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Substance Abuse - The Scene in Hong Kong

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BRINTELLIX TAKES CARE OF MORE THAN MOOD

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- Brintellix is efficacious in treating all the symptoms of depression (assessed by MADRS) across a range of patients[2-5]
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  - **These include: concentration difficulties, poor attention, problems with memory and difficulty planning**[6-8]
- Brintellix is well tolerated[4,5,10-12]
- Patients (18-65 yrs) can start, stay and stop on Brintellix 10 mg once daily[4]

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Disclaimer

The Cover Shot

This beautiful bird was photographed by Dr. Patrick Kwong in early Dec 2015 at Nam Sang Wai, New Territories. It has become quite the celebrity at this location, attracting hordes of bird photographers.

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Editorial

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Editor

Dr Ronnie PAO

I would like to thank Dr. YK Ng and the editorial board of the Federation of the Medical Society of Hong Kong for their kind invitation to serve as editor of this Substance Abuse issue. It is a great honour for me and a tremendous learning experience.

In our society, addiction is a more common problem than we may think. Some of these addictions are less harmful to ourselves and our surroundings (such as caffeine) whilst others take its toll on the users' health and society. Historically in Hong Kong, opiate based drugs such as heroin have been the most popular drug of abuse, although other psychotropic substances, such as ketamine, are becoming extremely popular, especially amongst the younger generation. According to the Central Registry of Drug Abuse in Hong Kong, ketamine is the most common abused substance, but this data may be only the tip of the iceberg since it does not include unregistered and thus hidden users in society.

I am extremely thankful that Professor CN Chen, the most respected and prominent psychiatrist in the field of addiction in Hong Kong, has agreed to provide an article for this edition. Many substances have been used therapeutically in the medical field before they became a substance of abuse. His insightful article on ketamine not only summarises the adverse effects of ketamine, but also argues that ketamine has its place in the management of various medical conditions.

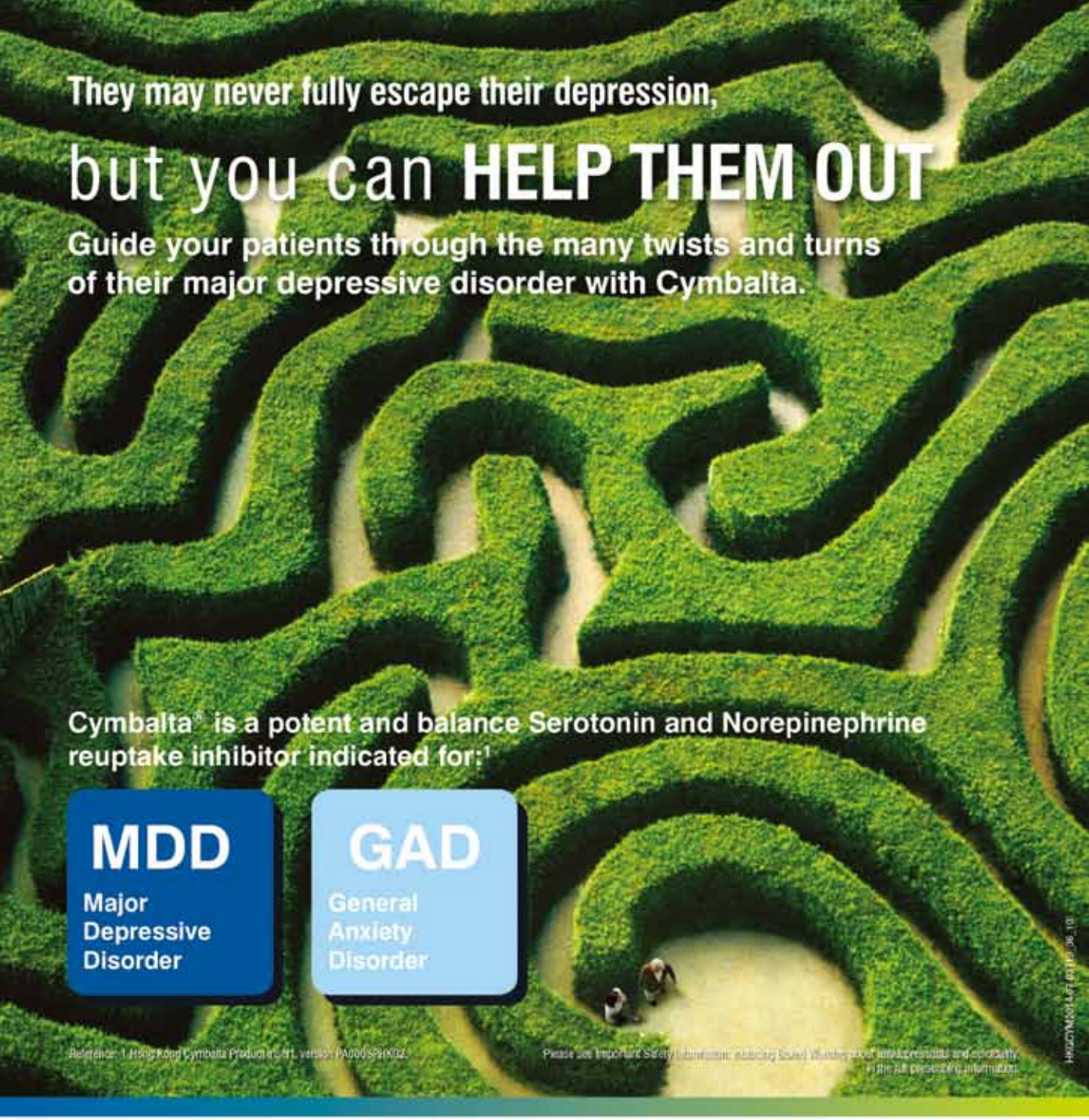
Professor WK Tang and his team from the Chinese University of Hong Kong provide an invaluable insight into ketamine and its effects in the local setting, specifically highlighting the effects it has in our society. Their article provides valuable information for us to understand more about the consequences and ways of combating ketamine abuse, with which all medical professionals should be familiar.

Alcohol use is culturally specific. In a place like Hong Kong where the culture is influenced by both western and eastern concepts, alcohol use can be perceived as normal and usually associated with happy events and collective celebrations. Dr. WH Cheung has kindly provided an article on alcohol-induced blackout. His summary of this topic reminds clinicians that it is a commonly seen phenomenon and it is important to educate our patients about the effects of alcohol on memory.

The management of addiction is no doubt multidisciplinary. Not only should we appreciate the involvement of neurotransmitters in the mesolimbic pathway between the ventral tegmentum in the midbrain and the nucleus accumbens that plays a large part in pleasure and addiction, we should also value the environment and social aspects of its aetiology. Mr. Kay Lam provides an occupational therapy perspective and its role in the rehabilitation process. His article focuses on the enhancement of life role in this group of clients, an important aspect of rehabilitation but unfortunately commonly overlooked.

Addiction bears the characteristics of any chronic medical illness including its effect on different body systems, the social impairment it causes, and its relapsing course. It is an exciting field of medicine that will face many new challenges in the future. I hope this edition will rekindle the readers' interest in addiction and substance abuse.

Finally, I must express my heartfelt thanks to Dr. P.K. Kwong for his generous provision of a stunningly beautiful cover photo and Dr. Isaac Yip for his sharing of an intriguingly captivating European trip. I am sure they will enthuse those who have not already visited these picturesque areas to plan their next holiday.



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Ketamine: Friend or Foe?

Prof Char-Nie CHEN

Prof Char-Nie CHEN

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 March 2016.

Introduction

In Hong Kong, according to CRDA[1], ketamine has emerged since 2001 as the most common abused substance (2,166 ketamine abusers in 2014). With widespread abuse, the USA (1999), Hong Kong (2000), Taiwan (2002), Canada (2005), UK (2001/2014), India (2013) and Australia (2015) have enacted laws to contain ketamine abuse[2-4]. Others are still considering[3]. Since 2006 the WHO has repeatedly been requested to list ketamine under international control5, but to no avail6. The main objections have come from anaesthetists and veterinary doctors who find ketamine important in their work, as well as from concerns that, if ketamine is strictly controlled, poor countries might be deprived of the only effective anaesthetic to which they have access[5-7].

Ketamine as a Chemical Substance

Ketamine is an arylcycloalkylamine developed from phencyclidine (Sernyl®). In 1970 it was approved by the FDA as an anaesthetic and marketed in racemic form as Ketalar, which has two enanthiomers[8]. The S(+) isomer (as Ketanesth® in Europe) is the active enanthiomer. It is twice as strong as the racemic form and four times stronger than the R(-) isomer.

Pharmacokinetics

Ketamine is both water- and lipid-soluble. It is available in liquid form for intramuscular (i.m.i.) and intravenous (i.v.i.) injection, in powder for intranasal use, and in tablet and elixir for oral use. Its bioavailability varies from 17% (oral) to 100% (i.v.i.)[9-10]. In children, effects are evident 45 seconds after 2mg/kg i.v.i., and 4 minutes after 3mg/kg i.m.i.[11] Recovery occurs 18 minutes after i.v.i., and 25 minutes after i.m.i. Its half-life is 2.17 hours. Ketamine (80%) is metabolized to its active metabolite, norketamine that accumulates for longer (5 hours) than ketamine9. Biotransformation of ketamine depends on CYP3A4, CYP2B6 and CYP2C9[12]. Norketamine is finally hydroxylised to 6-hydroxynorketamine before excretion through bile and urine.

UNDESIRABLE OR TOXIC EFFECTS OF KETAMINE

Cognitive Effects

Ketamine's cognitive side effects include concrete thinking, impaired episodic memory deficits, reduced verbal recall, reduced attentional performance, impaired performance on tests of vigilance, acute amnesic effects, poor recognition verbal memory, inhibition of long-term potentiation, impaired verbal frequency and perseveration.[13] In another review[14], the acute effects were listed as impaired episodic memory, semantic memory, attention, working memory, and procedural learning and the perceptual representation system. The chronic effects of ketamine are similar to the acute effects, but more marked on semantic and episodic memory. They may be dose-related and persistent, although ex-abusers report improved cognitive impairment[15].

Psychedelic Effects

These symptoms may be related to the 'emergence phenomenon' described by anaesthetists that have been reported in 5-30% of cases at a dose of ketamine 600-1,100 ng/ml in anaesthesia or 50-200 ng/ml in healthy volunteers, and are dose-dependent[16]. They include alterations in mood state and body image, floating sensation, vivid dreams or illusions, occasional frank delirium[17], out-of-body experience (k-hole or near-death experience), visual hallucinations, synaesthesia, anxiety, grimacing, lip-smacking, facial exploration[18], exotic imagery, confusion, euphoria, unpleasant feelings, dreams, and nightmares[19]. A higher dose may cause anxiety, paranoid ideas or even delirium[9]. They may also be due to ketamine's cognitive, psychedelic and psychotomimetic side effects.

