Coordinate Ketogenic Diet Programme
- Coordinate Vagal Nerve Stimulator Implantation Programme
- Support Ongoing Epilepsy Patient Care
- Strengthen Nursing Care in Refractory Epilepsy

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30 May	Effective Clinical Communication and Transition to Palliative Care	Dr. Rico K.Y. LIU Associate Director Comprehensive Oncology Centre Hong Kong Sanatorium & Hospital
6 Jun	Handling of the Dying Phase	Dr. Steven Wai-kwan SIU Associate Consultant Department of Clinical Oncology Queen Mary Hospital
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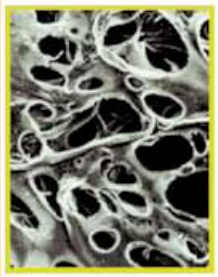

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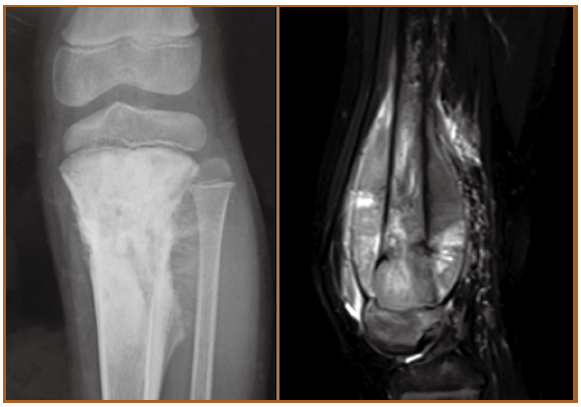
Radiology Quiz

Dr Bruce LEE

Department of Radiology, Queen Mary Hospital



Questions:

1. What are the imaging findings?
   (two different patients with the same disease)
2. What is your diagnosis?
3. What are the classification and potential underlying causes?
4. What is the role of MRI?

(See P.36 for answers)

Treatment Modalities for Paediatric Refractory Epilepsy: Epilepsy Surgery, Ketogenic Diet and Immunotherapy in Landau Kleffner Syndrome. Current Evidence and Local Experience

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 30 April 2016.

According to the NICE Guideline, children or adolescents with refractory epilepsy should be referred to tertiary services for further assessment as soon as possible if they fulfill any of the following:

- Epilepsy uncontrolled after two years of pharmacotherapy
- Management unsuccessful after therapy with two drugs
- Aged under 2 years
- At risk of unacceptable side effects from medication
- There is a unilateral structural lesion
- There is psychological and/or psychiatric comorbidity
- There is diagnostic doubt as to the nature of seizures and /or seizure syndrome
- The patient has a specific condition for which surgery is remedial. For example Sturge-Weber syndrome, Rasmussen's encephalitis, hemispheric syndrome and hypothalamic hamartoma[1]

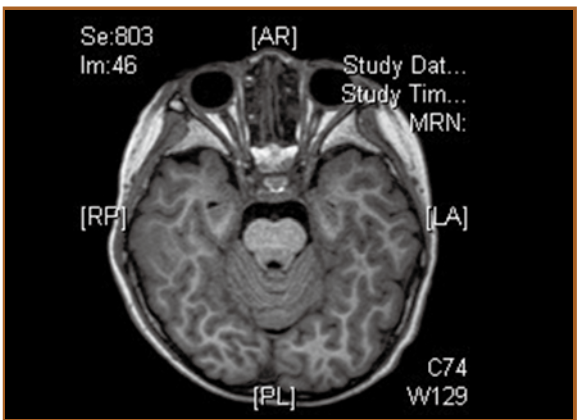
Pre-surgical evaluation in children

Presurgical evaluation should be noninvasive[2] wherever possible. At the author's centre, evaluation includes MRI that follows a specific epilepsy protocol and long term video EEG monitoring to document seizures. Detection of temporal lobe abnormalities in temporal lobe epilepsy require thin coronal cut and FAIR sequence.

Ictal and Interictal SPECT may be useful in providing information about the seizure onset zone, if reviewed in conjunction with MR data and video EEG. Interictal PET is more likely to demonstrate abnormalities related to structural defects, and may be particularly useful in infants where incomplete myelination may restrict structural information provided by MRI[2]. Neuropsychology testing plays a major role in determination of verbal and nonverbal function in older children, and in the determination of cerebral dominance. Functional MRI for determination of language or the motor cortex may enhance such evaluation, although it is limited at present to older non-sedated children. Although the aims of pre-surgical evaluation in children remain similar to those in adults, the children who present are a diverse group, and the aims and likely outcome of surgery require careful discussion with the family. [2]

Location of Epileptic Surgery and Seizure Outcome

According to a large scale study of seizure outcome following paediatric epilepsy surgery at the Cleveland Clinic, a seizure free outcome was more likely following temporal resection (56 of 72, 78%) than extratemporal or multilobar resection (26 of 48; 54%; 41 of 48 with a focal lesion on magnetic resonance imaging).[3] A comparable result was shown in a clinical audit from a local referral center where 84% of patients were reported to be seizure free after temporal lobe epilepsy surgery compared with 50% following extratemporal lobe epilepsy surgery.[4]



Picture 1. MRI brain of a child with refractory epilepsy reveals cortical dysplasia over the right lateral temporal neocortical region. The patient remained seizure free following resection and tailing down of anticonvulsant therapy. Histology of the surgical specimen confirmed focal cortical dysplasia type II B.

Aetiology/pathology in children who undergo surgery for refractory epilepsy

The Cleveland Clinic review of paediatric refractory epilepsy surgery revealed that surgery was performed for cortical dysplasia in 31.52% of cases and for low grade tumour in 44.82%. Hippocampal sclerosis was rare. In a small scale local clinical audit of 38 children who underwent surgery for refractory epilepsy age

at seizure onset ranged from 0.1 to 6 years (mean: 4.6 years). Aetiology/pathology of children was as follows: cortical dysplasia 39%; mesial temporal sclerosis 24%; developmental tumor/ low grade glioma 15%; hypothalamic hamartoma 7%; vascular lesion/ cavernous malformation 5%; tuberous sclerosis 5%; hemimegacephaly with tuberous sclerosis 3%, Porencephalic cyst 2% [5].

