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The Cover Shot

Taking off

Gazing through the window in a lazy afternoon, I spotted this heron just airborne. The flight was frozen in a split second and yet as remarkable as it is in photography, the action seemed to burst beyond this minuscule moment - this magnificent bird continued to glide effortlessly along in the air, determined and with an obvious target in mind.

Hong Kong has just broken ground for its Children's Hospital that is scheduled to be ready by 2018. Hope that it be like this bird in the air, majestically and smoothly gliding on, riding out the turbulence and convicted to serve our needy children with a world-class service, paediatric neurosurgery among them!

This photograph was taken with a Zoom-Nikkor 80-200 mm 1:4.5 on Nikon D800.

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Paediatric Neurosurgery

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Editor

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At birth, the central nervous system of the human being is relatively under developed. Compared to other mammals like a pony that would take just a couple of hours to start pacing with the mother, we humans take more than a year! Yet our brain continues to pick up, grow, expand and getting more and more sophisticated at least for the subsequent decade or two. During this time, the changes that take place in the brain are immense. That explains why a paediatric brain is not an adult brain in miniature but one that differs in terms of anatomy and physiology and thus even the management in time of diseases differs. A young child cannot give a precise history nor express clearly the exact symptoms. These are usually left to the vigilance of the carers or even the family.

Diseases that affect a child's central nervous system that is still growing very often lead to a life long impact of his ability to learn. Clinicians have to consider the long-term effects on the child when suggesting the best treatment option. In their long road to recovery, the education system and the community at large play key roles in fostering a truly successful rehabilitation.

Neurosurgeons who dedicated themselves in dealing with children soon find themselves adopting a very different mentality. Not only do they have to work very closely with fellow collaborators in dealing with a different spectrum of neurosurgical conditions, but also the families of these children, striving to seek the best of their rehabilitation and outcome.

So as neurosurgery evolves, paediatric neurosurgery is the first subspecialty that is established worldwide. The International Society of Paediatric Neurosurgery, as a forum for professional exchanges among paediatric neurosurgeons all over the world, has a history of 41 years. Locally, medical service is of a world standard and yet we still lack a children's hospital. Paediatric workload is shared among all neurosurgery departments making expertise difficult to attain. Paediatric neurosurgery has therefore not got the recognition it deserves. As it is now finally decided that Hong Kong is going to have its first hospital for children in 2018, it is high time that we prepare ourselves for it. This issue of the Medical Diary aims to give a brief introduction to some of the key specialties that support a comprehensive paediatric neurosurgery service.

A single issue of the Medical Diary would not possibly cover every aspect of paediatric neurosurgery but only serves to give readers a glimpse of the subspecialty. I am thankful to the contributors, a group of close collaborators in running such a service - apart from surgeons, we have our closest ally in the operating theatre, the neuroanaesthesiologists; and within the hospital, the neuro-oncologists and without, the development paediatricians who continue to look after our patients in their journey to recovery. Without their dedication, this issue would not be possible. The article by Mr CK Ng, a nurse consultant in neurosurgery who enjoys hiking in his leisure time also reminds us how beautiful the world is and how one can be reenergised from a hike out in nature after all the tension at work supporting us! I am sure you will all enjoy it like I do.

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Paediatric Neurosurgery – An Overview

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Dr Sui-to WONG

Development of Paediatric Neurosurgery into a Distinct Subspecialty

Paediatric neurosurgery has been well established as a distinct subspecialty in the Western world and in some of our nearby countries[6,10]. The first paediatric neurosurgery service began nearly 90 years ago at the Boston Children's Hospital. To propagate and expand the knowledge on the foundation created by the pioneers, the European Society for Paediatric Neurosurgery was founded in 1967, its American counterpart and the International Society both in 1972, the Japanese Society in 1973, and the Australian Society in 2002. Time and time again, all over the world, the development of paediatrc neurosurgery has gone through the same well-trodden paths: at the beginning, paediatric cases are handled by general neurosurgeons, usually the most senior surgeons. Then the need for a free-standing service is appreciated, but the sentiment is often met with resistance. Eventually, full-time paediatric neurosurgical practices in free-standing children's hospitals emerge and quickly become indispensable[4]. Nowadays, most well-known children's hospitals worldwide have a free-standing paediatric neurosurgery service staffed by full-time paediatric neurosurgeons[6,10]. In the USA, among approximately 3500 neurosurgeons, 235 have their primary practice devoted to children, and 136 are full-time paediatric neurosurgeons[1,3,5]. This slow but steady evolution of paediatric neurosurgery, into its current high status as a distinct subspeciality, is well founded on sound and pragmatic reasons.

Peculiarities of Paediatric Neurosurgery

Children have immature and developing body systems. Their immature cranial and spinal anatomy and physiology necessitate modifications of neurosurgical strategies and techniques customarily used in adults. To mention a few obvious examples: the proportionately larger heads, thin skulls, and sometimes open fontanelles in young children require special equipment even at the beginning steps of positioning and skull fixation; their delicate skulls and vulnerable dural sinuses are dangerous traps to the untrained; their small total circulating blood volume requires meticulous haemostasis and planned availability of blood products when excising vascular tumours or lesions; their immature nervous system and axial skeleton pose challenges at surgery ranging from intra-operative electrophyiological monitoring to instrumentation; their predilection to infection requires vigilant and "ritualistic" protocols in order to achieve good results[12].

Children are affected by a radically different spectrum of neurosurgical conditions compared to adults, most conspicuously, a large number of congenital and developmental malformations, a distinct hierarchy of brain tumours, and different prevalences of cerebrovascular diseases. Congenital conditions also include genetic disorders with slowly progressive symptoms such as neurofibromatosis and skeletal dysplasia. Even tumours with similar histological imprints to their adult counterparts may behave differently in the child, often carry a better prognosis, and children more often than not react and respond differently to standard therapies originally designed for adults[7].

The relatively low incidence of neurosurgical conditions in children calls for concentration of cases in centres. It has been shown repeatedly in the neurosurgical literature that clinical outcome correlates with caseload[17]. Locally, in the 3-year period from 2011-2013, there were altogether 820 operative paediatric neurosurgical cases in the public hospitals. This averages to 275 operative cases per year, a cohort size expected from a population base about half that of Hong Kong. This case number will likely go up with centralisation and reorganisation of clinical services. Centralizsation should also improve outcome through the salutary effects of larger caseloads, and inevitably an escalation of the level of competence in the providers. In a resource allocation point-of-view, centralisation is also more cost-effective[17]. More importantly, a comprehensive treatment centre allows paediatric neurosurgeons to work closely with experts in other paediatric specialities, thereby enhancing communication and collaborations and promoting research.

Children with neurosurgical problems require continuous medical, physical, psychological and social support, frequent re-evaluation, and rehabilitation. A multi-disciplinary team approach to their problems should be the norm; the team should consist not only of conventional therapists but unconventional ones such as play therapist and therapy pets (Figure1).

Figure 1: Dr. Hobbes, the therapy dog at the Department of Paediatric Neurosurgery, Kaiser Permanente Oakland Medical Centre, visiting a child in the intensive care unit (Courtesy of Mr. James Mitchell, Oakland, CA, USA).

Disease Categories and Common Disease Entities

I will highlight a few points in each of the disease categories and use examples to illustrate several basic principles in paediatric neurosurgery.

I. CONGENITAL AND DEVELOPMENTAL CONDITIONS OF THE HEAD

Common examples include encephalocoele, Chiari malformation, craniosynostosis, arachnoid cyst, hydrocephalus, and aqueductal stenosis[10].

Craniosynostosis –The management of craniosynostosis illustrates the importance of taking into account normal growth and development while treating young children. Craniosynostosis is the premature closure of cranial sutures leading to disproportionate growth of the cranial bones, resulting in unsightly craniofacial deformities, visual axis mal-alignment, and, in children with multi-suture involvement, intracranial hypertension and neurological consequences. Affected children usually have skull and facial abnormalities at birth. The standard treatment is cranial reconstruction, which involves the exposure of a large area of the cranium, various patterns of craniectomies depending on the suture(s) involved, re-shaping of the disassembled bone plates, and, finally, reassemblage of the bony jigsaw into a more pleasing and commodious head shape. One major concern of this surgery is blood loss because of the extensive soft tissue dissection and bone cuts. Thus, the older the child at the time of surgery, the larger the pre-surgical blood volume reserve, and the safer the operation (Figure 2)[16]. The counter argument against delayed surgery is that the brain grows most rapidly in the first year of life (Figure 2). It is therefore convenient to perform the operation early and make use of this brisk expansile phase as an inherent gauge to guide proper remolding of the reconstructed skull from the inside, as it were, and attain a better result (Figure 3). Another argument for early surgery concerns the possible deleterious effects on the developing brain of raised intracranial pressure, with which certain types of multiple sutures synostosis syndromes are undoubtedly associated. The timing of surgery is therefore a balance of all these considerations and more, and must be individualised according to the specific situation and the experience of the craniofacial team[14] .

Figure 2: Graph showing normal growth of the brain, and the total circulating blood volume in the first 6 years. Notice the brain grows rapidly in the first year. (Modified from: http://humanorigins.si.edu/human-characteristics/brains and reference 16.)

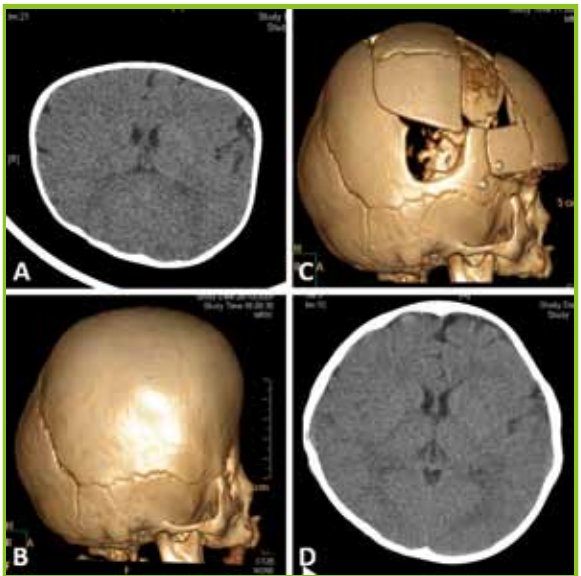

Figure 3: A patient with bilateral coronal suture synostosis presented to us with severe brachycephaly at 7-month-old. A &B: Pre-operative CT scan images showing markedly shortened anterior-posterior (AP) diameter of the skull, and "flattened" frontal lobes. C: CT at 3-week after reconstruction showing corrected AP diameter of the head, and the arrangement of the bone flaps as at surgery. D: CT at 1-year after operation showing the re-expanded frontal lobes.

II.CONGENITAL AND DEVELOPMENTAL CONDITIONS OF THE SPINE

Common examples include spinal dysraphism, craniovertebral junction anomalies, and other vertebral anomalies[10].

Spinal dysraphism – The management of dysraphic lesions exemplifies the need for paediatric neurosurgeons to learn embryology.

