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Dear Friends and Colleagues,

The Section of Neuropsychiatry is delighted to present its second newsletter at a very exciting time in the Section’s development. The Section’s aim is to increase the awareness and understanding of the full range of neuropsychiatric topics and this has proceeded very successfully. The broad spread of neuropsychiatric interests can be judged from this newsletter. Since the last newsletter the Section hosted an extremely successful “Institute day” at the Royal College meeting in Liverpool, attended by a big audience, which included the President and the Treasurer. The Section meeting on the 25th September, delightfully and helpfully described below by Norman Poole, was a “sell out”. The multidisciplinary nature of neuropsychiatry was well demonstrated by the presentations and we are hoping to develop NICE guidelines in relation to the management of somatoform disorders/unexplained medical symptomatology. Perhaps the first thing that’s needed is some agreement on what to call these disorders. Eileen Joyce and other members of the executive committee have been working tirelessly to prepare future meetings and you will already know that the symposium on memory disorders (10th February 2010) convened by Michael Kopelman (who chairs the memory disorders working group), is going to be a 5 star day. A number of neuropsychiatric symposia have been accepted for the College meeting in Edinburgh in June and the next section day in September has been planned. We are also working on a joint conference with the Faculty of the Psychiatry of Learning Disability.

However the section is not just about having meetings, there is a huge amount of work behind the scenes by members of the executive committee. The working groups, on epilepsy, led by Michael Kerr, sleep disorders, with Hugh Selsick, traumatic brain injury, now headed up by Mark Upton, have all been invaluable resources when specific questions have been asked by the College and by other bodies about their particular topics. The Section has developed worthwhile relationships with most of the College Faculties, perhaps particularly the learning disability faculty, the liaison faculty and the old age faculty but also the general adult and the child and adolescent faculties. It seems to me hugely important that we nurture these relationships and that the Section of Neuropsychiatry is seen as being a collaborative group which is highly supportive of all of the College’s activities. To this end, regular attendance by myself, our Secretary (Niruj Agrawal), our Treasurer (Rafey Faruqui), Eileen Joyce, Shoumitro Deb, Michael Dilley and many other members of the executive at the full range of College committees is of vital importance and I would like, here, to thank all of the executive committee members for their time, effort and particularly their enthusiasm.

I think that it has been particularly important over the last few months that we have moved forward with a number of initiatives relating to service developments; with neuropsychiatry included in both the specialised neuroscience service and specialised mental health service definition sets thanks to the work of Simon Fleminger. This should result in real improvements in neuropsychiatric funding, along with working towards proper tariffs for neuropsychiatric work within the National Health Service. We are ensuring quite a bit of neuropsychiatric input into the new ICD-11, currently there is a very valid feeling that neuropsychiatry misses out in a number of areas. Educational aspects, particularly the application to PMETB for subspecialty status and input to work-force planning committees has been an important focus for the Section of Neuropsychiatry as we are very keen to ensure a burgeoning future for the sub speciality.
The Section of Neuropsychiatry is now closely involved in the various initiatives set up by the Joint Neurosciences Council, including a recent meeting with the specialist health service commissioners and a very active input into the e-learning modules being developed by the JNC.

Our financial situation at present seems pretty secure after a couple of very successful meetings and this allows us to think more widely about future projects, particularly training weekends and residential meetings. We are also planning the development of a neuropsychiatry prize for trainees.

My message this time has been somewhat business like, but I think it very important, for those who have managed to read this far, that the readership of the newsletter should get a feel for the sort of work which is going on, in addition to the meetings which are the outward and physical sign of the Section. Our aim is both to support the College in all its endeavours (therefore having to read through huge numbers of lengthy policy documents for many members of the executive) whilst at the same time taking every opportunity to further the cause of neuropsychiatry nationally, within the colleague, within the universities, within our local health services and anywhere else that we can think of. Our Section is only as good as its members and its leadership and we certainly wish all members of the Section of Neuropsychiatry to feel fully involved in what is happening here. For instance, I have developed, with the approval of the executive committee, a paper on the role of the Consultant Neuropsychiatrist (this will be printed in the next newsletter). This has been put forward to the Central Executive Committee of the Royal College of Psychiatrists and forms part of the President’s paper on the role of Consultant Psychiatrists in general. I welcome comments regarding all of the work of the Section of Neuropsychiatry.

Finally I want to thank, most heartily, Alex Mitchell for editing this splendid second edition of the Section of Neuropsychiatry newsletter, which I know you will find fascinating.

May I wish everybody seasonal greetings and look forward to an exciting new year for neuropsychiatry.

Yours sincerely

Jonathan Bird, Chair, Section of Neuropsychiatry
2. SoN Working Group: Epilepsy in Neurosurgery Populations

By Ekkehart F.A. Staufenberg

This article has been prepared on behalf of the Royal College of Psychiatry’s Section of Neuropsychiatry’s Epilepsy Sub-Group. We hope to be able to both share clinical, and educational aspects in the field of epilepsy specific neuropsychiatry. This first letter is focusing on the epilepsy neurosurgery candidate populations.

**Historical developments to date:**

The history of a clinical recognition of a link between psychological and psychiatric health and seizures, and/or epilepsies, go back to the first descriptions in Greek Medicine by Hippocrates. The nineteenth century formulated more particular seizure semiologies and associated neurobehavioural changes. The twentieth century, however, witnessed an increasing professional and hence clinical distancing and divergence of the (hypothesised) neurobiological mechanisms of epilepsy from behavioural and psychological phenotypes. It took another fifty years from the development of the EEG by Hans Berger in the early 1930’s, and the description of the Landau phenomenon in the 1960’s to gradually dissipate the then prevailing conventional views that any psychotic or affective symptomatologies in the individual with epilepsy phenomena largely secondary to psychosocial adversity, medication, traumatic brain injury, or cognitive/other neurodevelopmental disorders.

The concept of a shared aetiology of neurostructural and neurofunctional pathology accounting for both seizure and epilepsy genesis on the one hand, and the generation, mediation or modulation of specific seizure or epilepsy syndrome linked neuropsychiatric syndromes, took until the late twentieth centre to become more generally accepted. Neuro-anatomical studies have been providing the seminal knowledge-base, to rejuvenate the re-discovery of the inter dependence of the neurophysiological and neuroradiological, first via Papez’ early structural works, then McClean’s and most recently Heimer’s integrative with neurodevelopmental, psychological observations, and neuropsychiatric phenomenology.

