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For the product’s safety, contraindications and side effects and toxic hazards, please refer to the package insert. Further information is available upon request.
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## The Cover Shot

**The happiness of the Ferris wheel 幸福摩天輪**

**Ferris wheel** is Hong Kong’s iconic tourist attraction. Each cabinet of this Ferris wheel may hold an individual, a family, or a group of friends. As a whole, the Ferris wheel represents the Hong Kong community.

Nowadays, being a mental health worker is akin to being a maintenance worker of a Ferris wheel. We need to broaden our horizons. Not only do we need to take care of the mental wellbeing of individuals, we should also pay attention to the mental health of the entire Hong Kong society. Mental health workers are not just limited to psychiatrists, who count on collaborative support of other medical professionals as well as allied health professionals. May we join hands to promote the mental health of Hong Kong and may our Ferris wheel keep wheeling bringing to us hope and happiness!

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**Dr Ivan WC MAK**
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In 2017, the World Psychiatric Association (WPA) and the Lancet Psychiatry have commissioned a team of medical professionals, researchers, and service users around the globe to draft a new commission on the future of psychiatry. It is believed that in the first quarter of this century, psychiatry is at the cusp of major changes. In this commission, several priority areas in the 21st century in the field of psychiatry have been identified and addressed. Among them, the importance of knowledge on the anatomy and physiology of the brain as it relates to complex behaviours, thoughts and emotions and more importantly, the effect of social factors on these processes, have been identified as being essential in understanding the pathophysiology of psychiatric disorders. Further advances in neuroscience would likely transform the way psychiatric disorders are being diagnosed and treated.

In this commission, the role of psychiatrists in society has also been stressed, particularly in the way of working with the communities. These could involve general mental health promotion and prevention activities, or focus on particular groups at risk. Digital psychiatry is another priority area that has been addressed in the commission. Given the exponential pace of technological advances, the commission would offer immense potential for radical changes in terms of service delivery as well as the development of new alternatives for psychiatric treatment.

In the light of these streams of ideas, I treasure this opportunity being offered as the issue editor in making this issue of Medical Diary a platform for the psychiatric specialty in various horizons including medical science, treatment and societal aspects. I have the honour to invite a few researchers and clinicians to contribute their expert and invaluable knowledge and experiences over these areas.

I have invited Professor Michael Wong, who has been running a territory-wide specialised clinic on neuropsychiatric disorders, to give us an overview in the concepts, history and development, as well as the development of new alternatives for psychiatric treatment. Furthermore, I have invited Professor Michael Wong, who has been running a territory-wide specialised clinic on neuropsychiatric disorders, to give us an overview in the concepts, history and development, as well as the development of new alternatives for psychiatric treatment. I have invited Professor Michael Wong, who has been running a territory-wide specialised clinic on neuropsychiatric disorders, to give us an overview in the concepts, history and development, as well as the development of new alternatives for psychiatric treatment.
psychosis, has kindly shared her precious experience in utilising the platform of informative technology in her iPEP internet-based psychoeducation programme to exemplify the possibility of telepsychiatry in the future. While we have exploration in the field of digitalisation and telepsychiatry in the modern era, Dr WC Yan has given a review on a film on shamanism in the lifestyle section, to let us visualise resemblance of psychiatric practice at the other end of spectrum in the more ancient and traditional aspect of our culture.

After witnessing what has been going through in our society in the past few months, which I had never imagined while drafting the initial contents of this theme issue in mid-2019, I have also invited Dr Ivan Mak, Chairman of the Public Awareness Committee of the Hong Kong College of Psychiatrists, to give a comprehensive review and update on the topic of post-traumatic stress disorder. The review has been written as an additional token of the contribution of the psychiatric specialty to the medical profession and to the society of Hong Kong as a whole.

References
Neuropsychiatry in the 21st Century

Prof Michael TH WONG

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INTRODUCTION

For decades if not centuries, neurological disorders have been regarded as disorders of the brain and psychiatric disorders those of the mind. Nowdays they are increasingly viewed as being on the same neuropsychiatric spectrum. Common neuropsychiatric disorders include those with prominent emotional, cognitive, perceptual and behavioural symptoms such as neurodegenerative disease, epilepsy, cerebrovascular disease, traumatic brain injury, movement disorders and autoimmune disorders such as anti-NMDAR encephalitis.

At one end of the spectrum are those with predominantly motor disturbance such as Parkinson’s Disease (PD) while those at the other end exhibit predominantly mental disturbance such as schizophrenia, bipolar disorder, depression and anxiety. Between them are those with various combinations of motor and mental disturbance such as multiple sclerosis, epilepsy and stroke with different degrees of gross neuropathology and/or pathophysiology. Overlaps of motor and mental symptoms are indeed common along the entire spectrum. Up to 36% of patients with PD show some evidence of cognitive impairment at disease onset, and another 40% of these patients developed depression over time.1,2 Four types of overlap have been observed – neurological disorder presenting with psychiatric symptoms, psychiatric disorder presenting with neurological symptoms, a neurological disorder causing a psychiatric reaction and psychiatric disorder causing a neurological reaction.3

Neuropsychiatrists focus on disorders of the brain and mind which are associated with gross brain pathologies and/or dysfunctions in neural circuits and neurotransmitters. These disorders manifest not only as a disturbance in psychomotor, cognitive, emotional and volitional processing but also as the impairment of a person’s interaction with other persons and the world. Unlike people with healthy brains and minds who display flexible thought and behaviour in meeting the demands of daily life, patients with these disorders struggle to adapt to various challenges in their physical and social environment.4

Neuropsychiatry is therefore not Consultation Liaison (CL) Psychiatry which deals with the psychiatric aspects of various medical and surgical conditions and is, therefore, broader in her concern and more eclectic in her approach. Neuropsychiatry is also not Behavioral Neurology which limits her concern to abnormal behaviour or experience with demonstrable brain pathology and is hence not inclusive of all psychiatric disorders. Those who are involved in Biological Psychiatry are not neuropsychiatrists but are essentially neuroscientists specialised in various biological aspects of psychiatric disorders such as genomics, neurochemistry, histology, electrophysiology, endocrinology, immunology, pharmacology and brain imaging. Many neuropsychiatrists, however, are also clinical neuroscientists with active research involvement in one or more areas of biological psychiatry.

Neuropsychiatry is not a new discipline. Trimble dated the origin of neuropsychiatry to the Renaissance and the Enlightenment when exploration of brain anatomy noted that brain lesions and pathology had something to do with conditions such as epilepsy, syphilis, movement disorders and hysteria.5 Neuropsychiatry was very active in the 19th century. Alois Alzheimer who identified pre-senile dementia was a psychiatrist and neuropathologist. This interest in and the work on the study of the brain in the practice of psychiatry was however eclipsed around the same time by the psychodynamic theory of Sigmund Freud on unconsciousness and dreams which led to psychiatry that split the mind from the brain. It was not until around the 1980s when the interaction between mind and brain in the expression of psychiatric symptoms was given the attention that it deserves again.6 Neuropsychiatry in her present form is, therefore, a modern endeavour, dealing with clinical presentations at the interface between psychiatry, neurology and general medicine. In this narrow sense, it represents the clinical discipline which manages patients with the psychiatric manifestation of neurological and general medical and surgical disorders. In the broader sense, it represents the practice of psychiatry informed by various advances in clinical neuroscience.

LATEST DEVELOPMENTS IN NEUROPSYCHIATRY

Research in clinical neuroscience over the past two decades have provided support to the notion of the neuropsychiatric spectrum. Postmortem pathological changes have been identified in the hippocampus in schizophrenia7 and the anterior cingulate cortex in major depressive disorder.8 Decreased amygdala-hippocampal reactivity follows administration of selective serotonin reuptake inhibitor (SSRI) and increased dorsolateral prefrontal cortex, and cingulate gyrus activation follows that of norepinephrine.
Neuropsychiatry, however, sees psychiatric disorders as better framed as disorders of distributed interconnected brain networks involving the prefrontal-subcortical brain circuits rather than as disorders of discrete localised pathology as commonly found in neurological disorders. Prefrontal regions include the dorsolateral prefrontal cortex, anterior cingulate cortex and orbitofrontal cortex each with discrete basal ganglia and thalamic connections and primarily involved in higher-order cognitive and affective functions. Impairments of anterior cingulate cortex-subcortical circuit cause the motivational deficit, orbitofrontal cortex-subcortical circuit disinhibited behaviour, and dorsolateral cortex-subcortical circuit dysexecutive symptoms.

It is therefore very important to distinguish clinical discrete lesion-based psychopathology such as post-stroke depression from those limited circuit based pathologies such as frontal network syndromes as seen in patients with dementia and traumatic brain injuries as well as those widely distributed network-based dysfunctions such as default mode, salience, attention, emotional processing, cognitive control, social cognitive, memory and visceral-somatic processing which can be potentially targeted by emerging treatment modalities such as Transcranial magnetic stimulation (TMS) and DBS.

A recurrent theme among neuropsychiatrists and clinical neuroscientists is that we have to move beyond simplistic localisation toward a model of distributed modular functions integrated through structural and functional connectivity, adding neural circuitry to the classic biopsychosocial formulation and integrating research findings in neural plasticity and epigenetics to provide mechanisms that mediate organism-environment interactions in the context of neurodevelopment, including the notion of risk and resilience and intervention that may alter illness trajectory.

The National Neuroscience Curriculum Initiative has been set up to develop this culture of embracing cognitive-affective neuroscience and neuropsychiatry to expedite the “bench-to-bedside” translation of brain-symptom relationship to help guide clinical thinking and future innovative therapeutic interventions through more precise brain-behaviour correlate and informed prediction, prevention, rehabilitation and recovery of psychiatric disorders. To achieve this aim, the ability to synthesise and translate research findings and put them into a patient’s individual context is required, without making the error of reductionism (that is to depict a person as nothing but just the brain) and oversimplification (that is localising specific mental function to a single brain structure or specific region rather than to a neural network). Clinical judgment is needed to decide when a worry has become anxiety, unhappiness depression, enthusiasm mania and memory dysfunction dementia and when to formulate a personalised narrative that is more therapeutic than medications like in a grief reaction and when to carefully document cognitive and functional impairment in order to organise a psychosocially appropriate care as in Alzheimer’s Disease.

NEUROPSYCHIATRIC SPECTRUM – A TALE OF THREE LADIES

Here are three case vignettes to illustrate what the neuropsychiatric spectrum is like in real life. They all have been de-identified to ensure confidentiality.

A is an 18 year-old waitress who presented with mixed depressive and anxiety symptoms. She has been well all along until four years ago when she became increasingly distractible and forgetful. She started to have episodes of staring look for up to 10 minutes. Her personality changed from being outgoing to socially withdrawn. Two years later she started to throw things around unprovoked. Her father described her as if she was “in another world”. These temper tantrums occurred nearly weekly, and all were quite similar and without warning. She would turn quiet and sometimes walked away and 10 minutes later she would become fearful and tremulous, saying “don’t go away … I’m scared...”. Her parents usually have to hug her to calm her down. She cried every time and took up to 30 minutes to calm down. She had to lie down to rest afterwards and only had a vague recollection of what had happened. During and after these episodes she heard voices which were numerous, male and female, talking to her directly, asking her to kill herself. She was initially diagnosed as having depression before her psychotic symptoms became apparent; she was put on an antidepressant but she became manic. Her mania was controlled with antipsychotic medications, but she started to hear voices all the time and even between episodes – multiple, male and female, third person, doing a running commentary on her thoughts and actions. Six months later she tried to jump from a height but was stopped in time. She developed the unshakable beliefs that people were following her and trying to kill her. She required multiple hospital admissions. Examination showed no localising neurological signs but there were persistent auditory hallucinations and delusions of reference and persecution. Her executive functions were reduced in speed but her cognition was otherwise normal. EEG on two occasions showed generalised slow waves and multifocal spikes. MRI brain scan was normal. She was diagnosed as having complex partial epilepsy. Her psychosis was initially ictal and postictal and later inter-ictal. She was put on the anticonvulsant Sodium Valproate and the antipsychotic Amisulpride. She showed noticeable but only partial improvement.

B is a 55 year-old single unemployed woman who presented with her first episode of psychosis. She had a stroke (dominant lobe) when she was 40 complicated by epilepsy, which responded to the anticonvulsant Sodium Valproate. She underwent renal transplant when she was 42 but had to start peritoneal dialysis two years later and switched to haemodialysis seven years after that. On one occasion, when her urea and potassium levels worsened, she developed delusion of reference that people everywhere were talking about her...
for no good reasons. She had no perceptual disturbance and was in clear sensorium. She became clinically anxious and depressed. She did not have any localising neurological signs. She had nominal dysphasia, impaired 3-minute recall and reduced speed in executive function. Her cognitive function was otherwise normal. She was diagnosed to have delusional disorder. EEG showed non-specific slow waves but no spikes. Her symptoms partially remitted with the improvement of her electrolytes and commencement of the antipsychotic Risperidone. She declined antidepressant. Her mental state worsened later when she stopped her antipsychotic but improved again when she agreed to go back on it.