Aetiology of refractory epilepsy and seizure outcome

According to the local study discussed above, seizure outcome following surgery varies with pathology/ aetiology. Epilepsy surgery was performed in 38 children aged 0.8 to 26 years (mean 10 years) with mean follow-up of 5.3 years. Following surgery, 68% of patients were seizure free/Engel Class I; 11% were Engel II; 8% Engel III; and 13% Engel IV. Different aetiologies were associated with different seizure outcomes following epilepsy surgery. The best seizure outcome was achieved following surgery for developmental tumour/ low grade glioma (n=5): Engel IA 80%; Engel ID 20%. Other results were as follows:

Focal cortical dysplasia (n=15): Engel I 80%; Engel II 7%; Engel III 13%.
The subgroup of focal cortical dysplasia type IIB (with balloon cell) (n=6) Engel I: 83%; Engel III 17%
Mesial temporal sclerosis (n=9): Engel I 66%;Engel II 11%; Engel III 11%; Engel IV 11%
Vascular lesion/Cavernous Hemangioma (n=2): Engel ID50%; Engel IV 50%
Hypothalamic hamartoma (n=3): Engel I 33%; Engel III 33%; Engel IV 33%.
Tuberous sclerosis (n=3): Engel IC 33%; Engel III 33%; Engel IV 33%
Porencephalic cyst (n=1): Engel 2B

Engel Classifications of postoperative outcome[6]
Class I: free of disabling seizures
A. Completely seizure free since surgery
B. Non-disabling simple partial seizures only since surgery
C. Some disabling seizures after surgery, but free of disabling seizure for at least 2 years
D. Generalised convulsion with AED withdrawal only
Class II: rare disabling seizure ('almost seizure free')
A. Initially free of disabling seizures but has rare seizures now
B. Rare disabling seizures since surgery
C. More than rare disabling seizures after surgery, but rare seizures for at least 2 years
D. Nocturnal seizures only
Class III: worthwhile improvement
A. Worthwhile seizure reduction
B. Prolonged seizure free intervals amounting to greater than half the follow-up period, but not less than 2 years
Class IV: no worthwhile improvement
A. Significant seizure reduction
B. No appreciable change
C. Seizure worse

Patient age and surgery outcome

In the 136 patients reviewed in the Cleveland Clinic, seizure outcome following surgery was similar for infants, children, and adolescents, and comparable with the results from adult series. [3]

Benefits of Epilepsy Surgery in children

In a local clinical audit study, 47% of children were both seizure and medication free after temporal lobe resection surgery.[7] If the patient became seizure free after epilepsy surgery, they could be weaned off their anticonvulsant drug therapy. In the author's experience, although not all patients who undergo surgery become seizure and medication free, the majority can be seizure free with a reduced amount of anticonvulsant medication and consequent fewer side-effects and improved quality of life. For these patients, epilepsy surgery can result in epilepsy that is pharmacoresponsive instead of pharmacoresistant. In addition to improved seizure control, development, psychosocial function and overall health-related quality of life significantly improved following epilepsy surgery.[7]

Timing of epilepsy surgery

In infants and young children with intractable epilepsy and with regression in or plateau of development, epilepsy surgery should be performed as soon as possible. It is hoped that surgery will decrease or eliminate the detrimental effects of seizure activity on the developing brain, and restore normal brain development. Apart from its effects on physical health, refractory epilepsy affects patient's self-esteem and psychosocial well-being. The benefits of seizure elimination or reduction by epilepsy surgery in drug-resistant focal epilepsy prior to adolescence are self-apparent.[2]

Ketogenic Diet: Current Evidence

The ketogenic diet has been successfully and widely used since the 1920s to treat patients with refractory epilepsy. The NICE guidelines recommend referral of children who have not responded to appropriate antiepileptic drugs to a tertiary paediatric epilepsy specialist for consideration of a ketogenic diet.[8]

A randomised controlled trial at Great Ormond Street Hospital in London of a ketogenic diet in childhood epilepsy involved 145 children aged between 2 and 16 years who had at least daily seizures (or more than seven seizures per week), had failed to respond to at least two antiepileptic drugs, and had not been treated with a ketogenic diet and modified Atkin diet programme.[9] Children were assigned to the ketogenic diet (n=73) or the control group (n=72). Data from 103 children were available for analysis: 54 on the ketogenic diet and 49 controls. Of those who did not complete the trial, 16 children did not receive their intervention, data were inadequate in 16, and ten withdrew from the treatment before the 3 month review, six because of intolerance.[9]

After 3 months, the mean percentage of baseline seizures was significantly lower in the diet group than in controls (62.0% vs 136.9%, 75% decrease, 95%CI

42.4-107.4%; p<0.0001), and 28 children (38%) in the diet group had greater than 50% seizure reduction compared with four (6%) controls (p<0.0001). Further, seizure reduction greater than 90% was achieved by five children (7%) in the diet group compared with no controls (p=0.0582). There was no significant difference in the efficacy of the treatment between symptomatic generalized or symptomatic focal syndromes. The most frequent side effects reported at 3 month review were constipation, vomiting, lack of energy, and hunger.[9]

Ketogenic Diet and Modified Atkin Diet: Local Experience

At the author's unit, 13 paediatric patients with refractory epilepsy were commenced on the Ketogenic Diet or Modified Atkin Diet. Age at start of diet ranged from 0.3 to 11 years (mean age: 3.1 years) and mean duration of follow up was 5.26 years. Seizure reduction >90% was achieved in 46% of patients with etiologies that included succinic semi-aldehyde dehydrogenase deficiency, hypoxic ischemic encephalopathy, Leigh's disease (mitochondrial cytopathy complex I & IV deficiency), congenital CMV infection, and two patients with suspected neuro- metabolic disease.

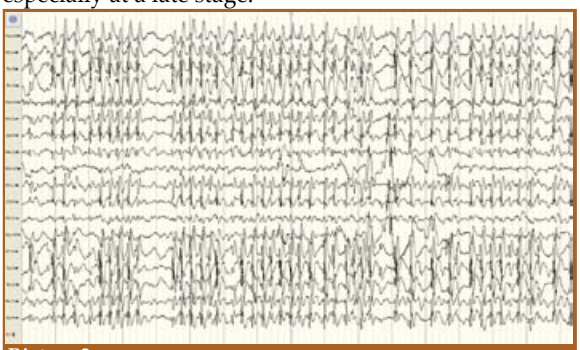
Seizure reduction by 50-90% was achieved by 23% of patients with the following aetiologies: Ohtahara's disease, Lennox Gastaut Syndrome, symptomatic generalized epilepsy.