**The current professional environments:**

Tertiary and some secondary Specialist Epilepsy service-based teams of neurologists and neuropsychiatrists acknowledge the complementary professional skills required for a detailed, shared, patient needs focused formulation of the epilepsy syndromes’ aetiologies and their biological, social, and neuropsychiatric/psychological clinical imperatives and therapeutic pathways. For both adult and paediatric clinical epilepsy services and the pre-neurosurgery neuropsychiatric assessments, as well as post-surgery therapy programme, the integration of behavioural neurology/neuropsychiatry and neuropsychology within the multidisciplinary clinical team practices require increasing specialised knowledge and training of neuropsychiatrists. Familiarity with the neurofunctional correlates and behavioural phenotypes manifestations relating to individual seizure semiologies, seizure syndromes, and entire epilepsy syndromes are a sine qua non.

Although not the focus of this Newsletter, any paediatric epilepsy neurosurgery programme – and the pre-surgical evaluation of associated functional (neuropsychiatry, developmental and cognitive) disabilities and impairments require the consideration of an additional clinical judgement-based risk benefit analysis. The evaluation of the child’s history and the genesis of the seizures, not only inform the potential surgery approaches, but are taken in context more pronounced and sometimes difficult to predict aspects of neuroplasticity, as well as considerations...
for potential secondary beneficial outcomes for both child and family throughout the young persons’ formative years.

What may be the implications for training in neuropsychiatry? Common to those neuropsychiatry in the transitional and adult epilepsy services and in paediatric neurosurgery programme remains the specialised training imperatives in the recognition, delineation and formulation of pre-, intra-, post-, and inter-ictal neurobehavioural syndromes, associated neuropsychiatric disorders, and resulting therapeutic options. This specialised expertise will not only improve patient (and carer) prognosis, and hence measurable outcomes (including health economic variables) but also contribute to the reduction of (apparent) dichotomies in shared team working, between neurosurgery, neurology and neuropsychiatric epilepsy services. The different service models which are currently formally or informally working via clinical network in the UK are currently subject to a survey which forms part of an MD thesis by one of the co-authors of this newsletter. Clinical training and service development opportunities for Epilepsy specific Neuropsychiatry will emerge and present the Section of Neuropsychiatry with the chance to prepare appropriate curricula for psychiatric (and neurology) trainees based on the clinical imperatives.

The role of neuropsychiatric epilepsy specific pre-neurosurgical assessment and treatment:
This brief introduction cannot serve to comprehensively review the components of an epilepsy specific neuropsychiatric assessment process and our professional roles. Neurosurgical programmes in the UK are aware that limitations of capacity translates into a potentially very significant number of potential candidates for epilepsy neurosurgery (both in paediatric but certainly in adult populations) who currently do not progress to, or beyond a pre-neurosurgical assessment. Specific assessments pre-neurosurgery and post-neurosurgical treatments take place under various arrangements or models, including individual referrals, operationalised protocols, and/or embedded within a comprehensive epilepsy neurosurgery programme. Other models include more devolved neuropsychiatry specific clinical pathways where neuropsychiatric expertise in epilepsy is based in a secondary or tertiary but working functionally in close collaboration with the pre- and post-surgery pathways.

Epilepsy neurosurgery programme leads’ referrals and (justifiable) expectation of such neuropsychiatrists assessments and post-neurosurgical therapies, may conventionally comprise the following clinical domains and therapeutic avenues:

Practical summary of Pre-and / or post-epilepsy neurosurgery evaluation:

- To specifically identify and define the presence or absence of a diagnosable psychiatric illness or associated neuropsychiatric syndrome.
- The neuropsychiatric assessment is to differentiate psychological and neuropsychiatric resilience protective and vulnerability for risk factors, as they relate to an individual’s inherent predispositions. This component of any neuropsychiatric assessment would characteristically need to include personality traits, psychosocial vulnerability versus resilience variables, and neurocognitive and social adjustment, amongst others.
- To formulate any diagnosed psychiatric illness within classificatory systems. We need to be aware of and specifically identify the significant limitations of both the current ICD-10 and DSM IV classificatory systems in their ability to formally delineate (possibly pre-existing) psychiatric pathology from seizure semiology or epilepsy syndrome specific, associated neuropsychiatric
syndromes (such as inter-ictal dysphoric disorder, peri-ictal psychotic illness episodes, and post-ictal versus inter-ictal psychotic syndromes with particular attention to the ‘lucid interval’).

- To carefully examine the surgical candidates psychiatric and medication (both anti-epileptic drugs and psychotropic) history in relation to individual psychiatric phenomenologies and their (potential) episodic, recurrent pattern.
- Finally to define the neuropsychiatric phenomenologies and place them in the context of seizure semiology (i.e. pre-, intra-, post-, and inter ictal temporal relationships).
- The post-ictal psychosis within/after a so-called ‘lucid interval’ continues to be easily overlooked, leading to a false diagnosis and possible treatment of a psychotic episode as ‘schizophrenia’ despite these issues having been highlighted for some years, most recently comprehensively in the Neurobiology Working Party of the International League Against Epilepsy chaired by Professor Michael Trimble at the time.

The differential diagnostic formulation of pre- or post-surgical presence of non-epileptic attacks, somatoform and/or conversion syndromes. The post-neurosurgery outcome, and the management of expectations, both in the multidisciplinary team and, certainly for the surgical candidate and their relatives/carers also form part of a wider systemic neuropsychiatric formulation and care planning. Depending upon the nature of the seizure syndromes and the epilepsy syndromes, the neuropsychiatrist may also consider and discuss with his neurosurgical team colleagues alternative therapeutic avenues, including Vagus Nerve Stimulation (VNS).

**Summary:**

The neuropsychiatrist’s role affiliated to, working with, or embedded within an epilepsy neurosurgery programme, therefore, would appear to be in a unique position, given the right training and expertise, to integrate the diverse neurobiological (including cognitive) and emotional, psychosocial variable, and personality attributes in their specific assessment, and to effectively communicate his or her diagnostic formulation to our epilepsy neurosurgery programme team colleagues.

The Royal College of Psychiatrist’s Section of Neuropsychiatry is in the process of working towards a clarifying definition, within epilepsy services and epilepsy neurosurgery clinical programmes, of the roles and resulting training needs for psychiatrists in the specific clinical diagnostic and therapeutic (including pharmacological) responsibilities of working as part of a multidisciplinary assessment programme for (potential) neurosurgical candidates.