Spinal dysraphism, an umbrella term used to designate all forms of spinal neural tube defects, literally means defective midline fusion during the formation of the embryonic spinal cord. In truth, the formation of the spinal cord is more complicated. It begins with gastrulation, when the exceedingly early 2-layered embryo turns into a 3-layered (ecto-, meso-, and endoderm) embryo with a distinguishable longitudinal axis. The dorsal layer of the embryo along its longitudinal midline, called the neural plate, then goes through the sequential processes of primary neurulation, namely, its lateral sides roll up dorsally, their free-edges meet in the midline, then fuse together to form a tubular structure called the primary neural tube. Lastly, in secondary neurulation, the caudal end of the primary neural tube accumulates primitive pluripotential mesenchymal stem cells that subsequently transform in situ into a solid epithelial secondary neural tube (mesenchyme-epithelial transformation). According to this schema, the seemingly disparate crop of spinal dysraphic malformations can be classified under the relevant stage of embryogenesis in which the impugned insult occurs. Thus, a malformation may result from: 1) failure of gastrulation, such as split cord malformation, type I and II; 2) failure of primary neurulation, such as open neural tube defect (myelomeningocoele), limited dorsal myeloschisis, dermoid sinus tract and cyst, dorsal spinal cord lipoma; 3) failure of secondary neurulation, such as terminal myelocystocoele, retained medullary cord, terminal lipoma, and filum abnormalities; and 4) combined abnormality of both primary and secondary neurulations, such as transitional and perhaps chaotic spinal cord lipomas. With better understanding of embryology, new dysraphic malformations are described and new terms are coined: limited dorsal myeloschisis and retained medullary cord are recently described entities whose embryogenetic mechanisms have been well worked out. Also, old myths are debunked and erstwhile terms become obsolete and are discarded. Old terms such as leptomyelolipoma, lipomyelocoele, lipomyelomeningocoele, and lipomyelocystocoele supposedly designated different lesions, but are in fact fodders of over-zealous taxonomy, for we now know they all represent the same core embryological entity with minor variations in the fringes. They have been replaced by a simple, pragmatic term of spinal cord lipoma. Clinical observations and laboratory studies have verified these new revelations[9,15].

Besides improving nomenclature and stimulating academic research, a robust knowledge of embryology also serves the practical purpose in helping the neurosurgeon to decipher impossibly complex neuroimages of certain malformations, and in imbuing confidence to navigate safely and gainfully through the undulating and deceptive tissue planes at surgery. In lipoma surgery, for example, the appreciation of the morbid anatomy of the lipoma types - dorsal, transitional, chaotic, or terminal – verily dictates the correct surgical strategies in tackling these lesions. In the dorsal and transitional types, the surgeon is always able to find a fibrous white plane separating the dorsal surface of the spinal cord from the lipoma, enabling the surgeon to achieve total or near total excision of the fat (Figure 4). In the chaotic type, profound disruption of secondary neurulation leads to the admixture of fat throughout the substance of the neural placode and spillage of fat into the ventral surface of the cord, precluding complete resection. Treatment for chaotic lipomas is therefore "restrained" resection and expansile duraplasty rather than aggressive extirpation. On the flip side of labourious lipoma surgery, removal of a terminal lipoma requires no more than a sheer vertical "guillotining" at the well-demarcated fat-conus interface [15].

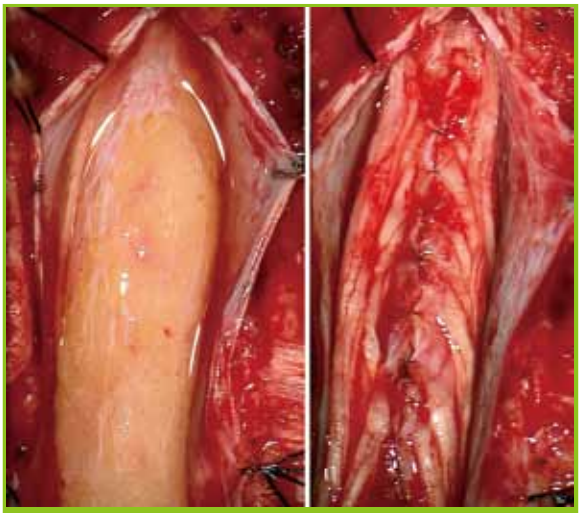

Figure 4: Spinal cord lipoma: Intra-operative photographs showing a transitional spinal cord lipoma (left panel), and the spinal cord after total excision of the lipoma and neurulation with microsutures (right panel).

III.TUMOURS

Common paediatric nervous system tumours include medulloblastoma, ependymoma, astrocytoma, germ cell tumours, craniopharyngioma, choroid plexus tumour, neuroblastoma, and nerve sheath tumours[10].

Perhaps more than for any other disease categories, the care of children suffering from nervous system tumours embodies the absolute need for a team approach. Though surgery remains the mainstay of treatment for most paediatric brain tumours, the comprehensive list of key health providers in the management of these children includes, besides the paediatric neurosurgeon, the paediatric neuro-oncologist, radiation oncologist, nurse practitioner, paediatric anaesthetist, intensvist, paediatric neurologist, paediatric physiatrist, social worker, and allied health providers. A successful paediatric neurooncology programme should be both protocol-driven and driving for new protocols. Describing such a programme deserves its own separate treatise beyond the scope of this overview.

IV.VASCULAR DISEASES

Common cerebrovascular diseases include arteriovenous malformation, cavernous malformation, moyamoya

disease, vein of Galen malformation, and intraventricular haemorrhage of the preterm[10,18].

An overview on vascular diseases is presented in another article. Molecular biology has greatly enriched our understanding of cerebrovascular lesions[2,11].

V.FUNCTIONAL DISORDERS

Common disorders include epilepsy, spasticity, and movement disorders[10].

Intractable epilepsy – Epilepsy surgery in children aims at eliminating medically intractable seizures and maximising the developmental potential of the growing brain. Epileptic discharges shut down normal brain activities, cause shunting of blood flow away from normal brain tissue, and temporarily deprive the brain of oxygenated blood. Repeated epileptic attacks are detrimental to the developing brain; the insults are additive and the adverse effects are cumulative.

A non-inclusive list of specialists for a paediatric epilepsy programme includes paediatric epileptologists, paediatric neurosurgeons, radiologists, nuclear medicine physicians, nurse practitioners, electrophysiologists, child psychologists, developmental paediatricians, and allied health workers.

VI.TRAUMA

Common scenarios include acute head and spine trauma, non-accidental injury, and brachial plexus injury[10].

The enormous differences in biomechanics between the adult and juvenile vertebral columns are highlighted in the landmark article defining the entity **spinal cord injury without radiological abnormality (SCIWORA)** in 1982. Due to the inherent elasticity and other age-related anatomical peculiarities, the spinal column in a young child can be deformed to a great extent without fracture or dislocation and consequently also without x-ray abnormality, but often at the expense of the underlying spinal cord[13].

Similarly, in the management of **severe head trauma**, the mechanisms of injury, physiological reactions of the brain to blunt forces, response to treatment, and outcome are very different in children compared to adults. There is very little overlap between paediatric and adult data in both spinal and cranial trauma[8].

VII.INFECTION

Common infections include bacterial meningitis, brain abscess, and subdural empyema[10].

VIII.HYDROCEPHALUS

The management of children with hydrocephalus exemplifies the need for long term monitoring of paediatric neurosurgical patients.

Shunting procedures and endoscopic third ventriculostomy have been the mainstays of treatment for childhood hydrocephalus. The two benchmark indices for shunting procedures are the shunt infection rate and the length of shunt survival, the latter relates closely to the correct positioning of the tip of the ventricular catheter. In treating children with hydrocephalus, additional challenges are the open sutures and the growing head and body. Over-shunting in children leads to spontaneous fusion of the cranial sutures, secondary microcephaly, and severe alteration of brain compliance. This can result in the dreaded "slit ventricle syndrome" and recurrent shunt occlusion, which may ultimately become untreatable. The distal catheter may pull out of the abdomen because of longitudinal growth, or the ventricular catheter may become extraventricular because of normal enlargement of the cranium, resulting in shunt failure at an older age. Even the newest programmable valves only address a limited aspect of the whole problem (Figure 5). On the same note, late deaths have been reported in children who had successful third ventriculostomy years ago, presumably due to sudden late occlusion of the ventriculostomy opening. Regular long term follow-up of children with shunts and ventriculostomy is therefore mandatory.

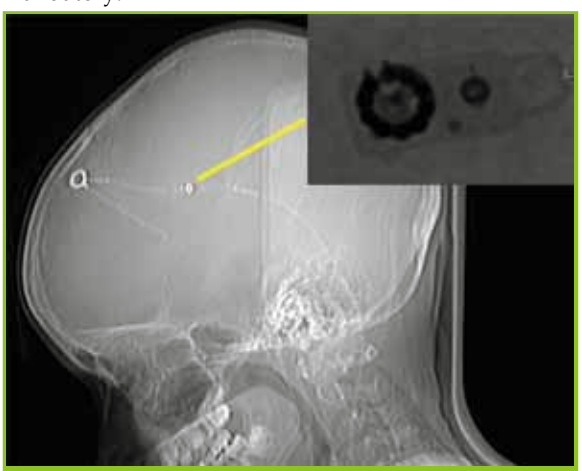

Figure 5: X-ray showing a programmable ventriculo-peritoneal shunt and a magnified view of the programmable component.

Multi-disciplinary Team Approach

From what has been presented here, it is easy to appreciate the merits of the team approach while one contemplates a centre for paediatric neurosurgery or neuroscience. The benefits of the team approach in present-day paediatric neurosurgery is readily shown when one accesses the websites of any of the prominent paediatric neurosurgery services in the world. For Hong Kong, bringing all relevant people to work in a children's hospital is a great step forward, but only a first step. In a true eutopian centre of paediatric neuroscience, the core members of the clinical team from diverse though related disciplines should be put under a single departmental governance. Management of complex diseases such as brain tumours and many dysraphic malformations automatically mobilises the entire team, jostling with lively discussions and disputations while planning and executing treatment together, with a single-minded and common goal of seeking and providing the best possible care to the patient, minus petty political games and machinations of personal egos. Because this arrangement breaks all traditional paradigms, it can only be realised by an unswerving resolve and ceaseless efforts on the part

of the supplicating clinical leaders, but ultimately it is the wisdom, courage, and foresight to embrace the unconventional on the part of the institution that consummate the project.

Conclusions

"Surgery of the developing nervous system", an alternative term for paediatric neurosurgery, seems to convey concisely and precisely the fundamental difference between caring for children and adults with neurosurgical problems. The implication of this difference is enormous. Devoted practitioners are required if the best care for these children is the goal.

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Paediatric Neuroanaesthesia

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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 2014.

Introduction

Paediatric neurosurgery presents the anaesthetist with a unique set of interrelated challenges, including the imperatives of the intracranial pressure-volume relationship and the limitations on the anaesthetic technique implied by intraoperative neurologic monitoring; satisfying these in the context of the paediatric patient's vulnerable physiologic and developmental milieu requires forethought, assiduous planning, and a readiness to adapt and respond to events as they unfold in the operating theatre. The anaesthetist will always call to mind the tight coupling of metabolic supply and demand in the central nervous system: disproportionately high requirements for substrate, the absence of any significant metabolic reserve, and a lack of functional redundancy in the underlying organ system collectively underscore the cardinal importance of supporting adequate end-organ metabolism.

In this review we will briefly survey the unique operative and monitoring requirements of the neurosurgical operating theatre, before attempting to devise an idealised neuroanaesthetic, one that optimises the relationship between cerebral blood flow and metabolism, that facilitates intraoperative neurologic monitoring, and that, if not protective of, is at least not deleterious to neuronal survival and development.