There was no significant change in seizure occurrence in 30% of patients in whom etiologies included symptomatic focal epilepsy with venous malformation, symptomatic generalized epilepsy, symptomatic focal epilepsy, one due to focal cortical dysplasia IIB and the other due to focal cortical dysplasia IIA. In the latter two patients, epilepsy improved after resective epilepsy surgery.

Side effects of the Ketogenic Diet and Modified Atkin Diet included iron deficiency anaemia that required iron therapy; infection; hypercalciuria that improved following oral potassium citrate, none of patients had renal stone; elevated serum LDL that normalized after change from Ketocal to Calogen; hypertriglyceridemia that normalized following calorie restriction.[10]

Landau Kleffner Syndrome (LKS)

LKS, also called acquired epileptic aphasia, is a rare childhood neurological disorder characterized by language regression (after the child has already shown a period of normal language development) with sudden or gradual loss of language comprehension (auditory verbal agnosia) and verbal expression (aphasia). LKS usually begins between 3 and 7 years of age. Awake EEG shows slow wave, spike and slow wave over the centro-temporo-parietal region that becomes bilateral synchronous and generalised during sleep, and in some is associated with continuous electrical status epilepticus of slow sleep.[11] LKS is an epileptic encephalopathy, attributed to epileptiform activity that is often subclinical, and associated with long term cognitive, behavioural and motor control impairment.[11] Clinical seizures, most often absence seizures, are noted in most patients. The anticonvulsants of choice include sodium valproate and a benzodiazepine. Reversal of the aphasia and abnormal EEG is difficult and immunotherapy such as steroids or intravenous immunoglobulin is usually required. Some resistant cases have been reported to improve following MST surgery (Perisylvian Multiple Subpial Transections deep into the sylvian fissue). LKS has occasionally been misdiagnosed as autism, especially at a late stage.[11]



Picture 2
Sleep EEG shows electrical status epilepticus in sleep in a child with LKS

In a local case study, a toddler with pharmacoresistant LKS developed language regression despite prescription of sodium valproate and Clonazepine. The child responded well to high dose oral prednisolone and language returned to normal. Nonetheless language ability began to regress on commencement of steroid withdrawal. The patient was switched to monthly pulsed intravenous methylprednisolone with the aim to minimize steroid side effects. Because of concern about long term steroid side effects, the patient was then changed to monthly intravenous immunoglobulin (IVIG). In this patient we were able to compare the effectiveness of three medical treatments by measuring the duration of disease (language regression) remission and monitoring side effects.

1) Continuous high dose oral prednisolone 2mg/kg/day for 2 months then tailed off;
2) Monthly pulse intravenous methylprednisolone 30mg/kg/day for 2-4 days;
3) Monthly intravenous immunoglobulin 2g/kg.

Although all three different treatments could achieve remission of disease (language regression), they differed in terms of time taken to achieve remission and side effects. Continuous high dose oral prednisolone took the longest time (i.e. 26 days), was least tolerable and associated with most side effects e.g. Moon face, Cushing´s features, myopathy. Monthly pulse intravenous methylprednisolone took a shorter time (i.e.7 days) with speech improvement and fewer side effects e.g. mild Cushing features with moon face when compared with high dose oral prednisolone. Monthly IVIG was the best tolerated therapy with least side effects e.g. transient headache and took a shorter time (i.e.7 days) with language improvement[10].

Conclusion

The success of seizure control following surgery for

refractory epilepsy is influenced by the aetiology of the epilepsy. Surgery for temporal lobe epilepsy achieves better seizure outcome than surgery for extratemporal lobe epilepsy. Surgery may 'cure' patients of their epilepsy. Nonetheless where this is not possible, in a significant proportion of patients, epilepsy can change from being pharmacoresistant to being pharmacoresponsive with a lower dose requirement of antiepileptic drugs. The ketogenic diet or modified Atkin Diet also offer safe and effective treatment for refractory epilepsy. Although there are side effects, most can be corrected.

Landau Kleffner Syndrome is an acquired epileptic aphasia, sometimes misdiagnosed as autism. A high index of clinical suspicion and sleep EEG is required to make an early diagnosis. Early intervention with immunotherapy including steroid or IVIG is required to optimize the long term outcome of the disease.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Treatment Modalities for Paediatric Refractory Epilepsy: Epilepsy Surgery, Ketogenic Diet and Immunotherapy in Landau Kleffner Syndrome. Current Evidence and Local Experience" by Dr Mario CHAK 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 30 April 2016. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

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

1. Patients with epilepsy that is uncontrolled by one antiepileptic drug should be referred to a tertiary center for further evaluation
2. Pre-surgical evaluation for epilepsy surgery includes MRI, longterm video EEG monitoring, Ictal SPECT, PET, and neuropsychological assessment
3. Seizure free outcome is more frequent following extratemporal resection than after temporal resection
4. According to a large scale study at the Cleveland Clinic, cortical dysplasia and low grade tumour were the most common causes of intractable epilepsy
5. According to a local clinical audit, the seizure outcome is good in refractory epilepsy children with etiology of developmental tumor, cortical dysplasia and mesial temporal sclerosis
6. Engel class 1 postoperative outcome indicates the patient is completely seizure free since surgery
7. The NICE guidelines recommend referral of children who do not respond to appropriate antiepileptic drugs to a tertiary paediatric epilepsy specialist for consideration of a ketogenic diet
8. Landau Kleffner syndrome, also called acquired epileptic aphasia, is characterized by language regression with auditory verbal agnosia and verbal expression
9. Landau Kleffner Syndrome can be misdiagnosed as autism
10. The effective treatment of Landau Kleffner Syndrome includes steroid and IVIG

ANSWER SHEET FOR APRIL 2016

Please return the completed answer sheet to the Federation Secretariat on or before 30 April 2016 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Treatment Modalities for Paediatric Refractory Epilepsy: Epilepsy Surgery, Ketogenic Diet and Immunotherapy in Landau Kleffner Syndrome. Current Evidence and Local Experience

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Answers to March 2016 Issue

Ketamine: Friend or Foe?