C is a 25 year-old cleaner who started to hear voices and see visions a year ago. She was a victim of sexual assault by a stranger and of physical and emotional abuse by her mother when young. She was a frequent target of bullying at school. She had a problem paying attention in class, especially when she was picked on by other students or confronted by her teachers. Her mind just “went numb” although she “could still hear and see but could not feel a thing”.

Sometimes the passage of time seemed to slow down. She had to pinch or scratch herself to get out of that unpleasant state. More recently, she started to see and hear at times characters from movies or comic books and at other times her abusive mother and bullies back in her school days; such experiences made her very angry and wanted to cut herself. She felt that these characters were there to harm her. She could not be sure if these voices and visions were in her head or not, but they were very vivid and real to her. She could no longer cope with work. She became depressed and anxious, losing interest, motivation and energy and having a problem with her sleep. She felt suicidal but did not have any plan. Neurological and cognitive assessment were both normal. EEG and brain scan were normal. She was diagnosed to have major depressive disorder. Her perceptual disturbance and her mind going numb are actually the consequence of her previous traumas which have sensitised her to stress and that in turn makes her go into a dissociative state. She has responded to antidepressant and has found praying with her friends at church very comforting, but her dissociation requires further post-traumatic counselling.

These three patients experience anxiety and depression with disturbance of their perception and with ideas or beliefs that people are talking about them and/or planning to harm them. From the brain-symptoms correlate perspective, their depressive symptoms can be partially localised to the anterior cingulate cortex and related striatal-thalamic subcortical components. Their negative cognition is suggestive of impaired modulation of negative mood states and is compatible with changes at the subgenual cingulate cortex and the amygdala. Their anhedonia can also be localised to the anterior cingulate cortex-subcortical circuit, particularly the ventral striatum/nucleus accumbens. Their cognitive slowing suggests a mild dysexecutive syndrome of lateral prefrontal dysfunction localisable to the dorsolateral prefrontal cortex. Their mixed anxiety and depressed presentation are compatible with the fact that these two symptoms share the frontolimbic substrate. Their individual clinical histories, however, have their own particular biopsychosocial features and require more than brain-mind correlate to make full sense. The epilepsy in A is the precipitating and now the perpetuating factor for her psychosis. The previously well-controlled epilepsy and the brain compromised by an old infarct in B have likely been destabilised by her electrolyte imbalance precipitating a psychotic episode which improves with treatment but deteriorates when antipsychotic was stopped. The childhood traumatic experience of C has predisposed her to cortical hyperexcitability which could have been misinterpreted as epilepsy and psychosis without a careful neuropsychiatric assessment and meticulous clarification and detailed documentation of psychopathology which uncovers very different pathophysiology that benefits from spiritual support and requires psychotherapy in addition to pharmacotherapy.

THE FUTURE OF NEUROPSYCHIATRY

Despite the advocacy of Engel29 for a biopsychosocial approach to psychiatry in general and that of Lishman a balanced and comprehensive perspective for neuropsychiatry in particular, psychiatry continues to tend to go from one extreme to the other, what Eisenberg20 calls the unfortunate swing from “brainlessness” (neglecting neurobiology) to “mindlessness” (over-focusing on neuroscience) and on top, her ongoing ambivalence to and negligence of culture, religion and spirituality.21

As long as we are mindful of these potential pitfalls in the practice of neuropsychiatry and clinical neuroscience as discussed above, we should be able to go from strength to strength. In the area of nosology, we should be able to stay clear from either reductionism (nothing but the brain) or dualism (brain and mind are separate and different) and have an integrative approach to the disorders of the brain and mind. The never-ending advances in neurobiology and cognitive science will continue to refine our explanation of psychopathology. Seeing psychiatric disorders as not only just brain disorders but also disorders of social relationship and environmental/ecological adaptation will open up opportunities for us to develop a concept of illness that upholds a sophisticated understanding of the complex interplay between symptoms, behaviour and function in the experience and expression of mental illness and health and wellbeing. The fact that we have evidence for neuroplasticity and that mental forces can transform brain matter, such as cognitive-behavioural therapy (CBT) can change brain patterns to the benefit of patients with obsessive-compulsive disorder (OCD) and depressive disorders22 or childhood abuse and bullying and acute and chronic stress may have a permanent impact upon brain organisation and behavior23, there is a good chance for new therapeutics that goes beyond medications and surgery to non-invasive psychosocial interventions which can fine-tune our brain and mind. Last but not least any further and ongoing dialogues between neuropsychiatry, clinical neuroscience and philosophy of science and psychiatry will facilitate the formulation of psychiatric ethics that is informed by neuroscience of ethics which not only explain our brain more but also help us understand our mind better.
References


INTRODUCTION

Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis was first described in a case series of 12 women with ovarian teratomas by Dalmau and colleagues in 2007. In his group of patients reported, a wide range of symptoms, including prominent psychiatric symptoms, amnesia, dyskinesia, autonomic dysfunction, and decreased level of consciousness, were found. This autoimmune condition was later found to be not exclusively paraneoplastic, and subsequent studies reported up to 70% of patients afflicted without any tumour involvement.

In 2012, the book “Brain on Fire: My Month of Madness”, an autobiography written by a young New York Post journalist suffering from this illness, was published. The book was further adapted into a motion picture in 2016, which made this disorder known even to the general public subsequently.

Anti-NMDAR encephalitis was found to be the most common form of autoimmune encephalitis. It was also reported to have surpassed the frequency of any individual viral cause of encephalitis in young persons. Since its discovery, the impact of this disorder in both specialties of neurology and psychiatry has been remarkable. People started pondering if there had been cases of such being misdiagnosed and thus mistreated as psychiatric conditions like psychosis or mood disorders in the past.

A CLINICAL SCENARIO

Miss X was a 20 year-old university student. She had complaint of abdominal pain and swelling for more than one month before the diagnosis of a right ovarian teratoma was subsequently ascertained. The operation was soon performed including right salpingo-oophorectomy (with the frozen section of right tube and ovary showing immature teratoma), followed by left ovarian cystectomy, as well as right pelvic and para-aortic lymphadenectomy. Subsequent sessions of chemotherapy were undertaken as well.

Since around the time of her operation, Miss X was aware of mild thought disturbance subjectively though the problem was not marked and did not cause any obvious disturbance in her normal life initially. More significant symptoms were, however, reported six weeks after the operation, with obvious abnormal mental state observed by her parents as well since then. After discussion of her problems with the gynaecologist, and subsequent assessment by a clinical psychologist, the care team decided to refer the patient to a consultation-liaison psychiatrist in the gynaecology ward for further assessment of possible psychiatric disorder.

During the psychiatric assessment, Miss X reported subjective difficulty in comprehension of others’ speech. She often felt her thoughts being stuck. She found it hard to respond with appropriate answers during conversations. Her parents also noticed she was being inattentive and had a problem in carrying out a conversation. Her condition fluctuated from time to time. At one time when her mental state appeared to be most disturbed, she seemed to have difficulty in telling even the correct name of her mother.

Miss X also recalled experiences of hearing some vague voices coming from her mind on and off, though she could not describe their content clearly. She also experienced abnormal visual perceptions from time to time, like seeing changes in colours of objects around. She further reported difficulty in distinguishing events happening in reality versus in dreams at times.

Miss X described her mood being low at times after the operation but was not persistently depressed. She had worrying thoughts about her abnormal experiences and symptoms. She reported subjective difficulty in concentrating on mental activities such as reading, and she experienced decreased interest in her usual hobbies. She failed to continue with her study and worried that she would be unable to do so in the long run. Her quality of sleep had been suboptimal with easy awakening. Her appetite remained normal otherwise. She did not have any suicidal idea.

Miss X was noticed by her parents to have some facial twitching on and off. There was an episode of fainting which occurred at home days before the psychiatric consultation, and she was admitted to the medical ward. Investigations including blood tests and CT brain revealed no significant abnormality, and she was discharged after a brief stay.

During the interview by the psychiatrist, Miss X appeared calm, polite and cooperative. She had fair concentration and was able to give accurate yet brief general answers to questions on factual demographic and autobiographic information. When asked about more complex and abstract questions, she had more obvious difficulty to respond and tended to give more vague answers such as “I’m not sure”. Her mood was
stable and described by herself to be mildly low. She otherwise did not have any negative thoughts or suicidal idea. She did not report any hallucination, abnormal perception, or any delusional belief at the time of assessment. She demonstrated satisfactory orientation. However, upon further screening for cognitive function using the Montreal Cognitive Assessment Hong Kong version (HK-MoCA), she scored 22/30 only (Normal cutoff 26/30).

After the psychiatric assessment, in view of her recent history of teratoma, fluctuating nature of cognitive function impairment and abnormal perceptions reported, and her fainting episode with seizure attack being a possibility, suspicion of a diagnosis of autoimmune encephalitis, particularly anti-NMDA receptor encephalitis, was raised. Other possible psychiatric differential diagnoses, including depressive disorder and at-risk mental state of psychosis, were also taken into consideration though.

A check of serum anti-NMDAR antibody was found to be negative. Other basic blood tests revealed no obvious abnormality. A brief generalised convulsion was however observed during her stay in the gynaecology ward. Miss X was then referred to the neurology team for further assessment and investigation. Electroencephalography (EEG) revealed slow activities over left temporal lobe, with no other abnormality. Lumbar puncture showed normal CSF glucose (3.9 mmol/L) and CSF protein (0.32 g/L). CSF anti-NMDAR antibody was however found to be positive. Cultures, MRI brain and PET-CT were all negative.

The neurology team prescribed a course of treatment of IVIG for the auto-immune condition and levetiracetam for seizure prophylaxis. No more seizure or twitching movement was noted. The patient reported gradual improvement in her cognitive function and resolution of her psychotic or mood symptoms. She was able to resume her study at the university after four months.

**DISCUSSION**

Anti-NMDAR encephalitis is a predominantly female condition (with a female to male ratio of around 8:2), with a median age of 27 years at presentation. The frequency of occurrence is observed to be greatly reduced after 40 years of age. Psychiatric symptoms to a certain extent, are reported in up to 95% of patients. The psychopathology is found to be polymorphic in nature encompassing catatonia, mood, behaviour and psychosis domains.²

Clinical features of anti-NMDAR encephalitis are divided into three stages.⁵ In 70% of patients, there could be a prodromal period, averaging five days but could be up to 2 weeks, of a viral-like illness with symptoms of headache, fever, malaise, myalgia, upper respiratory discomfort, nausea and diarrhoea.⁷ Whether the prodromal symptoms are part of early immune activation or have resulted from a non-specific infection that facilitates the crossing of the blood-brain barrier by the immune response, is not clear.⁷ In the second stage, early features are characterised by cognitive dysfunction, psychiatric features and seizures.⁶ Seizure is a common presentation of anti-NMDAR encephalitis in children and young patients, in up to approximately 70% of patients.⁵ The third stage is characterised by late features including reduced consciousness, involuntary movements, and autonomic dysfunction, which can occur as early as 10-20 days after the early features.⁸ According to this scheme, our patient presented with mainly second stage early symptoms at the time of her psychiatric assessment.

In pathophysiological perspective, glutamate receptors (type NMDA) are located in the post-synaptic membrane and form heteromeric ligand-gated cation channels composed of NR1 and NR2 subunits.¹⁰ Autoantibodies in the serum or CSF of patients with anti-NMDAR encephalitis were shown to bind specifically to an epitope located in the extracellular domain of the NR1 subunit.⁷ There are striking similarities between anti-NMDAR encephalitis and other models of NMDAR-specific disruption, such as experimental and recreational use of phencyclidine and ketamine.¹¹ In other psychiatric conditions such as post-partum psychosis, there are also similarities in clinical presentations identified, thus shedding light into possible mechanism of reversible NMDAR-dominant receptor-mediated network disruption in these conditions secondary to alternative non-immune mechanisms.²

Diagnosis of anti-NMDAR encephalitis is based on the characteristic clinical symptoms, as well as supporting investigations including the brain MRI, EEG and CSF studies. Possible differential diagnoses include psychiatric conditions such as psychosis, affective disorder and substance-induced disorder. Other medical conditions such as infectious encephalitis (especially HSV) and other autoimmune etiologies (e.g. limbic encephalitis with autoantibodies against Hu, Ma2, CV2 and amphiphysin) should also be considered.¹⁰

In patients with anti-NMDAR encephalitis, CSF studies may review lymphocytic pleocytosis or oligoclonal bands. CSF protein can be elevated or normal, as in the case of our patient.¹² The diagnosis of anti-NMDAR encephalitis is confirmed by the detection of CSF antibodies against the GluN1 subunit of the NMDAR. Assays for serum anti-NMDAR antibodies are not as sensitive as CSF studies, with false-negative results in up to 14% of cases.¹³ This was indeed the case found in our patient. Her serum anti-NMDAR was negative in the initial screening. Only in subsequent CSF studies performed was anti-NMDAR found to be positive.