Neurological Effects

In rats, NMDAR antagonists such as phencyclidine or ketamine produce vacuoles in nerve cells, the so-called 'Olney's lesion'[20]. In recent years, ketamine has been reported to cause numbness, burns, impaired consciousness, gait disturbance, muscle weakness, ulner nerve compression, dizziness, vertigo, headaches, sweating, muscle spasms/twitches, sudden jerky movements, tremor, and increased intracranial pressure in humans[13].

Recreational Use

A typical recreational dose18 of ketamine is 200-300 mg (oral administration), 75-125 mg (i.m.i.), 60-250 mg (intranasal snorting), or 50-100mg (i.v.i.). A mega-dose of over 250 mg may cause unconsciousness.

In Hong Kong, ketamine is mainly abused by youths individually (at home or in a school toilet) or in small groups (parties, clubs, parks, recreational places). Abuse in a first-year primary school student has been reported[21]. In Taiwanese nightclubs, people compete in snorting contests. Ketamine powder is lined up on a table several feet long and individuals snort the powder as quickly as possible. Deaths have been reported[22].

Addiction Liability

Ketamine tolerance increases rapidly. Although DSM-5 describes phencyclidine/ketamine as being free from withdrawal symptoms[23], symptoms have been reported in Hong Kong in both primary ketamine abusers and ketamine-related polydrug abusers. Symptoms include fatigue and excessive yawning; irritability; anger, hostility or acting aggressively; sleep difficulty; and depression[24].

Acute Emergency Symptoms

In a review of 233 ketamine abusers from 15 public hospitals in Hong Kong[25], abusers were commonly 13-19 years old (range: 13-60 years), and twice as many were male. In most instances, ketamine was snorted. Co-administered substances included alcohol, MDMA, methamphetamine, benzodiazepines and zopiclone. Common symptoms involved neurological, cardiovascular, gastrointestinal, urinary and respiratory systems. Powder was sometimes visible in the nose and fever was sometimes present.

Psychiatric Symptoms

In 1959 phencyclidine-induced psychotomimetic effects were reported[27], and a glutamatergic model for schizophrenia involving phenycyclidine and ketamine[28] was later proposed (see previous review[29]).

In a community study[30] of 230 teenage ketamine abusers, 48.3% reported childhood adversities prior to the age of 16 years. They included physical abuse, parental divorce/separation, parental substance use, mental disorders or sexual abuse. The frequency of ketamine use correlated positively with total score of Beck Depression Inventory, and the duration of ketamine use with the Hospital Anxiety and Depression Scale.

In a study[24] of 95 ketamine and ketamine-related polydrug abusers, based on DSM-IV criteria, co-morbid psychiatric disorders included mainly depressive disorder, drug-induced psychosis, and phobic anxiety disorder. In another study[26] of 124 ketamine abusers, based on ICD-10 criteria, 55.6% had co-morbid psychiatric disorders that included drug-induced psychosis, schizophrenia, other psychosis, depressive episodes, dysthymic disorder, adjustment disorder, personality disorder and others.

Urological Symptoms

Symptoms vary and include increased frequency of small volume micturition, urine leakage or incontinence, dysuria, nocturia, supra-pubic pain, painful haematuria, and urinary strangury. The frequency of taking ketamine has been associated with the Pelvic Pain, Urgency, Frequency Symptoms Score, Bother Score and total score[30]. 47.8% of abusers develop symptoms of an overactive bladder. For each extra day of drug abuse, the probability of overactive symptoms increases by 0.5. Both duration and frequency of drug use are inversely correlated with maximum urinary flow rate. Hydronephrosis, found on ultrasound, is also correlated with the dosage of ketamine.

Gastro-intestinal Symptoms

K-cramp was described by Jansen (2000)[31]. Among 37 ketamine users, 76% had upper gastrointestinal symptoms[32]. Post-endoscopy diagnoses were mainly of gastritis. Abdominal pain did not respond to any treatment, but abstinence was associated with relief of symptoms. The odds ratio of symptomatic relief for abstinence versus continued use of ketamine was 12.5.

Other Symptoms

Other symptoms reported have included dilatation of the common bile duct[33], liver dysfunction[34], increased ocular pressure[35], and overdose or accidental death[22,36].

DESIRABLE EFFECTS OF KETAMINE AS AN ANAESTHETIC AND ANALGESIC

Ketamine is included in the W.H.O. Model List of Essential Medicines[37]. It is 'a safe, rapid-acting parenteral anaesthetic and analgesic agent...' [38], and 'one of the most commonly used anaesthetic agents in developing countries (readily available, easy to use and inexpensive)'[39]. It is widely used for induction and maintenance anaesthesia[40] in paediatric surgery[41] and veterinary medicine[42], especially in poor countries. It is also used in burn cases, cardio-thoracic and obstetric surgery, out-patient surgery and the critically or elderly ill38. It can be used as an effective analgesic for post-operative pain[43] and complex regional pain syndrome[44], and to assist in reducing preoperative anxiety[45] and acute agitation[46]. It has the following pharmaco-therapeutic effects.

Analgesic effects

Ketamine is a non-competitive antagonist of the N-methyl-D-Aspartate receptors (NMDAR). NMDAR are glutamatergic receptors that bind to excitatory amino acids such as glutamate, aspartate, and glycine. They are responsible for processing sensory information, including nociceptic impulses, at the spinal, thalamic, limbic and cortical levels. At 200 ng/ml (plasma) ketamine reduced heat pain score and abolished the activated response to noxious stimuli, with a 61% reduction in the measured fMRI signal (parameter estimate from 97.8 to 36.9)[47]. The reduction in the brain occurred significantly in the thalamus and insula, with a non-significant reduction in the anterior cingulate cortex.

In cats, ketamine induced an alternating pattern of delta-wave activities and low voltage, fast activities in the thalamus and neocortex, but not in the hippocampus or midbrain reticular formation[48]. Instead, typical theta activity appeared in the hippocampus. Thus, incoming information flows to the neocortex through the thalamus, but not to the limbic system through sensory association cortex. This was confirmed by another study with rhesus monkeys[49]. Ketamine is therefore known as a dissociative anaesthetic.

Ketamine also binds to opioid receptors (μ, δ, κ). Since it is not antagonised by naloxone[50], it is likely an indirect effect. Ketamine may also inhibit transporters of monoamines (noradrenaline, dopamine and serotonin), leading to increased monoamines in the synaptic clefts[51-52]. These may also contribute to the analgesic effects of ketamine at both the central and spinal levels, as well as the cardiovascular and psychotomimetic effects.

Hypnotic effects

Ketamine increases slow-wave sleep (SWS) in rats[53]. Nonetheless it has been argued that the hypnotic effects of ketamine are not mediated by the NMDAR[54]. Instead, the S(+) isomer of ketamine is a potent inhibitor of cloned hyperpolarization-cyclic-nucleotide (HCN1) channels and of hyperpolarization-activated pacemaker current (Ih) in cortical pyramidal neurons. This is supported by another study with HCN1 knock-out mice[55].

Sympathomimetic Effects

Unlike other anaesthetics, ketamine increases heart rate, cardiac output and blood pressure. These sympathomimetic effects are caused by direct stimulation of the CNS structures[38] Therefore ketamine is a useful anaesthetic in patients with hypovolaemia and haemodynamic instability, especially trauma cases[56]. Nonetheless ketamine has a direct depressant effect on the myocardium, hence it is contraindicated in patients with hypertension and coronary artery disease. The undesirable effect can be reduced by benzodiazepine[17].

Respiratory Effects

Ketamine is a smooth bronchial muscle relaxant with minimal effect on central respiratory drive and relative preservation of swallowing, cough, sneeze and gag reflexes. It may increase salivation or produce bronchospasm[38].

KETAMINE AS A POSSIBLE ANTIDEPRESSANT

Intravenous infusion of 0.5 mg/ml ketamine has been reported to reduce depressive symptoms in seven depressed subjects within a 72-hour period[57]. Such improvement was significant for condition-by-time but not for condition or time alone. It is supported by another study[58] of major depression with a randomized, double-blind crossover design. Significant improvement occurred in 71% of cases 2 hours later, and 29% met remission criteria. 35% maintained a response for at least 1 week. Similar improvement was shown in patients with bipolar I or II depression[59]. Nonetheless ketamine has not yet been approved for routine clinical treatment of depression. Further confirmation with a larger sample and longer duration of treatment is necessary. It is particularly important to find out the mechanisms for such rapid but short-lived antidepressive effects, bearing in mind that ketamine has acute amnesic and psychedelic side effects.

KETAMINE AS AN ADJUVANT IN PSYCHOTHERAPY

LSD psychotherapy was reported in the 1950s[60]. Following the establishment of the Multidisciplinary Association for Psychedelic Studies[61], psychotherapists have attempted to make use of several psychedelic/hallucinogenic drugs including LSD, psilocybin, MDMA, mescaline, phencyclidine, and ketamine. The rationale for its use was to 'aid emotional insight by lowering psychological defences'[62]. With the use of fMRI in a placebo-control study[62], psilocybin was found to activate memory vividness and visual imagery. Ketamine-assisted psychotherapy has since been applied to alcoholism[63], heroin addiction[64], death anxiety[65] and perhaps others.

CONCLUSION

All drugs should be properly managed before use. This review has shown that ketamine is a useful anaesthetic and analgesic; but like anxiolytic benzodiazepines, it is liable to be abused and lead to dependency and is ultimately dangerous to health. The Convention on Psychotropic Substances of 1971 provides Schedules, I to IV, from most to least restrictive control, under the United Nations[66]. It is unclear if the current debate is about whether or not ketamine should be scheduled, or whether it should be listed under Schedule I or IV[6]. Such confusion might have caused the debate to be based more on emotions than on facts. The facts are that many countries have made laws to control ketamine abuse. If WHO experts refuse to examine the facts, what is the purpose of the WHO Convention? Veterinary doctors and anaesthetists are worried about restrictions on use if ketamine is scheduled, but this relates more to a national government's efficiency than a socio-medical matter.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Ketamine: Friend or Foe?" by Prof Char-Nie CHEN and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 March 2016. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

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

1. Ketamine is used by anaethetists and veterinary doctors in their line of work.
2. Ketamine can only be used via intranasal method.
3. Ketamine's cognitive side effects includes a reduction in verbal recall.
4. Ketamine does not cause paranoid ideas.
5. Ketamine is free from withdrawal symptoms.
6. Hydronephrosis is correlated with the dosage of ketamine.
7. Ketamine is likely to have a direct effect on opioid receptors.
8. Ketamine is free from any respiratory effects.
9. There is poosibly antidepressant effects with ketamine.
10. Ketamine is routinely used as an adjuvant in psychotherapy in modern days.

ANSWER SHEET FOR MARCH 2016

Please return the completed answer sheet to the Federation Secretariat on or before 31 March 2016 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Ketamine: Friend or Foe?

Prof Char-Nie CHEN

1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

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

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

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

Answers to February 2016 Issue

Drugs Prescription in Patients with Kidney Disease

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

Ketamine Misuse in Hong Kong

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Abstract

Ketamine has ranked as the top abused psychotropic drug in Hong Kong since 2000. The epidemiology, physical harm, psychiatric comorbidities and cognition deficits associated with ketamine abuse, and strategies to combat ketamine abuse are reviewed based on local literature.