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

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Nutrition Information

	Per 100g Powder	Per 100 kcal*	Per 100 ml**
Energy kJ	3011	413	602
kcal	730	100	146
Protein g	15.25	2.1	3.1
Carbohydrate g	3	0.4	0.6
as sugars g	0.59	0.08	0.12
Fat g	73	10	14.6
of which saturates g	16.2	2.2	3.2
monounsaturates (cis fatty acids) g	17.4	2.4	3.5
polyunsaturates (cis fatty acids) g	10.9	1.5	2.2
total trans fatty acids g	25.3	3.5	5.1
% LCT	100		
Ratio n6: n3 fatty acids	11:1		
% energy from linoleic acid	13.8		
% energy from a linolenic acid	1.3		
Fat: protein + carbohydrate	4:1		
Fibre g	nil added		

* approximately 19.7g powder
** 20g made up to 100ml

Typical Amino Acid Profile	g / 100g Powder
L-Alanine	0.49
L-Arginine	0.52
L-Aspartic Acid	1.05
L-Cystine	0.35
L-Glutamic Acid	3.2
Glycine	0.26
L-Histidine	0.44
L-Isoleucine	0.8
L-Leucine	1.45
L-Lysine	1.2
L-Methionine	0.41
L-Phenylalanine	0.74
L-Proline	1.5
L-Serine	0.78
L-Threonine	0.85
L-Tryptophan	0.4
L-Tyrosine	0.78
L-Valine	0.96
L-Carnitine	0.04
Taurine	0.03

Carbohydrate Profile	g / 100g Carbohydrate	g / 100g Powder
Dextrose	1.7	0.05
Lactose	3.3	0.1
Sucrose	9.3	0.28
Maltose	5.3	0.16
Maltotriose	8	0.24
Raffinose	4.7	0.14
Higher Saccharides	67.7	2.03

Typical Fatty Acid Profile	g / 100g Fatty Acids
C12:0	0.1
C14:0	0.1
C16:0	12.1
C18:0	10.3
C18:1 cis	25.0
C18:1 trans	34.3
C18:2 cis	14.5
C18:2 trans	1.6
C18:3 cis	1.2
C18:3 trans	0.3
C20:0	0.3
C22:0	0.2

Vitamins	Per 100g Powder	Per 100 kcal*	Per 100 ml**
Vitamin A µg RE	380	52.1	76
IU	1265	173	253
Vitamin D µg	5.2	0.71	1
IU	209	28.5	41.8
Vitamin E mg α T.E.	7.4	1	1.5
IU	11	1.5	2.2
Vitamin C mg	45	6.2	9
Vitamin K µg	30	4.1	6
Thiamin mg	0.7	0.1	0.14
Riboflavin mg	0.75	0.1	0.15
Niacin mg	7.5	1	1.5
Niacin equivalent (mg NE)	14.2	1.9	2.8
Vitamin B6 mg	0.75	0.1	0.15
Folic Acid µg	110	15.1	22
Vitamin B12 µg	0.8	0.11	0.16
Biotin µg	20	2.7	4
Pantothenic Acid mg	3	0.41	0.6
Choline mg	250	34.3	50
Inositol mg	20	2.7	4

Minerals	Per 100g Powder	Per 100 kcal*	Per100 ml**
Sodium mg	500	68.5	100
mmol	21.7	3	4.3
Potassium mg	800	110	160
mmol	20.5	2.8	4.1
Chloride mg	750	103	150
mmol	21.4	3	4.3
Calcium mg	430	58.9	86
Phosphorus mg	430	58.9	86
Magnesium mg	110	15.1	22

Trace Elements	Per 100g Powder	Per 100 kcal*	Per100 ml**
Iron mg	7.4	1	1.5
Copper µg	600	82.2	120
Zinc mg	6	0.82	1.2
Manganese mg	0.65	0.09	0.13
Iodine µg	90	12.3	18
Molybdenum µg	30	4.1	6
Selenium µg	22	3	4.4
Chromium µg	15	2.1	3

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Art as a Language for Children in Need

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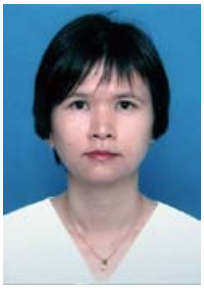

Prof Sophia LAW

When people think of art, masters and masterpieces often spring to mind. In this article, art is considered in its fundamental sense - as a language written in images. Art involves visual thinking and the use of imagery rather than language for the construction, articulation, and expression of thoughts and feelings. For children whose language skills are still developing, image writing is the best means of expression. This article reveals the significance of image thinking and the biological origin of art making. A case study is used to illustrate how art can help children in need.

Sight Comes Before Words

Of the five senses, neuroscientists have found vision to be the most complex and richest (Restak, 1994). In his book Descartes' *Error*, Damasio (1994) explains in detail the vibrant role played by visual perception in our mind in terms of cognitive and emotional aspects. 'Images are probably the main content of our thoughts (p. 107),' he claims, 'both words and arbitrary symbols are based on topographically organized representations and can become images. Most of the words we use in our inner speech, before speaking or writing a sentence, exist as auditory or visual images in our consciousness (p. 106).' Art making is image making, and as our thoughts exist mainly in imagery, artistic creation is undeniably a valuable way to represent both thoughts and feelings. This is particularly so for children whose cognitive and emotional development largely depends on their visual perception.

Young children, given a pen, appear compelled to draw. They get excited about exploring lines and forms on a surface. Anthropologists hypothesize that art making has a biological origin in human evolution. Alland (1997) claims that art evolved as a kind of exploratory play in which the external world is searched and manipulated by the inner world of the imagination. Art making not only enhanced our visual acuity and hand-eye coordination, which were significant in the evolutionary development of Homo Sapiens, but also supported a highly developed capacity for pattern recognition and symbol association – both of which are crucial to the appearance of written languages (pp. 22-26). Likewise, Dissanayake (1995) redefines art, as found in rituals, as a unique human behaviour of "making special". She explains that art and ritual in primitive cultures can be understood as a way that ancient people allayed their anxiety in response to life's uncertainties. Modern philosophy of art and aesthetics also hints at the biological origin of art appreciation. Dutten (2009) notices that specific elements in a landscape such as open spaces of grasses, the presence of water and a distant horizon evoke intrinsic pleasure in humans across different cultures. These shared responses to 'preferred landscapes' are signs of atavism. The preferred landscape is characterized by two features, coherence and legibility, that promote orientation and invite exploration. Dutton also argues that art appreciation appeared first as an evolutionary adaptation. All these aforementioned hypotheses and arguments indicate that art is not exclusive for the talented, but rather an innate behaviour of which we are all capable.