Surgical Imperatives: Intracranial Relaxation

Although neurosurgery presents the anaesthetist with an abundance of physiologic challenges to distinguish it from other surgical environments, these can, on a purely technical level, be reduced to modulation of the intracranial pressure-volume relationship. In particular, profound limitations on the intracranial compliance described by Monroe and later Kellie imply unfavourable increases in the intracranial pressure, and corresponding decreases in the cerebral perfusion pressure, with even small changes in the intracranial volume.[1,2] While the interval before closure of the intracranial sutures offers some increased compliance in infants, the practical protection on an anatomic level is small, and easily overwhelmed; certainly, the anaesthetist should not discount the possibility of intracranial hypertension in infancy.[3] The classical view of the intracranial compartment advanced by Monroe and Kellie and expanded on by Burrows decomposes its volume into three distinct entities: brain, blood, and cerebrospinal fluid; the anaesthetist will be called on to modulate each of these to some degree.[4]

Intravenous administration of osmotic agents will, in the setting of an intact blood-brain barrier, establishes an osmotic gradient whereby free water flows out of the brain and into the capillary blood. The obvious practical limitations of the technique are implied by the osmotic agents themselves. As an osmotic diuretic, mannitol dehydrates not only the brain, but the entire compartment of total body water; these osmotic losses, in turn, must be repleted, typically with crystalloid, leading, ultimately, to re-equilibration of intracranial free water, thereby undermining the very osmotic effects that had been sought at the outset. Although mannitol remains a valuable part of the anaesthetist's armamentarium, its use is perhaps best confined to those situations where a definite end-point for anatomic relief of intracranial hypertension is envisioned. Hypertonic saline's greater clinical versatility and more durable effects on intracranial pressure result from osmotic activity at the blood-brain barrier and simultaneous lack of osmotic diuretic activity at the nephron.[5] Far from depleting the intravascular compartment, hypertonic saline disproportionately increases the intravascular volume by drawing free water out of the interstitium; with some kinetic limitations surrounding re-equilibration of sodium in the periphery, hypertonic saline can be seen as a resuscitation fluid.[6] The modest systemic hypernatraemia induced by rational administration of hypertonic saline is generally well-tolerated by patients; although its desiccant effects theoretically make it poorly suited to administration through peripheral veins, short term administration through peripheral intravenous lines has been described without complications.[7,8] When longer term administration of hypertonic saline is contemplated,central venous access, ideally via the left subclavian vein or a peripherally inserted central catheter, should be sought.

Immediate preoperative placement of a cerebrospinal fluid drain, typically in the form of an external ventricular drain, will frequently precede posterior fossa craniotomy for tumours; up to 80 percent of children presenting with posterior fossa tumours will

manifest preoperative obstructive hydrocephalus owing to the anatomic proximity of the tumour to the fourth ventricle, and extraventricular drain placement is common in this setting. The anaesthetist will commonly drain incremental quantities of cerebrospinal fluid through the drain following craniotomy and prior to dural opening.

Of the three components of the intracranial compartment, the anaesthetist is best equipped to modulate cerebral blood volume through changes in the cerebral blood flow. Tight coupling of the arterial carbon dioxide tension and cerebral blood flow offers the anaesthetist a ready mechanism with seductively rapid kinetics to reduce cerebral blood flow through increased minute ventilation; that this relationship is preserved under a wide variety of anaesthetising conditions only adds to its considerable allure. Yet the anaesthetist should perhaps regard hyperventilation with some skepticism, in that the decrease in the cerebral blood flow produced by falling arterial carbon dioxide tension does not, in and of itself, invite a corresponding decrease in cerebral metabolism. Hyperventilation instead raises the ominous threat of decreased cerebral oxygen delivery relative to metabolic demand, a threat borne out by clinical data indicating an increase in arterio-venous oxygen gradients across the cerebral circulation even at mildly hypocapnoeic levels (32 mmHg/4.3 kPa), and concomitant worsening tissue hypoxia measured by transcranial monitors.[9] Although we naturally focus on the cerebral blood flow relationship in considering changes in arterial carbon dioxide tension, we may also recall that hyperventilation shifts the oxygen-haemoglobin dissociation curve to the left according to the effect first described by Bohr, thereby further degrading delivery of oxygen at the cellular level.[10] In short, the powerful effects of hyperventilation on cerebral blood flow and volume are best reserved for intracranial hypertensive emergencies, and even erstwhile "mild" hypocapnia deserves considerable caution.

Far more palatable than hyperventilation are the various anaesthetic agents in the regulation of cerebral blood flow. The potent volatile anaesthetics will, in general, increase cerebral blood flow while decreasing cerebral metabolism, a so-called "uncoupling" that seemingly renders these agents poorly suited to neuroanaesthesia. Although this dose-dependant uncoupling effect pervades the entire class of volatile agents, it is least prominent in sevoflurane, while isoflurane and desflurane appear to produce relatively higher cerebral blood flow in paediatric subjects; this quality, together with the various other advantages of sevoflurane in paediatric anaesthesia, make sevoflurane the agent of choice when a volatile anaesthetic is applied in the paediatric neurosurgical setting.[11] Responsiveness to changes in carbon dioxide tension are preserved under sevoflurane anaesthesia, and induction of mild hypocapnoea (for example to a carbon dioxide tension of 35 mmHg/4.7 kPa) in the face of unfavourable conditions on the surgical field is a plausible method for normalising cerebral blood flow during volatile anaesthesia. Nitrous oxide increases cerebral blood flow, albeit in a less dose-dependent fashion than the volatile anaesthetics, while leaving global cerebral metabolism effectively unchanged; its emetogenicity, particularly with exposures longer than two hours; its tendency to irreversibly oxidise cobalt the prostheses of cobalamin, thereby disrupting methionine and folate metabolism; and its lasting deleterious effects on the environment notwithstanding, nitrous oxide may be valuable in certain clinical situations such as electrocortography where neurologic monitoring considerations predominate.[12, 13, 14, 15]

In contrast with the inhalation anaesthetics, intravenous anaesthetics, particularly those acting on the gamma aminobutyric acid (GABA) receptor system, tend to decrease cerebral blood flow and cerebral metabolism in a tightly coupled fashion.[16] Propofol's effects on cerebral blood flow and metabolism epitomise this tendency; taken together with its predictable haemodynamic effects and, compared with other intravenous agents such as sodium thiopental and midazolam, its relatively short context-sensitive half-time, propofol's apparently favourable modulation of the cerebral blood flow/metabolism relationship would appear to be that of the idealised neuroanaesthetic.

As an NMDA antagonist, ketamine stands apart from the GABA-ergic intravenous agents, and its effects on intracranial haemodynamics are less certain. Although it has commonly been supposed to increase cerebral blood flow and metabolism, some clinical data suggest that ketamine may be safely administered to neurosurgical patients without increasing the intracranial pressure.[17] Certainly, ketamine at subanaesthetic doses may have a useful role in the analgesic management of certain paediatric spine surgery procedures, and in facilitating monitoring of motor evoked potentials in these same procedures; with other compelling alternatives available, the existing evidence is insufficient to recommend its routine use for intracranial neurosurgery.

Dexmedetomidine's relative novelty implies less study and scrutiny of its neuroanaesthetic properties. Nevertheless it appears, like propofol, to decrease cerebral blood flow and metabolism in a coupled fashion, suggesting alpha-2 agonism may have a significant value in neuroanaesthetic techniques.[18] Its haemodynamic effects may be less predictable, or in any event less familiar, than those of other routinely used anaesthetics. Although a relatively low baseline sympathetic tone in small children may blunt some of the haemodynamic changes associated with dexmedetomidine administration, bolus dosing can nevertheless produce transient hypertension and bradycardia, while infusion will typically result in relative, albeit mild, bradycardia. The mean arterial pressure may fall, but is in general better-preserved than with equipotent infusion of propofol.[19] Although these haemodynamic changes are generally manageable, their significance to the cerebral perfusion pressure must be integrated into the anaesthetic planning. On a practical level, perhaps the most significant obstacle to the routine use of higher doses of dexmedetomidine lies in its relatively prolonged elimination kinetics, which may impede immediate postoperative emergence and neurologic examinations, particularly in children under two years of age; lower dose infusions, in the order of 0.3 mcg/kg/hr, can typically be used as part of a multimodal anaesthetic technique without detrimental effects on emergence.[20]

The intravenous opiates are comparable to other intravenous agents in that they too decrease cerebral blood flow and metabolism at clinically relevant doses. While the short context-sensitive half-time of remifentanil makes this an appealing agent for intravenous infusion, induction of hyperalgesia remains of particular concern, and fentanyl or sufentanil may be more appropriate choices, particularly for surgeries of greater durations or those associated with a relatively higher burden of postoperative analgesia.[21] Both fentanyl and sufentanil can be successfully infused in this context as long as care is taken, particularly with fentanyl to use judicious doses and to discontinue the infusion promptly at the beginning of dural closure.

Neurologic Monitoring

A multitude of neurologic monitoring techniques allow for intraoperative assessment of the integrity of the central nervous system. Four essential techniques comprise the bulk of routine intraoperative neurophysiologic monitoring: Electromyography (EMG) allows for monitoring of cranial nerves and spinal nerve roots by measuring depolarisations in the corresponding muscle groups; Somatosensory evoked potentials (SSEPs) and their cousin the brainstem auditory evoked response (BAER) tests the integrity of the afferent sensory nervous system including, in the case of SSEPs, the dorsal columns of the spinal cord, by applying a peripheral stimulus and measuring the cortical (or subcortical) response; Motor evoked potentials (MEPs) interrogate the pyramidal nervous system by applying a cortical stimulus and measuring corticospinal (inclusive of the anterior spinal cord) and corticobulbar motor responses in the periphery; and Electroencephalography (EEG) offers an indicator of cerebral cortical function contingent on anaesthetic depth and cerebral perfusion.

Although each modality exhibits differential responsiveness to different anaesthetic techniques, SSEPs and MEPs are most sensitive to the selection of the anaesthetic agent. Volatile anaesthetics decrease the amplitude and increase the latency of SSEPs in a dose-dependant fashion; propofol and dexmedetomidine, by contrast, have relatively little impact on SSEPs.[22] Both the volatile and intravenous agents depress MEPs, but this reduction in amplitude is more readily overcome through the application of multiple pulses in the case of propofol and dexmedetomidine than with volatile agents. While MEPs may not be obtainable at volatile anaesthetic concentrations exceeding 0.75 MAC, up to 0.25 MAC of the volatile anaesthetic will allow for MEP monitoring in most conditions.[23] MEPs are more difficult to elicit in small children, perhaps due to the immaturity of the pyramidal tracts, but have been successfully demonstrated in infants as young as five months of age under propofol and opiate anaesthesia.[24,25] The intravenous opiates have little effect on evoked potential monitoring.[26] Regardless of the anaesthetic agent selection, the voltage threshold for eliciting a response to MEP stimulus will gradually increase during the course of an anaesthetic, reflecting a "fade" phenomenon unrelated to the anaesthetic technique.[27]

The significance of intraoperative neurologic monitoring on selection of the anaesthetic technique can seldom be formulated in terms of absolutes. Oft-heard blanket statements to the effect that "we are doing motors so we need TIVA" should be regarded with skepticism. Absent baseline myelopathy, a balanced anaesthetic technique integrating up to 0.25 MAC of a volatile agent is almost always compatible with robust monitoring of motor evoked potentials in children greater than two years of age; smaller children may require a totally intravenous anaesthetic during MEP monitoring. Ultimately, it is incumbent on the anaesthetist to understand the neurologic monitoring imperatives of a given procedure, and to effectively partner with the neurophysiologist to ensure these imperatives are fulfilled. The mere act of monitoring does not guarantee the integrity of the central nervous system throughout the course of surgery. A sound understanding of how to contextualise, troubleshoot and respond to changes in evoked potentials will ultimately be more important than the act of eliciting these potentials.