Future updates on this, as well as the paediatric perspectives on the role of psychiatry and neuropsychiatry within such programmes, will outline specific issues in relation to the younger epilepsy neurosurgery candidate populations in the subsequent newsletter.

**Ekkehart F.A. Staufenberg on behalf of the Epilepsy Subgroup**

*Consultant in Epilepsy and Neuropsychiatry*

*Dept. Neurology, Norwich Epilepsy Clinic, Norfolk & Norwich University Hospital NHS Foundation Trust*
“Don’t waste your time on hysteria, it doesn’t exist!” declared a consultant supervisor during my training. Perhaps. But on the 25th of September the Royal College of Psychiatrists was so crammed with aspirant delegates to the one-day conference on the topic that many had to be turned away, and of those able to secure entry some had to content themselves with a spot on the steps. Before anyone had even begun talking it became clear the diagnosis has shed some of its former disrepute.

Alan Carson kicked off with an erudite introduction to assessment and diagnosis. A potentially dry subject was enlivened by his delineating continuities and discontinuities from Sydenham via Charcot to our own time, challenging some modern assumptions on the way. The incidence has not waned over the years; rather it has stubbornly remained the same. The symptom profile does not appear to alter across time or cultures. However, even if patients’ presentations have been more stable than previously supposed, medical thinking has been anything but. Thomas Sydenham is clearly something of a hero for Dr Carson, who advocates a return to the early theoriser’s integrationist approach. Research should focus on providing a causal explanation for physical symptoms while their meaning and the patient-doctor dynamic needs to be better understood.

Having established the legitimacy of the diagnosis Dr Carson handed over to Richard Kanaan, who has qualitatively researched neurologists’ opinions on conversion disorder. We learnt that for neurologists the apparently theory neutral ‘medically unexplained’ designates something distinct from conversion disorder. This problem of nomenclature was to become a recurrent theme of the day. Conversion is suspected when functional inconsistencies are elicited or psychological factors strongly implicated in the aetiology. But neurologists are conflicted by the possibility of feigning. Dr Kanaan’s timely research should be of great interest to psychiatrists. The neurologists seemed certain that assessment and management lay outside their range of interest and expertise. In a climate of cut backs the prospect for psychiatry of an unwanted complex and chronic population is tantalising.

Not all neurologists are uninterested in conversion however, as our next speaker Dr Mark Edwards, a movement disorder specialist, made abundantly clear. Although organic movement disorders are increasingly identified, such as paroxysmal kinesigenic dyskinesia – an epilepsy of the basal ganglia, the concept of psychogenesis necessarily endures. Conversion is suspected when the abnormal movement appears atypical, can be entrained and improves with distraction. Intriguingly psychogenic myoclonus can even be positively diagnosed because a Bereitschaftspotential (cortical readiness potential) is present, which is lacking in organic myoclonus. Dr Edward’s fascinating talk confirmed why a neurologist should be integral to a service for conversion disorder, and was followed by a joint presentation from the multi-disciplinary team at Queen Square.

The team, comprised of a physiotherapist (Sarah Edwards), occupational therapist (Jo) and psychologist (Linde), presented their model illustrated by a successfully treated case. They advocated a rigorous cognitive behavioural approach heavily based on the models of Chalder and Brown. They described goal setting; grading and pacing; limit setting on maladaptive behaviours; and facilitation of useful coping strategies and personal resources. It was interesting to hear professionals from such diverse backgrounds talk in the same language and surely this unity accounts for some of their success. This intensive multi-disciplinary approach may be the current gold standard,
but could lack practical relevance to those in the crowd aiming to provide a service with more limited resources to hand.

The Epilepsy Working Group’s discussion of non-epileptic attack disorder (NEAD) that followed lunch aimed to provide guidance to the interested generalist. Prof Michael Kerr made the case that NEAD (he made it quite clear he would not countenance any other name) is common, overlaps with other psychiatric diagnoses and usually responds to psycho-education and support. The NEAD group has therefore been formed to raise awareness and improve outcomes. If NEAD was beginning to seem relatively straightforward, Dr Jonathan Bird came on to highlight the complexities. His videos of patients undergoing video EEG at the Burden Centre demonstrated that a lifetime’s experience with epilepsy and NEAD are sometimes required to discriminate between them, and that classical symptoms of hysteria, like pelvic thrusts, can confound. Dr Manny Bagary presented the assessment and management of a traumatized young woman with NEAD and co-morbid anorexia nervosa who struggled to accept the diagnosis. This section of the day underlined that psychiatrists are uniquely skilled to provide services that assess and manage medically unexplained symptoms.

The Memory Working Group had forgotten to put the event in their diary, and so instead Dr Bird once again took to the stage, this time presenting on forensic aspects of sleep disorders. It is rather a shame the continuity of the day was disrupted, but the talk was nevertheless fascinating. Memorably, in response to a question posed by Dr Bird regarding an accused somnambulist, Dr Joanna Herrod interjected that the accused must be guilty of the grisly murder for he had ejaculated in his pyjamas.

The day was concluded with all the speakers returning to the stage for a forum discussion. Much energy was expended on terminology without, unsurprisingly, a consensus being reached; though everyone appreciated the wry suggestion “dysfunctional neurocognitive adaptation”. The lack of robust trials for dynamic therapy makes its inclusion in guidelines problematic. However, a psychodynamic understanding of the patient and systems should not be dismissed. Then came the vexatious issue of abreaction. Most agreed that any response is probably a placebo, though Alan Carson saw no problem with harnessing this. The forum concluded with an agreement to develop guidelines for the UK on management of medically unexplained neurological symptoms.

Dr Kanaan had earlier drawn our attention to Freud’s obituary of Charcot. His greatest achievement, Freud asserts, was to have thrown the weight of his authority behind the diagnosis, thereby legitimizing the sufferers. It is only a small exaggeration to claim this much-needed service was repeated on September 25th, for which Professor Joyce, the organizer, and all the speakers are to be commended.

Norman Poole,
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“Translational research” has been a common theme that we hear about these days. There are a considerable number of years before basic medical research results have any clinical use (Cooksey Review, 2006; Bell, 2002). The major funding bodies, the MRC and Wellcome have started investing millions into translational research—but what do we mean by translational research. When I attended the annual SSBP conference (the Society for the Study of Behavioural Phenotypes) in Cambridge, October 2009, it started to become clearer.