The Quality of 'Aboutness'

According to the neuroscientist Churchland (1995), 'the vast majority of our cognitive activities take place at levels well below the conscious level' (p. 182). Spontaneity induces flows of thought that can slip under the guard of our consciousness. Artistic creativity of any kind involves a different degree of spontaneity, and thus often provides information that reveals the unconscious. Nonetheless art making is a self-directed process. The dynamic flow between the conscious and the unconscious during a creative process is described by Allen (1977): 'Our response to art is a unique combination of emotion and subconscious cognitive activity. Art makes the subconscious symbolic system real and objectively present' (p. 20). Art is primarily about emotion rather than rationality. It relies on senses rather than intellectual ability and thus applies to all walks of life. Take drawing as an example. The artist's colours, lines, forms choices and the final composition are all directed by his or her state of mind at the time of execution. Whatever is within is transformed into kinetic forms of expression through brushstrokes, line rhythms, colour intensity and tones and compositional arrangement - with or without the artist's consciousness. Art making is indeed a symbolic practice that has a quality of 'aboutness' (ibid 1995). Once an image is made, what was once internal has been physically manifested. It can be seen, and thus prompts a change in the artist from an unconscious to a conscious state (Schaverien 1988).

Art and Childhood Trauma

Children are less able than adults to face adversity. This is particularly so for children who sustain traumatic experiences such as family violence and chronic illness. Given that their cognitive ability and language skills are still developing, many of the issues arising from

their situation are incomprehensible. These children are often confused and living with complex emotions - fear, anger, guilt and sadness - that are literally 'unspeakable'. These accumulated repressed emotions can affect their development, particularly their self-regulation, self-concept and interpersonal functioning (Cloitre et al., 2009). Without early intervention, many of these children will develop personality and behavioural disorders as they grow older (Bennett et al. 2005; van der Kolk 2005). A fundamental focus of intervention for these children is to help them liberate and visualize their inner feelings, and art in the forms of play and image writing is best suited to this purpose.

'Children need to have the opportunity to process traumatic experiences in a manner consistent with their cognitive and emotional development (Arvidson et. al. 2011, p.38).' As a form of play, art creates a non-verbal platform for children to disclose and develop their abstract thinking and feelings in images. The process of art making provides them with a self-directed, joyful platform that is safe and natural to project their feelings under the guise of play. The ways in which they create, and the final products, reveal their perceptions of the world. These images can then facilitate our understanding of their developmental needs, allowing us to help them release the repressed emotions – a process that enhances their self-esteem and promotes their ability to manage feelings and problem-solve.

Visualization of What's Within

A one-year art therapy programme was conducted for eight child victims of family violence in 2013. Y was an 11-year-old girl whose parents had separated, and she was living with her father when she enrolled in the programme. Two weeks before the programme started, Y's father committed suicide and she had to move back in with her mother who had previously abused her. Given the very intense and complex reality, she had great difficulty understanding and expressing herself. The programme helped her vent and visualize her hidden emotions, and gain a better understanding of her reality. The focus of this article is not the overall art therapy process, but rather some images of Y's work that illustrate how art helped her to voice her otherwise muted inner emotions.

Y appeared to be calm in the first few individual sessions, and it was decided that she was in denial of her father's death.[1] In the first session, she chose to do free drawing and started looking for a black pen, explaining that black was her favourite colour. No black pen was available, so Y picked purple and mixed it with other colours to outline her image (Drawing 1), a monster with two heads sharing one body, with four feet that were webbed like ducks'. She then drew a bold line dividing it in half, one half coloured in purple and the other in blue. She narrated that the monster had been split by a cut, and although both halves survived, she stressed that the splitting had really hurt. Later, she added a patch of substance right below the purple head, concluding that this half had a running nose and the separation was a good thing.

Drawing 1: A monster with two heads sharing one body and four feet

Drawing 2: A zoo getting ready to move to a new site

Drawing 3: The new zoo

In the second session, Y drew a story about a zoo during the time when things were being moved to a new site (Drawing 2). She started with a black marker and drew a cage inhabited by a 'Giraffe' (aged 59) and a small 'Hamster' (1 month old) in separate compartments. Y narrated that all animals except these two were leaving. Giraffe had been abandoned because it was too big to be moved. Hamster, as Giraffe's best friend, had decided to stay. Hamster became agitated whenever humans came close. In the right upper corner, Y drew an 80-year-old person pushing a grumbling lion toward a truck. Interestingly, Y did not put Giraffe and Hamster next to one another. Instead, they were distanced by an empty compartment in between. Finally, Y made some 'food' with yellow playdough to feed the animals. In the following session, Y worked on a new zoo in water colours (Drawing 3). In her own narrative, the bottom

[1] *When her case social worker asked her how she felt about her father's death, Y answered: "I'd take it as he would be sleeping for a very long period."*

green area was a land filled with spikes and the blue stood for Mother tortoise and her baby son. Mother Tortoise had been wounded by the spikes and was bleeding and crying as her baby tried to comfort her with hugs. On the right was a light green giraffe, and Y did not specify whether it was a new giraffe or not. She said it was crying and used her fingers to paint the tears in blue dots.