The Idealised Neuroanaesthetic: Optimised End-Organ Metabolism and Neuroprotection

With the foregoing discussions in mind, can we devise an ideal neuroanaesthetic, one allowing for favourable modulation of the cerebral blood flow/metabolism relationship, while facilitating adequate neurologic monitoring? At first glance, propofol would appear to stand out as the neuroanaesthetic agent of choice. Of course some caveats are necessary. The anaesthetist will recall that, particularly with longer, higher-dose infusions, propofol acts as a metabolic poison, inhibiting mitochondrial fatty acid metabolism and precipitating metabolic acidosis, bradyarrhythmia and cardiovascular collapse, collectively termed the propofol infusion syndrome; propofol should be used with caution in very long anaesthetics, and in patients with suspected mitochondrial disease.[28] Also, a growing body of data suggests that propofol, along with ketamine, the inhalation anaesthetics, and indeed nearly every anaesthetic agent, has pro-apoptotic effects in animal models of neuronal development, with correspondingly negative impacts on cognitive development.[29,30] These findings have obviously inspired considerable introspection in the paediatric anaesthesia community, even as their clinical significance in human subjects remains unclear. Population cohort studies offer some hope of better understanding the practical significance of anaesthetic exposure on the developing human central nervous system, although the effects of anaesthesia remain difficult to disentangle from those of surgery and any underlying comorbid disease; while some studies appear to indicate a deleterious effect of early childhood anaesthetic exposure on later measures of cognitive function, other cohorts suggest no durable cognitive impact of a single anaesthetic exposure in infancy, while environmental factors, including parental education, appear to have a comparatively far more powerful effect on long-term outcomes.[31,32] Nevertheless, one would obviously hope to avoid even the suggestion of neuroapoptosis in anaesthetising a developing child, and dexmedetomidine may offer some hope in this regard; animal studies suggest that not only does the alpha-2 agonist not have any intrinsic neuroapoptotic effect, but it protects against the neuroapoptotic effects of the potent volatile anaesthetics.[33] That

dexmedetomidine similarly protects against emergence agitation and delirium behaviuors following volatile anaesthesia naturally raises the question of a common underlying neuroprotective mechanism.[34]

Neuroprotection is a fraught topic, one littered with an abundance of experimental ideas that, while compelling in animal models, failed to demonstrate significant clinical benefit. Even the most robust examples may offer an advantage in one setting while failing to translate to another; the favourable experience with moderate hypothermia in resuscitation of asphyxiated newborns and survivours of out-of-hospital cardiac arrest, without evidence of a corresponding benefit in neurosurgical patients, underscores these limitations.[35,36] Accordingly it is with some caution that we consider the apparently unfavourable effects of anaesthetics on neuronal survival, even as a parallel body of data suggests that volatile anaesthetics may, paradoxically, be neuroprotective in certain clinical settings. Indeed, among cardiac surgical patients randomised to propofol or sevoflurane anaesthesia, those patients who received sevoflurane fared better than the group randomised to propofol, not only in terms of neurocognitive outcomes but integrating global postoperative complications; this was particularly true among patients who sustained hypoxic events.[37]

Here we must return to the relationship between cerebral blood flow and metabolic rate and ask whether the tight coupling of these parameters under propofol anaesthesia is as valuable as we initially supposed. While most of the literature surrounding the neuroprotective effects of volatile anaesthetic agents revolves around their effects at the molecular level including, paradoxically, increased phosphorylation and thereby activation of antiapoptotic mitogen-activated protein kinases, perhaps the so-called "luxury perfusion" of volatile anaesthesia is not a matter of luxury but necessity when hypoxia or ischaemia is involved.[38] Clinical data appear to bear out this supposition, showing that, across a range of clinically relevant carbon dioxide tensions, jugular venous oxygen saturations fall to significantly lower levels in subjects anaesthetised with propofol than in those receiving inhalation anaesthesia; interestingly, some hyperventilated propofol patients showed a paradoxical increase in jugular venous oxygen saturation, indicating vulnerability to the Bohr effect; this same vulnerability was not evident in the inhalation anaesthesia group.[39]

How then are we to proceed, when called upon to anaesthetise a child for a neurosurgical procedure? At this juncture it would appear that we must humbly concede that there is no ideal neuroanaesthetic, no single agent that fulfils all of our anaesthetic, surgical and monitoring imperatives while protecting or, failing that, demonstrably not harming the developing central nervous system. Absent this, we must perhaps make tactical use of the various agents at our disposal, using each to fulfil a discrete clinical need. Opiate analgesics should be administered for noxious stimuli. Propofol can be used in moderation to facilitate adequate intraoperative neurologic monitoring while modulating the cerebral blood flow and metabolism to allow for satisfactory intracranial relaxation on the operative field. At the same time a low dose (0.25 MAC) of sevoflurane may offer some plausible neuroprotective effect against hypoxic and ischaemic insults while still allowing for adequate monitoring of evoked potentials in most circumstances. Finally, a growing body of data suggests that dexmedetomidine mitigates the neuroapototic tendencies of propofol and sevoflurane and, given its other valuable anaesthetic properties, deserves a routine place in paediatric neuroanaesthetic techniques.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Paediatric Neuroanaesthesia" by Dr Nicholas RIEGELS 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 2014. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please choose the best answer.

1. Hyperventilation:

a. reduces cerebral blood flow
b. shifts the oxyhaemoglobin dissociation curve to the left
c. decreases cerebral oxygen delivery
d. increases cerebral tissue hypoxia
e. all of the above

2. Potent volatile anaesthetics such as sevoflurane are associated with:

a. dose-dependent increases in cerebral blood flow
b. dose-dependent decreases in cerebral perfusion pressure
c. uncoupling of cerebral blood flow and cerebral metabolic rate
d. preserved responsiveness of cerebral blood flow to changes in carbon dioxide tension
e. all of the above

3. Coupling of the cerebral blood flow: cerebral metabolic rate relationship is observed with:

a. propofol
b. fentanyl
c. dexmedetomidine
d. sevoflurane
e. a, b and c only

4. With reference to somatosensory evoked potentials, sevofluane causes:

a. decreased amplitude and increased latency
b. increased amplitude and increased latency
c. decreased amplitude and decreased latency
d. increased amplitude and decreased latency

5. Motor evoked potentials:

a. cannot be obtained in infants
b. are not influenced by anaesthetic technique
c. cannot be obtained when volatile anaesthetics are used
d. can be obtained in small children, including infants, with thoughtful collaboration between the anaesthetist and neurophysiologist.

6. The propofol infusion syndrome:

a. entails impaired mitochondrial fatty acid metabolism
b. is associated with infusions of propofol at greater than 4mg/kg/hour, for longer than 24 hours
c. results in bradyarrythmia, lactic acidosis and cardiovascular collapse
d. has been reported after intraoperative propofol administration
e. all of the above

7. Exposure to potent volatile anaesthetics appears to:

a. provoke neuroapoptosis in animal models of neurodevelopment
b. to be neuroprotective in animal models of cardiac arrest
c. result in phosphorylation of antiapoptotic mitogen-activated protein kinases
d. to have an uncertain effect of human neurodevelopment
e. all of the above

8. The neuroapototic effects of potent volatile anaesthetics in animal models of development are mitigated by:

a. ketamine
b. propofol
c. nitrous oxide
d. dexmedetomidine

9. The ideal neuroanaesthetic is:

a. propofol-based totally intravenous anaesthesia
b. sevoflurane-based volatile anaesthesia
c. ketamine
d. a combination of agents selected to suit the anaesthetic, surgical and monitoring imperatives of a particular operation

10. Alexander Monro (secundus), of the Monro-Kellie doctrine:

a. was appointed, at age 21, to shared a conjunct professorship of anatomy in the College of Edinburgh with his father, also named Alexander Monro (primus)
b. later shared a conjunct professorship of anatomy in the College of Edinburgh with his son, also named Alexander Monro (tertius)
c. taught anatomy to George Kellie whilst the later earned his medical degree at Edinburgh
d. described the eponymous foramina allowing for communication of cerebrospinal fluid between the lateral ventricles
e. all of the above

ANSWER SHEET FOR APRIL 2014

Please return the completed answer sheet to the Federation Secretariat on or before 30 April 2014 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Paediatric Neuroanaesthesia

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Name (block letters):_____ HKMA No.: _____ CDSHK No.: _____

HKID No.: __ __ - __ __ __ __ X X (X) HKDU No.: _____ HKAM No.: _____

Contact Tel No.:_____ MCHK No.: _____ (for reference only)

Answers to March 2014 Issue

Breast Ultrasound Imaging

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

CERTIFICATE COURSE FOR DENTAL NURSE

• CME/CNE Course • Course No. C240

Jointly organised by

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The Hong Kong Association of Oral and Maxillofacial Surgeons Limited

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2 May	Introduction to Oral and Maxillofacial Surgery	Dr. Mike Yiu-yan LEUNG Specialist oral and maxillofacial surgery Clinical Assistant Professor, Oral and Maxillofacial Surgery Faculty of Dentistry, The University of Hong Kong
9 May	An overview to minor oral surgical procedure	Dr. Julianna Cho-hwei LIEW Specialist oral and maxillofacial surgery Dental Officer, Oral Maxillofacial and Dental Unit Queen Mary Hospital
16 May	An overview to dental implant surgery	Dr. Raymond Lop-keung CHOW Specialist oral and maxillofacial surgery Private Practice
23 May	Peri-operative nursing in oral surgical procedures	Ms. Lai-har LEUNG Perioperative Nurse, (Registered Nurse – instrument nurse) Operation Theatre, Queen Mary Hospital Ms. Chui-lin NG Specialty Trained Nurse Operation Theatre, Queen Mary Hospital
30 May	Sedation in dental clinic	Dr. Gary Lee-ka TAM Specialist Anaesthesiologist Private Practice
6 June	Medical emergency in dental clinic	Dr. Jeni Lai-in HO Advanced Trainee in oral and maxillofacial surgery United Christian Hospital

Date : 2 May 2014 – 6 June 2014 (Every Friday)
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Strokes in Children

Dr Dawson FONG

MBBS(HK), FRCS(Edin), FCSHK, FHKAM(Surgery)

Chief of Service and Consultant Neurosurgeon
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Dr Dawson FONG

Stroke, often referred to as 'cerebrovascular accident' or even 'brain attack'[1], is a rapid loss of cerebral function due to either the occlusion of arteries to the brain leading to ischaemia or a breach of integrity of the vascular wall resulting in haemorrhage. Undoubtedly, strokes are more common in adults and older individuals and yet children are not immune. Strokes can strike as congenital vascular anomalies that affect even neonates, as haemorrhages in the central nervous system or even as ischaemic strokes in the very young.

Congenital Conditions and Strokes

Congenital anomalies of the cerebral vasculature can be classified into four categories – arteriovenous malformation (AVM), cavernous haemangioma (CH), venous malformation and capillary telangiectasia. The former two are more common and their nature better known whereas venous malformation and capillary telangiectasia are of little clinical significance.

Although these congenital lesions are formed early in the embryo, they would generally remain asymptomatic early in life except for the very large ones. As the child grows, lesions with significant arteriovenous shunting would be under increasing challenges from the higher blood pressure of adulthood until the fragile abnormal vascular wall ruptures and spontaneous haemorrhage ensues. The incidence of bleeding from congenital arteriovenous malformations therefore rises gradually from the early teens and peaks at about the second and third decades of life. With the advent of neuroimaging, such lesions are very often identified incidentally. (Figure 1)

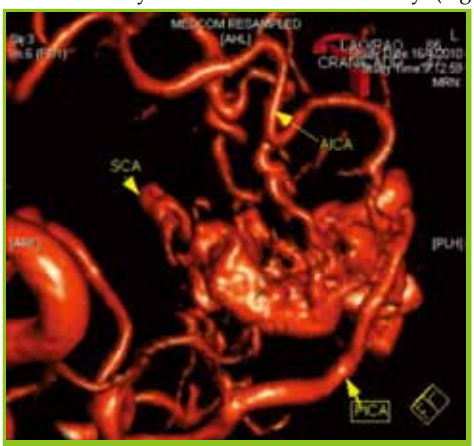

Figure 1. 3D CT angiogram of a girl of 8 following an incidental finding of and AVM on brain CT after a minor head injury.