Introduction

Ketamine was first synthesized in the 1960s and can mediate profound analgesia and amnesia without any slowing of heart rate or breathing (Morgan, Curran, & Independent Scientific Committee on, 2012). It was approved by the USA Food and Drug Administration in 1970 for anesthesia in children, adults, and the elderly (Ross, 2008). Nonetheless non-medical use of ketamine has become increasingly common since the 1990s when it appeared on the rave scene (Dalgarno & Shewan, 1996). Ketamine is one of the most frequently used drugs, particularly among young people and club-goers in the USA (Gahlinger, 2004). In the United Kingdom, 33.8% of respondents in an online survey reported ketamine use within the last year (Winstock, Mitcheson, Gillatt, & Cottrell, 2012).

Ketamine is a non-competitive N-Methyl-D-aspartate (NMDA) receptor antagonist that interferes with the transmission of excitatory amino acid glutamate and aspartate, and also with other monoamines such as dopamine and serotonin (Olney, Newcomer, & Farber, 1999). These pharmacological actions lead to psychiatric symptoms and cognitive dysfunction (Krystal et al., 1994; Wolff & Winstock, 2006), such as hallucinations (Reich & Silvay, 1989), delusions (Lahti, Koffel, LaPorte, & Tamminga, 1995) and impaired memory (Morgan, Muetzelfeldt, & Curran, 2009; Morgan, Muetzelfeldt, & Curran, 2010).

The route of ketamine abuse varies widely and includes intranasal, intravenous, oral, intramuscular and rectal solution (Radvansky, Puri, Sifonios, Eloy, & Le, 2015). Nonetheless for ketamine abusers in Hong Kong, the principal route of administration is intranasal (Joe-Laidler & Hunt, 2008). In the United Kingdom, the average daily consumption of ketamine in frequent users (more than four times a week) is 3.8 grams (Morgan et al., 2009). The average weekly consumption of ketamine in a group of 297 users in Hong Kong has been reported to be 17.9 gram, with the majority (79%) claiming daily use. The amount of ketamine consumed per use was 2.8 gram (G. L. Wong et al., 2014)

Epidemiology of ketamine misuse in Hong Kong

Ketamine misuse in Hong Kong has been popular in the last two decades. The frequency of ketamine abuse increased from less than 1% in 1999 to being the most commonly abused drug in 2011 (Narcotics Division, 2015).

The problem of ketamine abuse is especially serious among young people who attend rave parties and social gatherings (S. Wong et al., 2009). In a survey conducted between January 2003 and May 2004, ketamine was the most frequently used drug by Hong Kong`s club-drug users, of whom the proportion lifetime use was 88.3% and use in last 12 months was 80.8% (Loxton et al., 2008). In Hong Kong, the ketamine user is typically a youth under the age of 21. In 1999, only 0.6% of drug users under 21 years reported abusing ketamine but this figure increased to a peak of 85.0% in 2008, then decreased slowly to 46.3% in 2014. In drug users aged over 21 years, 0.1% reported abusing ketamine in 1999, but this had increased to a peak of 22.4% in 2013, then decreased to 19.5% in 2014 (Narcotics Division, 2015).

Physical harm from ketamine abuse in Hong Kong

Due to the large number of ketamine misusers in Hong Kong over the last two decades, physical harm by ketamine misuse has been well described and assessed. There is a high demand for emergency medical care amongst this population. Ketamine abusers comprised 16% of all drug abusers who attended accident and emergency departments in the period July 2005 to December 2005, and the proportion increased to 40% in the period January 2008 to June 2008 (Ng, Tse, Ng, & Lau, 2010). The largest cohort study examined the acute clinical presentation of 233 ketamine abusers and found that the most common presenting symptoms were impaired consciousness (45%), abdominal pain (21%), lower urinary tract symptoms (12%), and dizziness (12%) (Ng et al., 2010).

Urinary tract damage is one of the most common physical impairments associated with ketamine misuse. Chu et al. first reported bladder contraction in 10 ketamine misusers in Hong Kong in 2007 (Chu et al., 2007). Another study recruited 66 community dwelling ketamine users and found that those with a heavier (more than 3 times a week) and longer duration (more than 24 months) habit were more likely to have impaired

bladder function. These early functional changes could resolve after 1 year of ketamine abstinence (Mak et al., 2011). A 3-month longitudinal study tracked urinary symptoms after cessation of ketamine use in female misusers and concluded that the severity of symptoms was inversely correlated with the duration of cessation although some symptoms persisted after cessation (Cheung et al., 2011).

Gastrointestinal abnormalities are also reported in ketamine abusers. A study reviewed 26 ketamine misusers from 2008 to 2014 in Hong Kong and indicated that 18 (69%) had fusiform dilatation of the common bile duct and the degree of dilatation was correlated with duration of ketamine misuse (Yu et al., 2014). This finding was also supported by several previously published case reports of dilatation of the bile duct in ketamine users (Lo, Krishnamoorthy, Freeman, & Austin, 2011; Lui, Lee, & Li, 2014; Seto et al., 2011). In addition, 9.8% of chronic ketamine abusers had liver injury, and in some cases significant liver fibrosis (G. L. Wong et al., 2014).

Toxicity is another major concern. Ketamine was the fourth most common poison amongst the Hong Kong Poison Information Centre (HKPIC) poisoning data in 2010 (Chan, Tse, & Lau, 2012). HKPIC have analyzed the medical data of 188 acute and 96 chronic ketamine poisoning cases from June 2008 to July 2011. Among the 188 acute cases, 90 cases (48%) presented with neurological features such as confusion and drowsiness; 60 (32%) with lower urinary tract symptoms; 50 (27%) with cardiovascular features such as hypertension and tachycardia; 49 (26%) with abdominal pain and 19 (10 %) with acute psychiatric problems such as acute psychosis. The 96 chronic cases had a mean duration of ketamine abuse of 8.6±4.1 years, and the majority (88 cases, 92%) presented with features of cystitis while 63 (66%) and 40 (42%) of them presented with chronic abdominal pain and nasal problems respectively (Chan, 2012).

Psychiatric comorbidities of ketamine misuse

Several local studies have investigated the psychiatric comorbidities in ketamine abusers over the last decade (Table 2) (Chen, Chan, Chen, & Tang, 2005; Liang, Tang, Chan, Ungvari, & Tang, 2015; A. Tang, Cheung, Liang, Ungvari, & Tang, 2011; W. K. Tang, Liang, Lau, Tang, & Ungvari, 2013; W. K. Tang et al., 2015). These local studies found that mood disorders were the most common psychiatric comorbidity, followed by psychotic disorders. Notably, the ketamine abusers in these studies were long term and frequent users.

Ketamine abuse, both acute and chronic, has wide-ranging effects on memory systems (Morgan & Curran, 2006). In acute use, ketamine mainly impairs episodic memory, while in chronic use there are marked effects on semantic as well as episodic memory (Morgan & Curran, 2006). In Hong Kong, a study of cognitive impairment found that chronic ketamine poly-drug users displayed predominantly verbal and visual memory impairment, even after a period of abstinence from ketamine (Liang et al., 2013). Another study indicated that cognitive impairment was found only in current ketamine users and cognitive function in ex-ketamine users was comparable with that of healthy controls (W. K. Tang et al., 2013).

Fighting ketamine in Hong Kong

Ketamine abuse has been a serious social problem in Hong Kong for the last two decades. The government and all sections of society have joined forces to combat the problem. Educational professionals and the narcotics division have impelled promotion of the Healthy Campus Plan since the 2011/12 academic year. The plan aims to encourage students to avoid drugs and motivates those who abuse drugs to seek help or treatment. In the coming 2015/16 academic year, there will be more than 90 schools involved in this project.

Medical professionals are carrying out a community screening study and longitudinal treatment research to help solve the physical comorbidities associated with ketamine abuse, for example the community study of Uro-Psycho-Physical changes in young adults using ketamine, a school-based survey of bladder dysfunction symptoms and the efficacy of treatment for ketamine-induced voiding dysfunction. There are also ongoing projects exploring the brain damage and changes in bio-markers caused by ketamine use. All these projects or studies aim to not only determine better treatment but also help the youth and society to be better informed about the problems associated with ketamine abuse.

There are also many facilities that are taking part in the anti-drugs project in support of the narcotics division of Hong Kong government or providing services against drugs, including Counselling Centres for Psychotropic Substance Abusers (11), Centers for Drug Counselling (2), Substance Abuse Clinics, Youth Outreach Teams (19), Young Night Drifters (18), Drug Treatment and Rehabilitation Centers (39), Drug Addiction Treatment Centers (4), and Methadone Treatment Program. The Three-year Plan on Drug Treatment and Rehabilitation Services in Hong Kong (2015-2017) commenced in July 2015 and will continuously help drug abusers. In the coming years, monitoring of ketamine abuse, publicity, education and effective treatment services will be required to control the problem in Hong Kong.

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Table 1. Recent studies of the psychiatric comorbidities of ketamine abusers in Hong Kong

	Pain-specific risk factors	Setting	Substances abuse	The most common diagnosis
Chen et al., 2005#	n= 95 (male 73, female 22)	Nightclubs and drug counselling centers.	24 (25%) primarily ketamine users; 71 (75%) ketamine poly-drug users.	12.6% depressive disorder; 6.3% drug induced psychosis.
Tang et al., 2011#	n=32 female	residential treatment center	27 (84%) dependent on ketamine; 12 (38%) dependent on MA.	47% substance-induced mood disorder; 16% substance-induced psychotic disorder.
Liang et al., 2015*	N = 129 (male 66, female 63)	Substance abuse clinic.	98 (76%) dependent on ketamine only; 12 (9.3%) dependent on MA; 11 (8.5%) dependent on cocaine.	31.8% substance-induced psychotic disorder; 27.9% depressive disorder.
Tang et al., 2015#	N = 200	Drug Counselling Center	100 primarily ketamine users; 100 ketamine poly-drug users.	80.4% mood disorders ; 33.3% anxiety disorders; 7.8% psychotic disorders.

Note: diagnosis criteria by * ICD-10 or # DSM-IV; MA: methamphetamine.

Alcohol-Induced Blackout

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Introduction

Alcoholic beverages are readily accessible in Hong Kong. In 2014, the per capita alcohol consumption in Hong Kong was estimated to be 2.80 litres (of pure alcohol) [1].

Alcohol contributes to more than 60 types of disease and injuries, and accounts for 4.2% of the global risks for burden of disease as measured by disability-adjusted life years (DALYs). Its burden is particularly high in some high-income countries (6.7%) [2].

Alcohol is a known central nervous system (CNS) depressant: acute alcoholic intoxication not only impairs motor coordination and judgement, but also causes memory deficits. Alcohol-induced blackout is a common phenomenon experienced by moderate and excessive drinkers, once thought to affect only alcoholics. Nonetheless research has established that it is also common in the social drinker[3-6].