EEG abnormalities may be identified in terms of focal or diffuse slow or disorganised activities. Epileptic activities may be revealed sometimes.¹⁴ Extreme versions of the “delta brush pattern” in EEG, which are transient patterns characterised by a slow delta wave with superimposed fast activities, appeared to be unique to anti-NMDAR encephalitis and may suggest a more prolonged illness. It, however, was only seen in less than one-third of patients.¹⁵ The EEG of our patient revealed only non-specific slow activities over left temporal lobe without other more specific abnormality. MRI is normal in 50% of the cases of anti-NMDAR encephalitis, as in our patient. Those with
abnormalities shown in MRI mostly involve non-specific T2 hyperintensity in the hippocampus, frontal and insular cortex.16

With regard to treatment, in those patients with teratoma being identified, tumour resection results in a noticeable neurological improvement in days or weeks in some of them. Immunotherapy is the treatment of choice with or without the presence of a tumour and involves trials of corticosteroids, intravenous immunoglobulins, or plasma exchange.17

Early identification and treatment of the condition are associated with better outcome.9 Approximately 50% of patients are able to achieve full recovery. Yet more than 40% of patients continue to have mild to severe deficits. Up to 25% of patients may have severe deficits or even death.7 Substantial, persistent cognitive impairments are found in some patients, particularly in the areas of executive function and memory. This observation is in line with the proposed pivotal role of NMDARs in the human brain. The good cognitive long-term outcome also depends on early and aggressive treatment, following which the patients are found to have less frequent hippocampal damage.17

A recent study has attempted to construct a grading score to predict neurologic function after diagnosis of anti-NMDAR encephalitis. Features including intensive care unit admission, treatment delay for more than 4 weeks of symptom onset, lack of clinical improvement within four weeks of starting treatment, abnormal brain MRI, as well as elevated CSF WBC count (> 20 cells/µL) are found to be independent predictors of poor functional status one year after symptom onset.18

As for our patient, the resection of her teratoma apparently failed to halt the progress of further development of the disorder. It is not clear if this early removal of the tumour led to relatively less severe symptoms observed in her case. To a certain extent, it was fortunate that her teratoma had been identified early due to relative prominent symptoms of abdominal pain and distension. This important piece of clinical information raised early suspicion of anti-NMDAR encephalitis as a possible diagnosis when only relatively non-specific psychiatric and cognitive symptoms were present at the early phase of her illness. The early confirmation of the diagnosis with appropriate investigations including CSF studies, and subsequent early initiation of appropriate intervention with immunoglobulin therapy likely contributed to quicker remission of the disorder and satisfactory recovery of her cognitive function.

CONCLUSION

As discussed, anti-NMDAR encephalitis demonstrates a possible common mechanism of receptor-mediated network disruptions via different aetiology in neurological and psychiatric disorders. In the case of autoimmune encephalitis, “yellow flag” symptoms serving as warning signs of the condition have been suggested including decreased level of consciousness, abnormal postures or movements (orofacial, limb dyskinesia), autonomic instability, focal neurological deficits, aphasia or dysarthria, rapid progression of psychosis (despite therapy), catatonia, hypoaesthesia, headache, and presence of other autoimmune disease (e.g. thyroiditis).19 As part of the assessment of patients with new-onset psychotic symptoms, vigilance is required to watch out for other medical conditions which may result in similar neurophysiological disruption disguising as a psychiatric condition. The emergence of atypical symptoms are alarming features in particular.

References
Patients with MDD* often lose pleasure in those everyday activities they used to love.

Which of the following concern do you commonly see in MDD* patients?

Help patients rediscover their love for life.

Consider Bupropion in MDD* patients because,

- The only dual acting antidepressant to target noradrenaline and dopamine.
- Greater improvement in sleepiness and fatigue scores compared to SSRP.
- No association with weight gain compared to placebo.
- Lower incidence of sexual side-effect compared to SSRIs.

Bupropion is associated with a dose-related risk of seizures. Wellbutrin XL should be discontinued in patients who experience a seizure while on treatment.

ADVERSE REACTIONS

Wellbutrin XL has been associated with a dose-related risk of seizures. Recommended by a guideline as 1st line drug for patients with MDD.

References:
Pharmacological treatment and psychotherapy are the two main streams of treatment modalities in modern psychiatry. However, there are limitations to each kind of treatment modality, including the side effects of the medications, the labour intensity and cost-ineffectiveness of psychotherapy, and the non-responsiveness or inadequate response to both modalities. Therefore, new options for treatment modality has become a pressing issue in the field of psychiatry.

Looking back at psychiatric history, invasive psychosurgery where a specific brain area is targeted was once viewed as a cure for mental illness in the mid-20th century. However, inadequate understanding of neuroscience and the invasive nature of psychosurgery resulted in poor treatment outcomes, and thus this treatment modality gradually faded out. Only electroconvulsive therapy (ECT), which requires general anaesthesia, remains commonly used in the clinical setting for treatment-resistant depression and psychotic disorders. In contrast, deep brain stimulation (DBS) for refractory depression and obsessive-compulsive disorder (OCD) is rarely used in the clinical setting.

In recent decades, non-invasive brain stimulation (NIBS) has been under the spotlight in mental health research. The most common forms of NIBS are transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). With the advancement of neuroscience, mental illness nowadays is viewed as a kind of brain disorder. Specific brain areas or networks are believed to contribute to the pathophysiology of those disorders. Therefore, if we could target specific brain abnormalities, it would be possible to cure or alleviate the symptoms without causing generalised side effects from non-specific approaches. The recent development of neurostimulation and neuro-navigation techniques has allowed us to target specific brain areas or networks with direct electric current, induced electric current or ultrasound shock wave, to modulate activities at the cellular level and change the neuroplasticity. Another advantage of this treatment modality is that it is usually well-tolerated without the need for general anaesthesia. Some patients might experience mild and transient physical discomfort such as headache, itchiness and redness, but only a few people have experienced a seizure in TMS.

TMS uses an electromagnetic field to induce an electric current within the brain. Repetitive TMS (rTMS) delivers a train of pulses at the same intensity over time at a particular region of the brain. It either stimulates or suppresses neuronal activity, depending on the frequency and the inter-train interval between pulses. The effect of rTMS is mediated by modulating the neuronal activities of the targeted dysfunctional area. Distributed modulation of brain activities via a specific brain network may also be achieved. The clinical effects of rTMS may be affected by many factors, including the number of sessions, the intensity level of pulses, the interval between sessions, and the position and shape of the coil.

TMS has been most studied in the treatment of depression. For adults with depression and on whom antidepressants have failed, application of high-frequency rTMS over the dorsolateral prefrontal cortex (DLPFC) for 4-6 weeks daily, usually five times per week, is a standard treatment protocol approved by the U.S. Food and Drug Administration (FDA) in 2008. In recent years, the FDA has approved a newer treatment parameter of TMS using theta-burst stimulation, which has the advantage of shorter treatment time.

Apart from depression, the FDA also approved deep TMS as a treatment of OCD in 2018, based on a randomised, multi-centre study of 100 patients. In the study, 38% of OCD patients had more than 30% reduction in symptoms, according to the Yale-Brown Obsessive-Compulsive Scale (YBOCS). The specific TMS coil was used to achieve deep penetration in the brain. High-frequency deep TMS was applied to the medial prefrontal cortex (mPFC) and the anterior cingulate cortex (ACC) region to target the dysfunction of the cortico-striato-thalamo-cortical (CSTC) circuit, which is the underlying pathophysiology of OCD.

TMS has also been applied to patients with cognitive impairment, anxiety disorder and post-traumatic stress disorder (PTSD) with promising results in some studies. However, conflicting evidence has been noted. Further larger-scale studies are needed to provide more evidence.

tDCS AND ITS APPLICATION

tDCS is a neurostimulation technique where a mild direct current (1-2 mA) is induced through the cerebral cortex via electrodes placed on the scalp, which in turn modifies cortical excitability, depending on the polarity of the applied current.
directions. It is easier and cheaper to administer compared to TMS, and it can also be delivered at home. Its effects are possibly exerted through modulating cortical excitability. Long-term plasticity is enhanced, with modulations in the rate of neurotransmitter release.

The application of tDCS in clinical use is less supported by evidence. To date, there is not yet any FDA approval for tDCS for the treatment of any mental illness. tDCS studies are most commonly done on depression. In a recent meta-analysis of six randomised controlled trials (RCTs) with 289 adult patients, tDCS was shown to be significantly superior to sham-control treatment in response, remission and improvement in depression. In most studies, the left DLPFC was stimulated, and the effect size was comparable with those reported for antidepressant drug treatment and rTMS. However, a recent large international RCT of adult patients with depression showed no difference in reducing depressive symptoms between active and sham stimulation in patients with unipolar or bipolar depression. The conflicting results warrant further high-quality RCTs to determine the efficacy of tDCS.

Moreover, studies of tDCS on patients with schizophrenia, substances abuse disorders and OCD also showed promising initial results. Further larger-scale studies and meta-analysis, however, would be necessary to inform the effectiveness of tDCS in a clinical setting.

EMERGING NIBS TECHNIQUE

Besides the aforementioned two most commonly investigated NIBS, there are new NIBS which use transcranial ultrasound to modulate brain activity. Compared with TMS and tDCS, this technique has a higher spatial resolution and can reach deeper structures. Its safety profile is comparable to that of the other NIBS techniques. There are various modalities and names for this new technique, including transcranial focused ultrasound and transcranial pulse stimulation (TPS), also known as low-intensity extracorporeal shock wave therapy (Li-ESWT).

The underlying mechanism of this technique is mechanotransduction. It is a biological pathway through which the cells convert the mechanical TPS stimulus into biochemical responses. It could affect neurons and induce neuroplastic effects through several pathways, including increasing cell permeability, stimulation of mechanosensitive ion channels, release of nitric oxide resulting in vasodilation; increased metabolic activity and angiogenesis, stimulation of vascular growth factors (VEGF); and stimulation of brain-derived neurotrophic factor (BDNF).

Most of the existing studies have been done on healthy volunteers to test the neuromodulation effect in different parts of the brain, including the thalamus and basal ganglia. With the adjustment of stimulation parameters, it can cause the suppression or facilitation of neural activity.

Such a technique has also been applied to five patients with unresponsive wakefulness syndrome, resulting in significant improvement in their vigilance and oropharyngeal motor functions. Another application is on Alzheimer’s disease (AD). TPS was obtained with CE marking in 2018 for the treatment of the central nervous system (CNS) in patients with mild to moderate Alzheimer’s disease (AD). In a recent study, TPS was applied to elderly patients with AD in three sessions (6000 pulses each) per week for 2-4 weeks, either over classical AD affected sites such as the dorsolateral prefrontal cortex, areas of the memory and language network, or overall accessible brain areas (global brain stimulation). Significant improvement in the CERAD (Consortium to Establish a Registry for Alzheimer’s Disease) score was demonstrated (immediately as well as 1 and 3 months after stimulation). MRI also showed significantly increased connectivity within the memory network.

Since this NIBS technique is still relatively new, further studies should be done with various disease groups before it can be applied in a clinical setting.

CONCLUSION

NIBS is a group of non-invasive neuromodulation techniques which are potentially useful to treat various mental illnesses such as depression, OCD, schizophrenia, substances abuse disorders and dementia. Their administration is easier, and their safety profile is better than traditional psychosurgery or deep brain stimulation. Their efficacy in some conditions such as depression and OCD has been proven to be comparable to pharmacological treatment. NIBS has great potential to be a new treatment option for the 21st century, as monotherapy or adjuvant therapy to existing treatment. Further effort in researching this area will provide more evidence of the effect of these NIBS techniques in clinical use.

References


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**Dermatology Quiz**

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**This 51 year-old Caucasian lady who was totally asymptomatic came to the clinic to have mole checking. Incidentally, a pigment patch was found on left sole. Further asking revealed the patch had been present for around three years. Since the patch was totally asymptomatic, the patient did not pay much attention to it. She just knew that the patch seemed increasing in size slowly. Physical examination revealed a 2.5 cm irregular pigmented patch on the left sole which was heterogeneous in colour and with suspected satellite lesions. The surface was smooth without any ulceration or erosion. (Fig. 1)**

**Questions**

1. What are the differential diagnoses of his skin lesion?
2. What investigation are you going to order?
3. How do you treat this patient?

*(See P.36 for answers)*
Well-being and functioning Improvement

Recommended 1st line treatment in MDD*

Minimal drug-drug interaction

Wide dosing range

* SNRIs, the class of drugs to which Pristiq® belongs, is one of the first-line recommendations for pharmacotherapy for MDD.

MDD = major depressive disorder. SNRI = serotonin and noradrenaline reuptake inhibitor.

References

PRISTIQ® ABBREVIATED PACKAGE INSERT

TRADE NAME: PRISTIQ®
PRESENTATION: 25mg, 50 mg and 100mg Extended-Release tablets
INDICATIONS: Treatment of adults with major depressive disorder (MDD)
DOSAGE & ADMINISTRATION: 50 mg once daily at approximately the same time, with or without food. The maximum recommended dose for patients with severe renal impairment or end-stage renal disease (ESRD) is 25 mg every day or 50 mg every other day. Supplemental doses should not be given to patients after dialysis. The 25 mg per day dose is intended for a gradual reduction in dose when discontinuing treatment.