Strokes in Newborns

Preterm babies run a high risk of developing ***intraventricular haemorrhages*** (IVH). It was estimated that 15% to 35% of infants of less than 34 weeks' gestational age and 1500 g birth weight develop IVH within the first few postnatal days.[2] The incidence of IVH is inversely related to the gestational age and likewise the parenchymal damage to birth weight. The site of haemorrhage is believed to be from the germinal matrix that is not mature until 32 gestational weeks. Blood in the ventricles might not only lead to hydrocephalus, sequelae also include diffuse hypoperfusion and secondary alterations in cortical genesis in the developing brain.[3]

Diagnosis is generally easy with ultrasound (Figure 2) and in case the ventricles appear enlarged in subsequent progress ultrasounds, we may have to resort to inserting a Rickham's reservoir for daily release of cerebrospinal fluid. In a good proportion of them, the clot would resolve in due course without hydrocephalus. Otherwise shunting would be indicated in those who, by then, have grown much sturdier to withstand the impact of the procedure.

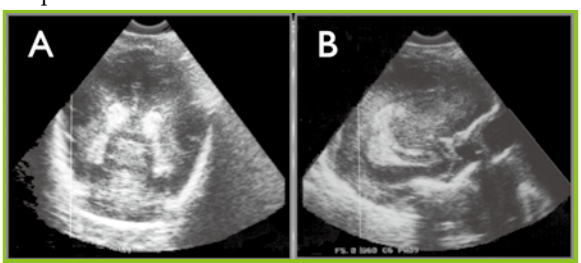

Figure 2. Ultrasound of a neonate with intraventricular haemorrhage. A Axial and B Sagittal view, showing blood in the ventricles as hyperechoic lesion.

Aneurysm of the Vein of Galen is a challenge to paediatric neurosurgeons. It presents as a severe form of arteriovenous shunting (AV shunting) to the extent that the child is very often born with cardiac failure. The steal phenomenon from shunting of the cerebral vasculature may inflict extensive ischaemic damage and atrophy of the cerebrum even at birth (Figure 3). Possible interventions after alleviating the cardiac failure include endovascular embolisation or even surgical excision in the selected few. Generally, prognosis remains poor.

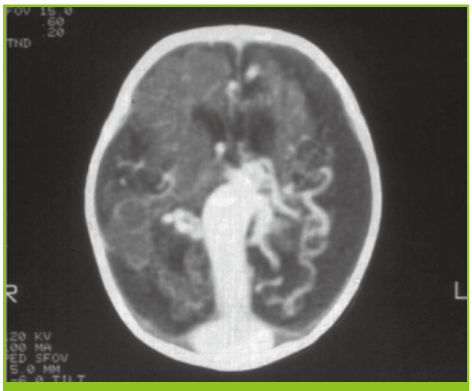

Figure 3. CT scan of a neonates with Aneurysm of the Vein of Galen showing the hugely dilated venous channels and markedly atrophy cerebrum due to steal phenomenon.

Haemorrhagic Strokes in Children

AVM (Figure 4) should be suspected in practically every case of spontaneous haemorrhage in young patients. As a congenital lesion, AVM grows as a child grows until it strikes in a hitherto healthy one. Presentation is mostly as haemorrhages, up to 60 to 70% or as epilepsy in 20 to 30% and the rest as neurological deficits from the steal phenomenon to functional areas close to the lesion. The annual bleeding rate of AVMs is estimated to be around 2 to 4% and thus for young children with such lesions, the rate of having an eventual haemorrhagic stroke is extremely high. It is for this reason that if at all possible, paediatric AVMs should be aggressively treated.

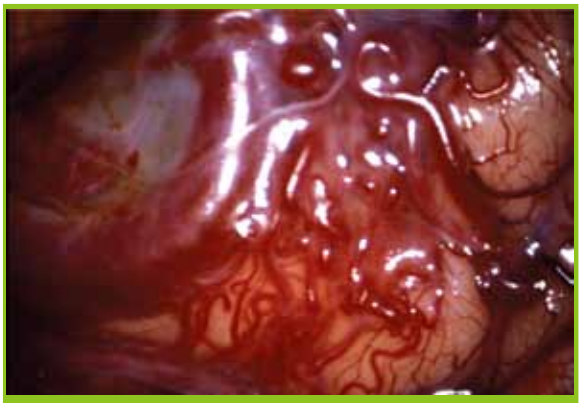

Figure 4. Cortical AVM with dilated, tortuous nidal arteries and dilated arterialized veins.

Sizeable clots have to be removed to relieve the high intracranial pressure. Based on thorough investigations with CT or digital angiography, a decision on further management can be made.

Total surgical excision is the ultimate cure precluding further strokes but in case of Spetzler-Martin high-grade lesions[4] - large or deep malformations in which eloquent areas are involved - other modalities or a combination of which have to be considered. In such incidents, treatment responses may take years to be seen.

Endovascular embolisation serves to protect flow aneurysms within an AVM to decrease the risk of haemorrhage or to shrink the lesion to facilitate other treatment options. Evidence seems to suggest that embolisation itself could lead to a cure by completely obliterating AVMs of a diameter of less than 3 cm in about 18%.[5] Long term outcomes however are still awaited.

Stereotactic radiosurgery delivered with a linear accelerator or Gamma Knife offers obliteration of deep-seated lesions, especially the smaller ones (<3 cm). However, radiation obliterates the AVM nidus by causing vascular endothelial proliferation and it takes time. Until complete obliteration, the bleeding rate remains unchanged.[6]

Thus for complex high grade AVMs, cure may resort to a combination of embolisation, radiosurgery and surgical excision.

Cavernous haemangioma, often referred to as a cavernoma, is another form of vascular malformations. Unlike AVMs, it is usually compact, consisting of sinusoidal vascular channels without direct AV shunting. It is therefore not demonstrated on angiography. Yet because of its nature of repeated minute bleeding within the lesion, diagnostic features of haemosiderin and calcification are often depicted on MR and CT.

Cavernomas are often multiple, could be found both in the cerebrum and the spinal cord. Presentation varies. Seizure is common followed by haemorrhage and neurological deficits. Incidental findings are also very common. Deep lesions in the midbrain and brainstem have a worse prognosis in that they tend to bleed more frequently. Because of the important structures nearby, patients are often rendered severely disabled or even death. Radiosurgery is not effective leaving only the surgical option. Excision is often rewarding if care is taken to avoid eloquent areas close by with neurophysiological monitoring (Figure 5). For deep lesions within the brainstem and midbrain, once they expand enough from repeated haemorrhages to open up to the surface, surgery should be offered and additional deficits are often insignificant.

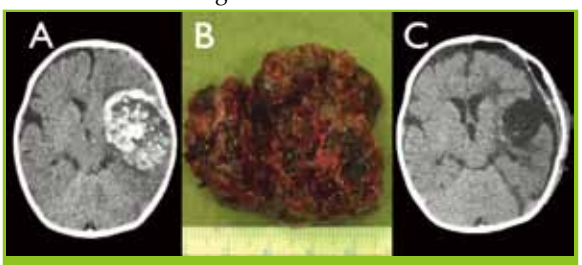

Figure 5. Cavernoma presented as a vascular tumour in a boy of 7 months old. A. Preoperative CT B. The excised lesion C. Postoperative CT.

Aneurysm is uncommon in the children population. It might be associated with systemic diseases such as tuberous sclerosis, the Ehlers-Danlos and Marfan's syndromes. Moyamoya disease, physical trauma and infection are other more common causes. Presentation is similar to that of adults in the form of subarachnoid haemorrahge. Management follows the same principle – thorough angiographic study followed by surgical clipping or endovascular intervention depending on the location of the aneurysm and expertise at hand.

Ischaemic Strokes in Children

It is uncommon for a child to develop ischaemic stroke. Such attacks may even be brief. For the very young, who may not complain, such episodes might well be missed or ignored unless the parents or carers are vigilant enough. Each of these cases deserves thorough investigations.

Familial conditions like protein C or protein S deficiency need to be considered. Once diagnosed, anticoagulation therapy is indicated, under the expert care of paediatric neurologists. More commonly, however, vasculopathy is the culprit and among which the Moyamoya disease is a typical example of how neurosurgery could help and alter the natural course of the disease.[7]

Moyamoya disease is the idiopathic chronic stenosis of the supraclinoid carotid arteries, while the *Moyamoya* syndrome refers to a similar vasculopathy due to an identifiable cause such as radiation therapy.[8] As stenosis develops gradually, patients present with repeated episodes of ischaemic stroke. Typically such neurological deficits are short-lived and these children soon recover evading early diagnosis. As they grow, tiny neovasculature is formed as collaterals. Yet these tiny and frail collaterals – thus the Japanese name from its angiographic appearance - would bleed as the patients grow into adulthood (Figure 6).[9] This bimodal course of ischaemia followed by haemorrhage in later years is typical of the disease.[10]

Figure 6. Moyamoya vessels seen on the lateral view of a left common carotid angiogram (blue arrow)

Patients with Moyamoya disease have disturbed CO2 reactivity.[11] Because of their acute response to low CO2 after hyperventilation, neurological deficits very often set in after a bulk of crying or temper tantrum. This phenomenon renders anaesthetists to assure that these patients be maintained normal or even slightly hypercapnic during general anaesthesia.

Diagnosis is suspected on MR and confirmed with cerebral angiogram. Perfusion studies with CT or MR are essential to assess the severity and extent of the disease. In bilateral disease, perfusion studies also help surgeons to decide which is the worse side that should be tackled first.

The brain of patients with Moyamoya disease is in a chronic ischaemic state. To counteract the pathology, augmentation of cerebral blood supply is the key. There are two ways to achieve this.

Direct extracranial-intracranial (EC-IC) anastomosis provides an immediate increase of blood supply. It is commonly done by anastomosing the superficial temporal artery (STA) to a distal branch of the middle cerebral artery (MCA). However it has a few limitations. The size of STA in children is around 0.5 mm and this makes the procedure technically difficult. If the extent of ischaemia goes beyond the MCA territory to involve the anterior or posterior cerebral arteries, those areas would not be benefited and would go on to develop infarcts. For advanced disease where the STA is already contributing to neovasulature through the vault, STA-MCA anastomosis is even contraindicated. Direct anastomosis is therefore more suited for adult patients with early and limited disease of the MCA territory.

Indirect revascularisation is achieved by laying vascular extracranial tissues over the cortical surface where the pia mater is widely opened. Many tissues have been tried and the STA, galea or temporalis muscle are the better choices. What tissue to use could be tailored to individual needs according to the pattern of ischaemia. Usually neovascularisation could be demonstrated as early as a couple of months after surgery. (Figure 7, 8) Perfusion studies confirm improvements of cerebral blood flow and better vascular reserve. Clinically, ischaemic episodes are no more and even for the haemorrhagic group, long term studies indicate that the prevalence of bleeding after surgery diminishes.[12]

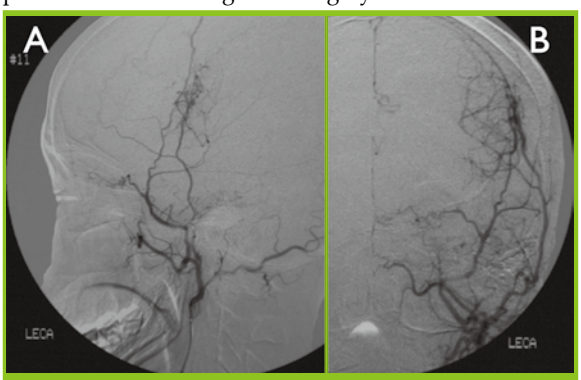

Figure 7. Left External Carotid Angiography after indirect revascularization using STA. A. Lateral and B. AP views showing neovascularization from the transposed artery.