Phenomenology of alcohol-induced blackout

Alcohol-induced blackout refers to the loss of memory of the events of any part of a drinking episode without loss of consciousness, and was first systematically described by Goodwin et al. (1969)[7]. Among the 100 randomly selected hospitalized alcoholics interviewed (85 males and 15 females), 64 reported memory loss associated with drinking. There are two qualitatively different types of alcohol-induced blackout: en bloc and fragmentary.

An en bloc blackout has a definite beginning and terminates with a feeling of "lost time" because the latter may be blurred by subsequent unconsciousness or sleep[8]. The amnesia is total and permanent. The feeling of apprehension is intense when the complete amnesia is realised. The person did not behave differently during this lost period of time compared with the remaining time for which memory is available.

In fragmentary blackout, the subject does not realize that events have been forgotten until they are later recalled spontaneously or brought to the person's attention by someone else. The recall, however hazy, is the rule. Most subjects who report en bloc blackout also experience the fragmentary kind.

Blackouts occur during intoxication in non-alcoholics and alcoholics. It is a kind of anterograde amnesia in which the ability to retain new information is markedly impaired[9].

Risk factors

Alcohol-induced blackouts are more common when gulping drinks, when alcohol is ingested without food or in the presence of head injury or illicit drug use[7]. It is more likely that an alcohol-induced blackout is a result of a rapid rise in blood alcohol level combined with innate factors.

Genetic factors have been found to contribute to blackouts. The heritability of lifetime blackouts has been reported to be 58%. Genes whose products have either direct or indirect involvement in γ-aminobutyric acid A (GABAA)-mediated transmission or the polymorphisms that affect the structure of N-methyl-D-aspartate (NMDA) receptors, their subunit composition or the degree to which various receptor types are expressed could be contributing genetic risk specific to blackouts. Genetic factors also contribute to individual differences in alcohol metabolism, for example genotype at the loci for alcohol dehydrogenase (ADH) ADH1B and ADH1C; gastric emptying speed and gastric ADH activity may also be a source of shared genetic variance[10].

In a 6-year longitudinal study that examined alcohol use and behavioural risks during the transition from high school through college, men with a maternal family history of alcohol problems were more likely to experience blackout[5]. Women were more likely to report blackouts than men[5,6,11]. The increased bioavailability of alcohol due to the substantially less gastric Class III Alcohol Dehydrogenase (ADH) and higher body fat/water ratio in women results in women experiencing a higher blood alcohol concentration and greater intoxication than men per given amount of alcohol, after controlling for body mass. The recovery of short-term memory function is also slower in women[11].

The environment plays a key role in determining current drinking habits, and the risk of blackout. In an e-mail survey of 772 college students and their experiences with blackouts, the likelihood of having a blackout in the previous year was positively related to consuming three or more drinks per occasion in the two weeks before the survey, and initiating drinking at age 16 years or younger[6].

Low plasma tryptophan levels[12] and concomitant drug use, including tricyclic antidepressants[13], may

predispose even the social drinker to alcohol-induced blackouts.

Many factors presumed to contribute to the occurrence of blackouts have subsequently been found to be unrelated to blackout risk, including cognitive dysfunction and duration of problem drinking; and organic brain dysfunction[11].

Effects of alcohol on memory

Memory formation and storage takes place in several stages. When one attends to sensory information, it is transferred from the sensory memory (which lasts only for a few seconds) to the short term memory. The short term memory (which lasts up to minutes) is likely to be encoded into the long term memory if it is rehearsed and processed through understanding and manipulation of the information.

In alcohol-induced blackout, the ability to encode the information from the short-term to long-term memory is impaired due to the complete blockade of the normal neural pathway for memory occurring in the medial temporal structures. Alcohol profoundly suppresses CA1 pyramidal cell activity in a dose-dependent manner[14]. CA1 contains a very high concentration of hippocampal pyramidal neurons that facilitate hippocampal communication with other brain regions. The hippocampus incorporates information from other brain regions to form new autobiographical memories, and CA1 pyramidal cells send the results of this processing from the hippocampus to the neocortex[15]. Apart from the hippocampus, other brain structures that are implicated in alcohol-induced cognitive and memory impairment include the frontal lobes[11,16], the ventral tegmental area[17]. New memory does not occur, while recall from previous memory is maintained.

The hypothesis that alcohol-related amnesia is a result of state-dependent effects of alcohol[7] was not substantiated in another study of fragmentary blackouts where participants had poor recall of the drinking event after returning to the intoxicated state[18].

The procedural memory and the retrieval of long term memory remained functional. Therefore the person is walking and talking without much difficulty during the blackout period[19].

Clinical implications

Buelow and Koeppel[20] reported that many students experienced alcohol-induced blackout after only one or several drinks. They were at higher risk of injury due to physical incoordination and lapse in impulse control and memory consolidation. They engaged in a wide range of high risk behaviour during the blackout period, including reckless driving, getting into fights, and having sex with people other than their usual partners. Blackouts lead to emotional stress and distress. They experienced intrusive thoughts about the event, trouble sleeping, and waves of strong feelings about the incident.

In another study of 954 college students at five university sites, there were 404 emergency department visits over a two year observation period for injuries that ranged from bone fractures to head and brain injuries requiring computed tomography. Blackouts were a strong predictor of emergency department visits for college drinkers[21].

Although the majority of individuals who experienced an alcohol-induced blackout were frightened by the amnesia and decreased their intake of alcohol, those who failed to modify their drinking behaviour were likely to have further blackouts. This could be construed as a sign of alcoholism[15].

In the study of 123 college students, 38.6% of them experienced alcohol-induced blackout. Students with a blackout history expected more positive benefits from drinking, i.e. a higher positive alcohol expectancy[3]. In another study, those who had en bloc blackouts[22] and drank heavily[23] also had more positive alcohol expectancies. The limited recall of the negative experiences at the later stage of drinking may bias the outcome expectancies towards the positive effects at the initial stage. These may have important implications in determining the future drinking behaviour of an individual.

It is important for clinicians to inform their clients of the phenomenon and risk factors of alcohol-induced blackout to facilitate informed decisions about alcohol use, to address subject concerns over their drinking experiences, and to help motivate clients to change their drinking behavior. Concern about alcohol-induced cognitive impairment and blackout can motivate a drinker to seek professional advice and consultation and represents an opportunity for a clinician to harness this concern to facilitate a change in drinking behaviour[11].

Legal implications

Alcohol-related amnesia is a common claim of criminal defendants. In court, the generic understanding and use of the phrase "alcohol-induced blackout" are not sufficient but must follow rules of scientific evidence. In the evidence-based analysis of 26 empirical studies by Pressman and Caudill[24], there is no objective or scientific method to verify the presence of an alcoholic blackout while it is occurring or to confirm its presence retrospectively. Since only the short term memory, not other cognitive functions, such as planning, attention, or long term memory to form the criminal intent, is impaired, an alcohol-induced blackout does not negate mens rea of the criminal behaviour.

In addition, there is no direct correlation between alcohol-induced blackout and crime. The criminal defendant claiming alcohol-induced amnesia for the period of the alleged commission of a crime is therefore to be treated as if the amnesia is of any other type (e.g. traumatic or psychogenic) with regard to determination of fitness to stand trial or assume criminal responsibility[25].

Conclusion

Alcohol-induced blackout is a common phenomenon experienced by moderate and excessive drinkers. It is

important for clinicians to inform their clients of the phenomenon and the associated risks, and to motivate them to change their drinking behaviour.

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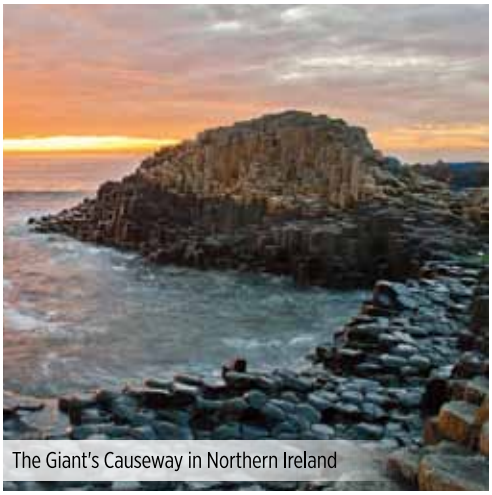
The Giant's Causeway in Northern Ireland

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Occupational Therapy Perspectives: Enhancing the Life Role of Substance Abusers

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Introduction

Hong Kong has long-adopted a multi-disciplinary approach to treatment and rehabilitation to cater for the divergent needs of substance abusers. The Substance Abuse Clinic (SAC) is a specialized service run by the seven service clusters of the Hospital Authority for those with substance abuse disorder and co-morbidity of psychiatric problems. A multi-disciplinary team of professionals including psychiatrists, nurses, clinical psychologists, social workers and occupational therapists collaborate to provide multifaceted services with their unique perspectives to best address the individual needs of substance abusers for abstinence and relapse prevention. Within the multi-disciplinary team, the occupational therapist is particularly positioned to assist people who are struggling to recover from substance abuse by coaching them to redesign their lifestyle, and to re-establish the role and identity most meaningful to them.

Occupational therapy perspectives

Hossack (1952) and Doniger (1953) suggested that balance of activity is the concern in substance abuse intervention. The issue raised by these two pioneers of occupational therapy in the field of substance abuse has remained a key component throughout subsequent literature. The practice guideline of occupational therapy for substance use disorder was published by the American Occupational Therapy Association (Stoffel & Moyers, 1997) to standardize the occupational therapy perspectives and role in a substance abuse service.

Occupation is a lifestyle structure, referring to performance in time and place, and everything that people do and experience in different contexts according to their own life role (Pierce, 2001). This is the central component of a persons' life, and the practice of occupational therapy working around it. An occupational therapist views health as not merely the absence of disease or maladaptive behavior, but a dynamic, functional state that enables an individual to perform their own daily occupations to a satisfying and meaningful level. An individual is viewed as healthy or functional when they have successful participation in their expected range of roles throughout life (Creek, 2003).

To define the complex of substance abusers from an occupational therapy perspective, the occupation and lifestyle of an active substance abuser is focused entirely on activities related to substance abuse, such as securing money for the substance, and obtaining and consuming the substance (Gutman, 2006). When commencing treatment, substance abusers experience gaps in their habits/daily routine that were formerly occupied by substance use, and the absence of a new habit can trigger relapse (Helbig & McKay, 2003; Martin et al., 2015). In both periods of pre- and post-treatment, abusers have lost their occupations, and even their function in the roles of their lives that they once considered meaningful. It is the focus of occupational therapy intervention to reconstruct and enhance the lost roles of the substance abuser without the addictive use and effect of substance.