CONTRAINDICATIONS: Hypersensitivity to desvenlafaxine succinate, venlafaxine hydrochloride or to any excipients in the Pristiq formulation. Use of MAOIs with Pristiq or within 7 days of stopping treatment of Pristiq. Use of Pristiq within 14 days of stopping a MAO.

WARNINGS & PRECAUTIONS: All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening, 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. Cautions on Serotonin syndrome; abnormal behavior, especially in children; increased risk of bleeding. Caution is advised to patients with pre-existing hypertension, cardiovascular, cerebrovascular conditions; angle-closure glaucoma; seizure; hyponatremia; interstitial lung disease & eosinophilic pneumonia; discontinuation syndrome.

INTERACTIONS: MAOI; other serotonergic drugs; drugs that interfere with hemostasis; drugs that are primarily metabolized by CYP2D6; avoid alcohol consumption. False-positive urine immunoassay screening tests for phencyclidine (PCP) and amphetamine have been reported in patients taking desvenlafaxine.

PREGNANCY AND LACTATION: There are no published studies on Pristiq in pregnant women. There are risks associated with untreated depression in pregnant women. The developmental and health benefits of breastfeeding should be considered along with the mother’s clinical need for Pristiq and any potential adverse effects on the breastfed child from Pristiq or the underlying maternal condition. Pristiq is not a controlled substance. See FULL PRESCRIBING INFORMATION AVAILABLE UPON REQUEST.
Impact to Psychiatry in the Age of Digitalisation: Disease of the Mind from Understanding, Treatment to Transformation

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INTRODUCTION
Since the coining of the term ‘Psychiatry’ by Johann Christian Reil in 1808, Psychiatry has been developing rapidly as a branch of modern medicine for the diagnosis, treatment and prevention of disease of the mind.

When we take some time to look back in the history of disease of the mind, as far back as the time of ancient Greece, around 400 BC, Hippocrates2, the father of medicine, had revolutionised medicine at that period, when his ideas and works were subsequently collected in the legendary Corpus Hippocraticum. By then, he and his followers had already been postulating about the brain as the organ of the mind as well as delineating diseases of the brain.

Two milleniums had passed.

With the dawn of the Scientific Revolution and the Age of Enlightenment, the discovery and understanding of the mind and the world embarked into a whole new world of evidence and rationality.

Among the many important theorisations, the conceptualisation of body-mind dichotomy by Descartes3 had been well received in subsequent generations as the mainstream model in understanding the human body and mind.

At about the same time, as modern medicine and technology drastically advanced, doctors and scientists were able to have a more accurate and detailed delineation of the human brain.

Human civilisation continued to bloom.

About one century ago, in addition to the perspective of philosophy, the conceptualisation of the self and the mind was gradually drifting towards the areas of Psychiatry and psychoanalysis. On top of the Freidian structural and topographic models of the mind4-5, Freud also postulated the possible role of intra-psychic conflicts in the formation of disease of the mind, with subsequent development of psychoanalysis as a form of therapeutic intervention.

Decades later in the twentieth century, the pioneering scientific understanding in the disease of the brain and mind, like the Dopamine Hypothesis of Schizophrenia in the causation of psychosis, and the progressive invention of evidence-based psychiatric treatment (including pharmacological treatment, Electro-convulsive Therapy (ECT) and psychotherapy), in combination with humanistic psychosocial interventions, has brought about promising improvement in treatment efficacy and quality of life of psychiatric patients.

In essence, such major and landmark leaps in Psychiatry have shifted the paradigm of Psychiatry from containing insanity to advocating hope and recovery, fundamentally converting the Age of Asylum to an Era of Treatment and Rehabilitation.

Concerning the birth and history of digitalisation, it was the ground-breaking ideas and works of Alan Turing around the time of World War II making him considered the father of theoretical computer science and artificial intelligence (AI)6. Subsequently, it only takes half a century for the world to realise the drastic impact of the Digital Revolution in the humankind.

When it comes to the first encounter of Psychiatry and digitalisation in human history, I think that it could have been the invention of MuCulloch-Pitts Unit7, a basic subunit of the computer simulating the basic subunit of the brain, in the 1930s. It was actually the embodiment of the first tangible connection of AI and neuroscience, while neuroscience is the mainstay for us to explore our mind and the disease of the mind, for which Psychiatry is about.

MEDICINE AND PSYCHIATRY IN THE AGE OF DIGITALISATION
After the dawn of the Age of Digitalisation in the twentieth century, understandably but unexpectedly, digitalisation and AI have been transforming the delivery of healthcare unprecedentedly in the past few decades.

Together with other ground-breaking advances in biotechnologies, like gene therapy and nanotechnology, the inevitable and increasing perusal of digitalisation in Medicine and Psychiatry, currently mainly in the form of algorithms, big data and robotics, should be originally intended for improving our health and quality of life, relieving our suffering and prolonging our lives at a more efficient, sophisticated and extensive as well as less expensive way with this best tool in our history.

To the other extreme, in less developed countries where basic healthcare delivery is still compromised by limitation in resources and accessibility, digital clinical management and online consultations can be one of the readily available short-term solutions to such an imminent need of basic rights and humanity.

In fact, all these advances can be attributed to the evolution of Artificial Neural Network (ANN), as well as the invention of human-created algorithms and evidence-based programmes by researchers and clinicians.

Currently, the major forms of digitalisation in Medicine and Psychiatry would include processing of big data...
in healthcare, telemedicine, telepsychiatry, as well as various digitalised tools in assessment, diagnosis, treatment and research.

**TWO SIDES OF THE SWORD**

Undoubtedly, the digitalisation of clinical care and healthcare delivery is bringing about many essential benefits to human beings, including the possibility of treating more patients as well as previously inaccessible patients, the delivery of personalised medical care with better efficacy and probably efficiency, the higher reliability in medical service delivery, the possibly better healthcare cost-effectiveness as well as the development of more quantified and co-ordinated healthcare systems.

These unprecedented advances in the history of modern medicine are due to the drastic improvement in terms of flexibility, speed, scale, decision-making and personalisation in the delivery of healthcare in the Age of Digitalisation.

On the other hand, the apparent disadvantage brought about by digitalisation that most people can readily think of would be the compromise of the human touch in the course of a therapeutic relationship, which is particularly indispensable in Psychiatry where empathy is therapeutic and essential.

At a deeper level, challenges to ethical issues could range from the handling of massive confidential healthcare data to the accountability for the clinical decisions made by AI.

There have already been even worries about the possibility of replacement of doctors by AI in future, which is essentially a perceived existential threat of our profession.

**UNIQUE IMPACT OF DIGITALISATION IN PSYCHIATRY**

Regarding the specific impact of digitalisation and AI to Psychiatry, due to various obvious reasons, it would be wider and deeper than other medical specialties, ranging from psychopathology, pathogenesis, presentation, diagnosis, nosology, treatment, clinical management and research, to ethics.

In Psychiatry, one of the major cornerstones in the understanding of the disease of the mind has been built on descriptive psychopathology, which involves phenomenology, observation and empathy with a process to achieve understanding and explanation. However far our digitalisation can go in the end, in the first place, it would be fundamentally impossible for any standardised assessment tools (like checklists and questionnaires) nor any invented digital diagnostic tools to comprehend and understand psychiatric patients in the way a psychiatrist does.

In terms of presentation and diagnosis in Psychiatry, we can take schizophrenia as an example. The salient feature of schizophrenia is the disturbance in the boundary of self and differentiation of reality and non-reality. The increasing infiltration of digitalisation in our self might weaken our self-boundary in general, and the interpretation about specific schizophrenic symptoms in patients, like passivity experience and thought alienation, would need more detailed delineation in our clinical practice in due course. Moreover, we can imagine that the increasing dominance of virtual reality in our daily living would actually pose increasing difficulty for us in validating the psychotic symptoms reported by our patients, like referential ideation and paranoia. What is more, with our increasing sense of omnipotence in the virtual world, the possible altered sense of salience and personal significance might be difficult to be differentiated from the primary symptoms like delusional atmosphere in patients who have schizophrenia.

For the aspect of nosology, there have been on-going debates about the aetiology and classification of addiction disorder related to internet use. We are still not sure whether it is a condition related to impulsivity or compulsion, or both. It is recently put as the condition for further research in the fifth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM V) as Internet Gaming Disorder. But the next question to ask would be, ‘would internet addiction still be regarded as a disease if every one of us needs to survive in a 24/7 internet world?’

For the pathogenesis of psychiatric disorders, psychosocial stressors have been identified as one of the major causes in the pathogenesis of different kinds of psychiatric disorders in a multi-factorial model. In such an increasing digitalised world as ours, it is also just natural that the stressors experienced in the virtual world can also cause mental problems in our ‘real’ or pre-existing world. Worse still, the influence of digitalisation would actually reach deep down to different levels of inner ‘Self’, which would be possibly causing profound adverse effects upon our mental health, needing further timely research.

Recently, there have been even postulations of harm to our brain by the process of multi-tasking, a common daily practice in the Age of Digitalisation, through the possible excessive release of cortisol and adrenaline as well as disturbance of dopamine addiction loop. Likewise, the possible resting function of the mind-wandering mode upon the brain might also be interrupted by the constant digital use and the immersion in the digital world.

About the proliferative new development of digital psychiatric management in recent decades, there have already been various kinds of digitalised systems of healthcare delivery, digitalised information processing and storage, digital monitoring tools and charts for individual’s instantaneous mental state, online consultations and standardised assessments, readily accessible information on psychiatric disorders and psychotropics in the internet, computer-based psychotherapeutic interventions like Cognitive Behavioural Therapy (CBT), digital drug ingestion tracking system, as well as GPS tracking for those patients at risk.

Regarding the further psychiatric research in our mind and brain and related disease in the Age of Digitalisation, it is being made further possible by the rapid development of digital tools like Functional MRI and different kinds of brain-mapping.

When it comes to the specific treatments in Psychiatry, the progressive advancement of therapeutic interventions in the brain, including Trans-cranial Magnetic Stimulation (TMS) and Deep Brain Stimulation (DBS), would not have been possible without the technological advancement brought about by digitalisation and AI.
As compared with these different aforementioned issues, the controversies in Psychiatry in the Age of Digitalisation can be even more intricate. Among them, the clinical data and human right issues in Psychiatry are becoming progressively complicated and sensitive in our daily practice in the Age of digitalisation, in a background of various issues of stigmatisation, accessibility and vulnerability. Further ethical considerations would definitely need more in-depth contemplation and deliberation, in order to protect our potentially vulnerable patients.

More ultimately, the essential and indispensable elements of human touch, empathy, transference and countertransference in daily psychiatric practice in Psychiatry can be seriously jeopardised if there would be indiscriminate digitalisation.

DISCUSSION

On one hand, it is getting more and more clear that the progression of digitalisation is inevitable in human history.

On the other hand, it is getting more and more necessary for Psychiatry to progress further and leap higher, in order to face the present and future challenges towards the mind of the mankind, a major one of which would definitely be the projected estimation of depression as the most important cause of global disease burden in 2030.

In the first glance to everyone, digitalisation and AI appear irresistible as a quick, timely and easy solution to the challenges of our time, before the related harms and threats are becoming more and more conspicuous subsequently.

The stakeholders in the facing the upcoming upheavals would be patients, doctors, information technology (IT) and healthcare business entrepreneurs, service providers, scientists, researchers and governments.

It would be an unstoppable pendulum of struggle and consensus and struggle in due course.

Among these stakeholders, needless to say, the individual and collective interests of patients should always be given the utmost priority. In this regard, psychiatrists should continue to play an indispensable role to maximise the benefits, minimise the harms, improve well-being and safeguard the safety of our potentially vulnerable patients.

CONCLUSION

As a specialty in Medicine which is about the human inner experience as well as individual and collective meaning, the Age of Digitalisation is actually bringing more fundamental changes in Psychiatry than it had been anticipated.

That might even be a high time we revisited even more ontological issues, like mental health in the Age of Digitalisation.

With the potential and unprecedented impacts at different levels brought about by digitalisation and AI, we psychiatrists should constantly gate keep and review the drastic development of Psychiatry, not only from the medical perspective but also from inter-disciplinary perspectives of philosophy, culture and history, which would be a beacon to shed light on our misty way ahead.

In particular, conceptually and practically, digitalisation and AI should always be regarded and perused as an invented tool to assist us humans to improvise and personalise our psychiatric service to meet the ever-increasing psychiatric healthcare need now and in future, instead of as a substitute of us.

That comes to the need for advocacy of psychiatrists working with AI-assisted Psychiatry.

More important still, in this on-going, dynamic and multi-level processes of digitalisation of Psychiatry after the Millennium, human touch, empathy, humanity, and the boundary between human and AI should never be compromised.

In the end, with our concerted and pro-active efforts, I believe eventually these important and fundamental qualities of human beings would be filtered and retained across time and space, despite the imminent and unprecedented transformation in Psychiatry into the future world.

After all, Psychiatry is about experience and meaning, which makes us human.