The society was set up by a group of scientists and clinicians who had been “investigating behavioural and emotional aspects of biologically determined syndromes associated with intellectual disability...” (http://www.ssbp.co.uk/ssbp/). The society holds annual conferences and this year’s conference was entitled “Listening to genetic disorders: from molecules to management”. It had many pre-eminent speakers (see website for programme) and the themes covered were from the molecular aspects of cognition and behaviour to the translating of that knowledge to inform clinical studies.

Genetic studies have identified the genes responsible for the defects of some neurodevelopmental syndromes and this has lead to studies in animals (especially mice) that have unravelled abnormalities of synaptic plasticity (and its underlying the biochemical pathways), and the cognitive and behavioural impairments that characterise the respective conditions.

Although tuberose sclerosis was the main topic of discussion at the conference, other neurodevelopmental syndromes were also covered. Tuberose sclerosis is caused by mutations of the TSC1 or TSC2 (tuberose sclerosis complex) genes (Crino et al., 2006). An important downstream protein target of TSC is mTOR (mammalian target of rapamycin), as the name suggests, is antagonised by a clinically used transplant drug, rapamycin. The mTOR pathway controls cell growth and protein synthesis. Mice that had been genetically engineered to express mutations of the TSC genes appear to recapitulate many of the features of tuberose sclerosis. As the TSC mutations result in the up-regulation of their downstream targets, it was predicted that an inhibitor at the mTOR site would reverse the phenotype by ameliorating the up regulation of the pathway. This indeed was the case for many of the tuberose sclerosis phenotypes in the mouse, and so this lead to the potential applicability of this drug in to treat the condition in humans with the mutation. The clinical research is still on-going but it is already showing some promise.

There are several other examples where the same genetic approach is beginning to yield results and hasten the journey from “mouse to the clinic”. Examples include Down’s syndrome, fragile X and neurofibromatosis (Ehninger & Silva, 2006). More interestingly, such treatments can reverse phenotypes even in the mature animals; a missed opportunity for early intervention may not deny one the possibility of treatment as an adult with a syndrome. Such examples illustrate a good case of translational research. Although clinical observations continue to help formulate hypotheses that can be tested in animal models, such genetic studies are helping to unravel the details of the biochemistry and cell biology that can inform potential clinical interventions.
Certainly research in the field of intellectual (or learning) disability has been largely ignored thus far, but finally this area of research is now producing great strides forward in knowledge. The progress in this field has been remarkable during this decade or so and is likely to continue; it is an exciting time indeed to be in the field.

The 2010 meeting of the SSBP is in Rome, Italy and I am looking forward to being there.

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Competing interests: I am a member of the SSBP and I encourage you to join!

References

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Deep Brain stimulation (DBS) is a well-recognised treatment for certain movement disorders. The most common reason for considering surgery is for patients with Parkinson's disease (PD) for whom drugs no longer control their symptoms. These are patients who have developed severe fluctuations in their condition (sudden off periods and short-lived on periods) and/or have marked uncontrolled exhausting movements of their limbs and trunk (dyskinesias). Other patients who may benefit from surgery are those with severe tremor, dystonias, and other movement disorders such as severe Tourette's Syndrome, tremor in multiple sclerosis and ballistic movements following stroke. The surgery is associated with much improved control of movements and, although it does not delay the progress of the underlying neurological disease, it greatly improves quality of life.

The Functional Neurosurgery Clinic at Kings College Hospital, London (KCH) is a multidisciplinary clinic primarily serving Southeast London and Kent, Sussex and parts of Surrey. It is staffed by neurologists, neurosurgeons, specialist nurses, neuropsychologists, neurophysiologists and supporting administrative and technical staff providing a tertiary assessment and treatment service for patients with complex movement disorders, resistant to medical treatment who may benefit from a neurosurgical intervention.

The clinicians have from the outset been very clear that all patients being assessed for surgery must have a neuropsychiatric assessment. This is because reported psychiatric complications of DBS, though uncommon, include transient aggression, hypomania and mania and more persistent depression, anxiety, apathy and suicide. Poor psychiatric outcome may be related to a range of psychiatric, social, neurological and surgical variables. Additional problems may be disorders of impulse control or dopamine dysregulation syndrome, pathological gambling and hypersexuality. Cognitive problems have also been reported. Good quality postoperative assessment and management of mood is important as reduction of dopaminergic medication, which may be an essential part of managing dyskinesia, can cause deterioration in mood.

Because of the specialist nature of the clinic and the intervention, it is important that the presurgical assessments and post surgical follow-up is done by a clinician experienced in the specific neuropsychiatric problems faced by patients with movement disorders, most commonly Parkinson's disease. The Neuropsychiatric Service of The South London and Maudsley NHS Foundation Trust (SLaM) is well placed to provide the neuropsychiatric input into this clinic and we have been doing that now for 5 years. The coming together of SLaM and KCH, together with the Institute of Psychiatry and Guy's and St. Thomas’ Trust as Kings Health Partners Academic Health Sciences Centre will further help our joint working to deliver the best care to these patients.

At present, our assessment includes current psychopathology, past psychiatric illness, screening for impulse control disorders and personal, social, forensic and family history, competency, carer support, expectations from surgery and a qualitative assessment of the patient's ability to cooperate with the demands of DBS and medication changes which would be required post-operatively should the patient proceed to surgery. The neuropsychiatric examination also includes a corroborative history from a spouse or carer, obtained with the permission of the
patient. The neuropsychiatric assessment is not, as usually has to be carefully explained to the patients, a “test” which they are required to pass. Rather, the screening is used to identify any current mental disorders requiring treatment or management; to help ensure that patients are aware of possible postoperative risks in the psychiatric domain; and perhaps most importantly to ensure that they and they carers can be offered prompt and timely interventions should any such problems occur.

When all assessments (neurological, dopamine responsiveness, imaging, neuropsychological and neuropsychiatric) have been completed the MDT meets to consider the suitability of the patient for surgical intervention and, if suitable, the options of surgical target. The commonest procedure is bilateral stimulation of the subthalamic nucleus for patients with motor complications of PD.

In addition to assessment and follow-up and treatment of any complications, the neuropsychiatry service has also been involved in the evaluation of the postoperative outcome of neuropsychiatric symptoms and overall patient and carer satisfaction with the procedure.