Enhancing life role as an important outcome

Life role is the pacemaker of personal life. It represents a high level of organization and expectation of occupations, lifestyle, behavior as well as personal causation, including volition, value and identity (Kielhofner, & Burke, 1980). Life role is the drive for a person to do everything in his/her daily life. For the substance abuser in pre-contemplation, contemplation and preparation stages in Trans-theoretical Model (Prochaska & DiClemente, 1983), life role is the motivation to cease substance use once the discrepancy between their substance abuse behavior and personal life role has been acknowledged through motivational interviewing and coaching. A meaningful life role comprises an organized and constructive lifestyle. For substance abusers in action and maintenance stage, it is the crucial element for relapse prevention to minimize the risk factors of loneliness, emptiness, non-engaged and chaotic lifestyle (Moyers, 1992; Suvisaari et al., 2009). Therefore, enhancing life role is an important outcome throughout the continuum of changing behavior of substance abusers.

Substance abuse services in western countries identify life role fulfillment as a sign of recovery for substance abusers. The United Kingdom Drug Policy Commission (UKDPC, 2008) determined that the process of recovery from problematic substance use was characterized by voluntary control over substance use that maximized wellbeing and participation in the roles and responsibilities. Similarly, the Substance Abuse and Mental Health Service Administration (SAMHSA, 2015) suggested that service to promote recovery supports the individual to adopt a productive life role in the community and maintain abstinence. Recovery of substance abusers involved connecting with life role.

Occupational therapy in substance abuse service

Several occupational therapy interventions have been described that help recovering substance abusers achieve a life role.

The functional assessment is commonly implemented at the engagement stage since an occupational therapist is concerned that disruption in the area of cognition, self-care, work and leisure functioning is secondary to substance abuse, and how substance abuse can cause dysfunction in the life role is most important (Morgan, 1994). This is an important element to consider, and incorporate in motivational interviewing (Miller & Rollnick, 1991), to promote self-reflection, planning, action and support, and to boost motivation for change (Stoffel & Moyers, 2004). Once the motivation is firmly established, the intervention focus is shifted to strengthening of self-management and life skills to prepare substance abusers to stay clean. An occupational therapist works with substance abusers to teach them relaxation strategies, coping skills, and communication and assertiveness skills for relapse prevention. Substance abusers have reported this to be a positive approach (Bell et al., 2015).

Fostering engagement in meaningful occupations according to the specific life roles of substance abusers is constantly mentioned in occupational therapy literature, as it serves as the key to recovery of substance abusers (Stoftel & Moyers, 2004; Ozechowski & Liddle, 2000; Creek & Lougher, 2008). To reconstruct the behavior, habit, environmental context and lifestyle for re-establishing a meaningful life role, the occupational therapist coaches the substance abuser to engage in work and leisure activities. Vocational engagement is the predictive factor for recovery and resilience of substance abuse (Platt, 1995). It has been suggested that vocational assessment, counseling, competitive employment and continuing support with adaptation of the Individual Placement and Support Model would be beneficial for substance abusers (Magura, 2003). Once vocational activity has been transformed into an attainable reward, substance abusers are motivated to begin work (Silverman et al., 2002). Leisure activities play a vital role in relapse prevention strategies (Hodgson & Lloyd, 2001; Hodgson & Lloyd, 2002). During intervention, the occupational therapist facilitates substance abusers to experience leisure activity through goal setting, and the positive feedback with gains in feeling of satisfaction have been noted (Bell et al., 2015). This is particularly important when the worker role is ineffective (Super, 1990).

Clinical practice in the local setting

Occupational therapy has been involved in the substance abuse service at SACs in certain local settings, and identified enhancement of the life role of substance abusers as the theme of intervention. In recent years, several projects have been implemented in divergent service pathways including in-patient, ambulatory and community outreach programmes and achieved positive outcomes.

For in-patients, occupational therapists at the North District Hospital worked alongside the Department of Surgery, Integrated Ambulatory Care Center and Christian Chaplaincy to offer a Crisis Accommodation Program for ketamine abusers in 2012 - 2015. The multidisciplinary team provided 5 days of in-patient intervention. During this short period, occupational therapists conducted functional assessments of cognitive function, eye-hand-foot coordination, and hand dexterity with the aim of raising awareness of substance abusers of the discrepancy between current functional performance and personal life role, and providing motivation to get clean. The participants were then coached to formulate a plan for relapse prevention and life role re-establishment. 172 ketamine abusers completed the program. Motivation for treatment was significantly enhanced, and ketamine consumption, stress, depression and anxiety level were significantly reduced. A maintenance effect was also evident 13 weeks after program completion.

For ambulatory service, the Substance Abuse Assessment Unit and Occupational Therapy Department in Kwai Chung Hospital provided an ambulatory service with the aim of enhancing the life role of substance abusers. R3 Project (Refuse drug, Redesign lifestyle and Re-integrate into the community) was conducted in 2010 – 2012 for substance abusers and provided a day- and out-patient service. An occupational therapist provided intensive coaching and experiential learning for lifestyle redesign according to the life role of participants. Among the 101 recruited participants, functioning, lifestyle engagement and drug-related problems improved.

For a community outreach service, occupational therapists from Castle Park Hospital and Tuen Mun Substance Abuse Clinic implemented a 2-year project from 2012 – 2014, namely Handy Occupational Therapy Service. It was the pioneer community outreach vocational rehabilitation service for substance abusers. The service addressed the problem of high unemployment (45.9%) among substance abusers in Hong Kong, and provided a vocational service for substance abusers in the form of a Drug Treatment and Rehabilitation Center (DTRC), Counseling Center for Psychotropic Substance Abusers (CCPSA), as well as community support. 125 substance abusers were recruited at DTRC and CCPSA to take part in a motivational interview-based training protocol that included vocational assessment, vocational counseling for goal setting, modular-based training in work-related and life skills, and job coaching. More than 79% of participants showed an improvement in drug avoidance self-efficacy, quality of life and employment readiness. Employment rate among the 125 participants improved from 19.2% to 90.4%, and employment status was sustained for more than one month. This was evidence that occupational therapy can contribute to the establishment of a worker role for substance abusers.

Way Forward

Enhancing life role is the core component of occupational therapy, and there is evidence that it can help substance abusers abstain from the substance, prevent relapse, and live beyond the substance. During the recovery journey, it was evident that substance abusers had a level of cognitive impairment, consistent

with previous findings on cognitive dysfunction for substance abusers (Fals-Stewart & Lam, 2010; Rogers, & Robbins, 2001). This cognitive dysfunction can be a barrier to recovery and life role establishment, due to its correlation with poor treatment compliance (Bates et al., 2006), treatment effectiveness (Turner et al., 2009), maintenance of abstinence (Fals-Stewart & Lam, 2010), and coping skills (Cooney et al., 1991). Cognitive rehabilitation and computer-assisted training offer a promising means to ameliorate cognitive impairment in substance abusers (Fal-Stwart & Lucente, 1994; Gorhman & Fals-Stewart, 2004; Fals-Stewart & Lam, 2010). Local occupational therapists are now applying their clinical experience of cognitive rehabilitation for people with mental illness along with research evidence to pilot a computer-assisted cognitive rehabilitation programme for substance abusers. Local data gained from this study will help facilitate the recovery of substance abusers in future.

Conclusion

The occupational therapist's unique view of disability involves understanding how maladaptive behavior affects life role performance. The life role of substance abusers can be encouraged with the support of various services that help with abstinence, relapse prevention, and future recovery. Occupational therapy has a considerable role to play in the rehabilitation of substance abusers and can make a major contribution to substance abuse services.

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Dermatological Quiz

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Fig.1: Pigmented nodule on left buttock

This 61-year-old lady complained of a solitary and rapidly growing mass on her left buttock for around three to six months. She recalled no history of injury or precipitating cause. There was mild itchiness and occasionally mild tenderness. Physical examination revealed a 1 cm roundish nodule on the left upper buttock. Colour was heterogenous and the surface was smooth (Fig. 1). There was no ulcer or erosion on the nodule.

Questions:

1. What are the differential diagnoses of this skin lesion?
2. What investigations would you order?
3. How do you treat this patient?

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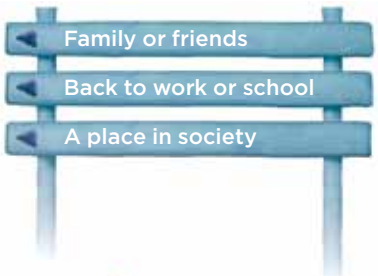

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ACTIVE INGREDIENT(S): Paliperidone palmitate. INDICATION(S): Acute and maintenance treatment of schizophrenia in adults. DOSAGE & ADMINISTRATION: I.M. use only. For patients naïve to oral paliperidone or oral or injectable risperidone, it is recommended to establish tolerability with oral paliperidone or oral risperidone prior to initiating treatment with INVEGA SUSTENNA. The recommended initiation dose is 150 mg on treatment day 1 and 100 mg one week later, both administered in the deltoid muscle. The recommended monthly maintenance dose is 75 mg; some patients may benefit from lower or higher maintenance doses within the recommended range of 25 to 150 mg based on individual patient tolerability and/or efficacy. Following the second dose, monthly maintenance doses can be administered in either the deltoid or gluteal muscle. Adjustment of the maintenance dose may be made monthly. CONTRAINDICATIONS: Known hypersensitivity to either paliperidone or risperidone, or to any of the excipients in the INVEGA SUSTENNA formulation. SPECIAL WARNINGS & PRECAUTIONS: INCREASED MORTALITY IN ELDERLY PATIENTS WITH DEMENTIA-RELATED PSYCHOSIS: INVEGA SUSTENNA is not approved for the treatment of patients with dementia-related psychosis. Cerebrovascular Adverse Events, Including Stroke, in Elderly Patients with Dementia-Related Psychosis: Increased incidence of cerebrovascular adverse events (cerebrovascular accidents and transient ischemic attacks, including fatalities). Neuroleptic Malignant Syndrome: It has been reported in association with antipsychotic drugs, including paliperidone. Manage with immediate discontinuation of antipsychotic drug and other drugs not essential to concurrent therapy; intensive symptomatic treatment and medical monitoring; treatment of any concomitant serious medical problems for which specific treatments are available. QT Prolongation: Avoid use with drugs that prolong QTc interval. Also avoided in patients with congenital long QT syndrome and in patients with a history of cardiac arrhythmias. Tardive Dyskinesia: Discontinue drug if clinically appropriate. Metabolic Changes: Atypical antipsychotic drugs have been associated with metabolic changes that may increase cardiovascular/cerebrovascular risk. Hyperprolactinemia: Prolactin elevations occur and persist during chronic administration. Orthostatic Hypotension and Syncope: Use with caution in patients with known cardiovascular or cerebrovascular disease and patients predisposed to hypotension. Monitoring of orthostatic vital signs should be considered in patients who are vulnerable to hypotension. Leukopenia, Neutropenia, and Agranulocytosis: Monitor complete blood count frequently during the first few months in patients with a history of a clinically significant low white blood cell count (WBC) or a drug-induced leukopenia/neutropenia. Consider discontinuation if clinically significant decline in WBC in the absence of other causative factors. Patients with clinically significant neutropenia should be carefully monitored for fever or other symptoms or signs of infection and treated promptly. Discontinue INVEGA SUSTENNA in patients with severe neutropenia. Potential for Cognitive and Motor Impairment: Use caution when performing activities requiring mental alertness. Seizures: Use cautiously in patients with a history of seizures or with conditions that lower the seizure threshold. Dysphagia: Use cautiously in patients at risk for aspiration pneumonia. Suicide: close supervision of high-risk patients should accompany drug therapy. Body Temperature Regulation: Appropriate care to patients experiencing conditions which may contribute to an elevation in core body temperature. Intraoperative Floppy in Iris Syndrome: Current or past use of drug should be made known to the ophthalmic surgeon in advance of surgery. SIDE EFFECTS: Injection site reactions, somnolence/sedation, dizziness, akathisia, and extrapyramidal disorder. Refer to full prescribing information for other side effects. PREGNANCY & LACTATION: Pregnancy Category C. Women receiving INVEGA SUSTENNA should not breast feed infant. INTERACTIONS: Centrally-acting drugs and alcohol. Levodopa and other dopamine agonists. Drugs that may cause orthostatic hypotension. Carbamazepine.