P.S. At the time of writing this article, I read in the news that artificial neurones had been already invented as ‘brain chips’ with the potential to repair our brain, like in Alzheimer Disease. It is probably a matter of karma, initially beginning from the invention of AI by simulating to our neurones about one century ago to the subsequent invention by AI to replace our lost neurones in the Millennium, which is actually a beautiful interplay of digitalisation, AI and Psychiatry. This is also the time when humanity should come into play.

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HONG KONG. WHERE THE WORLD CHOOSES TO MEET.

With an exceptional spectrum of MICE's unique advantages and authentic experiences, this is where the world gets connected and inspired in every way. Hong Kong. The World's Meeting Place.
A Web-based Psychoeducation for Caregivers of Patients with First-episode Psychosis in Hong Kong and Possibility of Telepsychiatry

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INTRODUCTION

The role of caregivers of patients with psychosis is crucial at all stages of the illness. Support from caregivers helps to improve medication adherence and social functioning and to reduce hospitalisation duration for patients with psychosis. However, this is often demanding to the caregivers, particularly during the initial phase of the illness. With very little understanding of psychosis, caregivers often feel shocked, frustrated and helpless when their relatives become unwell for the first time. Therefore, caregivers often experience distress with a high rate of depression, anxiety and subjective sense of significant burden. About one-third of the caregivers have been found to suffer from depression. Therefore, support and psychoeducation of the caregivers of patients with the first episode of psychosis (FEP) would impact the outcome of patients and overall health of caregivers.

Various international clinical guidelines (International Early Psychosis Association Writing Group, 2005) have suggested family intervention as one of the key psychosocial interventions. Studies have provided solid evidence on the effectiveness of a family-focused intervention on patients with FEP including a reduction in rate of relapse and hospitalisation, as well as an improvement in adherence and functional outcomes. Psychoeducation, aiming to provide knowledge on psychosis, and to improve caregivers’ communication skills and coping skills in caring for the patients, is one of the key components of the family-focused intervention. The effectiveness of the caregivers’ psychoeducation on its own has been demonstrated. The role of the family as an informal source of care for patients with psychosis is particularly significant in Hong Kong in view of the family-oriented Chinese culture, and as more than 80% of patients are living with their family of origin.

Despite the stated evidence on the effectiveness of the caregiver psychoeducation, implementation of these programmes in the real world has been challenging. Most of the programmes are in the form of short-term face-to-face family group intervention. The operation of this format is restricted by the high case-load of the healthcare professionals in Hong Kong and the low accessibility of the service. Furthermore, the caregiver stigma, multiple roles and responsibilities of caregivers and the lack of flexibility of the service have all restricted the participation of caregivers in programmes in its traditional face-to-face format. In view of the limitation, telepsychiatry has been suggested as a possible format in delivering psychoeducation to caregivers in Europe with demonstrable effectiveness in improving knowledge on the illness among the caregivers. Within this context, internet-based psychoeducation programme for caregivers of psychosis (iPEP) (www.ipep.hk) was established in 2013, funded by the Health Care Promotion Found (HCPF) to fill the gaps in the relevant services in Hong Kong.

DEVELOPMENT OF iPEP 2013-2019

The internet-based psychosis education programme for caregivers (iPEP) was established in collaboration with several local non-governmental organisations (NGOs) with the aim to provide up-to-date online information about psychosis and available resources. The needs of users with a wide age range were taken into consideration during the process of development. An example in point is the design of a simple operating interface. The content of iPEP comprises three components:

1) 34 short YouTube videos prepared and recorded by the healthcare professionals including psychiatrists, social workers and clinical psychologists ranging between 2 to 5 minutes each to provide detailed information about psychosis, such as aetiology of psychosis, different treatment modalities, recovery, relapse, side effects of medication, risk management and necessary skills as caregivers (Table 1). An article of the same content in PDF format is also available for downloading;

2) Compendium of local mental health services provided by both NGOs and the government, including services in hospitals, vocational training, residential care services and financial aids (17 articles) is provided as a one-stop resource centre; and

3) An interactive on-line forum to facilitate communication between the caregivers and healthcare professionals. Caregivers need to join as members of the website in order to access the forum, where they can post questions that would be answered by the professionals. Updated information will also be uploaded on the forum from time to time. Caregivers can also post questions directly to the website administrator through the ‘Contact Us’ function of the website. Response will be provided by the administrator or the healthcare team.

During the funding period (2013-2015) with active promotion, including public talks, there were 809
caregivers registered as iPEP members with a monthly average page view count of 3204 based on Google analytics (http://www.google.com/analytics/). Evaluation of the members’ experience of the use of iPEP has been conducted and revealed 85.2% of member caregivers subjectively agreed that the website improved their knowledge on psychosis; over 80% of them would recommend the website to others.17 After the funding ended in 2015, there are no further active promotion and publicity about the iPEP. However, the number of iPEP members has further increased to 939 with a cumulative pageview of 606,566 based on Google analytics at the end of 2019. Caregivers may get to know the website either via recommendation by others including healthcare providers or via their own online search. This feedback suggested that there is a need for comprehensive online resources for the caregivers. A total of 98 questions have been posted by the caregivers through the forum and the ‘Contact Us’ function over the last six years; most often-asked questions are medication-related, both of which suggest that there is a need for an anonymous online consultation platform for the caregivers.

FUTURE DEVELOPMENT OF iPEP AND POTENTIAL OF TELEPSYCHIATRY

Telepsychiatry is the use of communication technologies to provide psychiatric services from a distance and has been around for half a century. One of the main formats to provide psychiatric services from a distance has been the videoconferencing-based telepsychiatry, which has enabled and empowered service delivery, such as the chatbots, which allows for anonymity and accessibility. Although the effectiveness of using chatbots to assist the diagnosis of mental health disorders is still at its embryonic stage,19 its usage in providing health-related information in physical conditions20 and in delivering positive psychology21 has yielded some positive outcomes. The experience of iPEP suggested that there is a need for providing information about mental health illness and lending support to the caregivers online. This iPEP platform could form the basis for the future development of chatbots service for caregivers of psychosis and other mental health patients in Hong Kong.

CONCLUSION

There is a service gap in psychoeducation and in support to caregivers of patients with psychosis. The development of iPEP and its usage over the last six years have suggested that an online information resource and communication platform between caregivers and healthcare professionals could be a possible approach to fill the gaps of existing service. Our experience gathered from iPEP could pave the way for the future development of chatbots service for caregivers of patients with psychosis and other mental health. However, the integration of such services with the existing mental health service would be crucial for a comprehensive and cost-effective multidisciplinary service for patients with psychosis and other mental health conditions.

Table 1. Content and structure of iPEP website (Developed by author)

<table>
<thead>
<tr>
<th>Basic knowledge</th>
<th>Treatment</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. What is psychosis?</td>
<td>9. What are the treatments for psychosis?</td>
<td>15. What is recovery?</td>
</tr>
<tr>
<td>3. What is a hallucination?</td>
<td>11. How about treatments other than medication?</td>
<td>17. Can patients with psychosis go to work?</td>
</tr>
<tr>
<td>4. What is a delusion?</td>
<td>12. What are the uses and side effects of the medication?</td>
<td>18. What are the risk factors for relapse?</td>
</tr>
<tr>
<td>5. What are the negative symptoms?</td>
<td>13. How can side effects be managed?</td>
<td>19. How can relapse be prevented?</td>
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<tr>
<td>6. What are the stages of psychosis?</td>
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<tr>
<td>7. How is psychosis being diagnosed?</td>
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<tr>
<td>8. What causes psychosis?</td>
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Caregivers’ self-support

<table>
<thead>
<tr>
<th>Caregivers’ self-support</th>
<th>10. What might be caregiver be feeling?</th>
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<tbody>
<tr>
<td>11. What expectations should I have?</td>
<td></td>
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<tr>
<td>12. Am I stressed?</td>
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<tr>
<td>13. How can I relax myself?</td>
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<tr>
<td>14. How should I express praise and care?</td>
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<tr>
<td>15. How should I express discontent?</td>
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Tips on caring for patients

<table>
<thead>
<tr>
<th>Tips on caring for patients</th>
<th>1. Why is family support important?</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Suggested skills and attitude towards patients with psychosis?</td>
<td></td>
</tr>
<tr>
<td>3. Should I look after the patient long-term?</td>
<td></td>
</tr>
<tr>
<td>4. What should I do if the patient is reluctant to see the doctor or take medications?</td>
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<tr>
<td>5. What should we do when the patient is experiencing delusions or hallucinations?</td>
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<tr>
<td>6. Should I do if the patient lacks motivation?</td>
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<tr>
<td>7. What should we do if the patient is suicidal?</td>
<td></td>
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<tr>
<td>8. What should we do if the patient attempts to harm other people?</td>
<td></td>
</tr>
<tr>
<td>9. What are the procedures for involuntary admission?</td>
<td></td>
</tr>
</tbody>
</table>

References


INTRODUCTION

Traumatic man-made incidents and natural disasters can occur at any time in life, possibly resulting in post-traumatic stress disorder (PTSD), the onset and persistence of which tends to vary by trauma type, e.g. war, violence, an accident, or the death (or witnessing death) of a loved one. Furthermore, about half of PTSD patients have also been diagnosed with major depressive disorder. This article provides some clinical insights on the effective management of PTSD in the Hong Kong context and discusses the latest developments in scientific research as well as international guideline recommendations. Also, in view of the recent social unrest and encompassing psychiatric burden, it is hoped that this article will increase the awareness and preparedness of mental health professionals in handling related PTSD incidents.

TRAUMAS AND PTSD

In the World Health Organisation World Mental Health (WMH) Surveys that were conducted in 24 countries (2001-2012; n = 68,894), 70.4% of respondents had experienced lifetime traumas, at an average of 3.2 traumas per respondent. While disaster-related PTSD is relatively uncommon in high-income countries, it is worth noting that disasters caused by humans were associated with significantly higher odds of PTSD vs natural disasters (OR = 3.3, 95% CI: 1.1–9.7). In Hong Kong, preliminary findings of the Hong Kong Mental Morbidity Survey (2010-2013) reported that 65% of 4,644 adult participants had a traumatic experience, which was associated with higher Trauma Screening Questionnaire (TSQ) scores, higher psychological distress, lower social support, and lower life-functioning. Previous studies of the 2003 severe acute respiratory syndrome (SARS) epidemic showed that the cumulative prevalence of any psychiatric disorder among survivors was as high as 58.9%, and remained at 33.3% at 30 months; for PTSD, the prevalence was 25.6% in this group.

As our understanding of PTSD continues to evolve, the American Psychiatric Association has made several changes to the classification of PTSD in the 5th Edition of The Diagnostic and Statistical Manual of Mental Disorders (DSM-5; published in 2013). Notable changes include:

i) the creation of a new diagnostic category named “Trauma and Stressor-related Disorders”, which removes PTSD from the anxiety disorders category, indicating that PTSD is distinct in character from other anxiety disorders;

ii) in Criterion A: emotional reactions to the traumatic event (e.g. fear, helplessness, horror) are no longer a part of the criterion because it is noted that the emotional presentation of an individual varies. The definition of a traumatic event was further refined: stressful events without immediate threat to life or physical injury (e.g. divorce or job loss) are excluded, and medically-based trauma was limited to sudden catastrophe, which excludes non-immediate illnesses (e.g. terminal cancer) and medical incidents involving natural causes (e.g. heart attack). A fourth exposure type (A4) was added, which involves repeated or extreme exposure to aversive details of a traumatic event, which applies to work-related exposure as part of professional responsibilities (e.g. military mortuary services or child abuse investigations), but not to exposure through the mass media. Specifically, criterion A4 “does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work-related,” likely because of a lack of direct association between exposure to media reports, which is highly common in the society, and PTSD.

iii) An increase in the number of symptom groups from three to four: in addition to experiencing, avoidance and arousal, the group of negative cognitions and mood (i.e. Acute Stress Disorder), the International Classification of Diseases ICD-11, which was released in June 2018 and will come
into effect on January 1, 2022,9 has removed acute stress from the mental disorders section. The ICD-11 reclassified acute stress as a reaction to trauma and placed it in a section on factors influencing health. The intention is to recognize a non-pathological category of transient emotional, cognitive, behavioural and somatic reactions in response to trauma. Besides narrowing the definition of PTSD, the ICD-11 created a new complex PTSD category that comprises of six symptom clusters: they include not only the three clusters of experiencing, avoidance and arousal, but also difficulties in emotion regulation, problems with self-concept such as feelings of shame, guilt or failure, and disturbances in interpersonal relationship functioning.10 This new PTSD category describes the disturbance in self-organisation that may result from multiple, chronic or repeated traumas.11 The classification helps to distinguish patients whose conditions are focused on the trauma itself from those who experience extensive difficulties throughout their lives.