Dr John Moriarty
Consultant Neuropsychiatrist
Maudsley Hospital and Kings College London

ABN Annual Meeting
11-14 May 2010
BIC, Bournemouth

Key note speakers to include:
Yves Agid, France
Stephen Hauser, USA
Jerry Posner, USA

www.abn.org.uk
Association of British Neurologists
27-Boswell Street, London, WC1N 3JZ
Summary
We report an interesting clinical case of recurrent psychotic events caused with Chiari I malformation in adults. The mediosagittal reconstruction of MR image of the brain and cranio-cervical region is the most important diagnostic approach in finding the cause of epileptiform events, neurological signs and psychotic future by Chiari malformation.

Introduction
Chiari I malformation, a congenital abnormality in which deformed cerebellar tonsils are displaced downward through the foramen magnum, commonly present in patients with headache or symptoms of dysfunction of the cerebellum, brain stem, lower cranial nerve palsies and cervical spinal cord. (1)

Arnold-Chiari type I malformation was a rare finding among young children (<6 years) (Wisoff & Abbott, 1997). However, with the increased use of magnetic resonance imaging (MRI), specially with medio-sagital reconstruction of MR image, this craniocervical malformation is being identified more often in children and young adults with neuropsychiatric symptoms such as headache, neck pain, ataxia, sensory deficit, lower cranial nerve palsies, speech disorders, altered consciousness (psychosis), burning sensations in body and arms, nystagmus and other oculomotor disturbances. Syncope, epileptiform seizures (in EEG), involuntary movements of arms, paralysis of arms, torticollis, scoliosis, sleep apnea, and sudden death resulting from Arnold-Chiari type I malformation also have been reported (Ali, Russell, Awada, & McLean, 1996; Dure, Percy, Cheek, & Laurent, 1989; Strayer, 2001, Ilankovic, 2002). (2,3,4)

Case Study
Our patients was a young women (30 years old) with 4 repetitive acute psychotic episodes (in last 3 years), with many neuropsychiatric symptoms and signs: altered consciousness (schizophrenia-like states), confusion, disorientation, agitation, insomnia, dysgnosia (prosopagnosia), dysphasia, dysarthria, dysphagia (N VII, IX, X, XII), tinnitus, vertigo, nystagmus (N VIII), hypotonia and ataxia (cerebellum), epileptiform events (short loss of consciousness with atonic crisis).

All the psychotic episodes (Schizophrenia-like) happened during premenstrual and menstrual period: the first two happened in summer on the sea-side, the third one at home and the fourth in our clinic (both in springtime). There was no infection, head injury, ethanol and/or drug abuse (repeated laboratory tests were negative). In first two episodes she had uncomfortable movements in the neck caused by swimming. The patient reported headache and neck pain. In the last two episodes we could detect water retention in the body (face and body swelling).

All the laboratory tests were without any pathological change. The EEG indicated irritative dysfuntions above centro-temporal regions. The MR investigation of cranio-cerebral and cranio-cervical regions showed (in medio-sagital reconstruction of images) the signs of Chiari I malformation!

The therapy was: 1. AED (Valproates), 2. Benzodiazepins (diazepam, lorazepam), 3. Neuroleptica (small doses of haloperidol), 4. Diuretic (furosemid small doses) and 5. Water and salt intake restriction. The recovery in all episodes was complete after few days, with amnesia on psychotic episodes. The recovery of signs of cerebral dysfunction in EEG was complete, too.
Discussion

The increasing use of MRI to diagnose neurological problems is leading to a greater recognition of the Chiari I malformation. Still, many patients have symptoms years before the problem is identified. Patients with the Chiari I malformation display one or more signs and symptoms of brainstem, cranial nerve, cerebellar or spinal cord dysfunction including: headache (especially cough headache), neck pain, disordered eye movements, epileptiform events, “drop attack”, disorder of consciousness (psychosis), trigeminal dyesthesia, sensorineural hearing loss, tinnitus, vertigo, nystagmus, ataxia, dysphagia, dysarthria, dizziness, weakness (greater in the upper than lower extremities), dysesthesias/numbness sensory loss, cenestopathias, dysmorphophobias, snoring, sleep apnea, memory disturbances, affective disorders, etc. All this happens because of the compression not only of the neuronal tissue and the nerves but the blood vessels as well. Vascular symptoms include syncope, drop attacks, vertigo, intermittent periods of confusion and altered consciousness, episodic weakness, and transient visual and hearing disturbances. Vertebrobasilar ischemia may be provoked by movements or by changing of head position. Because the spinal cord is flexible and therefore susceptible to intermittent compression, several types of lesions at this level can cause neuropsychiatric symptoms that vary from patient to patient and that can be intermittent!

Symptom Alleviation Methods for Chiari patients:

1. Wear highly cushioned, support shoes to reduce neck, cerebellum pounding. 2. Avoid neck-stressing activities (football, soccer, basketball, wave pools, diving, tennis, roller coasters, other amusement park rides of high G forces, backpacking, falling asleep in chairs, extended reading with head bent etc.) 3. Get plenty of rest and sleep (6hr minimum). Pillows and sleep position are very important. Soft, small pillows with fiberfill are very good for this, i.e. little or no"push" back. Most Chiarians are sidesensitive. Use large pillows etc. to prevent rollover onto the affected side. A head-down position should be avoided. 4. Stop eating and drinking three to four hours before bedtime to reduce the need to get up at night. Avoid caffeine, alcohol, aspirin and/or high salt diet if tinnitus is a major symptom or if any of these dietary items increase other symptoms. 5. Stay in excellent physical condition via walking, exercise bikes and other non-neck stressing activities. 6. Sit in soft recliners with high backs and foot rests. 7. Support reading material with elbows on your knees/thighs or chair arms. Read “straight” ahead. Use book holders or music stands. Look at computer monitors straight ahead. 8. Drive if you have to but use wide vision mirrors and get seats with high backs. 9. Relax and avoid stress and noise. Don’t tighten the neck muscles. Stay “cool”. 10. Straining during bowel movements should be avoided. Eat plenty of roughage and eat at regular times. Drinking herbal tea containing senna may help give pain-free relief from constipation. 11. Do crossword puzzles to assist short-term memory retention. 12. Avoid cervical traction. 13. Lumbar punctures, spinal taps or epidurals can be dangerous for Chiarians. 14. Brushing teeth or gargling can result in sneezing. Minimize head motion during brushing and not bending the neck when gargling. 15. Avoid chiropractor adjustment! 16. Eliminate as much neck stress as possible. 17. Use the special tray that most beauty shops have for washing hair for people who cannot lean back on blunt sink rims. Chiarians should never lean back on the edges of sinks. 18. Avoid neck “jerking”/stressing activities such as football, basketball, tennis, weight/furniture lifting, wave pools, roller coasters, backpacking, extended reading or sleeping with the head bent down or up, etc. 19. Avoid too much salt in food (especially women in premenstrual period). 20. The preventive use of diuretic drugs can be helpful (N. Ilankovic).
If the preventive intervention and the pharmacotherapy are not sufficient, the posterior fossa and upper cervical decompression need to be considered. This neurosurgical intervention provides more room around the lower brainstem and promotes improved neuropsychiatric functioning.