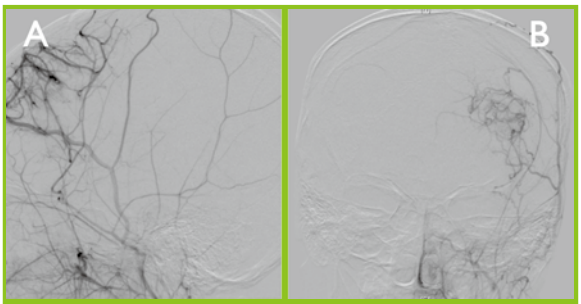

Figure 8. Left External Carotid Angiography after indirect revascularization using galea. A. Lateral B. AP views showing neovascularisation supplying the anterior lobe affected by Moyamoya Disease.

Summary

Both ischaemic and haemorrhagic strokes happen in children. Vigilance of carers and clinicians is essential in getting to an early diagnosis and timely treatment. As we understand more about the peculiar spectrum of diseases that causes strokes in children, appropriate treatment proves fruitful in making a difference in these young ones as well as their loving families.

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Paediatric Neuro-oncology: Exploring the New Frontier

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Introduction

Central nervous system (CNS) tumours are the commonest solid malignant tumours in children and one of the leading causes of cancer death[1]. The presentation is often non-specific in children which sometimes leads to late diagnosis and long-term morbidity. Despite the advances in modern medicine, the treatment combination with neurosurgery, radiotherapy and chemotherapy remains similar in the last decade. The overall survival and morbidity of these patients have improved slowly compared to other childhood cancers especially acute leukaemia. Moreover, these young patients often suffer from various degrees of neurological impairment, growth failure and endocrine dysfunction, either resulted from the primary disease or treatment toxicity. New research and clinical trials are emerging rapidly since the completion of The Human Genome Project. Translation of the latest understanding and laboratory research findings is important to enhance the clinical outcome. Multi-disciplinary care is essential for both their acute treatment and subsequent long-term rehabilitation.

Background

In the United States, the incidence of primary paediatric CNS tumours was 3.33 per 100,000 children yet it contributed to 45.8% of total primary CNS tumours in all ages[2]. In Hong Kong, the incidence of CNS tumours ranges from 21 to 41 cases per year, approximately 3.3 cases per 100,000 children in year 2012[3]. The most common childhood brain tumours are ependymoma, primitive neuroectodermal tumours (PNET) including medulloblastoma and different grades of astrocytoma[2]. For Asian populations, there is a higher proportion of intracranial germ cell tumours (GCTs) among all childhood brain tumours, 7.9% in China[4], 7.8% in Japan[5], 11.2% in Korea[6], whereas only 2-3% in North America[7]. However, a recent review of 4 registries showed similar incidence of intracranial GCTs per 100,000 in Japan (males = 0.143, females = 0.046) and United States (males = 0.118, females 0.030)[8].

45-60% of childhood CNS tumours are located at the infratentorial region, including ependymomas, PNETs / medulloblastomas and astrocytomas[9]. Their presentations are often subtle and non-specific leading to delayed diagnosis. Those presented with headache, nausea, vomiting and visual changes led to earlier diagnosis[10]. Other presenting symptoms depend on the site of the lesions, e.g. enuresis due to cranial diabetes insipidus caused by suprasellar GCTs[11], ataxia and diplopia in cerebellar medulloblastoma[12], anorexia and emaciation in the diencephalic syndrome related to hypothalamic tumours[13]. A rare but interesting sign, the Parinaud's syndrome (failure of upper gaze, pupils which react better to accommodation than light, lid retraction, and convergence or retraction nystagmus) points to tumour in the midbrain or pineal region[14].

Conventional Management

Most of the childhood brain tumours are treated with surgery aiming at complete resection with the preservation of residual normal brain function. The extent of surgical excision is important for the prognosis as shown in PNET / medulloblastoma[15] and ependymoma[16]. Some patients require further chemotherapy and radiotherapy post-operatively which depends on the tumour pathology. Radiotherapy is commonly used in older children for local control, spinal prophylaxis and also in palliative care. Moreover, prolonged and intensified chemotherapy is particularly useful for treating young children (less than 3 years old) with brain tumours to spare or defer the use of radiotherapy[17].

Late Effects in Children with Brain Tumours

Apart from presenting neurological deficits caused by CNS tumours, these patients may suffer from other morbidities related from surgical intervention, chemotherapy and radiotherapy. Childhood brain tumour survivors are at a significantly increased risk for several adverse endocrine (hypothyroidism, growth hormone deficiency, need for medications to induce puberty and osteoporosis) and cardiovascular (arrhythmia, stroke, blood clots, and angina-like symptoms) late effects, particularly if they were treated with radiation and chemotherapy[18]. A study for adult survivors of childhood brain tumours showed long-term sequelae of hearing impairment, seizure disorders, coordination, and motor problems[19]. For children received cranial radiation for posterior fossa tumours, they have lower short-form IQ and slow information progressing speed[20]. They are also more likely to report symptoms of depression, somatisation and anxiety[21]. Therefore, long-term follow up for late effects and rehabilitation are essential after the acute management of brain tumours.

Exploring the New Frontier

Hence, there are two major goals for future development in treating childhood CNS tumours. Firstly, improve overall survival for those aggressive lesions despite giving combination of intensified treatment. Secondly, minimise the treatment-related toxicity for long-term survivors with tumours having excellent prognosis.

Intensified induction chemotherapy followed with myeloablative chemotherapy with autologous haemopoietic progenitor cell rescue (AuHCR) is occasionally used for aggressive or recurrent CNS tumours. A minority of children with newly diagnosed CNS atypical teratoid / rhabdoid tumours (ATRT) treated in Head Start III trial may become long-term survivors without irradiation[22]. However, intensified chemotherapy and AuHCR have doubtful benefits for supratentorial ependymomas with residual disease after surgery and those infratentorial ependymomas without irradiation[23].

The Human Genome Project was completed in April 2003, which started a new page for molecular understanding and linkage of genotype for of many diseases. It is useful to provide information for diagnostic, prognostic and therapeutic aspects in treating cancers. For example, childhood medulloblastoma is currently stratified using clinical factors (age, presence of metastasis, and extent of resection) and histological subgrouping (classic, desmoplastic, and large cell / anaplastic histology). However, diagnosis and classification using histology alone may be inadequate nowadays. The latest international consensus divided medulloblastomas into 4 molecular subgroups, Wnt, Sonic Hedgehog (SHH), Group 3 and Group 4. They showed distinct demographic, transcriptional, genetic and clinical differences[24].

For infants and very young children, i.e. less than 36 months old, with CNS tumours, they are at risk of serious toxicity including neurotoxicity, endocrinopathy, growth failure and leukoencephalopathy[25]. To reduce long-term radiation related toxicities, recent advances in radiotherapy may be beneficial in children and other subgroups of patients. Image-guided radiation therapy (IGRT) is based on computer tomography (CT) / magnetic resonance (MR) imaging which delineate the target for computer-aided planning and simulation of treatment before the actual treatment takes place[26]. Protons beam radiation therapy is superior to convention photons methods in terms of the missing exit dose and the steep distal dose gradient characteristics, which provides better conformity of the highest doses and potentially lower doses to normal tissues[27]. However, more long-term follow-up studies are needed to demonstrate clinical advantages for children.

Despite the advances in chemotherapy and radiation therapy, neurosurgery is still the mainstay of treatment for childhood brain tumours. Advances in neurosurgical techniques such as image-guided surgery and intraoperative neurophysiological monitoring may achieve a more complete and safer resection for tumours especially when operating on the little brains in children.

Conclusion

CNS tumour is the most common solid tumour in children. It carries significant neurological and psychosocial impacts in affected children, long-term survivors and their families. Its variable presentation, histopathology and treatment modalities require multi-disciplinary management for both acute treatment and long-term rehabilitation. Future advances of surgical technique, chemotherapeutic regimen and radiotherapy would be able to salvage patients with aggressive and recurrent lesions while minimise treatment-related toxicities. A structured rehabilitation programme is also essential for long-term survivors. The establishment of the Hong Kong Children Hospital in 2018 could provide an excellent platform for all concerned health care providers to deliver patient-centred management, research and education.

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Serving Children with Cerebral Palsy- from Surgery and beyond Collaboration between Sectors: Child Assessment Service and Department of Neurosurgery, Tuen Mun Hospital

Dr LY WONG

Child Assessment Service
Department of Health
HKSAR

Dr LY WONG

Introduction

Cerebral Palsy (CP) is the most common physical disability in childhood.[1] The point prevalence for children aged six to twelve years with cerebral palsy was estimated to be 1.3 per 1000 in Hong Kong.[2] Spastic CP remains the most common type.

Service for Children with Cerebral Play: The Role of the Child Assessment Service

Many children with cerebral palsy have ongoing medical, educational and therapy needs. In addition to physical challenges, they may also suffer from epilepsy, behavioural problems, learning difficulties, language and communication problems, visual and hearing impairment.[3,4] They often require a wide range of services and would benefit from continued and coordinated multidisciplinary health care services.

Physical Neurorehabilitation (PNR) is one of the subspecialties within CAS. Our team members are Developmental Paediatricians, Physiotherapists, Occupational Therapists, Clinical Psychologist, Speech Therapists and Social Workers. The role of the PNR team is for diagnosis, rehabilitation and educational planning for children with physical impairments such as cerebral palsy. Our assessment is on functional approach and is based on the International Classification of Function, Disability and Health Model (ICF).[5,6] The team learn from and support each other in the vision to further develop assessment and rehabilitation protocols.

Selective Dorsal Rhizotomy

Children with Spastic CP require treatment for their spasticity. There are different treatments targeted on spasticity reduction aiming at various sites in the reflex arc. Selective Dorsal Rhizotomy (SDR) reduces the excitatory influence from the dorsal root and is shown to be effective in reducing spasticity in children with spastic diplegia. First pilot local data were available in 1999.[7] The surgery was also shown to have a small positive effect on gross motor function.[8] A local study also reported improvement in short term functional outcome after SDR.[9] Data for long term functional improvement post SDR are lacking though there is evidence in long term tone reduction.[10,11] Timing of the surgery also seems to have an effect on long term outcome, evidences suggest that it should be done before the age of ten.[12]

Local Scene

Over the past decades, there were much changes in the management of children with cerebral palsy in Hong Kong, particularly in the management of spasticity. The use of Botulinum toxin with ultrasound guidance, is now considered a standard practice especially in early childhood was introduced to Hong Kong in the early 90s', with better understanding on the importance of patient selection[13,14] and with the modification of technique, the surgery is considered safe and effective in permanent tone management. The concept of single event multilevel surgery (SEMLS) has significant impacts on the orthopaedic care in these children, surgeries are now being planned to aim at various levels in one goal and preferably at a later age; saving the child's time but require expertise in planning on the timing and extent of the surgery. Its intermediate term effect on the improvement of gait was also well established.[15] The use of 3D Instrumental Gait analysis[16] as an objective tool in monitoring treatment progress has already been widely used over the territory and with the more recently available rehabilitation aid like robotics-assisted locomotor training, all these improve our patients' care and outcome.

Collaborative Work between Sectors

Child Assessment Service seems to have no close relationship with Neurosurgery, both in the clients served or the format of service delivery. But it is through the bonding from children with cerebral palsy, this unusual collaboration has then begun.

With the conjoint effort from Dr Dawson Fong and Dr Catherine Lam, the Selective Dorsal Rhizotomy (SDR) Clinic was first established in 1997 as a joint venture between the CAS and the Department of Neurosurgery, Tuen Mun Hospital (TMH) to provide comprehensive assessment and care for children with CP. The clinic was held in the Tuen Mun Child Assessment Centre which was stationed within the premises of TMH. The aim of the clinic was to provide a platform for clinicians to share experience in the management of cerebral palsy and to discuss management plan for potential clients whom might benefit from SDR. Initial core members included Paediatricians and Physiotherapists from CAS and TMH, Neurosurgeons and Orthopaedic Surgeons from TMH. School Therapists and referrers were the clinic's other members.

