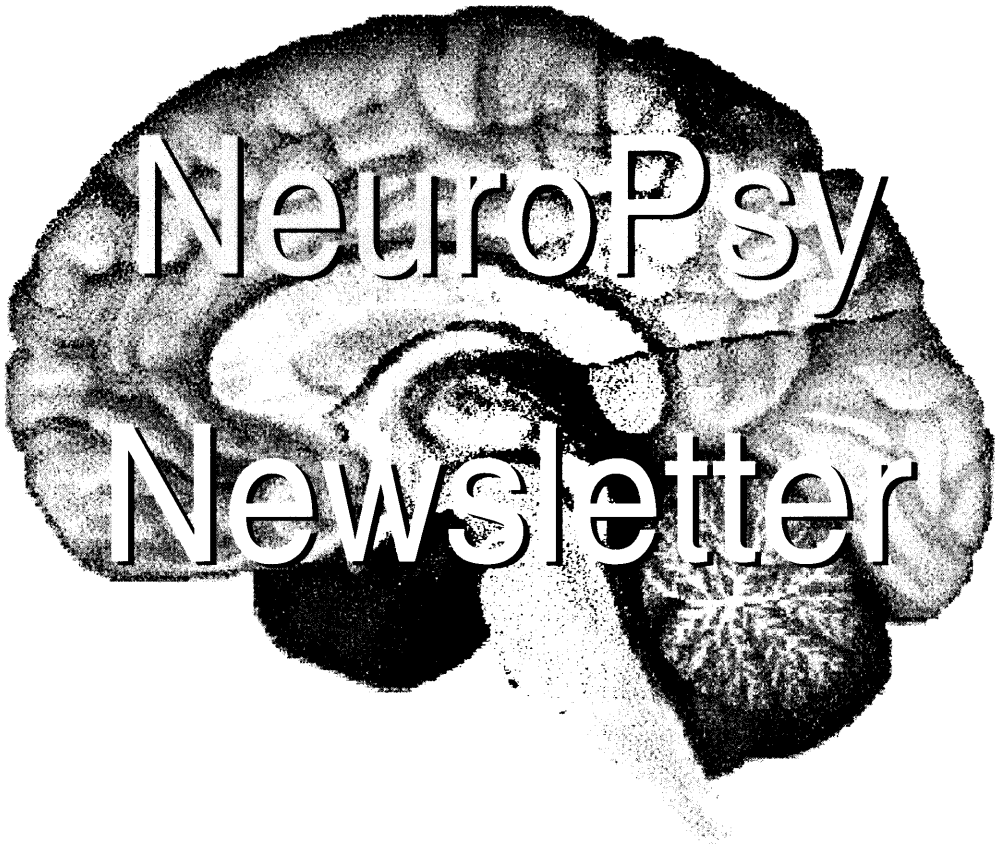
Hong Kong Neuropsychological Association

c/o Clinical Psychology Unit, Children's Habilitation Institute.
The Duchess of Kent Children's Hospital at Sandy Bay,
12 Sandy Bay Road, Hong Kong.
Tel.: 2974-0351 Fax: 2974-0366

April 2002



About Hong Kong Neuropsychological Association

The HKNA was established in October 1998. It aims to promote the advancement of knowledge, to encourage and facilitate clinical and theoretical research in neuropsychology in Hong Kong, and to promote and facilitate communication with relevant professional organizations within the local community, mainland and overseas. The activities of the Association include publication of newsletter, organization of interest/discussion groups, arranging seminars/workshops run by local and overseas speakers, and conducting research. Interested parties please fill in application form which can be obtained from the association.

Recent Activities

18/1 - 21/1 2002 Developmental Neuropsychology (Child Assessment Service, Department of Health), Mr. Tim Hannan

2/2/2002 Neuropsychological Phenomena of Obsessive Compulsive Disorder (Mary Wong, Clinical Psychologist, PYNEH)

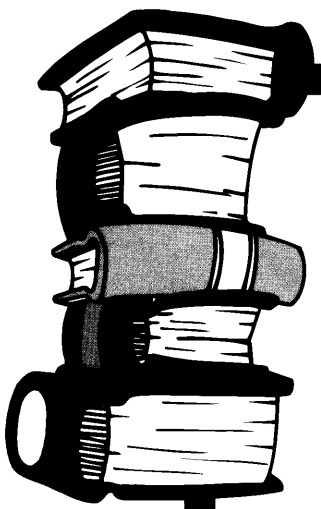
Coming Activities

Date: 4th May 2002 (Sat.)
Time: 2: 30 p.m.
Venue: 1/F, Block B, Queen Elizabeth Hospital (to be confirmed)
Topic: Evidence-based Cognitive Rehabilitation Recommendations for Clinical Practice (1)