PLEASE REFER TO FULL PRESCRIBING INFORMATION BEFORE PRESCRIBING. (version Aug, 2013)

References:

1. Emsley R, Medori R, Koen L et al. Long-acting injectable Risperidone in the treatment of subjects with recent-onset psychosis. J Clin Psychopharmacol 2008;28(2):210-213
2. Invega® Sustenna® Package insert (version Aug, 2013)
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4. Zang F, Si T., Chiou CF et al. Efficacy, safety, and impact on hospitalizations of paliperidone palmitate in recent-onset schizophrenia. Neuropsychiatr Dis Treat 2015;11: 657-668
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MKT-SUS-HK-0015

European Trip

Dr Isaac YIP

MBBS(HK), MResMed(HKU), FHKAM(Psychiatry), FHKCPsych

Associate Consultant, Kwai Chung Hospital

Dr Isaac YIP

It is my pleasure to write this column for the first time. I will describe a European trip that I made 2.5 years ago. The sixteen-day trip started in Berlin, Germany, then took us through Prague in the Czech Republic, Bratislava in Slovakia, Vienna and Salzburg in Austria, and finally back to Germany to Wurzburg, Rothenburg ob der Tauber, and Munich. What was remarkable was not the number of cities we visited, but the experience of the "once-in-a-century" (if not more) flooding in Central Europe.

Our first stop was Berlin. For many, Berlin is probably most famous for the Berlin Wall, and the history of East and West Germany. The Berlin wall was covered in graffiti by local and international artists and expressed their views on politics, freedom, separation and reunification. We also visited the DDR museum that offered a realistic and multi-dimensional exhibition of everyday life in the former East Germany – including a display of a living room and a video of school life, TV programs and film clips, original cinema seats, and numerous everyday items. The exhibits also provided historical details, for example information about the political system, a model that demonstrated how the Berlin Wall and various components of the "Death Strip" prevented East Germans from defecting to the West, and stories about various creative ways by which people tried to escape to the West.

Leaving Berlin, our journey by train to Prague took 4.5 hours. Compared with Berlin and its heavy and historical atmosphere, Prague is a much lighter place with many tourist attractions. The Prague Castle dates back to the 9th century, and impressed us with its size, representation of various architectural styles, and functional diversity (including a Cathedral, residence of the President of Czech Republic, various museums and galleries). Another attraction was the Old Town Square and the Old Town Hall with it Horologe – a medieval astronomical clock that still works. From the Old Town Hall, one can enjoy a panoramic view of the surrounding area with its magnificent buildings, numerous orange roofs of houses so characteristic of Prague, and beautiful scenery.

We travelled by bus from Prague to our next stop – Bratislava, capital of Slovakia, at a considerably lower cost than the train. Although Czech and Slovakia used to be one country (the Czechoslovakia), the atmosphere of Prague and Bratislava was quite different. Despite the very central location of our hotel, there appeared to be few pedestrians along the main street. The place felt a bit old and cold, and there were less street lights than expected. We planned to explore the main street after dinner (there was a bar where traditional Slovakian music was being performed) but finally gave up because we did not feel safe. Nonetheless the hotel, food and drinks were quite inexpensive in Bratislava: a very decent hotel room in the city center cost around 80 Euro, and a glass of wine in a decent restaurant cost around 3 Euro.

After visiting the Bratislava Castle, we left for Vienna by train (about 1 hour). We hoped to be able to see the Vienna State Opera without prior online reservation – perhaps unsurprisingly we could not. But we did visit Hofburg Palace – the former imperial palace in the center of Vienna, well worth the visit.

After enjoying the nice weather in Vienna, we travelled by train to Salzburg, known by most as the birthplace of Mozart, and where the classic musical "The Sound of Music" was filmed. We took the funicular railway to visit the Hohensalzburg Fortress, and booked dinner and a Mozart concert there in the evening. Disappointingly, both the dinner and concert were rather commercial and targeted mostly tourists. It rained all day and when we returned to our hotel we heard news of the record breaking rainfall and flooding in central Europe. Worse still, the train from Salzburg to Munich (our next stop) was suspended as the tracks were flooded. We arrived at the train station early next morning hoping for a contingency plan. Although there were no English public announcements, repeated enquiries established that we could take an alternative route (transiting at a small and distant station) to Munich. We managed to squeeze into the train together with many other tourists and finally arrived in Munich to transit to Wurzburg, without any delay.

Wurzburg is a beautiful city in Central Germany (especially after the successful escape from Salzburg). located on both sides of the River Main with a connecting bridge. Together with many others, we enjoyed several glasses of wine in the wine bars located near the bridge and enjoyed walking on the bridge and taking pictures. In the evening, we went to the Marktplatz that was busy with locals and tourists enjoying the German food, beer and wine sold at the many different stalls.

As I am running out of space here, I would just like to highlight a popular spot in Fussen around Munich – the Hohenschwangau and Neuschwanstein Castle. The Neuschwanstein Castle was a palace commissioned by King Ludwig II of Bavaria as a retreat, but also a project to realize his enthusiasm for the opera of Richard Wagner. Beyond the magnificent castle, the story of King Ludwig II, the proclamation that he was mentally ill and unfit to rule the country, and the mysterious death of him and his psychiatrist in the lake were also remarkable!

I could go on and on about this amazing sixteen-day trip. For those who are interested, I would definitely recommend that you go and see it for yourself!

Certificate Course for All Healthcare Professionals and General Public | Course No. C280 | CME/CNE Course

Certificate Course on
Paediatric Surgery 2016

Objectives:

Surgical management plays an important role in the management of common childhood conditions. This course is designed to provide an overview of surgical intervention in common paediatric conditions. It provides an update in management in common paediatric surgical and urological conditions

Jointly organised by

The Federation of Medical Societies of Hong Kong

The Hong Kong Society of Paediatric Surgery

Date	Topics	Speakers
10 Mar	1) Urinary incontinence in children	Dr Kenneth Chung Associate Consultant Department of Surgery, Queen Elizabeth Hospital
	2) Constipation and fecal incontinence	Dr Nicholas Chao Consultant Department of Surgery, United Christian Hospital
24 Mar	1) Update in management in cleft lip and palate	Dr Kelvin Liu Consultant Department of Surgery, United Christian Hospital
	2) Common lesions of head and neck in children	Dr Paula Tang Associate Consultant Department of Surgery, Queen Elizabeth Hospital
31 Mar	1) Current management in vascular anomalies in children	Dr Clarence Liu Specialist in Paediatric Surgery United Christian Hospital
	2) Common surgical emergency in children	Dr Michael Leung Consultant Department of Surgery, Queen Elizabeth Hospital
7 Apr	1) Surgical oncology	Dr Jennifer Mou Associate Consultant Department of Surgery, Prince of Wales Hospital
	2) Thoracic surgical condition in children	Dr Kenneth Wong Clinical Associate Professor Department of Surgery, The University of Hong Kong
14 Apr	1) Common urological problem I - penile & scrotal	Dr Kristine Pang Specialist in Paediatric Surgery Prince of Wales Hospital
	2) Common urological problem II - bladder & kidneys	Dr Ivy Chan Associate Consultant Department of Surgery, Queen Mary Hospital
21 Apr	1) Neonatal intestinal obstruction	Dr Edwin Chan Associate Consultant Department of Surgery, Prince of Wales Hospital
	2) Surgical jaundice	Dr Patrick Chung Clinical Assistant Professor Department of Surgery, The University of Hong Kong

Date : 10, 24, 31 March 2016 and 7, 14, 21 April 2016 (Every Thursday, skip 17 March 2016)
Time : 7:00 p.m. – 8:30 p.m.
Venue : Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Language Media : Cantonese (Supplemented with English)
Course Fee : HK$750 (6 sessions)
Certificate : Awarded to participants with a minimum attendance of 70%
Enquiry : The Secretariat of The Federation of Medical Societies of Hong Kong
Tel.: 2527 8898 Fax: 2865 0345 Email: info@fmshk.org

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

Medical Diary of March

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		★ HKMA Yau Tsim Mong Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice? ★ HKMA Kowloon West Community Network - Update on the Management of Chronic Hepatitis B ★ HKMA Council Meeting ★ FMSHK Officers' Meeting **1**	★ HKMA Central, Western & Southern Community Network - Common Eye Conditions: A Problematic Approach & Ocular Emergency Requires Urgent Referral **2**	★ HKMA Hong Kong East Community Network - Shingles Prevention: A Geriatrician's View ★ HKMA New Territories West Community Network - Rosacea and Related Dermatosis **3**	**4**	**5**
6	★ Renal Artery Aneurysm: The Contemporary Approach **7**	**8**	★ Hong Kong Neurosurgical Society Monthly Academic Meeting –Familial Brain Tumours - An Update **9**	★ HKMA Structured CME Programme with HKS&H Session 2: Current Management of Hyperhidrosis & Osmidrosis **10**	**11**	★ 11th International Symposium on Healthy Aging "Science and Aging: An Era of Discovery" ★ CME Lecture - Refresher Course for Health Care Providers 2015/2016 **12**
★ 11th International Symposium on Healthy Aging "Science and Aging: An Era of Discovery" ★ Open Lecture for Public: Managing Sleep Apnea: Medical and Surgical Perspectives ★ 1st HKMAPS Seasonal Photo Competition 2016 **13**	**14**	★ HKMA Kowloon West Community Network - Knee Injury: ACL Deficiency **15**	★ HKMA Central, Western & Southern Community Network - Psoriatic Arthritis: Is it a Skin or Joint Disease? **16**	★ The Economist Events' Health Care Forum: War on Cancer ★ HKMA Hong Kong East Community Network - What is New in Diabetic Nephropathy anagement? ★ FMSHK Executive Committee Meeting **17**	★ HKMA Yau Tsim Mong Community Network - What's New in COPD Management? **18**	**19**
★ HKMA Football Day 2016 **20**	**21**	**22**	**23**	**24**	**25**	**26**
27	**28**	**29**	**30**	**31**		