**Conclusion**

Not only the horizontal slices, but the mediosagital and mediocoronar reconstruction of MR image of brain and cranio-cervical region and the MR angiography are very important diagnostic approach in finding the cause of many psychotic disorders, epileptiform events and unclear neurological signs. The known ethiopathogenesis (Chiari I and II, Mesiotemporal sclerosis, Hypocampal atrophy, multinfarcts, leuconcephalopathy, AV malformations, sinus thrombosis, CNS infection-toxoplasmosis, neurosyphilis, abscessus, etc.), relative simple convert the so called “functional schizophrenia” into symptomatic schizophrenia-like psychosis. Finally, it is the opportunity to introducing the more targeted pharmacotherapy and the preventive actions.

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Do you have an interesting case-report for further discussion? If so send it to me ajm80@le.ac.uk
6. Evidence Based Practice

Psychogenic non-epileptic seizures – quo vadis?

By Markus Reuber

Background

Psychogenic non-epileptic seizures (PNES) superficially resemble epileptic seizures, but are not associated with ictal electrical discharges in the brain. PNES can be thought of as episodes of paroxysmal impairment of self-control associated with a range of motor, sensory and mental manifestations, which represent an experiential or behavioral response to distress. The overwhelming majority of PNES are considered as beyond patients’ voluntary control. However, it is recognized that seizures are occasionally simulated for personal gain or to attract attention.

Whilst PNES are a form of human behaviour, which appears as if it should have been around for a long time, diagnostic uncertainty increases the further back in history one tries to pursue the phenomenon. Nevertheless we can be fairly sure that most of the patients nineteenth century physicians like Charcot, Gowers or Freud thought of as having “hysterical seizures” or presenting with “la grande hysterie” had attacks we would today consider as PNES. In fact, there is no convincing evidence that the prevalence of PNES (as compared to epilepsy or other “medically explained” neurological disorders) has varied much over the last century: the figures published in Gowers’ “Epilepsy and Other Chronic Convulsive Diseases” (1881) or the breakdown of diagnoses made in the outpatient department of the Salpetrière are no different from recent studies (Goetz, et al. 1995). However, research interest in PNES has not mirrored the unchanging prevalence levels – the last 150 years have seen two major peaks in activity separated by a long trough.

The first peak was closely associated with Charcot. Having made major contributions to our understanding of the functioning of the spinal cord, multiple sclerosis and motor neurone disease, Charcot set out to apply his clinicopathological method to “crack” hysteria in the 1860s, producing dozens of publications on the subject. Aided by his patronage and inspired by his enthusiasm most eminent neurologists included chapters on hysteria in their textbooks well into the 1920s. Some of this work (especially by Janet) remains unsurpassed today. In parallel, Freud and other members of the psychoanalytic movement made important contributions to the field during this period. The British, American (and to a lesser extent German) historiography of hysteria received a particular boost during World War I when it became clear that the disorder was not a phenomenon confined to French boudoirs or the “belle etage” of Viennese town houses.

From 1920 to 1980 scientific work on PNES went through a long hiatus in some ways encapsulated by Slater in the 1960s who suggested that the diagnosis of hysteria was almost always due to diagnostic error and that the phenomenon had essentially gone away (Slater 1965). Many mainstream psychiatrists still seem to adhere to this view today, and indeed, today’s patients with “functional” neurological symptoms are happy to confirm their erroneous impression by staying away from their clinics! However, the introduction and increasing use of video-EEG since the 1970s has forced neurologists to face up to the reality that PNES still exist. As it turns out, they are
Triggered by the sudden and undeniable visibility of PNES, a steady trickle of papers about PNES in the early 1980s soon turned into a flood. Even six years ago, it still seemed possible to summarise the (English language) literature of the previous two decades quite comprehensively (Reuber and Elger 2003). More recently it has become much more challenging to maintain a firm grasp of all the latest development in this modest field of medicine. Several more recent reviews have dealt with the glut of publications by focusing on more specific aspects, such as the aetiology of PNES (Reuber 2009), the diagnostic process (Cragar, et al. 2002, Cuthill and Espie 2005, Hoerth, et al. 2008) or the treatment of PNES (Baker, et al. 2007, LaFrance and Barry 2005, Reuber, et al. 2005).

Whilst the increased quantity or research should be good news for patients with PNES, progress in those areas which probably matter most (such as aetiology and treatment) has only been modest. We can now diagnose PNES with much greater certainty but it still takes more than five years on average to make the diagnosis. The vast majority of patients are still treated inappropriately with antiepileptic drugs initially, and some come to serious harm in the process (Reuber, et al. 2004). Nearly seventy years after his death, the psychodynamic theories which underpin much of modern thinking about PNES can still be traced back to Freud’s writings on the subject. An integrative non-dualistic psychobiosocial model of PNES which goes beyond hardware/software analogies and which embraces our understanding of the scope and limitations of neuroplasticity and neuronal modulation in health and disease remains elusive (Reuber 2008).