Y's choice of black and her narration of a zoo and animals are unusual images for a child. They were full of negative implications – darkness, splitting, separation, segregation, loneliness, anger, wounds and tears - all of which elicit vivid, tragic feelings. Confusion and conflict were also expressed by the lack of definite heads and tails, and the extreme contrasts in size and age among her characters. The food she made might imply nurturing, specifically a fulfilment of her emotional needs. Given Y's reality, her stories were undeniably connected to her suffering. Her narration of the 'separation' was symbolic of the pain and confusion created by her own loss that was too difficult for a child of her age to articulate. Her creations helped her channel her inner feelings in a detached way. Neuroscientists have found that repressed memories have strong sensorimotor and highly visual qualities. Thus, the best way to access traumatic memories is through non-verbal visual means such as drawing, given their visual and spontaneous nature (Greenburg & van der Kolk, 1987). This was evidenced by another of Y's work - a paper clay cat. She became increasingly silent and totally absorbed in the process of smoothing the surface of her cat. Only when she realised that the session was close to ending did she quickly made a bed, a red pillow, a black blanket and a ball. Drawing 4 is her presentation, a cat in a bed on a red pillow with a ball stuffed in its mouth. In the following session, Y admitted that when she was making the cat, memories of her father's funeral entered her mind. She remembered standing still in a very cold room where her father was lying in his coffin on a red pillow. Her mother had stuffed something into her father's mouth. It was the first time Y had talked about the funeral in such detail. Before the art sessions, neither she nor the art therapist would have expected a breakthrough such as this. It was probably the action of smoothing the paper clay that triggered thoughts of caressing and the associated emotional memories of her father.

Clinical reports of child abuse in which drawing is adopted as part of treatment, confirm that 'art work can provide a vehicle for bringing even deeply repressed trauma to the surface where it can be balanced by the outer world' (Stember & Halpert 1980, pp.59-63). Y's images helped her to visualize and acknowledge her inner feelings, and this is the very first step to help children whose suffering is difficult to articulate. Her case shows how art can help children in need.

Drawing 4: The cat lying on a red pillow

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戲劇普通話班

香港醫學組織聯會基金在1月8日至2月5日逢星期五舉行戲劇普通話班， 為小學生提供既有趣，又能增長語言能力的課外活動。以下是課程導師簫老師對活動的簡介。

一、 課程摘要

英國的Allardyee Nicoll曾在《西歐戲劇理論》中說過“戲劇是生活的摹本、習俗的鏡子、真實的反應”，能令大眾感同身受，這種生動、有趣的呈現形式更深受學生歡迎。我身為教育工作者，將戲劇融入到教學中去，對學生的學習、發展具很大的意義，同時對學生學習普通話更有很大的幫助。

二、 戲劇教學課程介紹

推廣創造性的戲劇是值得讚賞的，“美國藝評家Kenneth.L Grahani指出學生戲劇至少有五種價值，分別是娛樂、心靈的成長、教育的發展、美學的欣賞及未來觀眾的培養。”戲劇教學是藉由一個故事，用生動、有趣的戲劇表演方式進行主題教學。可使兒童從中認識多元文化，更能訓練兒童語言表達能力，瞭解自我，發展潛能，學習人際溝通，尊重、關懷與團隊合作，以及獨立思考，解決問題的能力。學生透過角色扮演投入戲劇表演，理解事物關係。過程中，學生要以說話、聆聽、眼神等各樣感觀和肢體表達所思所感，因此“戲劇是訴諸多種溝通媒介的學習模式。” 我希望通過從戲劇教學的設計到實施，過程中能幫助學生更容易理解授課內容，享受學習的樂趣，同時培養學生語言表達的完整性和邏輯性。

考慮到以上因素，本人設計課程內容主要包括：普通話漢語拼音、戲劇作品欣賞及朗讀劇場。希望學生修畢此課程之後能夠：

1. 學習、掌握及運用漢語拼音，提升普通話表達能力；
2. 透過戲劇作品欣賞進而加強閱讀理解、欣賞作品的能力；
3. 最後在朗讀劇場的過程中，將以上所學加以鞏固和運用；
4. 運用戲劇教學激發學生學習語言（普通話）的興趣；
5. 運用戲劇教學提高學生的說話（普通話）能力；
6. 運用戲劇教學增強學生學習語言的自信心；

三、 課程特色

課程的特色包括：摒除沉悶、冗長的教學方法，採取生動、有趣的教學模式，從而走出傳統教學的限制，幫助學生提高普通話說話能力，進一步激發學生語言學習的興趣，培養學生良好的語言學習習慣，輕鬆、愉快地學習、掌握。在整個教學過程中，所有同學的表現都非常積極、投入。透過作品分析、理解，朗讀劇本和表演等環節，組織學生分組討論，鼓勵他們自由發揮。其中朗讀訓練也是教學的重要環節之一，“「朗讀」是劇場的靈魂。朗讀者必須結合聲音及感情，把劇本清楚讀出來。”所以我認為戲劇教學就是幫助母語非普通話學生透過聆聽、訓練、表演等形式來提升學生語言的理解、模仿、表達能力，激發學生學習和表達的動機。同時更能幫助學生學習提高讀理解文章的能力，對寫作訓練也有相當大的幫助。

四、 總結

課程結束後，當同學們提及課堂感受，都不約而同地認參加此課程時不但開心，而且同時提高了彼此之間的合作能力、互相欣賞能力，更有效地提高了作品的鑑賞能力。更令我欣慰的是所有家長也對這個課程相當滿意，更提出什時候可以再開班的問題。

Spring Dinner 2016

Everything is beautiful in spring! The Federation of Medical Societies of Hong Kong held its Spring Dinner 2016 on 24th February to celebrate the festive season. The executive committee members and federation colleagues enjoyed the dinner very much. All of us at the Federation send our readers good wishes for a prosperous year of the Monkey!