Date: 3rd August 2002 (Sat.)
Time: 2:30 p.m.
Venue: 1/F, Block B. Queen Elizabeth Hospital (to be confirmed)
Topic: Evidence-based Cognitive Rehabilitation Recommendations for Clinical Practice (2)

Workshop on Developmental Neuropsychology, Mr. Tim Hannan

The Commissioned Training for the Department of Heath (Child Assessment Service) between Jan 18 - 21, 2002 was given by Mr. Tim Hannan, an experienced clinical neuropsychologist who worked in a children hospital in Sydney, Australia. On the first day of the training, Mr. Hannan provided a 1-day workshop for the staff about developmental cognitive neuropsychology which included overview about theories and methods drawn from cognitive neuropsychology in the analysis of developmental and acquired cognitive disorder in childhood. Furthermore, the workshop provided a comprehensive introduction to the application of these theories and methods in clinical practice with children. The other 3 days of the training were about specific assessment and rehabilitation on common children problems such as attention deficit (hyperactive) disorder, specific learning difficulties and traumatic brain injuries in children. In addition, updated neuropsychological assessment on intelligence, language, memory and executive were shared.

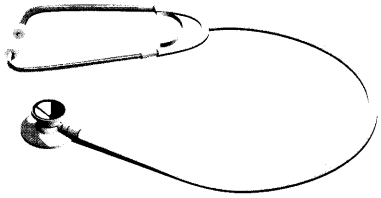


Book Review

A Man Who Mistook His Wife for A Hat - Oliver Sacks

(Ho Yim-chi, postgraduate student in psychology, CUHK)

A man who was not physically blind mistook his wife's head for a hat; a woman's mind was separated from her body and so she could never know what her body was doing without consciously monitoring it; another man tried to tear his own leg off his body because he did not feel it as part of him; remote memories that had been "forgotten" several decades ago could suddenly be retrieved with great details; a mentally retarded individual could memorize every details of an opera after a single hearing, and etc. These stories sound interesting like funny jokes, mysterious like ghost stories, unbelievable like a pack of lies, but they are real life experiences of Dr. Sacks's patients with various neurological disorders. After reading the clinical tales, one could not only gain a deeper understanding about the innocence, difficulties, frustration and horror of the patients and their families, but also appreciate the ways they struggled to live with their disorders, the outstanding talents of their spared functioning, and even the wisdom of them to take advantage out of their disadvantages.



Neuropsychological Phenomena of Obsessive Compulsive Disorder

(Mary Wong, Clinical Psychologist, PYNEH)

According to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), Obsessive Compulsive Disorder (OCD) is characterized by repetitive experience of unwanted and recurrent or perseverative thoughts (obsessions) and / or a compulsion to repetitively do certain acts that are severe enough to be time consuming (more than 1 hour per day) or caused marked distress or significant impairment (interference with normal routine, work, social activities or relationships). At some point during the course of this disorder, the patient recognizes that the obsessions or compulsions are excessive.

Obssessions are defined by the following 4 criteria: (1) recurrent, persistent thoughts, impulses, images as intrusive and inappropriate and that caused marked anxiety or distress; (2) not just excessive worries about real-life problems; (3) attempts to ignore or suppress these; (4) recognizes these are a product of his/ her mind. Obsessions usually have themes of concern with contamination (thus, associated with cleaning and washing rituals) and intrusive unwanted unpleasant (e.g. violent and/or sexual) images.

In DSM-IV, compulsions are defined by both (1) repetitive behaviors (e.g. hand washing, checking) or mental acts (e.g. praying) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly; (2) behaviors or mental acts aimed at preventing or reducing distress or preventing some dreaded event, but are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive.

OCD has a one-year prevalence of 1.5 - 2.1%, and a life-time prevalence of 2.5%. It is often associated with depression and other types of anxiety problems.

OCD could be conceptualized in neuropsychological terms as disturbances in emotion regulation (heightened arousal, excessive anxiety), impulse control (urge to perform compulsive and perseverative motor responses) and memory (e.g. a patient keeps checking whether a stove has been turned off because he cannot remember if he has already locked it). Thus, the limbic system (which mediates memory, emotion, learning, attention, and arousal) and the orbital frontal areas (which gates and inhibits limbic system activity) could be involved in OCD. Within the basal ganglia, the corpus striatum (consisting of several complex motor correlation centres - the caudate, and the putamen which modulate both voluntary movement and autonomic reactions), amygdala, and hypothalamus (which regulates appetite, sexual arousal and thirst) have been hypothesized to be particularly implicated in OCD.