Much of the research conducted into PNES has been reactive or opportunistic – often based on data collected routinely in epilepsy centres in the course of evaluating patients for epilepsy surgery. Hypothesis driven studies examining the mental or psychodynamic processes leading to PNES remain the exception (Bakvis, et al. 2009), and are greatly outnumbered by studies describing visible or behavioral phenomena. Far too many studies have used patients with epilepsy as the only controls. Most of our knowledge is derived from retrospective studies open to all the biases associated with “data trawling”. Whilst this is disappointing, it also means that there is still much to be learned about PNES by testing the many hypothesis, which have been generated by retrospective studies and psychodynamic thinking through carefully constructed prospective studies. Fortunately, there are several research groups in the UK, which now focus on PNES (for instance the group around Rod Duncan and Maria Oto in Glasgow or the group around Laura Goldstein and John Mellers in London). What is more, there are now nuclei of collaborative research networks in both the UK (www.shef.ac.uk/nest/index.html) and the US, which may produce the large studies this heterogeneous disorder calls for (Hall-Patch, et al. 2009, LaFrance, et al. 2006). A patient group has formed which may help to support research in the future (www.neadtrust.co.uk). So far, only a very small number of UK liaison psychiatrist and neuropsychiatrist have interpreted the gaps in our knowledge about PNES as an opportunity to use their own research expertise in this area. Many of the new investigational methods available to cognitive neuroscientists remain unused in this area. The same is true of the rapidly developing field of psychogenetics. Having said this, it is unlikely that we will be able to benefit fully from these investigational methods without a clearer understanding of the clinical phenomenology of PNES and its many subtypes. I hope this article has encouraged some of its readers to get in on the action. The author is happy to advise...
References


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7. History of Neuropsychiatry  
Freud’s View of Conversion Disorders

By Norman Poole

The idea that emotion can be converted into symptoms suggesting physical disease is attributed to Freud and Breuer and elucidated in their Studies in Hysteria. The concept gained such traction in psychiatry that both ICD 10 and DSM IV cite conversion as the hypothesised psychological mechanism at the root of conversion disorder: “the unpleasant affect, engendered by the problems and conflicts that the individual cannot solve, is somehow transformed into the symptoms”. However, not accidentally the concept of conversion in the manuals is a mere simulacrum of Freud’s original model, which itself evolved over his career. Given the prevalence of conversion disorder in neuropsychiatry it may be beneficial to quickly survey Freud’s most important thoughts about this condition.

In keeping with the postulate of his contemporary Koch – that each disease has a specific cause – Freud developed the aetiological model of hysteria with the following structure: “(a) preconditions; (b) specific causes; (c) subsidiary or contributory causes”. The preconditions are the hereditary factors that were held to be crucial by Pierre Janet but Breuer and Freud downplayed their significance in hysteria. For example, in the case history of Anna O in Studies in Hysteria her “intelligence, remarkably acute powers of reason, and a clear-sighted intuitive sense” are emphasised. This is not to say that heredity plays no role but it is no longer a sufficient factor. The contributory causes (physical exhaustion, illness, heightened emotional state) need not be present but if so make the development of neurosis, but not necessarily hysteria, more likely. It is in relation to the specific cause that Freud extended Charcot’s ideas, quarrelled with Breuer then discarded, reluctantly, his own central thesis, from the remnants of which psychoanalysis emerged.

Charcot thought physical trauma that caused a powerful emotion in conjunction with a particular idea, such as “I’ve been paralysed”, specifically caused hysterical symptoms. In the joint Preliminary Statement to Studies in hysteria Freud and Breuer modify this basic premise. The “ideogenic” component is abandoned and it is now the memory of the trauma – as opposed to the trauma itself – that is thought to cause the hysterical reaction. The traumatic memory is repressed and dissociated from its affect, which is converted into physical symptoms. Sometimes the symptom is associated in time with the original trauma (they illustrate by imagining an emotionally wrought girl who, while tending a sick bed, rests her arm over the back of a chair, which becomes dead and numb. When reminded of that time these same sensations return); in other cases the symptom directly symbolises the cause (moral disgust and vomiting); though frequently no relationship is apparent. Primary gain is the active repression of painful memories from conscious awareness.
However, the Preliminary Statement masked irreconcilable differences. Anna O, the case Breuer contributed to studies in hysteria, had noticed that her symptoms resolved when she remembered the context of their first appearance. Breuer argued that concurrent with a traumatic event an altered psychic state, which he termed hypnoid, must also be present. The memory for the event, being laid down in this hypnoid state, is dissociated from the rest of conscious life and can only be recalled in a similar state – induced by hypnosis. Breuer christened the process whereby dissociated memories are emotionally recalled during hypnosis, catharsis. Freud conceded to this view in the “Preliminary Statement” but distanced himself in his chapter “On The Psychotherapy of Hysteria” and rejects it outright in his case history of Dora.

Freud had found many of his patients poorly hypnotizable and so evolved the method of free association to gain access to pathogenic memories. This involved the patient concentrating on the symptom and relating, without censor, each and every emerging thought. In accordance with the physiology of the day, Freud believed every idea is associated with another in a deterministic fashion. By tracking each seemingly irrelevant idea further back he could ultimately recover a repressed memory of sexual trauma in every case. The difficulty in recalling these memories indicated there must be some resistance against doing so, which further suggested this resistance might be functioning as a defence against recollection. By this method a significant proportion of his patients revealed a history of childhood sexual trauma convincing Freud he had unearthed a casual factor in the genesis of hysteria. And this factor was specific, in the model outlined above, because the nature of the experience held aetiological significance. If accompanied by fear and revulsion hysteria was the outcome, whereas an “obsessional neurosis is conditioned by the same accompanied by pleasure”. Freud is usually criticised for denying the existence of childhood sexual abuse but in fact attempted to alert society to this hitherto unidentified problem, the reward for which was ostracisation from the medical community. Breuer vacillated but ultimately could not accept the seduction theory, as it came to be known, so abandoned the subject and his friend.

Undeterred, Freud hypothesized progressively earlier phases of development when the abuse must have occurred, eventually settling on the infantile period. Hysteria though does not generally develop until adulthood. Freud attributes this delay to the increase in libidinal drive from puberty onwards. The libido increases the energy attached to the memory of sexual trauma causing “unpleasure”, which by means of repression is converted to a hysterical symptom. However, his repeated lack of therapeutic success; incredulity that abuse could be so widespread; and a growing realization the unconscious had no criterion for signifying reality lead him to suspect the seduction theory must be false (Letter 69 in Letters to Fleiss). Contemporaneously Freud was engaged in a self-analysis that brought to light an infantile desire for his mother and hostility towards his father that he could also discern in his patients’ dreams. The sexual memories recovered during free association and hypnosis were now understood as repressed wishes rather than true events. Thus, the Oedipus complex was conceived.