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
					1	2
3	4	★ HKMA Council Meeting ★ FMSHK Officers' Meeting 5	★ MPS Workshop – Mastering Your Risk 6	★ MPS Workshop – Mastering Adverse Outcomes 7	8	★ CME Lecture - Refresher Course for Health Care Providers 2015/2016 9
10	★ Young Man with Huge Abdominal Mass 11	★ HKMA Kowloon West Community Network - SGLT2 inhibitors - A Novel Mechanism for the Management of Type 2 Diabetes ★ HKMA Tai Po Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice? 12	★ Hong Kong Neurosurgical Society Monthly Academic Meeting –Primary Intracranial Germ Cell Tumours ★ HKMA Central, Western & Southern Community Network - Better LUTS Management, Better Day for Your Patients ★ MPS Workshop – Mastering Professional Interactions 13	★ HKMA Hong Kong East Community Network - Certificate Course on Eye Diseases (Session 1) - Common Retinal Disease: Diabetic Retinopathy and Age-related Macular Degeneration (AMD) ★ HKMA Kowloon East Community Network - Certificate Course on Diabetes Mellitus (Session 3) – Achieving Therapeutic Goals ★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2016 – Oocyte Freezing ★ MPS Workshop - Mastering Difficult Interactions with Patients ★ FMSHK Executive Committee Meeting 14	15	★ MPS Workshop – Mastering Adverse Outcomes 16
17	18	19	★ MPS Workshop - Mastering Your Risk 20	★ MPS Workshop - Achieving Safer and Reliable Practice ★ FMSHK Foundation Meeting 21	22	★ HKMA KECN, HKCFP & UCH – CME Course for Health Personnel 2016 (Session 1): Common Gynecological Problems ★ MPS Workshop - Achieving Safer and Reliable Practice 23
24	25	26	27	★ HKMA Hong Kong East Community Network - Certificate Course on Eye Diseases (Session 2) - Latest Advances in Cataract Surgeries and Glaucoma Treatment ★ HKMA Kowloon East Community Network - Nutritional Strategies for the Primary Prevention of Allergies 28	29	30

Date / Time	Function	Enquiry / Remarks
5 TUE 8:00 PM	**HKMA Council Meeting** Organiser:The Hong Kong Medical Association; Chairman:Dr. SHIH Tai Cho, Louis; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
8:00 PM	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Gallop, 2/F, Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
6 WED 6:30 PM	**MPS Workshop – Mastering Your Risk** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. LEE Wai Hung, Danny; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
7 THU 6:30 PM	**MPS Workshop – Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. HUNG Chi Wan, Emily; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
9 SAT 2:15 PM	**CME Lecture - Refresher Course for Health Care Providers 2015/2016** Organiser: The Hong Kong Medical Association; Speaker: Dr. LAM Wing Wo; Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Point
11 MON 7:30 PM	**Young Man with Huge Abdominal Mass** Organiser: Hong Kong Urological Association; Chairman: Dr Eric LI; PYNEH; Speaker: Dr. Jeffrey YU, PYNEH; Venue:Multi- disciplinary Simulation and Skills Centre, 4/F, Block F, QEH	Ms. Tammy Hung Tel: 9609 6064 1 CME Point
12 TUE 1:00 PM	**HKMA Kowloon West Community Network - SGLT2 inhibitors - A Novel Mechanism for the Management of Type 2 Diabetes** Organiser: HKMA Kowloon West Community Network; Chairman:Dr. LEUNG Kin Nin, Kenneth; Speaker: Dr. YIP Wai Man; Venue: Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA Tai Po Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?** Organiser: HKMA Tai Po Community Network and Primary Care Office of the Department of Health; Chairman:Dr. CHOW Chun Kwan, John; Speaker: Dr. FUNG Wai Han, Amy; Venue: Chiuchow Garden Restaurant(潮江春) Shop 001-003, 1/F, Uptown Plaza (新達廣場), No.9 Nam Wan Road, Tai Po	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
13 WED 7:30 AM	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Primary Intracranial Germ Cell Tumours** Organiser: Hong Kong Neurosurgical Society; Chairman: Dr ZHU Xian Lun; Speaker: Dr CHAN Sik Kwan; Venue: M Block Ground Floor Lecture Theatre, Queen Elizabeth Hospital	Dr. Michael LEE Tel: 2595 6456 1.5 CME Point
1:00 PM	**HKMA Central, Western & Southern Community Network - Better LUTS Management, Better Day for Your Patients** Organiser: HKMA Central, Western & Southern Community Network; Chairman:Dr. POON Man Kay; Speaker: Dr. YIP Wai Chun, Andrew; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
6:30 PM	**MPS Workshop – Mastering Professional Interactions** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. LEE Wai Hung, Danny; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
14 THU 1:00 PM	**HKMA Hong Kong East Community Network - Certificate Course on Eye Diseases (Session 1) - Common Retinal Disease: Diabetic Retinopathy and Age-related Macular Degeneration (AMD)** Organiser: HKMA Hong Kong East Community Network; Chairman:Dr. AU Chi Lap, Simon; Speaker: Dr. YUEN Hsu, Leonard; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA Kowloon East Community Network - Certificate Course on Diabetes Mellitus (Session 3) – Achieving Therapeutic Goals** Organiser: HKMA Kowloon East Community Network; Chairman:Dr. SHIU Ka Lok, Ivan; Speaker: Dr. TING Zhao Wei, Rose; Venue: V Cuisine, 6/F., Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tseung Kwan O	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
2:00 PM	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2016 – Oocyte Freezing** Organiser: The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital; Speaker: Dr. TANG Oi Shan; Venue: Function Room A, HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
6:30 PM	**MPS Workshop - Mastering Difficult Interactions with Patients** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. FUNG Shu Yan, Anthony; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
8:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
16 SAT 2:30 PM	**MPS Workshop – Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. LEE Wai Hung, Danny; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
20 WED 6:30 PM	**MPS Workshop - Mastering Your Risk** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. LEE Wai Hung, Danny; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
21 THU 6:30 PM	**MPS Workshop - Achieving Safer and Reliable Practice** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: Holiday Inn Golden Mile, 50 Nathan Road, Tsim Sha Tsui	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point

Date / Time	Function	Enquiry / Remarks
21 THU 8:00 PM	**FMSHK Foundation Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
23 SAT 1:30 PM	**HKMA KECN, HKCFP & UCH – CME Course for Health Personnel 2016 (Session 1): Common Gynecological Problems** Organiser:HKMA Kowloon East Community Network & Hong Kong College of Family Physicians & United Christian Hospital; Chairman:Dr. SHA Kwok Yiu, Edmund; Speaker: Dr. LAM Wai Cheung, Mona; Venue: 1. Lecture Theatre, G/F, Block K, United Christian Hospital (UCH), 130 Hip Wo Street, Kwun Tong, Kowloon 2. Conference Room, G/F, Block K, UCH (video conference)	Ms. Hana YEUNG Tel: 2527 8285 1.5 CME Point
2:30 PM	**MPS Workshop - Achieving Safer and Reliable Practice** Organiser: The Hong Kong Medical Association & Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 2.5 CME Point
28 THU 1:00 PM	**HKMA Hong Kong East Community Network - Certificate Course on Eye Diseases (Session 2) - Latest Advances in Cataract Surgeries and Glaucoma Treatment** Organiser: HKMA Hong Kong East Community Network; Chairman:Dr. AU YEUNG Shiu Hing; Speaker: Dr. YUEN Shi Yin, Nancy; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA Kowloon East Community Network - Nutritional Strategies for the Primary Prevention of Allergies** Organiser: HKMA Kowloon East Community Network; Chairman:Dr. MA Ping Kwan, Danny; Speaker: Dr. HUNG Chi Wan, Emily; Venue: V Cuisine, 6/F., Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tseung Kwan O	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point