Date / Time	Function	Enquiry / Remarks
1 TUE 1:00 PM	**HKMA Yau Tsim Mong Community Network - Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?** Organiser:HKMA Yau Tsim Mong Community Network & Primary Care Office of the Department of Health; Chairman:Dr. LAM Siu Keung; Speaker: Dr. FOK Oi Ling, Annie; Venue: Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA Kowloon West Community Network - Update on the Management of Chronic Hepatitis B** Organiser:HKMA Kowloon West Community Network; Chairman:Dr. CHAN Siu Man, Bernard; Speaker: Dr. FUNG Tang Tat, Konrad; Venue: Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
8:00 PM	**HKMA Council Meeting** Organiser:The Hong Kong Medical Association; Chairman:Dr. SHIH Tai Cho, Louis; Venue:HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
	FMSHK Officers' Meeting Organiser:The Federation of Medical Societies of Hong Kong; Venue:Gallop, 2/F, Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
2 WED 1:00 PM	**HKMA Central, Western & Southern Community Network - Common Eye Conditions: A Problematic Approach & Ocular Emergency Requires Urgent Referral** Organiser:HKMA Central, Western & Southern Community Network; Chairman:Dr. YIK Ping Yin; Speaker: Dr. LIANG Chan Chung, Benedict; Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
3 THU 1:00 PM	**HKMA Hong Kong East Community Network - Shingles Prevention: A Geriatrician's View** Organiser:HKMA Hong Kong East Community Network; Chairman:Dr. KONG Wing Ming; Speaker: Dr. DAI Lok Kwan, David; 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
	HKMA New Territories West Community Network - Rosacea and Related Dermatosis Organiser:HKMA New Territories West Community Network; Chairman:Dr. MOK Kwan Yeung, Matthew; Speaker: Dr. CHAN Kam Tim, Michael; Venue: Pearl Ocean, 1/F., Gold Coast Yacht and Country Club, 1 Castle Peak Road, Castle Peak Bay, Hong Kong	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
7 MON 7:30 PM	**Renal Artery Aneurysm: The Contemporary Approach** Organiser: Hong Kong Urological Association; Chairman: Dr CHAN Chun Ki, PMH; Speaker: Dr NG Chi Yuen Alex, PMH; Venue: Multi- disciplinary Simulation and Skills Centre, 4/F, Block F, QEH	Ms. Tammy HUNG Tel: 9609 6064 1 CME Point
9 WED 7:30 AM	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Familial Brain Tumours - An Update** Organiser: Hong Kong Neurosurgical Society; Chairman: Dr CHAN Tat Ming, Danny; Speaker: Dr ZHUANG Tin Fong ; Venue: M Block Ground Floor Lecture Theatre, Queen Elizabeth Hospital	Dr. LEE Wing Yan, Michael Tel: 2595 6456 1.5 CME Point
10 THU 2:00 PM	**HKMA Structured CME Programme with HKS&H Session 2: Current Management of Hyperhidrosis & Osmidrosis** Organiser:The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital; Speaker: Dr. Kwan Kin Hung, Vincent; 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
12 SAT 9:00 AM *(13)*	**11th International Symposium on Healthy Aging "Science and Aging: An Era of Discovery"** Organiser:Research Centre of Heart, Brain, Hormone & Healthy Aging, Li Ka Shing Faculty of Medicine, HKU; Chairmen:Dr Tommy T CHEUNG; Dr George L Tipoe; Venue: 3/F Ballroom. Sheraton Hong Kong Hotel & Towers	Ms. Phoebe CHOW Tel: 3918 9866
2:15 PM	**CME Lecture - Refresher Course for Health Care Providers 2015/2016** Organiser:The Hong Kong Medical Association; Speaker: Dr. Lam Wing Wo; Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Point
13 SUN 11:00 AM	**Open Lecture for Public: Managing Sleep Apnea: Medical and Surgical Perspectives** Organiser:Hong Kong Society of Sleep Medicine; Speaker: Dr. Birgitta WONG (ENT surgeon); Dr. Fan Hon CHEUNG (Medical Physician); Venue: Activity Room 1, Hong Kong Central Library, Causeway Bay	Dr. K.W. TO Tel: 2632 2785
2:00 PM	**1st HKMAPS Seasonal Photo Competition 2016** Organiser:The Hong Kong Medical Association; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Heiman CHAN Tel: 2527 8285
15 TUE 1:00 PM	**HKMA Kowloon West Community Network - Knee Injury: ACL Deficiency** Organiser:HKMA Kowloon West Community Network; Chairman:Dr. CHAN Ching Pong; Speaker: Dr. KWOK Ken, Grace; Venue: Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
16 WED 1:00 PM	**HKMA Central, Western & Southern Community Network - Psoriatic Arthritis: Is it a Skin or Joint Disease?** Organiser:HKMA Central, Western & Southern Community Network; Chairman:Dr. TSANG Chun Au; Speaker:Dr. YU Ka Lung, Carrel; Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Ms. Hana YEUNG Tel: 2527 8285 1 CME Point
17 THU 8:00 AM	**The Economist Events' Health Care Forum: War on Cancer** Organiser:The Economist Events; Speakers:Gerardo V. BAYUGO, Assistant Secretary of Health, Office for Technical Services, Department of Health, Republic of the Philippines; CHIOU Shu-Ti, Director-general, Health Promotion Administration, Ministry of Health and Welfare, Taiwan; Venue: The Ritz-Carlton, Millenia Singapore	Ms. Gloria WONG Tel: 2585 3839 Fax: 2802 7007
1:00 PM	**HKMA Hong Kong East Community Network - What is New in Diabetic Nephropathy Management?** Organiser:HKMA Hong Kong East Community Network; Chairman:Dr. NGAN Sze Yuen, Silas; Speaker: Dr. HO Chung Ping, MH, JP; 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
7:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898

Date / Time	Function	Enquiry / Remarks
18 *FRI* 1:00 PM	**HKMA Yau Tsim Mong Community Network - What's New in COPD Management?** Organiser:HKMA Yau Tsim Mong Community Network; Chairman:Dr. HO Fung; Speaker: Dr. TAI Kian Bun; Venue: Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME Point
20 *SUN* 12:00 PM	**HKMA Football Day 2016** Organiser:The Hong Kong Medical Association; Venue: Stanley Ho Sports Centre, 10 Sha Wan Drive, The University of Hong Kong	Mr. Ian KWA Tel: 2527 8285

Upcoming Meeting

13/4/2016	**Better LUTS Management, Better Day for Your Patients** Organiser: HKMA Central, Western & Southern Community Network; Speaker: Dr. Yip Wai Chun Andrew (Urologists); Venue: HKMA Central Premises	Ms. Hana YEUNG 2527 8285 1 CME Point

FMSHK Medical Diary Article on Expressive Art Therapy

"Expressing stories from Children's heart through arts" is an Expressive Arts program for grieving and bereaved children. It is sponsored by the HKFMS Foundation to provide a therapeutic service to children and families in need. Through creative interventions guided by expressive arts therapist, Ms. Snowy Lam, participants journey through various art-related activities. It aims to improve the functioning of the bereaved, as well as to strengthen coping abilities through the artmaking process and the art products.

The use of the arts in psychotherapy is a burgeoning area of interest, particularly in the field of bereavement. Grieving and bereaved children experience intense and complex emotions that may be difficult to verbally express or present in actions. By using an art framework and a coherent structure, a conceptual and relational scaffold is created for an artistically inclined grief therapy. Art activities provide an alternative way for children to express deep buried emotions, and also help them to experience and reconfirm the feeling of being loved, cared for, supported, and accompanied. In this Expressive Arts program children meet and get to know others with similar experiences and situations. This helps them by providing emotional and cognitive outlets to express themselves in an emotionally-safe and non-judgmental environment. It also provides an opportunity for the children to approach, construe, and manage their emotions, while building their own repertoire of coping skills through creative means.

With the adoption of a wide range of artistic modalities featuring music, visual arts, play and multi-modal practices, children **enjoy** the art making process, learn to **share** art materials and experience, **accept** differences among themselves, **express** their thoughts and feelings freely, **communicate** and **interact** with others by metaphor, imagination and use of arts. More importantly the children learn to seek help from one another, while parents and guardians better **understand** the inner world of their child. For instance, children manage to voice and express feelings through **storytelling**. This program has been highly praised and welcomed.

"The guardians of those participants gave positive feedbacks to this program, including improved parent-child relationship through arts media, finding a common interest in arts that facilitated communication, and enjoying the opportunity of experiencing expressive arts therapy.

The therapeutic interventions light up hope and activate inner resources for participants to move forward. They hope to explore more creative arts related activities and gain more benefits from the arts healing process in the future."

Public talk on Psychotic Disorders/Schizophrenia

On 12 December 2015, a public talk on Psychotic Disorders/Schizophrenia was held at the Lecture Hall. This event organized by the FMSHK Office successfully attracted many attendees, especially whose family members or friends had suffered from such problems. The Federation was pleased to have **Dr Gregory MAK**, Fellow of the Hong Kong College of Psychiatrists and **Ms. Zanonia CHIU**, clinical psychologist, as the speakers. The lectures gave an overview of Psychotic Disorders and Schizophrenia, described psychotic symptoms and introduced to the audience the treatments available to patients.

Healthcare Insurance Luncheon

On 30 January 2016, a luncheon on Healthcare Insurance「健康保障與理財之道」was held in the Penthouse at the Head Office of Hang Seng Bank. It was well attended by around 50 members from FMSHK member societies. We were privileged to have Mr Mark WAN & Ms Cheryl HO from the Hang Seng Bank investment team to share their insights on wealth management and Ms Veronica WONG from BUPA to deliver the talk on Health Insurance. We wish to express our sincere gratitude to Hang Sang Bank for their generosity in sponsoring the event. The Federation is planning more events in the future on topics of interest to our members.

Certificate Course for Medical, Nursing & Allied Health Professionals • CME/CNE Course • Course No. C279

Certificate Course on

PET-CT Imaging in Daily Clinical Practice

Jointly organised by

The Federation of Medical Societies of Hong Kong

Hong Kong Society of Nuclear Medicine and Molecular Imaging

Objectives:

PET-CT is one of the most widely used imaging modalities especially in clinical oncology. It is also useful in assessing various myocardial and neuro-psychiatric diseases. The course will give an overview of PET-CT status in Hong Kong, its basic principles, clinical application and recent advances. The attendees will learn how to apply PET-CT findings in daily clinical practice.