**PSYCHOLOGICAL MANAGEMENT**

Traumatic memory is a key concept in understanding the development of PTSD. Human beings have the ability to acquire, store and retrieve information, which helps our survival. Further, remembering fearful events may prime us to react more quickly to similar threats if they happen again. PTSD arises when the retention of fearful memories becomes extreme and persistent, which usually results from exposure to a traumatic event. Some authors suggested that the time between the traumatic event and the manifestation of PTSD are “golden hours”, during which memory of the trauma consolidates and spontaneous recovery takes place.12 The concept of golden hours is well-accepted in other areas of medicine, including, most famously, in cerebrovascular accidents where the administration of a thrombolytic agent is highly effective if given within the first three hours from the accident occurrence, but may otherwise be ineffective or even harmful. Applying an analogous concept of golden hours to PTSD provides some novel treatment insights. Interventions during these golden hours are aimed at preventing traumatic memory consolidation (i.e. secondary prevention) and not to deter spontaneous recovery.12

It is important to observe the patient during the first month after the occurrence of trauma before confirming the diagnosis and beginning treatment; such observation is termed watchful waiting or active monitoring. The United Kingdom’s National Institute for Health and Care Excellence (NICE) 2018 guideline recommends considering active monitoring within one month of a traumatic event (1.6.4 and 1.6.6).13

Of note, the NICE guideline (1.6.5) specifically discourages the use of psychologically focused debriefing for the prevention or treatment of PTSD.13 After the September 11, 2001, terrorist attacks on New York City, thousands of counsellors provided immediate debriefing services to survivors, which typically involved encouraging survivors to express their cognitive and emotional reactions, in addition to other advice and referral components.14 However, various studies generally showed that traumatised individuals who received psychological debriefing were doing significantly worse than those who did not, even years after the traumatic incidents.15 The process of debriefing may refresh and enhance the memory of the trauma and prevent spontaneous recovery from reducing it.15 Further, debriefing in group sessions may cause secondary traumatisation to the participants, where the emotional experiences of listening, understanding and caring for those who are traumatised can become a stressor.16

In recent years, the recommended practices during watchful waiting have shifted away from psychological debriefing toward psychological first aid (PFA).17,18 PFA aims to provide “basic, non-intrusive pragmatic care with a focus on listening but not forcing talk, assessing needs and concerns, ensuring that basic needs are met, encouraging social support from significant others and protecting from further harm.”19 Table 1 lists some of the basic components of PFA.

<table>
<thead>
<tr>
<th>Component</th>
<th>a. Contact and engagement</th>
<th>b. Safety and comfort</th>
<th>c. Protection from further harm and distress</th>
<th>d. Practical assistance</th>
<th>e. Reunion with family and loved ones (in person or by video/telephone)</th>
<th>f. Openness for sharing (but not forced)</th>
<th>g. Information on coping</th>
<th>h. Information or linkage with collaborative services</th>
</tr>
</thead>
</table>

For prevention of PTSD, besides watchful waiting/active monitoring, NICE also recommended individual trauma-focused cognitive behavioural therapy (TF-CBT) intervention to children, young people and adult who have clinical important symptoms of PTSD and have been exposed to trauma within the last month (1.6.6, 1.6.15). The core elements of TF-CBT include psychoeducation, coping strategies, gradual exposure, cognitive processing and caregiver participation, and robust study findings have supported its effectiveness.20 For the treatment of PTSD for children, young people and adult, i.e. those who have clinical important symptoms of PTSD who have presented more than one month after a traumatic event, NICE-recommended psychological interventions include TF-CBT (1.6.15-16) and eye movement desensitisation and reprocessing (EMDR; 1.6.18-19). In EMDR, re-exposure is combined with eye movements to allow for emotional processing of the patient’s excessively negative appraisals of the trauma.14 Results from a 2018 meta-analysis of 14 randomised controlled trials (RCTs) suggested that EMDR is better than CBT in reducing post-traumatic symptoms ($p = 0.006$; 11 studies) and anxiety ($p = 0.005$; 5 studies), but not depression ($p = 0.08$; 8 studies).21 Another meta-analysis of 11 RCTs suggested that EMDR may be more suitable than CBT for PTSD patients with prominent intrusion or arousal symptoms, while noting that the included studies were limited in both quantity and quality.22 While awaiting for further comparison...
studies, clinical applicability shall depend on the availability of proper training and expertise in these treatment areas.

Pharmacotherapy

In clinical practice, avoidance is commonly present among PTSD patients. Some patients decline or resist psychological therapy because it may touch on the traumatic experience, and prefer medication instead, especially when initiating treatment. For medication treatment, the NICE, American Psychological Association, and Maudsley guidelines generally recommend using selective serotonin reuptake inhibitor (SSRI) antidepressants (e.g. sertraline, fluoxetine or paroxetine) or the serotonin and norepinephrine reuptake inhibitor (SNRI) venlafaxine (modified release). These are usually given at a lower starting dose than when indicated for major depressive disorder (MDD). Patients who are resistant to SSRIs may be given mirtazapine, which also has a sedative effect to help with sleep. Anti-psychotics such as risperidone, which may help with flashbacks and nightmares (but are less helpful with avoidance and hyperarousal symptoms of PTSD), could be given at a very low dose.

However, benzodiazepines do not appear to be effective for sleep disturbances in PTSD, and some authors suggest that medication-induced deep sleep might interfere with the natural processing and extinction of traumatic memory. A 2016 meta-analysis of 6 RCTs suggested that the α-1-adrenergic receptor blocker prazosin produced moderate to large effects in reducing overall PTSD symptoms and nightmares, but similar results were not reproduced in a 2018 study with veterans (n = 304) vs placebo. The APA guideline also noted that the strength of evidence for prazosin in overall PTSD symptom reduction or remission is insufficient. Rat models and exploratory studies suggested that the β-adrenergic receptor antagonist propranolol may help to block traumatic memory reconsolidation, which will require further studies to support any clinical applications.

Note that due to the scarcity of evidence of an effect, the NICE guideline recommends against the use of drugs to: i) treat PTSD in children and young people aged < 18 years (1.6.14); and ii) prevent PTSD (1.6.24).

PTSD and Depression

The aetiology of PTSD and depression may be complex and interlinked: for example, a combination of high negative affect and low extraversion may constitute a pre-existing trait that confers vulnerability toward comorbid PTSD and MDD. Clinically, it is often difficult to differentiate PTSD from depression. While PTSD patients are often in a depressive mood, they may also have the depressive disorder as a comorbid condition. Statistically, while MDD is prevalent in about 50% of PTSD patients, it is not the case vice versa: a study of 1,346 MDD patients found that only 1.5% had comorbid PTSD. For differential diagnosis, the physician may observe the extent and severity of depression in the patient. For example, for a patient with comorbid depressive disorder, the experience of depression would tend to manifest outside of the traumatic experience, such as feeling useless or that survival is meaningless.

Studies on treatment for PTSD with co-existing depression are lacking. The NICE guideline recommends taking caution to treat the depression first if it is highly symptomatic or the patient poses a risk of self-harm (1.7.1). However, hypothetically, since the traumatic experience would likely aggravate the depressive mood, treating the PTSD first should help to alleviate the depression, too. Augmentation of antidepressant pharmacotherapy with low-potency antipsychotics have been documented in the literature, but cannot be regarded as evidence-based. Some preliminary data suggest that for PTSD patients with depression, trauma-focused interventions that include targeting comorbid depression may enhance intervention effectiveness and reduce dropouts.

Psychophysiological, neuroimaging, endocrinological and molecular genetic studies have identified biological markers for PTSD. For example, when compared with both healthy and trauma-exposed controls, PTSD patients demonstrated decreased grey matter volume within the subcortical (including the hippocampus and amygdala) and cortical brain regions. Decreased cortisol levels may also be observed, caused by “shutting-down” of the hypothalamic-pituitary-adrenal cortical (HPA) axis from negative feedback. Some studies suggested that antidepressant treatment may help reverse the stress-induced atrophy and promote neurogenesis of hippocampus.

CARING FOR HONG KONG PATIENTS

Traditionally, Hong Kong is regarded as one of the safest cities in the world, with low rates of homicides and disasters. Nevertheless, disasters may happen from time to time, such as the 2003 severe acute respiratory syndrome (SARS) epidemic, or the 2018 typhoon Mangkhut. Social unrests emerged in recent years, including the 2014 Occupy Central movement, 2016 Mongkok violence and the 2019 anti-Extradition Bill protests.

The Hong Kong FAMILY Cohort study reported that the prevalence of probable depression increased from 1.3% in 2011-2014 (n = 17,002) to 5.3% during the Occupy Central protests in 2014 and 9.1% in June-July 2019 (n = 1,269). Due to the extensive (active and passive) public involvement of the 2019 protests, citizens may have been exposed to potentially traumatic incidents in the form of threatened deaths, serious injuries, violence and/or related damages. The incidence of trauma- and stress-related disorders (and possibly some secondary traumatisation) would be expected to rise significantly.

During the 2013 civil unrest in Turkey, among 296 enrolled patients who sustained injuries from riot control and/or related exposure, 117 were psychiatically evaluated, and 43% were diagnosed with acute stress disorder, including some with subsequent PTSD and/or MDD. In the U.S., a survey of community members and police officers (n = 565) who were exposed...
to violence in the 2014 Ferguson, Missouri protests reported a high level of distress overall, and all assessed aspects of proximity to the violence (e.g., media, life interruption, and fear) were associated with mental health outcomes.57 When traumatic incidents occur, the uncertainties (for both patients and care professionals) and associated risks (e.g. to the patient’s loved ones) can also impose a heavy psychological burden on the society, with long-lasting effects.58 Consistent, protocol-based screening and referral of trauma patients could help to identify those who are at risk, allowing for the provision of timely monitoring and treatment if necessary.59

Trauma can produce intimate, practical consequences and psychological effects on one’s daily life. It is a common misconception that traumatic memories may be easily forgotten or even denied; on the contrary, PTSD patients often find it extremely difficult to forget these memories. For example, while SARS survivors were cured of the acute viral infection, in a ten-year follow-up study of a cohort of SARS survivors treated in the United Christian Hospital in Hong Kong with overall response rate of 69.3%, nearly one fifth (19.7%) still suffered from PTSD, followed by depressive disorder (14.7%) and panic disorder (4.9%). (unpublished data). Contrary to what one might wishfully expect of a “speedy recovery” from trauma, PTSD can be a long-term condition.50 In this regard, the physician must be extra-prudent in handling the patient’s memory of the traumatic incident, to maintain a trusting patient-physician relationship and to effectively manage this complex condition. It is hoped that this article may help to increase the awareness and preparedness of mental health professionals in handling PTSD cases. Special attention should be paid to the delicate and long-lasting nature of the condition, the need for watchful waiting, and the various recent developments in psychological and pharmacological management as discussed above. Voluntary screening and referrals of those who experienced trauma during the recent protests may facilitate case identification. As such, the foreseeable long-term psychiatric damages may be reduced.

References

Please read the article entitled “Time to Enhance Our Preparedness to Handle Trauma and Stress-related Disorders in Hong Kong” by Dr Ivan WC MAK 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 2020 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. Natural disasters were found to be associated with a significantly higher risk of development of post-traumatic stress disorder (PTSD) compared to disasters caused by humans.
2. In the 5th Edition of The Diagnostic and Statistical Manual of Mental Disorder (DSM-5), PTSD is indicated as being distinct in character from other anxiety disorders.
3. In the 11th Edition of International Classification of Disease (ICD-11), difficulties in emotion regulation, problems with self-concept, and disturbances in interpersonal relationship functioning are included as new clusters of symptoms.
4. The process of debriefing is strongly recommended for all survivors of traumatic events with proven evidence in significantly reducing the risk of subsequent development of PTSD.
5. The practice of psychological first aid (PFA) during the watchful waiting period is considered being “old-fashioned” and is not recommended in recent years.
6. When using antidepressant in the treatment of PTSD, a lower starting dose than for treating the major depressive disorder is recommended.
7. Benzodiazepines are found to be effective and beneficial for treating sleep disturbances in PTSD.
8. The UK National Institute for Health and Care Excellence (NICE) guideline does not recommend the use of medications to prevent the development of PTSD.
9. Patients with PTSD are found to have decreased grey matter volume within the subcortical and cortical brain region.
10. In a ten-year follow-up study of SARS patients in Hong Kong, only less than ten percent of them were still found to be suffering from PTSD.
Film Review
Shamans of the Blind Country –
A film by Michael Oppitz

Dr Wai-ching YAN
MBChB (HK), FHKCPsych, FHKAM (Psychiatry)
Specialist in Psychiatry
Associate Consultant, Department of Psychiatry, Kowloon Hospital

I well remember when we were in medical school, it was taught that for a proper physical examination, one should follow the sequence of inspection, palpitation, percussion and auscultation. The sequence cannot be altered (unless you want to fail your exam) and we practised over and over again with either our peers or real patients. When I was trained to be a psychiatrist, one of the first skills I was taught was the structure of a psychiatric history taking. I was asked to practice all the standard questions verbatim in order to elicit various psychopathologies. I memorised them hard enough to make sure I will not forget for the rest of my life.

After attaining my fellowship, I took up training as a psychotherapist. From the arrangement of my desk and chair, the way I and my client sit, to the intonation of my speech, every nuance is believed to influence the outcome of a session. I was coached by my supervisor to pay attention to every gesture of my clients, and to pick up important cues from my clients with appropriate reactions under the teaching of my psychotherapy school-of-thoughts. It took me a few years to master those skills before I became confident enough to lead a session with insightful communication with my clients.