In the past decade, overall evidence from nuclear brain-imaging techniques, psychopharmacological studies of OCD patients indicated the orbito-frontal and anterior cingulate-basal ganglia-thalamocortical circuits as representing the neuroanatomical substrate for OCD.

The following studies are some of which showing brain areas involved in OCD:

1) Amen (1998) used Single Photon Emission Computerized Tomography (SPECT; which allowed a 3-D depiction of cerebral blood flow and metabolic activity patterns in the brain). He found over-activation in the cingulate gyrus (on top of the corpus callosum). The anterior cingulate is concerned with emotional functioning and regulating autonomic and endocrine activities. The posterior cingulate is concerned with visual-spatial and tactile analysis, motor output and memory;
2) McGuire et al. (1994) used event-related functional MRI to study medication-free OCD patients' brain pattern when presented with an individualized hierarchy of contaminants which would elicit anxiety and a compulsion to handwash. They found significant correlation between symptom intensity and enhanced neural responses in the inferior frontal gyrus, caudate nucleus, putamen, globus pallidus, and thalamus on the right side and hippocampus, posterior cingulate gyrus, and cuneus on the left side.
3) Breiter et al. (1996), using fMRI and a symptom provocation model, found activation in medial orbitofrontal, lateral frontal, anterior temporal, and insular cortices in two-thirds of their OCD patients.
4) Hypermetabolism in caudate nucleus (Baxter et al., 1992, Rauch et al., 1994) and reduced caudate nucleus volume (Robinson et al.,1995) have also been reported.

In terms of treatment, medication involving brain serotonin systems and psychological treatment (cognitive and behavioral procedures, particularly exposure and response prevention) have efficacy in reducing the overactive parts of the brain, thus abating anxiety and reducing the OC symptoms.

For the minority of OCD patients whose symptoms were chronic (at least 2 years), severe, and non-responsive to medication and behavioral treatment, neurosurgery could be a last resort treatment. Procedures to interrupt the pathways from the frontal cortex to the basal ganglia, like cingulatomy, anterior capsulotomy, and orbital frontal leukotomy have been reported to be successful (Baer et al., 1995; Goodman, McDougle, & Price, 1992).

References

Amen, Daniel G. (1998). Change Your Brain, Change Your Life. NY: Times Books.

American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders, 4th ed. NY:American Psychiatric Association.

Baer L, Rauch SL, Ballatine T, et al. (1995). Cingulatomy for intractible obsessive-compulsive disorder. Arch Gen Psychiatry; 52; 384-392.

Baxter, L.R., Schwartz, J. M., Bergman, K.S., et al. (1992). Caudate glucose metabolic rate changes with both drug and behavioral therapy for obsessive-compulsive disorder. Arch Gen Psychiatry, 49, 681-689.

Joseph, Rhawn (1996). Neuropsychiatry, Neuropsychology, and Clinical Neuroscience: Emotion, Evolution, Cognition, Language, Memeory, Brain Damage, and Abnormal Behavior. Baltimore: Williams & Wilkins.

Goodman, W.K., McDougle C.J., Price, L.H. (1992). Pharmacotherapy of obsessive compulsive disorder, Journal of Clinical Psychiatry, 53(4 Supp), 29-37.

McGuire, P.K., Bench, C.J., Frith, C.D., Marks, I. M., Frackowiak, R.S.J., and Dolan, R. J. (1994). Graded activation of symptoms in obsessive compulsive disorder. British Journal of Psychiatry, 164, 459-468.

Rauch, S.L., Jenike M.A., Alpert, N.M. et al. (1994). Regional cerebral blood flow measured during symptom provocation in obsession-compulsive disorder. Arch Gen Psychiatry, 51, 62-70.

Robinson, D.L., Wu, H., Munne, R.A. et al. (1995). Reduced caudate nucleus volume in obsessive-compulsive disorder. Arch Gen Psychiatry, 52, 393-398.

An Overview of assessing malingering of memory disorder using multiple criteria measures

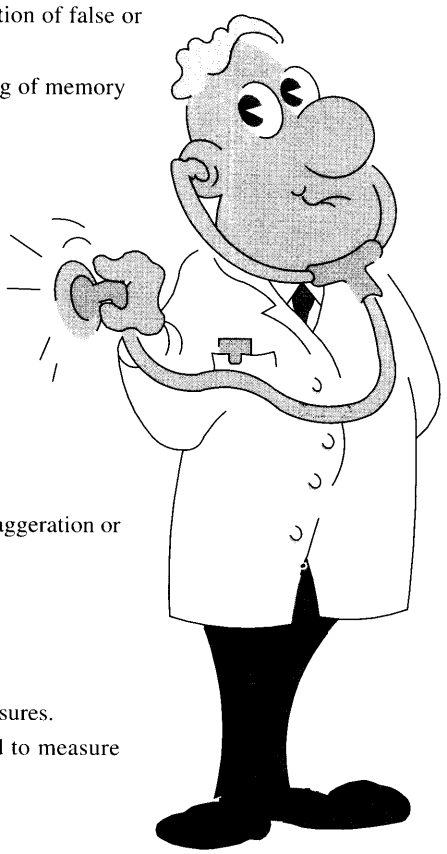
(Sonia Cheng, Clinical Psychologist, Kwai Chung Hospital)