Date	Topics	Speakers
1 Mar	Overview of PET-CT	Dr. LOK Chiu Ming Director Nuclear Medicine & PET Centre Hong Kong Baptist Hospital
8 Mar	PET-CT in Oncology I: Head & Neck Malignancy	Dr. CHOI Pak Tat, Frankie Consultant i/c Department of Nuclear Medicine Pamela Youde Nethersole Eastern Hospital
15 Mar	PET-CT in Dementia and Parkinsonism	Dr. Thomas KC CHENG Specialist in Nuclear Medicine Honorary Consultant in Nuclear Medicine Department of Nuclear Medicine & PET Hong Kong Sanatorium & Hospital
22 Mar	PET-CT in Oncology III: Abdominal Malignancy	Dr. Benz CP WONG Associate Consultant Nuclear Medicine Unit Queen Elizabeth Hospital
29 Mar	PET-CT in Oncology II: Thoracic Malignancy	Dr. AU YONG Ting Kun Consultant Nuclear Medicine Unit Queen Elizabeth Hospital
5 Apr	PET-CT in Lymphoma	Dr. WONG Kwong Kuen Consultant Nuclear Medicine & PET Centre Hong Kong Baptist Hospital
	PET-CT in Cardiovascular Disease	Dr. John KUNG Associate Consultant Nuclear Medicine Unit Queen Elizabeth Hospital

Date : March 1, 8, 15, 22, 29 and 5 April 2016 (Every Tuesday)
Time : 7:00 p.m. – 8:30 p.m.
Venue : Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Language Media : Cantonese (Supplemented with English)
Course Fee : HK$750 (6 sessions)
Certificate : Awarded to participants with a minimum attendance of 70%
Enquiry : The Secretariat of The Federation of Medical Societies of Hong Kong
Tel.: 2527 8898 Fax: 2865 0345 Email: info@fmshk.org

CME / CNE / CPD Accreditation in application
Application form can be downloaded from website: http://www.fmshk.org

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

(Effective from October 2009)

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

Classified Advertisement

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

Answers to Dermatological Quiz

Answer:

1. Malignant melanoma, pigmented basal cell carcinoma, secondary cutaneous metastasis, dysplastic naevus, blue naevus, squamous cell carcinoma, Spitz naevus, pigmented spindle cell tumour are possible differential diagnoses.

   There are numerous differential diagnoses but in light of the rapid growth the most likely or most important that needs to be excluded is malignant melanoma. The size is greater than 6mm. The colour is varied and appears to be spreading out from the edge. The ABCD mnemonic for malignant melanoma is fulfilled: A – Asymmetry / B – Border irregularity / C – Colour variation / D – Diameter >6mm.

2. Skin biopsy is required. Although no single histological feature is pathognomonic for melanoma, there are a few characteristic features such as cytological atypia with enlarged cells with numerous mitotic figures. A pagetoid growth with upward growth of the melanocytes so that they are not confined to the basal layer is commonly found. Immunohistochemical stains such as S-100 and homatropine methylbromide (HMB 45) can help to differentiate melanoma from other lesions.

3. Since the histological report of this patient showed the Breslow thickness to exceed 5mm, a surgical resection margin greater than 2 cm with sentinel lymph node biopsy and/or elective lymph node dissection should be offered for early stage disease. For advanced disease, chemotherapy should be considered. Biologic therapy can also be used in melanoma with BRAF mutation. Around 60% of melanoma have BRAF mutation and vemurafenib, dabrafenib, trametinib are biologics that have all been recently approved by the FDA for treatment of unresectable or metastatic BRAF mutation melanoma.

Dr. Chi-keung KWAN
MBBS(HK), MRCP(UK), FHKCP, FHKAM(Medicine)
Specialist in Dermatology and Venereology

5-HT NA

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Milnacipran

Peace of mind for your practice

Presentation: Milnacipran oral capsules. **Indications:** Treatment of major (i.e. characterised) depressive episodes in adults. Dosage: 50mg twice daily. **Dosage** should be reduced to 25mg twice daily in patients with creatinine clearance between 30ml/min and 60ml/min. Dosage should further be reduced to 25mg daily in patients with creatinine clearance below 30ml/min. **Contraindications:** Hypersensitivity to milnacipran or excipients. Concomitant use with non-selective MAO inhibitors, B-selective MAO inhibitors, digitalis, 5-HT1 receptor agonists (triptans), parenteral epinephrine or norephinephrine, clonidine, A-selective MAO inhibitors. Uncontrolled hypertension. Severe or unstable coronary heart disease. Lactation. **Precautions**: Patients with a history of suicide-related events. Children below 18 years of age. Renal failure. Patients with an obstruction to emptying the bladder. Hypertension or cardiac disease. Angle-closure glaucoma. Epilepsy. Patients taking diuretics or any other therapy known to cause hyponatraemia, and in cirrhosis or undernourished patients. Patients receiving concomitant treatment with oral anticoagulants, medicines acting on the platelet function such as NSAIDs and aspirin, or other medicinal products likely to increase the risk of bleeding. **Interactions:** Linezolid, alcohol, non-selective MAO inhibitors, MAO-A and MAO-B inhibitors, 5HT1D agonists, digitalis, adrenaline, noradrenaline, clonidine, lithium. **Undesirable effects:** Agitation, anxiety, depression, eating disorder, sleep disorder, suicidal behavior, headache, migraine, tremor, dizziness, dysaesthesia, somnolence, tachycardia, palpitations, hot flushes, hypertension, nausea, constipation, diarrhoea, abdominal pain, dyspepsia, vomiting, dry mouth, pruritis, rash, hyperhidrosis, musculoskeletal pain, dysuria, pollakiuria, ejaculation disorder, erectile dysfunction, testicular pain, fatigue. *For uncommon, rare and very rare undesirable effects, please refer to the full prescribing information.* **Preparation:** 25mg x 28's; 50mg x 28's. ***Full prescribing information is available upon request.***

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SANOFI

FINDING A NEW EQUILIBRIUM
IN MAJOR DEPRESSIVE DISORDER

Pristiq® 倍思樂
desvenlafaxine

- Recommended 1st-line treatment for MDD[1]
- Low potential for CYP2D6-mediated drug interactions[2-4]
- Discontinuations due to adverse events similar to placebo[5]
- Safety profile comparable to placebo[5-7]
- Long-term use significantly lowers rate of relapse compared to placebo[6]
- Improves well-being and functioning[8-9]

References 1. Law R, et al. Canadian Network for Mood and Anxiety Treatments (CANMAT) clinical guidelines for the management of major depressive disorder in adults. III. Pharmacotherapy. J Affect Disord 2009;117(Suppl 1):S26-43. 2. Preskorn S, et al. Comparison of the pharmacokinetics of venlafaxine extended release and desvenlafaxine in extensive and poor cytochrome P450 2D6 metabolizers. J Clin Psychopharmacol 2009;29:39-43. 3. Nichols AI, et al. Pharmacokinetics of venlafaxine extended release 75 mg and desvenlafaxine 50 mg in healthy CYP2D6 extensive and poor metabolizers: a randomized, open-label, two-period, parallel-group, crossover study. Clin Drug Investig 2011;31:155-167. 4. Preskorn SH, et al. Effect of desvenlafaxine on the cytochrome P450 2D6 enzyme system. J Psychiatr Pract 2008;14:368-378. 5. Pristiq® (desvenlafaxine) Prescribing Information. Pfizer Corporation Hong Kong Limited: version January 2011. 6. Rosenthal J, et al. Efficacy and safety of desvenlafaxine 50 mg/day for prevention of relapse in adult outpatients treated for major depressive disorder. Presented at: The 165th Annual Meeting of the American Psychiatric Association (APA), 5–9 May 2012, Philadelphia, USA. 7. Clayton A, et al. An evaluation of sexual function in employed outpatients with major depressive disorder treated with desvenlafaxine 50 mg or placebo. J Sex Med 2013;10:768–776. 8. Boyer P, et al. Efficacy, safety, and tolerability of fixed-dose venlafaxine 50 and 100 mg/day for major depressive disorder in a placebo-controlled trial. Int Clin Psychopharmacol 2008;23:243-253. 9. Soares CN, et al. Assessing the efficacy of desvenlafaxine for improving functioning and well-being outcome measures in patients with major depressive disorder: a pooled analysis of 9 double-blind, placebo-controlled, 8-week clinical trials. J Clin Psychiatry 2009;70:1365-1371

PRISTIQ® ABBREVIATED PACKAGE INSERT

TRADE NAME: PRISTIQ® **PRESENTATION:** 50 mg, light pink, square (pyramid-one sided) tablet debossed with "W" (over) "50" on the flat side **INDICATIONS:** Treatment of major depressive disorder (MDD) **DOSAGE & ADMINISTRATION:** 50 mg once daily at approximately the same time, with or without food. The recommended dose in patients with severe renal impairment or end-stage renal disease (ESRD) is 50 mg every other day. Supplemental doses should not be given to patients after dialysis. **CONTRAINDICATIONS:** Hypersensitivity to desvenlafaxine succinate, venlafaxine hydrochloride or to any excipients in the Pristiq formulation. Must not be used concomitantly in patients taking monoamine oxidase inhibitor (MAOI) or in patients who have taken MAOIs within the preceding 14 days. **WARNINGS & PRECAUTIONS:** All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening, suicidality, and unusual changes in behaviour, especially during the initial few months of a course of drug therapy, or at times of dose changes, either increases or decreases. Not approved for use in treating bipolar depression; Serotonin syndrome or Neuroleptic Malignant Syndrome-like reactions; Activation of mania/hypomania; Elevated blood pressure & abnormal bleeding; Serum cholesterol & triglyceride elevation; Narrow-angle glaucoma; Renal impairment; Seizure; Hyponatremia; Interstitial lung disease & Eosinophilic pneumonia; New symptoms and serious discontinuation symptoms were reported in discontinuation of treatment; Caution is advised to patients with cardiovascular, cerebrovascular, or lipid metabolism disorders; Co-administration of drugs containing desvenlafaxine & venlafaxine is not recommended. **INTERACTIONS:** Risk in combination with other CNS-active drugs; MAOI; Serotonergic drugs; Drugs that interfere with hemostasis; Alcohol; Concomitant use with potent inhibitors of CYP3A4 may result in higher concentrations of Pristiq; Minimal inhibitory effect of desvenlafaxine on CYP2D6. **PREGNANCY AND LACTATION:** Pregnancy Category C. Carefully consider the potential risks and benefits of treatment when treating a pregnant woman during pregnancy especially in the third trimester, labor and delivery. Only breastfeed if the expected benefits outweigh any possible risk as desvenlafaxine is excreted in human milk. **SIDE EFFECTS:** Most commonly observed adverse reactions in short-term fixed-dose studies were: nausea, dizziness, insomnia, hyperhidrosis, constipation, somnolence, decreased appetite, anxiety, and specific male sexual function disorders. Hypersensitivity, effects on blood pressure, abnormal bleeding, mydriasis, hypomania & mania, serum cholesterol & triglyceride elevation, and seizure were also reported. **DRUG ABUSE AND DEPENDENCE:** Not systematically studied in preclinical/clinical studies for its potential for abuse. Physicians should carefully evaluate patients for a history of drug abuse and follow such patients closely, observing them for signs of misuse or abuse of Pristiq. Limited clinical experience with desvenlafaxine succinate overdose in humans. Reference: HK PI (Version Date JAN2011) Date of preparation: JUL2012 Identifier number: PRIS0712

FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.

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