Upcoming Meeting

7/5/2016 9:00AM (7-8&14-15 May)	**BodyTalk Fundamentals Seminars** Organiser: BodyTalk Hong Kong; Speaker: Ms Angie TOURANI; Venue: All About You Wellness Center; Rm 1403, Kaiseng Commercial Centre, 4-6 Hankow Rd, TST, Kowloon, Hong Kong	Ms. Angie TOURANI Tel: 6683 5755 Email: angie@bodytalksystem.com.hk Website: www.bodytalksystem.com.hk
24/5/2016 6:30 PM	**How to Relieve Menstrual Cramps by Means of Traditional Chinese Medicinal Nursing** Organiser: Hong Kong College of Chinese Medicinal Nursing; Chairman: Ms HUI Yin Hing, Erika; Speakers: Mr LAU Pak Shing; Ms PANG Wai Sam, Nicki; Venue: Seminar Room 3, LG 1, Ruttonjee Hospital Wanchai, Hong Kong	Ms. Nicki PANG Tel: 9320 5076 3 CNE Point
23/7/2016 12:00 PM	**Hong Kong College of Health Service Executives Annual Conference 2016 - People, Technology, and Innovation** Organiser: HKCHSE; Venue: Shanghai room, Level 8, Cordis Hong Kong, 555 Shanghai Street, Mongkok	Ms Eva TSANG Tel: 2821 3514 Fax: 2865 0345

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

(Effective from October 2009)

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

Answers to Radiology Quiz

Answer:

1. X ray of the knee in this skeletally immature patient reveals increased ill-defined sclerosis over the metaphyseal region of the proximal tibia with cortical disruption and aggressive-looking periosteal reaction (Sunburst and Codman triangle appearance). The zone of transition is wide. Increased soft tissue swelling is also present.

   MRI T2 fat-saturated images of the other patient reveals a heterogenous T2 hyperintense signal over the metadiaphyseal region of the distal femur with areas of cortical destruction and also sunburst type of periosteal reaction. Areas of increased signal noted over the soft tissue of the posterosuperior margin of the lesion are suspicious of involvement as well.

2. Both images are in keeping with an aggressive bony tumour lesion, most commonly due to osteogenic sarcoma.

3. They are broadly divided into intramedullary (80%), surface or juxtacortical (10-15%) and extraskeletal (5%). They can be primary or secondary.

   Primary osteosarcoma: typically occurs in young patients (10-20 years) with 75% occurring before the age of 20 because the growth centers of the bone are more active during puberty/adolescence; slight male predominance.

   Secondary osteosarcoma: occurs in the elderly; usually secondary to malignant degeneration of Paget's disease, extensive bone infarcts, post-radiotherapy for other conditions, osteochondroma, and osteoblastoma.

4. MRI is proving essential for accurate local staging and assessment for limb sparing resection, particularly in evaluation for intraosseous tumour extension and soft-tissue and neurovascular bundle involvement. Assessment of the growth plate is also essential since up to 75-88% of metaphyseal tumours cross the growth plate into the epiphysis.

Dr Bruce LEE
Department of Radiology, Queen Mary Hospital

Support gut health with unique Synbiotics

Aside from nutrient digestion and absorption, the gut also plays a key role in immunity and other physiological functions important to the overall well-being of a baby.[1]

However, the gut is still immature during the first few months,[2-4] and can be prone to gastrointestinal symptoms,[5] which may cause distress to parents.[6-7]

The unique Synbiotics

Prebiotics

scGOS/lcFOS (9:1)

Patented and most well-studied for clinical benefits[8]

Probiotics

***Bifidobacterium breve* M-16V**

Most dominant bifidobacterial species in breastfed infants[9]

Clinically proven benefits:

- Stimulate the growth of *Bifidobacteria*[10]
- Help support natural defences[11,12]
- Help promote a healthy digestive system[10]

For healthcare professionals only

Important Notice:
Breastfeeding is best for babies and provides the best start in life. It is important that, in preparation for and during breastfeeding, pregnant women eat a healthy, balanced diet. Combined breast and bottle feeding in the first weeks of life may reduce the supply of mothers' own breast milk, and reversing the decision not to breastfeed is difficult. The social and financial implications of using infant formula should be considered. Improper use of an infant milk or inappropriate foods or feeding methods may present a health hazard. If mothers use infant formula, they should follow the manufacturer's instructions for use carefully – failure to follow the instructions may make their babies ill. It is recommended for mothers to consult doctors, midwives or health visitors for advice about feeding their babies.

References

1. Bischoff SC. *BMC Med*. 2011;9:24. **2.** Shamir R, et al. *J Pediatr Gastroenterol Nutr*. 2013;57 Suppl 1:S1-45. **3.** van Tilburg MA, et al. *J Pediatr*. 2015;166:684-689. **4.** Savino F, et al. *Acta Paediatrica*. 2005;94 Suppl 449:120-124 **5.** Vandenplas Y, et al. *Nutrition*. 2013;29:184-194. **6.** Vik T, et al. *Acta Paediatr*. 2009;98(8):1344-1348. **7.** Akman I, et al. *Arch Dis Child*. 2006 May;91(5):417-419. **8.** Boehm G, et al. *J Nutr*. 2008;138:1818S-1828S. **9.** Mackie RI, et al. *Am J Clin Nutr*. 1999;69:1035S-1045S. **10.** Moro G, et al. *J Pediatr Gastroenterol Nutr*. 2002;34:291–295. **11.** Arslanoglu S, et al. *J Nutr*. 2008;138:1091–1095. **12.** Arslanoglu S, et al. *J Biol Regul Homeost Agents*. 2012;26(S3):49–59.

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