Malingering is a substantial problem in forensic neuropsychological assessment. A diagnosis of malingering is a mixture of conceptual, philosophical and logical consideration when the issues of neuropsychological impairment and psychological factors as well as the stress inherent in the medico-legal proceedings are in play.

According to the DSM-IV (1994), the essential feature of malingering is the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives.

A set of criteria was proposed by Griffenstein et al. (1994) for the diagnosis of overt malingering of memory dysfunction. Malingering should be strongly suspected if the following is noted:

1. improbably poor performance on two or more neuropsychological measures
2. total disability in a major social life
3. contradiction between collateral sources and symptoms history
4. remote memory loss



Slick et al. (1999) had proposed the definition and criteria for Malingering Neuropsychological Dysfunction (MND). It takes into the consideration of the degree of certainty which divides into the category of Possible, Probable, and Definite MND. Its criteria include:

Criteria A

Presence of a substantial external incentive - At least one clearly identifiable external incentive for exaggeration or fabrication of symptoms is present

Criteria B

Evidence from Neuropsychological testing:

1. Definite negative response bias - Below chance performance on one or more forced-choice measures.
2. Probable response bias - One or more well-validated psychometric tests or indices designed to measure exaggeration.

3. Discrepancy between test data and known pattern of brain functioning - Marked discrepancy from currently accepted models of normal and abnormal functioning.
4. Discrepancy between test data and observed behavior - Performances on two or more neuropsychological tests within the same domain are discrepant with observed level of cognitive function.
5. Discrepancy between test data and reliable collateral reports - Performances on two or more neuropsychological tests within the same domain are discrepant with day-to-day cognitive function.
6. Discrepancy between test data and documented background history - Improbably poor performances on two or more standardized tests of cognitive functions within a specific domain that are inconsistent with documented neurological or psychiatric history.

Criteria C

Evidence from Self-report:

1. Self-reported history is discrepant with documented history. - Reported history is markedly discrepant with documented medical or psychosocial history.
2. Self-reported symptoms are discrepant with known patterns of brain functioning - Reported or endorsed symptoms are improbably in number, pattern or severity (e.g. claims of loss of autobiographical information after mild head trauma without LOC, claims of extended retrograde amnesia).
3. Self-reported symptoms are discrepant with behavioral observations.
4. Self-reported symptoms are discrepant with information obtained from collateral informants.
5. Evidence of exaggerated or fabricated psychological dysfunction - Well-validated validity scales or indices of self-report measures of psychological adjustment (e.g., MMPI-2) are suggestive of exaggerated or fabricated distress.

Criteria D

Behaviors meeting B or C are not fully accounted for by Psychiatric, Neurological or Developmental factors.

Definite MND

Presence of clear and compelling evidence of volitional exaggeration and absence of plausible alternative explanation:

1. Presence of a substantial external incentive (criteria A)
2. Definite negative response bias (Criteria B)
3. Criteria D.

Probably MND

Presence of evidence strongly suggesting volitional exaggeration and absence of plausible alternative explanation:

1. Criteria A
2. Two ore more types of evidence from neuropsychological testing, excluding definite negative response bias (2 or more of criteria B2 to B6); OR
3. One type of evidence from neuropsychological testing excluding negative response bias and one or more type of evidence from self-report (one of Criteria B2 to B6 and one or more Criteria C1 to C5).
4. Criteria D

Possible MND

Presence of evidence suggesting volitional exaggeration. Presence of criteria necessary for Definite or Probable MND except that other primary etiologies cannot be ruled out.

1. Criteria A
2. Evidence from Self-report (one or more of Criteria C1 to C5).
3. Criteria D; OR
   Meeting criteria for Definite or Probably MND except for Criteria D.

Detection of Malingering

1. Looking for Inconsistencies, e.g.,
   - Inconsistencies between the behavior during interview or casual interactions and the test behavior.
   - Inconsistency between test results from tests presumed to measure similar underlying constructs.
   - Inconsistency between obtained and expected scores is also an area frequently reported. Comparison between the test performance of a patient against the performance of known patient group illustrates implausibility of unusual test score.