Throughout the years, with better knowledge and accumulation of experiences, the purposes and also the

members of the clinic had slightly changed. Apart from the core members, we also have input from Urologists from TMH, in view that SDR might also improve bladder spasticity.[17] Though the name "SDR clinic" still holds, and we still screen and select candidates whom might benefit from SDR, the team also assess and recommend interventions if SDR is not the best option. With the availability of instrumental gait analysis in TMH, objective data are now available to assess patients with pre and post Botulinum Toxin injection, pre and post SDR or orthopaedic surgery. We have regular reviews for post-SDR clients for continual care and the clinic also serves to facilitate future screening, rehabilitation recommendations and look for long term data and outcome.

From 2003 through 2012, 61 patients had undergone SDR through referrals from the clinic. (Table 1) With cohort from 2003 through 2008, 80% of patients underwent SDR suffered from spastic diplegia (Table 2). The majority of them had Gross Motor Function Classification Scale (GMFCS) level one to three (Table 3) and most of them had their surgery at six to nine years of age. (Table 4)

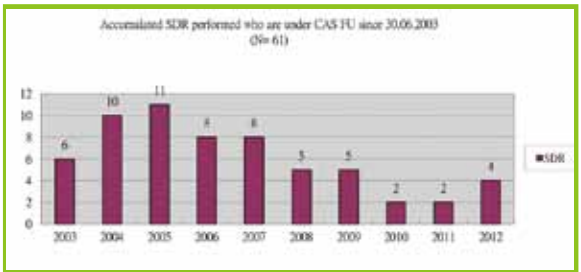

Table 1. Number of patients who had undergone SDR from 2003-2012

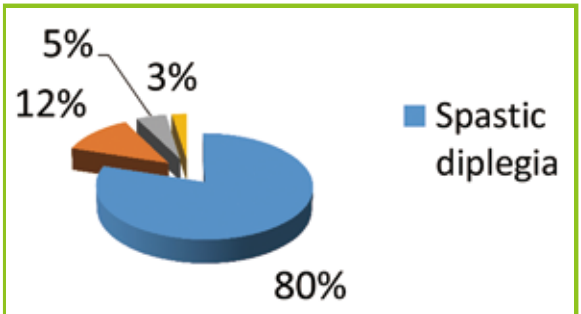

Table 2. Diagnosis of patients undergone SDR from 2003-2008
12% Asymmetrical spastic diplegia
5% HSP: Hereditary spastic paraparesis
3% Spastic quadriparesis

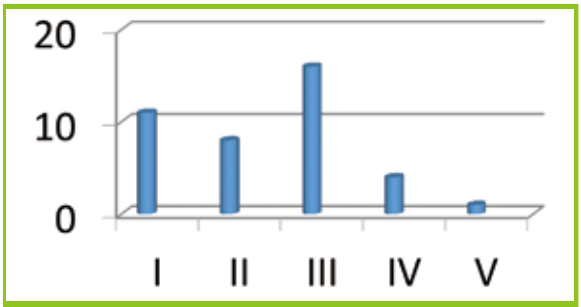

Table: 3. GMFCS level of patients undergone SDR from 2003-2008 GMFCS: Gross Motor Function Classification Scale

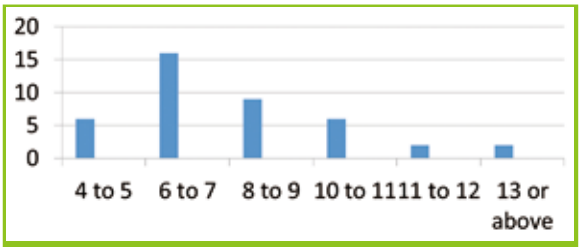

Table 4. Age range: Patients undergone SDR via SDR clinic from the year 2003-2007

With time, the Kowloon Physical Rehabilitation Clinic (KPRC), another conjoint clinic stationed in Kowloon, was established in 2009. Held in the Central Kowloon Child Assessment Centre, the clinic was aimed to serve patients with cerebral palsy living in Kowloon and also for easier access for some of the referrers. Approximately 30 patients were seen each year in the two conjoint clinics which was held once every two to three months. The clinic also invited overseas experts to join in every year as a platform for learning and advice concerning rehabilitation needs for complex cases.

Conclusion:

In our conjoint clinics, we demonstrate the coordination and integration of an overall care plan for our patients and within the clinic, we have excellent communication among specialists in various fields, the referrers and the school and primary care providers. We are now looking into the long term outcome of our patients especially in the third dimension of the ICF model, participation, and ultimately the quality of life. We wish our experience not only improves the care for children with cerebral palsy but can go further to other children with complex needs.

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Classified Advertisement

Please contact the Federation Secretariat at 2527 8898 for placement of classified advertisement.

The first hike at one of the "100 Famous Japanese Mountains " - Mt. Kiso-komagatake

Mr Cheung-kong NG

Nurse Consultant, Department of Neurosurgery, Tuen Mun Hospital.

Mr Cheung-kong NG

To begin hiking and expedition experience in Japan, most Hong Kong people will immediately think of the iconic Fuji Mountain. In fact, the Japanese hikers are keen on following the list from a book "100 Famous Japanese Mountains " (日本百名山 Nihon Hyaku-meizan), by mountaineer and author Kyūya Fukada. (深田久弥 1903 – 1971) in 1964.

The Chuo Alps or Central Alps (中央アルプス) are a mountain range mostly located in the Nagano prefecture (長野県). There are popular and challenging hiking courses demanding a higher level of mountaineering skills. Nevertheless, many of them are suitable for all hikers and require only a little earlier experience.

Mt. Kiso-komagatake (木曽駒ヶ岳 2956m) being the highest peak in the Chuo Alps, is also included on the list of "100 Famous Japanese Mountains." It would be a good option of interesting day trip climb for the beginners.

It takes an around 3.5 hours' ride in highway buses (through Chuo Expressway) or the railway (JR Okaya line岡谷線 & Iida line飯田線) from Shinjuku (新宿) to Komagane city (駒ヶ根). From there, visitors transfer to local buses for Shirabi-daira (しらび平 1661.5m), the bottom terminus of the Chuo Alps Komagatake Ropeway (駒ヶ岳ロープウェイ).

The ropeway provides all year round service since 1967. For sure, visitors will enjoy the spectacular views of mountain lines & waterfalls at the 7.5 minutes' "sky walk" to reach the highest altitude ropeway station in Japan – Senjojiki station (千畳敷 2612m).

Front - Senjojiki ropeway station, Right side back – Mt. Hokendake

The convenient ropeway trims 950m off the vertical ascent, leaving only around 300 more vertical metres from the top of the ropeway station to the summit of Mt. Kiso-komagatake. Visitors and hikers may stay overnight in the gorgeous Senjojiki Hotel (ホテル千畳敷) at the station, which has very nice views of Senjojiki cirque and the ridge trails. The Senjojiki Hotel gets extremely crowded at hiking and autumn red-leaves seasons. The Senjojiki cirque is a bowl-shaped terrain scraped away by ice in the ice-age 20000 years ago. It is famed as a treasure house of alpine plants in summer.

Wide view from Senjojiki station, Left – Mt. Kiso-komagatake shrine, Right – Senjojiki cirque

There are two trails leading off toward the left and right from the Mt. Kiso-komagatake shrine (駒ヶ岳神社) just outside the station. The right course is more popular, and it takes only 10 minutes walk from the station to Hatchozaka (八丁坂) - the starting point of a 40 minutes zigzag climb to the Norikoshi pure land (乗越浄土). The pure land is a 3-way junction with a mountain hut Hoken-sanso (宝剣山荘) in front. The mountain huts here provide all you needs for hiking including battery recharge for digital appliances. Behind the hut, there stands a giant eye catching rock - Tenguiwa (天狗岩) which looks like the face of a Japanese legend monster (天狗／てんぐ).

Round course of hiking trail

Continue to the right, you climb up past another hut to the top of Mt. Nakadake (中岳 2925m) and then down the other side to a saddle and a hut (頂上山荘). From there, it is a short climb to the summit of Mt. Kiso-komagatake,

where a lookout for panoramic view of both the North Alps and South Alps. Depends on the itinerary, hikers may stay in one of the huts to catch the sunset and sunrise.

Look back from Mt. Nakadake (Left - Norikoshi pure land, Front – Hoken-sanso 宝剣山荘 and Mt. Hokendake)

Summit of Mt. Kiso-komagatake

On return to Hoken-sanso, you may take the shortcut via the Mt. Nakadake winding road (中岳巻道). The narrow cliff pass astonishes you not only by the thorny rock configurations, but also a scary feeling of fall.

When arrived to Hoken-sanso, you may consider taking the trail back down to the Senjojiki, or traverse up and over Mt. Hokendake (宝剣岳 2931m). This trail is marked as a "dangerous" course. Mt. Hokendake is an interesting outcropping of granite rock with steep slopes and ridges. There are plenty of chains, ladders and paint marks to help you getting to the top. However, please don't forget to apply the "3-point holding" climbing technique. You will find your hardship pays off when seeing the truly stunning views from the top.

Most people will take the same route down but dare hikers challenge the way descending from the other side of the peak via terrible horseback ridges to the Sannosawa branch (三の沢分岐). With further walk to the Paradise of Flat (極楽平), a trail is shown up at the left and this is the way back down to the Senjojiki. In 2014, the last ride of ropeway from Senjojiki down is 17:07 in summer and 16:07 in winter.

On the following day, you may also enjoy another leisure day at the Komagane city for the thick cutlet pork-chop rice in special sauce (ソースかつ丼), Hayataro Onsen (早太郎温泉) and visits to the historic Temple of Light (光前寺) and the famous Yomeishu komagane Plant (養命酒駒ヶ根工場).

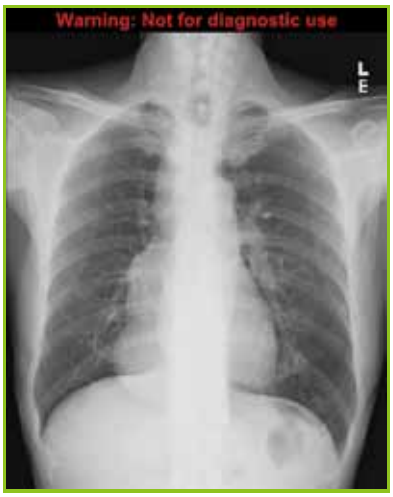
Chain climb at Mt. Hokendake

Summit of Mt. Hokendake

Radiology Quiz

Dr Katherine WONG

Department of Radiology, Queen Mary Hospital



Questions:

1. What is the abnormality?
2. What features suggest the location of the lesion?
3. What is the cause of the abnormality?

(See P.36 for answers)

"New paradigm in the management of chronic low back pain and osteoarthritis-related knee pain" Symposium

On 27 Feb 2014, a dinner symposium on pain management was held at the Mira Hong Kong, Tsimshatsui. The symposium was well attended by doctors, nurses, pharmacists and allied health professionals.

The lecture topics were "Chronic Low Back Pain and Pain Coping" and "Advances in Medications for Osteoarthritis". The Federation was privileged to have Dr Carina Ching-fan LI, Specialist in Anaesthesiology, and Dr Priscilla Ching-han WONG, Specialist in Rheumatology as our speakers; with Dr. Daniel Kam-hung NG, Specialist in Rheumatology, as our chairman for the symposium. Both speakers covered the topics extensively and provided valuable insight on the clinical management of these conditions. The Federation looks forward to organising further educational activities on various topics for our professionals in the near future.