From medical school to psychotherapy training, there have been loads of skills learning. The abilities to interact with patients properly, which we call soft skills, cannot be obtained from textbooks nor the internet. They were acquired through years of apprenticeship. So where did the apprenticeship begin? From where in history that a healer has started passing his skills to other people? Through what kind of ritualistic ceremony that skills of healing can be transferred from one generation to the next? If you are interested in tracing the origin, you could not possibly miss studying shamanism, which has been deeply rooted in almost every culture around the world.

Shamanism refers to the practice of a natural healer. Shamans have existed long before the birth of any civilisation and written language. Not many of us know what shamans exactly do because it is so varied across cultures. Moreover, very often, the training of being a shaman is through observation and oral teaching. The training details are difficult to be disseminated to outsiders. However, contrary to ordinary belief, the pathway to becoming a shaman can be extremely tedious. The chosen student has to go through a journey of rituals and trainings before he could be recognised as a shaman.

Because of self-protection as well as isolation from the modern world, it has been difficult for outsiders to study how a shaman is trained. Therefore, in order to have a glimpse of what shamanism is about, visual images are the best form of media that amateurs like us can depend on. Here I would like to recommend an epic documentary filmed by Michael Oppitz, who has provided the first vivid and in-depth look into this mysterious Shamanic training in a rural part of Nepal.

Michael Oppitz is an anthropologist and sinologist. From 1992 till 2008, he was a professor at the University of Zurich/Switzerland and Director of the Ethnographic Museum Zurich. Much early on in 1965, he came to Nepal for the first time and studied faith healers in a remote mountain region of north-western Nepal. Based on having spent years observing and living with the people there, he directed the four-hour documentary Shamans of the Blind Country in the 1970s with great impact on our understanding of shamanism. The film was first shown in Berlin Film Festival in 1981 with great success. Thirty-odd years later, a digitally remastered version was put on screen in the 64th Berlin Film Festival.

The documentary shows shamanism in a Himalayan community called Magar. The documentary follows the arduous process of initiation that each student has to go through before he or she can be chosen as healers. The film captures many rituals the shamans perform to cure their patients’ illnesses and to avert their misfortune. The film is both comprehensive and largely observational. Any facets of this shamanism are presented in a visual language that suits the Magar perspective, disregarding all theoretical explanation that usually happens in this kind of ethnographic films.
The documentary comprises two Parts. Part I displays a variety of healing rituals carried out by the shamans in the Dhaulagiri region. Part II focuses on the transmission of the shaman’s knowledge from master to student. The transmission is by the oral route, hence no books involved. It all happens in vicarious learning, where the student is requested to watch and imitate the master’s performance. The performance is complicated involving the manufacture of the required tools, preparation of a sacred spot or altar, conducting the operations in the right order, and above all, learning the mythical chants, the original stories and auxiliary chants, echoing the master line by line to the beat of his drum. You can imagine it takes a very long time to master those skills especially when there is no written language in the Magar community.

This four-hour documentary is definitely not the Hollywood type of movies which are filled with superheroes’ flying and fighting against each other. It demands both your attention span and patience. However, if you can immerse yourself in a culture of shamanism, you may try to compare their ritualistic practice with our modern medicine. It is always fun to compare and you will be amazed to see the similarities.
### Medical Diary of March

#### Sunday
- **1**
  - Primary Care Forum

#### Monday
- **2**
  - Colorectal Cancer Prevention and Treatment Updates
  - 1) Advanced in Neurorehabilitation
  - 1) Management of Hypertension with Vasodilating Beta-blockers
  - Update on the Management of Erectile Dysfunction

#### Tuesday
- **3**
  - HKMA-HKSHS CME Programme 2019-2020
  - Colorectal Cancer Prevention and Treatment Updates
  - 1) Advanced in Neurorehabilitation
  - 1) Management of Hypertension with Vasodilating Beta-blockers
  - Update on the Management of Erectile Dysfunction

#### Wednesday
- **4**
  - Certificate Course on Update in Clinical Sleep Medicine 2020
  - Certificate Course on Neurology
  - Certificate Course on Update in Clinical Sleep Medicine 2020

#### Thursday
- **5**
  - Certificate Course in Cardiology 2020
  - Certificate Course in Cardiology 2020
  - Certificate Course in Cardiology 2020

#### Friday
- **6**
  - Mastering Adverse Outcomes
  - Certificate Course in Cardiology 2020
  - Certificate Course in Cardiology 2020

#### Saturday
- **7**
  - Refresher Course for Health Care Providers 2020 in caring for elderly in family practice
  - Mastering Adverse Outcomes
  - Certificate Course on Lower Urinary Tract Symptoms (LUTS) Management
  - Better HT Management with the Right SPC from Control to Protection
<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Function</th>
<th>Enquiry / Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9:00 PM</td>
<td>Primary Care Forum&lt;br&gt;1. Incidence of Type II Diabetes in Chronic Obstructive Pulmonary Disease (COPD)&lt;br&gt;2. Opportunities to diagnose COPD at an earlier stage in primary care&lt;br&gt;3. Update in Asthma Clinical Practice Guidelines (GINA, 2019)&lt;br&gt;4. SABA: An appropriate reliever in asthma?&lt;br&gt;5. How to achieve high vaccination rate in primary care&lt;br&gt;6. Helicobacter pylori testing and treatment in primary care&lt;br&gt;7. Optimizing the management of IBS-C&lt;br&gt;Organiser: Hong Kong Medical Association; Speaker: Dr. Hugo TAN, Dr. YU Wai Cho, Dr. Thomas YW MOK, Dr. Matthew WONG, Prof. Fanny KO, Prof. Ivan HUNG &amp; Dr. Michael KS CHEUNG; Venue: Eaton Hotel</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;3 CME Points</td>
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<tr>
<td>3</td>
<td>1:00 PM</td>
<td>HKMA-HKS&amp;B CME Programme 2019-2020&lt;br&gt;Topic: Surgical Treatment of Urological Cancers&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Hong Kong Sanatorium &amp; Hospital; Speaker: Dr. YIU Ming Kwong; Venue: The HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;1 CME Point</td>
</tr>
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<td></td>
<td>9:00 PM</td>
<td>HKMA Council Meeting&lt;br&gt;Organiser: The Hong Kong Medical Association; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, HK</td>
<td>Ms. Christine WONG&lt;br&gt;Tel: 2527 8285</td>
</tr>
<tr>
<td>4</td>
<td>7:00 PM</td>
<td>Certificate Course on Update in Clinical Sleep Medicine 2020&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8858</td>
</tr>
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<td>5</td>
<td>7:00 PM</td>
<td>Certificate Course in Cardiology 2020&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8858</td>
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<td>7</td>
<td>2:15 PM</td>
<td>Refresher Course for Health Care Providers 2019/2020 - Caring for elderly in family practice&lt;br&gt;Organiser: Hong Kong Medical Association, HK College of Family Physicians &amp; H&amp;A-Our Lady of Maryknoll Hospital; Speaker: Dr. LAM Wing Wo; Venue: Lecture Halls A&amp;B, 4/F, Block G, Wong Tai Sin Hospital</td>
<td>Ms. Clara TSANG&lt;br&gt;2354 2440&lt;br&gt;2 CME Points</td>
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<td>10</td>
<td>1:00 PM</td>
<td>Colorectal Cancer Prevention and Treatment Updates&lt;br&gt;Organiser: HKMA-UTM Community Network; Speaker: Dr. CHOW Man Po; Venue: Crystal Ballroom, 2/F, The Cityview Hong Kong, 23 Waterloo Road, Kowloon</td>
<td>Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;1 CME Point&lt;br&gt;Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;2 CME Points</td>
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<td>1:00 PM</td>
<td>Certificate Course on Neurology&lt;br&gt;1) Dementia Journey From Practical Management to Case Sharing&lt;br&gt;Organiser: HKMA-Central, Western &amp; Southern Community Network; Speaker: Dr. LUI Wing Cheong, Victor; Venue: The HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td>7:00 PM</td>
<td>Certificate Course on Update in Clinical Sleep Medicine 2020&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8858</td>
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<td>12</td>
<td>1:00 PM</td>
<td>Hearing Loss - A Microscopic View&lt;br&gt;Organiser: HKMA-New Territories West Community Network; Speaker: Dr. CHOW Siu Wah; Jennifer; Venue: Pak Lo Chiu Chow Restaurant, Shop A316, 3/F, Yoho Mall II, 8 Long Yat Road, Yuen Long</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td>1:00 PM</td>
<td>Palliative Home Care in the Eye of Family Physicians&lt;br&gt;Organiser: HKMA-HK East Community Network &amp; Haven of Hope Christian Service; Speaker: Dr. CHAN Hung Wai, Patrick, Dr. TSANG Chi Yee, Luke &amp; Dr. KONG Wing Ming; Henry; Venue: The HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai</td>
<td>Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td>7:00 PM</td>
<td>Certificate Course in Cardiology 2020&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8858</td>
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<td>14</td>
<td>2:30 PM</td>
<td>Mastering Adverse Outcomes&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;3 CME Points</td>
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<td>18</td>
<td>1:00 PM</td>
<td>Certificate Course on Neurology&lt;br&gt;2) Prevention Strategies and Treatments for Migraine and Headache&lt;br&gt;Organiser: HKMA-Central, Western &amp; Southern Community Network; Speaker: Dr. TSANG Kin Lun; Venue: The HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;1 CME Point</td>
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<td>6:30 PM</td>
<td>Building Resilience and Avoiding Burnout&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Medical Protection Society; Speaker: Dr. FUNG Shu Yan, Anthony; Venue: The Cityview, 23 Waterloo Road, Kowloon, Hong Kong</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;3 CME Points</td>
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<td>7:00 PM</td>
<td>Certificate Course on Update in Clinical Sleep Medicine 2020&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8858</td>
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<td>19</td>
<td>1:00 PM</td>
<td>Legal Aspect of End of Life Care&lt;br&gt;Organiser: HKMA-HK East Community Network &amp; Haven of Hope Christian Service; Speaker: Ms. Olivia LIEUNG; Venue: The HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai</td>
<td>Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td>1:00 PM</td>
<td>Childhood Anxiety Disorder&lt;br&gt;Organiser: HKMA-KLN East Community Network; Speaker: Dr. LIN Hoy Yun, Candy; Venue: V Cuisine, 6/F, Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tsuen Kwan O</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>19 THU</strong> 7:00 PM</td>
<td><strong>Certificate Course in Cardiology 2020</strong>&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong&lt;br&gt;Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Vienna LAM&lt;br&gt;Tel: 2527 8898</td>
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<td><strong>FRI</strong> 8:00 PM</td>
<td><strong>FMSHK Executive Committee Meeting</strong>&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong&lt;br&gt;Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Nancy CHAN&lt;br&gt;Tel: 2527 8998</td>
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<td><strong>20 FRI</strong> 1:00 PM</td>
<td><strong>Dementia Revisited: New “Kids” on Board Helpful for Diagnosis and Treatment</strong>&lt;br&gt;Organiser: HKMA-KLN City Community Network; Speaker: Dr. CHAN Chun Chung, Ray; Venue: President's Room, Spotlight Recreation Club, 4/F, Screen World, Site 8, Whampoa Garden, Hunghom, Kowloon</td>
<td>Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>21 SAT</strong> 1:00 PM</td>
<td><strong>Certificate Course on Lower Urinary Tract Symptoms (LUTS) management</strong> 4. LUTS Management (Tips And Risk In Primary Care Setting) 5. LUTS And Anticholinergic Burden (ACB) 6. LUTS and Hearts Diseases&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Hong Kong Elderly Welfare Foundation; Speaker: Dr. MAK Siu King, Dr. Jennifer Ma Wai Wai MYINT &amp; Dr. WONG Tai Hung, John; Venue: Telemedicine and Conference Hall, 29/F, International Medical Centre, One Chinachem Central, 22 Des Voeux Road Central, Hong Kong (MTR Central Station Exit C)</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;2 CME Points</td>
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<td><strong>24 TUE</strong> 1:00 PM</td>
<td><strong>Certificate Course in Neurology</strong> 3) The Update of Parkinson’s Disease&lt;br&gt;Organiser: HKMA-Central, Western &amp; Southern Community Network; Speaker: Dr. TSANG Kin Lun; Venue: The HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>26 THU</strong> 1:00 PM</td>
<td><strong>Leading the Shift in Paradigm in T2D Treatments: Cardio-Protection with Glucose-Lowering Drug</strong>&lt;br&gt;Organiser: HKMA-New Territories West Community Network; Speaker: Dr. LAU Chun Leung; Venue: Atirum Function Rooms, Lobby Floor, Hong Kong Gold Coast Hotel, 1 Castle Peak Road, Gold Coast, Hong Kong</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>27 FRI</strong> 1:00 PM</td>
<td><strong>Non Surgical Treatment of Benign Thyroid Nodule</strong>&lt;br&gt;Organiser: HKMA-YTM Community Network; Speaker: Dr. KAN Mei Yee, Daisy; Venue: Crystal Ballroom, 2/F, The Cityview Hong Kong, 23 Waterloo Road, Kowloon</td>
<td>Ms. Candice TONG&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>28 SAT</strong> 2:30 PM</td>
<td><strong>Achieving Safer and Reliable Practice</strong>&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 21-22 Connaught Road, Central, Hong Kong</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;5 CME Point</td>
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<td><strong>31 TUE</strong> 1:00 PM</td>
<td><strong>Sarcopenia and Frailty</strong>&lt;br&gt;Organiser: HKMA-KLN West Community Network; Speaker: Dr. CHUANG Lai &amp; Dr. CHAN Fei; Venue: Fulum Palace, Shop C, G/F, 85 Broadway Street, Mei Foo Sun Chuen</td>
<td>Miss Antonia LEE&lt;br&gt;2527 8285&lt;br&gt;1 CME Point</td>
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<td><strong>31 TUE</strong> 6:30 PM</td>
<td><strong>Mastering Shared Decision Making</strong>&lt;br&gt;Organiser: Hong Kong Medical Association &amp; Medical Protection Society; Speaker: Dr. CHENG Ngai Shing, Justin; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong</td>
<td>HKMA CME Department&lt;br&gt;2527 8452&lt;br&gt;3 CME Points</td>
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That smile is not only for the kiss

Treatment of major depressive episodes in adults*
One tablet daily at bedtime

Valdoxan®, agomelatine
Re-emerging life

*In patients who have not responded adequately to at least one previous antidepressant therapy.
Dermatology Quiz

Answers to Dermatology Quiz

Answers:

1. Malignant acral lentiginous melanoma, Pigmented basal cell carcinoma, Secondary cutaneous metastasis, Dysplastic naevus, Squamous cell carcinoma and so on can be the possible differential diagnoses.