Certificate Course on Rheumatology

Course No. C241

Jointly organized by : The Federation of Medical Societies of Hong Kong and
The Hong Kong Society of Rheumatology

Date : 7, 14, 21, 28 May and 18, 25 June, 2014 (Every Wednesday, skip 4 and 11 June)
Time : 7:00pm – 8:30pm
Venue : Lecture Hall, The Federation of Medical Societies of Hong Kong,
4/F Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, HK
Course Fee : $750 (6 sessions)
Enquiry : 2527 8898

CME / CNE / CPD accreditation will be applied
Course details and application form can be downloaded from website: http://www.fmshk.org

DCH (Diploma in Child Health Examination) Written Examination (MRCPCH Foundation of Practice) 2014

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 2014 awarding DCH (HK) and DCH (International) to successful candidates.

The DCH Examination is divided into two parts, written (MRCPCH Foundation of Practice) (formerly known as Part IA) and clinical. The written examination is the same as the MRCPCH Foundation of Practice Examination, which is held three times a year in Hong Kong. The next DCH written examination will be held on **Tuesday, 10 June 2014.** The examination fee is **HK$4,500** for Foundation of Practice. Candidates who wish to enter the examination must hold a recognized medical qualification in Hong Kong.

Application: Candidates **must apply online** using the RCPCH website via the **member sign in** area https://www.rcpch.ac.uk/user. In order to access the online application form, you need to be a registered user. If you do not have an RCPCH online account, you will be required to create one using the following link: https://www.rcpch.ac.uk/user/signup Applications for all exams will open at 9.00am UK local time on the first day of the advertised application period and close at 4.30pm UK local time on the last day.

Please note that application is NOT confirmed until payment of examination fees is received in Hong Kong.

Candidates who wish to sit the examination in Hong Kong **MUST ALSO** apply through the Hong Kong College of Paediatricians (HKCPaed) by completing Form B (Application for entry to the MRCPCH Foundation of Practice & Theory and Science Examinations-Overseas Centres). For application details, please visit the HKCPaed website at http://www.paediatrician.org.hk/index.php?option=com_content&view=article&id=45&Itemid=46 or call the College Secretariat at 28718871.

Application Period: Monday 24 March 2014 - Friday, 11 April 2014

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:
http://www.rcpch.ac.uk/training-examinations-professional-development/assessment-and-examinations/examinations/clinical-e-3

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		★ MPS Workshop – Mastering Shared Decision Making ★ Council Meeting **1**	**2**	★ MPS Workshop – Mastering Difficult Interactions with Patients **3**	**4**	**5**
6	**7**	★ HKMA Tai Po Community Network – Computerizing Your Clinic and Your Options **8**	★ Hong Kong Neurosurgical Society Monthly Academic Meeting –Frontal Lobe Syndrome ★ HKMA Shatin Doctors Network - Do We Need So Many Drugs for Managing Diabetes? ★ HKMA Central, Western & Southern Community Network - Latest Treatment on GERD and Acid Pocket ★ MPS Workshop – Mastering Adverse Outcomes **9**	★ HKMA Hong Kong East Community Network - Dual Bronchodilation - A New Era for COPD Treatment ★ HKMA Kowloon East Community Network - Updates on Shingles Prevention ★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Diagnosis and Treatment of Headache **10**	★ HKMA Yau Tsim Mong Community Network - Update on Management of Atopic Dermatitis **11**	★ HKMA CME – Refresher Course for Health Care Providers 2013/2014 ★ MPS Workshop – Mastering Professional Interactions **12**
13	**14**	★ HKMA Kowloon West Community Network - Do Patient Characteristics Influence Choice of DPP-4 Inhibitor? ★ HKMA Tai Po Community Network - How Can Men Perform Better At Night **15**	**16**	**17**	**18**	**19**
20	**21**	**22**	**23**	★ HKMA Kowloon East Community Network - COPD – Common & Manageable Disease in HK ★ HKMA New Territories West Community Network - Coronary Artery Disease Survival Kit for the Primary Care Physician **24**	★ HKMA Yau Tsim Mong Community Network - When Should Antihypertensive Treatment be Started? The Update of 2013 ESH / ESC Guidelines on Hypertension **25**	**26**
★ Lantau Hiking Team (Route TBC) ★ 2014 Paediatric Update No.1 Paediatric Genetics / Genomics **27**	**28**	**29**	**30**			

Date / Time	Function	Enquiry / Remarks
1 TUE 6:30 pm	**MPS Workshop – Mastering Shared Decision Making** Organiser: Hong Kong Medical Association Medical Protection Society, Chairman: , 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	**Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. TSE Hung Hing, Venue: HKMA Head Office (5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
3 THU 6:30 pm	**MPS Workshop – Mastering Difficult Interactions with Patients** Organiser: Hong Kong Medical Association & Medical Protection Society, Speaker: Dr. CHENG Ngai Shing, Justin, Venue: Eaton Hotel	HKMA CME Dept Tel: 2527 8452 2.5 CME Point
8 TUE 1:00 pm	**HKMA Tai Po Community Network – Computerizing Your Clinic and Your Options** Organiser: HKMA Tai Po Community Network, Chairman: , Speakers: Dr. HO Chung Ping, 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	Miss CHU Tel: 2151 2823 1 CME Point
9 WED 7:30 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Frontal Lobe Syndrome** Organiser: Hong Kong Neurosurgical Society, Chairman: Hong Kong Neurosurgical Society, Speaker: Dr CHENG King Fai, Kevin, Venue: Seminar Room, Ground Floor, Block A, Queen Elizabeth Hospital	Dr. Gilberto LEUNG Tel: 2255 3368 1.5 CME Point
1:00 pm	**HKMA Shatin Doctors Network - Do We Need So Many Drugs for Managing Diabetes?** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Dr. TONG Chun Yip, Peter, Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin	Ms. Maggie LAU Tel: 9865 9377 1 CME Point
1:00 pm	**HKMA Central, Western & Southern Community Network - Latest Treatment on GERD and Acid Pocket** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. TSANG Chun Au, Speaker: Dr. LEE Yuk Tong, Venue: The HKMA Central Premises,	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
6:30 pm	**MPS Workshop – Mastering Adverse Outcomes** Organiser: Hong Kong Medical Association & Medical Protection Society, Speakers: Dr. Hung Chi Wan, Emily, Venue: Eaton Hotel	HKMA CME Dept Tel: 2527 8452 2.5 CME Point
10 THU 1:00 pm	**HKMA Hong Kong East Community Network - Dual Bronchodilation - A New Era for COPD Treatment** Organiser: HKMA Hong Kong East Community Network, Chairman: Dr. CHAN Hoi Chung, Samuel, Speaker: Dr. WAN Chi Kin, Raymond, 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 - Updates on Shingles Prevention** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. AU Ka Kui, Gary, Speaker: Dr. WU Yee Ming, Jimmy, Venue: Lei Garden Restaurant (利苑酒家) Shop no. L5-8, apm, Kwun Tong, No. 418 Kwun Tong Road, Kowloon	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Diagnosis and Treatment of Headache** Organiser: Hong Kong Medical Association & Hong Kong Sanatorium & Hospital,Speaker: Dr. Tsoi Tak Hong, 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
11 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network - Update on Management of Atopic Dermatitis** Organiser: HKMA Yau Tsim Mong Community Network, Chairman: Dr. LAM Wai Chi, Rocky, Speaker: Dr. CHIU Lai Shan, Mona, Venue: Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285
12 SAT 2:15 pm	**HKMA CME – Refresher Course for Health Care Providers 2013/2014** Organiser: Hong Kong Medical Association, HK College of Family Physicians & HA-Our Lady of Maryknoll Hospital, Speaker: Dr. Ko Po Wan, 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
2:30 pm	**MPS Workshop – Mastering Professional Interactions** Organiser: 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
15 TUE 1:00 pm	**HKMA Kowloon West Community Network - Do Patient Characteristics Influence Choice of DPP-4 Inhibitor?** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. TONG Kai Sing, Speaker: Dr. LEE Ka Fai, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
1:00 pm	**HKMA Tai Po Community Network - How Can Men Perform Better At Night** Organiser: HKMA Tai Po Community Network, Speaker: Dr. FUNG Tat Chow, Berry, 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	Miss Koko YAU Tel: 9322 3925 1 CME Point
24 THU 1:00 pm	**HKMA Kowloon East Community Network - COPD – Common & Manageable Disease in HK** Organiser: HKMA Kowloon Esat Community Network, Chairman: Dr. MA Ping Kwan, Danny, Speaker: Dr. CHU Chung Ming, Venue: East Ocean Seafood Restaurant (東海海鮮酒家), Shop 137, 1/F, Metro City Plaza 3, 8 Mau Yip Road, Tseung Kwan O	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
1:00 pm	**HKMA New Territories West Community Network - Coronary Artery Disease Survival Kit for the Primary Care Physician** Organiser: HKMA New Territories West Community Network, Chairman: Dr. CHEUNG Kwok Wai, Alvin, Speaker: Dr. KO Lap Yan, Ryan, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
25 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network - When Should Antihypertensive Treatment be Started? The Update of 2013 ESH / ESC Guidelines on Hypertension** Organiser: HKMA Yau Tsim Mong Community Network, Chairman: Dr. CHOI Siu Tong, Speaker: Dr. LEUNG Tat Chi, Godwin, Venue: Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
27 SUN 10:00 am	**Lantau Hiking Team (Route TBC)** Organiser: The Hong Kong Medical Association, Chairman: Dr. HO Chung Ping & Dr. SIN Pui Yee, Helena, Venue: Lantau Island	Mr. Benjamin CHAN Tel: 2527 8285
2:00 pm	**2014 Paediatric Update No.1 Paediatric Genetics / Genomics** Organiser: Hong Kong College of Paediatricians, Speakers: Dr. Brian CHUNG, Dr. Ming-luk HO, Dr. Stephen LAM, Dr. Josephine CHONG, Venue: M-Ground, Lecture Theatre, Queen Elizabeth Hospital, Kowloon	Ms. Lily LIN Tel: 2871 8782

Upcoming Meeting

1/6/2014	**FMSHK Annual Scientific Meeting 2014 – Care for the Older Population** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Ballroom, 3/F, Sheraton Hotel, 20 Nathan Road, Kowloon, Enquiry: FMSHK Secretariat Tel: 2527 8898
28-30/6/2014	**4th IDKD Intensive Course in Hong Kong "Musculoskeletal Diseases"** Organiser: IDKD, HKU & HKCR, Venue: Hong Kong Convention & Exhibition Centre (HKCEC), 1 Expo Drive, Wanchai, Registration: www.idkd.org
18-19/10/2014	**MSHP 30th Anniversay Conference & Exhibition** Organiser: Management Society for Healthcare Professionals, Speakers: Dr Wing-man KO, Dr Pak-yin LEUNG, Prof Gabriel LEUNG, Prof Francis CHAN, Prof EK YEON, Venue: Kowloon Shangri-la Hotel, Enquiry: Ms. A. CHEUNG, Tel: 2861 2668

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: 9.30 am - 5.30 pm 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

1. Right posterior mediastinal mass.

2. The heart border and the hilar vessels are not obscured by the mass, so the mass is not in the anterior or middle mediastinum. It extends below the diaphragm, suggesting it arises from posterior mediastinal structures.

3. The surgical clips in the bilateral mediastinal region and absence of the stomach bubble suggest that the patient has had oesophagectomy with resultant thoracic stomach.

Dr Katherine WONG
Department of Radiology, Queen Mary Hospital

SCHOOL OF PUBLIC HEALTH
THE UNIVERSITY OF HONG KONG
香 港 大 學 公 共 衞 生 學 院

MASTER OF PUBLIC HEALTH

Postgraduate Diploma & Postgraduate Certificate

2014/15

Public Health Practice

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Information Day

- *January 18, 2014 & March 8, 2014*
- *2:00 - 4:00 p.m.*
- *Seminar Room 6, LG1 Laboratory Block*
  Faculty of Medicine Building
  21 Sassoon Road, Hong Kong

Early Application Offer

15 Feb 2014

Application Deadline

30 Apr 2014

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