Malignant acral lentiginous melanoma is a subtype of malignant melanoma which is more commonly found amongst Asian and sub-Saharan African. It is due to the development of malignant melanocytes along the basal layer of the epidermis. These malignant cells may arise from an existing melanocytic naevus or more often, de novo, from previously normal-appearing skin.

2. Dermoscopy by a trained doctor can be helpful to find clues such as asymmetrical structure and colours, multi-component pattern, parallel ridge pattern and diffuse pigmentation of different shades of brown colours suggesting melanoma. Skin biopsy is still necessary for differentiating the above-mentioned differential diagnoses and confirming malignant melanoma. Although no single histologic feature is pathognomonic for melanoma, there are few characteristic features such as cytologic atypia with enlarged cells with numerous mitotic figures. Immunohistochemical stains such as S-100 and homatropine methylbromide (HMB 45) can help to differentiate melanoma from other lesions.

For small and primary malignant melanoma, wide excision is the mainstay of treatment. Sentinel lymph node biopsy should be offered if the melanoma is thick. For advanced disease, chemotherapy may be needed, especially for widespread disease. Nowadays, immunotherapy and biologicals such as ipilimumab and BRAF inhibitors, in which around 60% of melanoma has BRAF mutation, such as vemurafenib, dabrafenib and trametinib are all recently approved by FDA in treatment of unresectable or metastatic BRAF mutation melanoma are showing the promising result.

Dr Chi-keung KWAN
MBBS(HK), FRCP(Lond, Glasg), Dip Derm(Glasg), PDipID(HK), FHKCP, FHKÂM(Med)
Specialist in Dermatology and Venereology

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Tel: 2527 8898 Fax: 2865 0345

Dr Mario Wai-kwong CHAK 翟偉光醫生
Prof Bernard Man-yung CHEUNG 陳文勇教授
Dr Chun-kong NG 吳振江醫生

Mr Benjamin Cheung-mei LEE 李祥美先生
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President
Dr Raymond See-ki LO 劉思傑醫生

Immediate Past President
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The Hong Kong Medical Association
香港醫學會

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

THE HONG KONG MEDICAL DIARY
A FRESH STEP IN LUTS+ MANAGEMENT

*OAB: Overactive Bladder + LUTS: Lower Urinary Tract Symptoms
# α1-blockers are often considered the first line drug treatment of male LUTS+

ABCPharma Hong Kong Co., Ltd. Unit 1103-08, 11/F., Tower 1, Grand Century Place, 193 Prince Edward Road West, Mongkok, Kowloon.
Tel (852) 2377 9801 Fax (852) 2866 1440

THE 1ST β3-AGONIST FOR OAB+ PATIENTS WITH PROMISING SAFETY PROFILE
PLACEBO-LIKE DRY MOUTH(1.7%) SIDE EFFECT

YOUR 1ST STEP FOR MALE LUTS+ PATIENTS WITH PROMISING SAFETY PROFILE
PLACEBO-LIKE DIZZINESS(1.4%) SIDE EFFECT

Abbreviated prescribing information of Harnal® OCAS® 6.4 mg Tablets
Version: 001 Provision Date: 2023-03-15
Indications: Tamsulosin HCL Tablets are indicated for: lower urinary tract symptoms (LUTS) associated with benign prostatic hyperplasia (BPH). Dosage: 1 tab, daily, can be taken independently of food. Administration: Swallow whole, do not chew. Contraindications: Hypersensitivity to tamsulosin hydrochloride or any of the excipients. Special warnings and special precautions for use: As with other α1-adrenergic antagonists, a reduction in blood pressure can occur in subjects during treatment with Harnal® OCAS 6.4 mg Tablets, as a result of which, rarely, syncope can occur at the first signs of orthostatic hypotension (dizziness, weakness), the patient should sit or lie down until the symptoms have disappeared. Before therapy with Harnal® OCAS 6.4 mg Tablets is initiated, the patient should be examined in order to evaluate the presence of other conditions, which can cause the same symptoms as benign prostatic hyperplasia. Digital rectal examination and, if necessary, determination of prostatic specific antigen (PSA) should be performed before treatment and at regular intervals afterwards. Treatment of patients with a history of orthostatic hypotension should be approached with caution. The treatment of patients with severe renal impairment (creatinine clearance of <10 mL/min should be approached with caution, as these patients have been studied. The treatment of patients with severe hepatic dysfunction is not recommended. During perioperative assessment, surgeons and anesthesiologists should consider whether patients scheduled for cataract or glaucoma surgery are being or have been treated with tamsulosin in order to ensure that appropriate monitoring will be in place to manage the FS during surgery. Tamsulosin hydrochloride should not be given in combination with strong inhibitors of CYP3A4 in patients with prior myocardial CYP3A4 substrates. Tamsulosin hydrochloride should be used with caution in combination with strong and moderate inhibitors of CYP3A4. Caution should be exercised in patients with a history of traumatic head injury, as well as in patients with a history of psychiatric disturbances. A comprehensive list of drugs that inhibit CYP3A4 is available from the U.S. National Library of Medicine's Drug Information Online (www.nlm.nih.gov/medlineplus/druginformationonline.html).

Abbreviated prescribing information of Betmiga® prolonged-release capsules
Version: 001 Provision Date: 2023-04-22
Indications: Mirabegron Indications: Symptomatic treatment of overactive bladder (OAB) syndrome. Dosage: Adult incontinence: 50 mg once daily with or without food. Administration: Swallow whole with liquids. Do not chew/ divide/ crush. Contraindications: Mirabegron is contraindicated in patients with - Hypersensitivity to the active substance or any of the excipients. - Moderate to severe hepatic impairment (Child-Pugh Class B or C) - Concurrent treatment with strong CYP3A4 inhibitors, strong CYP3A4 inducers, or strong inducers of CYP3A4. - Active liver disease (including hepatitis and cirrhosis). - History of or current treatment with tamsulosin hydrochloride or other α1-adrenergic antagonists. - Concurrent treatment with a drug that inhibits CYP3A4 by >50%, or is a strong CYP3A4 inducer. - Patients with uncontrolled hypertension (systolic blood pressure >180 mmHg, diastolic blood pressure >110 mmHg). - Patients with a history of respiratory tract infections. - Patients with a history of preexisting cardiovascular disease. - Patients with a history of severe renal impairment (creatinine clearance <30 mL/min). - Patients with a history of uncontrolled glaucoma. - Patients with a history of uncontrolled hypertension. - Patients with a history of uncontrolled diabetes mellitus. - Patients with a history of uncontrolled hyperlipidemia. - Patients with a history of uncontrolled liver disease. - Patients with a history of uncontrolled pulmonary disease. - Patients with a history of uncontrolled lung disease. - Patients with a history of uncontrolled cancer. - Patients with a history of uncontrolled anemia. - Patients with a history of uncontrolled infectious disease. - Patients with a history of uncontrolled allergy. - Patients with a history of uncontrolled asthma. - Patients with a history of uncontrolled respiratory disease. - Patients with a history of uncontrolled cardiovascular disease. - Patients with a history of uncontrolled gastrointestinal disease. - Patients with a history of uncontrolled neurological disease. - Patients with a history of uncontrolled psychiatric disease. - Patients with a history of uncontrolled urological disease. - Patients with a history of uncontrolled ophthalmological disease. - Patients with a history of uncontrolled dermatological disease. - Patients with a history of uncontrolled hematological disease. - Patients with a history of uncontrolled infectious disease. - Patients with a history of uncontrolled respiratory disease.
Ketosteril®
Gain Precious Time

Ketosteril®-supplemented vegetarian very low protein diet (VLPD) could defer dialysis initiation by ameliorating metabolic disturbances associated with chronic kidney disease (CKD).

Name of the medicinal product: Ketosteril® film-coated tablets. Composition: One film-coated tablet contains: (DL)-3-methyl-2-oxovaleric acid (β-ketoanalogue to DL-isoleucine, Casval) 67 mg, 4-methyl-3-oxovaleric acid (β-ketoanalogue to isoviscine, Casval) 107 mg, 2-(R)-3-phenylpropionic acid (β-ketoanalogue to phenylalanine, Casval) 88 mg, 3-methyl-2-oxobutyric acid (β-ketoanalogue to valine, Casval) 96 mg, (DL)-hydroxy-2-methylbutyric acid (β-hydroxyalanine to DL-methionine, Casval) 59 mg, L-Lysine acetate 105 mg (+ 75 mg L-lysine), L-Phenylalanine 63 mg, L-leucine 23 mg, L-tyrosine 36 mg, L-tyrosine 30 mg, total nitrogen content per tablet 36 mg, calcium content per tablet 1.25 mmol = 50 mg. Excipients: Maize starch, crospovidone type A, talc, silica (collodial anhydrous), magnesium stearate (PH.EUR) (vegetable), macrogol 6000, quinoline yellow E104, basic butylated methacrylate copolymer, triacetin, titanium dioxide E171, povidone K 29-32. Therapeutic indications: Prevention and treatment of damages due to faulty or deficient protein metabolism in chronic kidney disease in connection with a limited dietary protein intake of 40 g/day or less (adult). Usually this applies to patients whose glomerular filtration rate (GFR) is less than 25 ml/min. Precaution and method of administration: If not otherwise prescribed the dose for adults (170 kg body weight) is 4 to 8 tablets three times daily during meals. The tablets must not be chewed. Ingestion during meals facilitates proper absorption and the metabolisation into the corresponding amino acids. Contraindications: Hypersensitivity to the active substances or to any of the excipients, hypercalcemia and disturbed aminoacid metabolism. Special warnings and precautions for use: The serum calcium level should be monitored regularly. A sufficient supply of calories should be ensured. No experience has been gained so far with the administration in paediatric patients. In the presence of hereditary phenylketonuria, attention should be given to the fact that Ketosteril® contains phenylalanine. Monitoring of the serum phosphate levels is needed in case of concomitant administration of aluminium hydroxide. Interaction with other medicinal products and other forms of interaction: Concomitant administration of calcium-containing drugs may cause or aggravate elevated serum calcium levels. Drugs that form hardly soluble compounds with calcium (e.g. tetracyclines, quinolines such as ofloxacin and norfloxacin as well as drugs containing iron, fluoride or strontium) should not be taken at the same time with Ketosteril® to avoid disturbed absorption of the active substances. An interval of at least two hours should elapse between the ingestion of Ketosteril® and these drugs. The susceptibility to cardiovascular glycosides, and hence the risk for arrhythmia will increase if Ketosteril® produces elevated serum calcium levels. Lactic acidosis improves under therapy with Ketosteril®. Thus, in case of aluminium hydroxide administration, the dose of this drug has to be reduced if necessary. Serum phosphate levels should be monitored for a decrease. Pregnancy and lactation: There are no adequate data from the use of Ketosteril® in pregnant women. Animal studies do not indicate direct or indirect harmful effects with respect to pregnancy, embryonal/fetal development, parturition or postnatal development. Caution should be exercised when prescribing to pregnant women. No experience has been made so far with the use during lactation. Undesirable effects: The intake of Ketosteril® may very rarely lead to hypercalcemia. If hypercalcemia occurs, the intake of vitamin D should be reduced. In case of persistent hypercalcemia, the dose of Ketosteril® as well as the intake of any other calcium sources has to be reduced. Overdose: No case of overdose has been reported. Special precautions for handling/storage: Do not use Ketosteril® after expiry date. Keep out of the reach of children. Do not store above 25°C. Store in the original package and keep the blister tightly closed to protect contents from moisture.

Reference code: 7946837/03