Freud’s conception of hysteria undergoes a significant change at this point. He rejected sexual abuse as a specific cause, therefore constitutional “preconditions” once more gained ascendency in aetiological importance. But for Freud the constitutional factor is the sexual drive rather than a tendency to dissociation as Pierre Janet asserted. Repression of the Oedipal conflict occurs in all healthy individuals but hysteria is marked by “exaggerated sexual
craving and excessive aversion to sexuality”. The case of Dora illustrates this point well. Dora at 14 was kissed by a married family friend, fled the scene in disgust then quickly developed a hysterical mutism. Freud interprets her disgust as excessive repression of her sexual feelings towards the older man who is identified with her father. The mutism is understandable because “speech had lost its value since she could not speak to him” . The nature of particular hysterical symptoms though becomes more complex in this new model. Previously, if symptoms could be connected to the trauma it was by association in time or through directly symbolising the symptom’s cause (ie disgust and vomiting). Now however, each symptom is to be understood as both symbolically representing the underlying conflict and as a direct somatic expression of sexual excitement. A lengthy quote illustrates this point well: “Not only is a large part of the symptomatology of hysteria derived directly from expressions of sexual excitement, not only do a number of erotogenic zones attain the significance of genitals during neuroses owing to an intensification of infantile characteristics, but the most complicated symptoms are themselves revealed as representing, by means of ‘conversion’, phantasies which have a sexual situation as their subject matter”.

However, even this is only one possible reading of many that a symptom may simultaneously convey. Each symptom represents a plethora of unconscious processes. Thus the body of the hysteric begins to be treated like a text, which yields meaning only to psycho-analysis.

Subsequently, a fascination with hysteria was usurped by the desire to explain all of human life with his newly acquired techniques. Freud’s conversion is thus from collaborator to iconoclast; seeker of specific causes to covert meanings; physician to interpreter.

**Norman Poole, Locum Consultant Liaison Psychiatrist**

*Kingston hospital*

*Kingston-on-Thames*
# 8. Trainee Corner => Recommended Neuropsychiatry Reading List

*By Niruj Argrawal*

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Recommended Neuropsychiatry Reading List (continued)

**General Neuropsychiatry Papers**

Recommended Neuropsychiatry Reading List (continued)

Royal College Online CPD Modules

1. The Bedside Assessment of Cognition, Dr Jordi Serra-Mestres & Dr Richard Perry,
   [http://www.psychiatrycpd.co.uk/learningmodules/bedsideassessmentofcognition.aspx](http://www.psychiatrycpd.co.uk/learningmodules/bedsideassessmentofcognition.aspx)

2. Brain injury, Dr Simon Fleminger & Dr Arsime Demjaha,
   [http://www.psychiatrycpd.co.uk/learningmodules/braininjury.aspx](http://www.psychiatrycpd.co.uk/learningmodules/braininjury.aspx)

3. Psychiatric problems in Parkinson's disease, Dr J. Serra-Mestres and Dr S. Mukhopadhyay,
   [http://www.psychiatrycpd.co.uk/learningmodules/parkinsonsdisease.aspx](http://www.psychiatrycpd.co.uk/learningmodules/parkinsonsdisease.aspx)

4. The neuropsychiatry of multiple sclerosis, Dr Kate Jefferies,
   [http://www.psychiatrycpd.co.uk/learningmodules/neuropsychiatryofmultiplesc.aspx](http://www.psychiatrycpd.co.uk/learningmodules/neuropsychiatryofmultiplesc.aspx)

5. Early onset dementias, Dr Kate Jefferies and Dr Niruj Agrawal,
   [http://www.psychiatrycpd.co.uk/learningmodules/earlyonsetdementias.aspx](http://www.psychiatrycpd.co.uk/learningmodules/earlyonsetdementias.aspx)

6. Neuropsychiatry of Epilepsy, Dr Suren Govender & Dr Niruj Agrawal
   [www.psychiatrycpd.org/learningmodules/neuropsychiatryofepilepsy.aspx](http://www.psychiatrycpd.org/learningmodules/neuropsychiatryofepilepsy.aspx)

7. Neuropsychiatry of Stroke, Dr Kate Jefferies & Dr Niruj Agrawal
   [www.psychiatrycpd.org/learningmodules/neuropsychiatryofstroke.aspx](http://www.psychiatrycpd.org/learningmodules/neuropsychiatryofstroke.aspx)

8. Acute Confusional State: a guide to diagnosis and management. BMJ Learning. Dr Brian Parsons & Dr Niruj Agrawal
   [http://learning.bmj.com/learning/search-result.html?moduleId=10007766](http://learning.bmj.com/learning/search-result.html?moduleId=10007766)

Learning modules

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Book Review: Lishman’s Organic Psychiatry: A Textbook Of Neuropsychiatry

Anthony David and colleagues - Wiley Blackwell £120

Last month I was kindly sent a copy of the much anticipated 4/e of Lishman’s Organic Psychiatry now “a textbook of neuropsychiatry” (ISBN 1405118601) by the British Journal of Psychiatry. I expect they felt I would be suitable reviewer, having published a single author textbook “Neuropsychiatry and Behavioural Neurology Explained” (ISBN 0702026883) in 2004.

My experience writing my book leaves me with no doubt about the about of time and effort needed to produce a textbook of over 900 pages. It probably not the best career decision for a single-handed author to take several years out of mainstream clinical or academic commitments in order to try to produce a seminal textbook! Of course the RAE / REF takes little account of such projects. Nevertheless, such textbooks are valuable for the community as a whole, especially trainees (see page 21 above) even if their dominance is under threat by dynamic online information.

I was very honoured when Alwyn Lishman agreed to write the forward to my 2004 book and it is excellent to see his seminal 1978 book continue to evolve. The main change is actually a change in authorship which will come as a surprise to many. The new editorial team have taken the difficult decision to convert Lishman’s single author volume into an (almost) entirely new text produced by five editors and thirteen authors all affiliated with the Maudsley hospital or Institute of Psychiatry. I am sure this was not an easy decision not least because there is a compromise if all the authors are limited to one institution just as there is a compromise if there is only one author. The biggest gain from multi-author works is an increase in the breadth and depth of expertise and this is evident here. That said, many such books take on encyclopedia type characteristics by allocating each topic to an expert in the field. Lishman 4/e tries to maintain some continuity in style by allocating only main chapters to individual authors, however this does leave each chapter author with a lot to do.

One way to examine the coverage of Lishman 4/e is to compare the text to the undoubted classic first edition from thirty years ago (which by the way cost £28!). The chapter list is very similar to the original with two omissions namely, differential diagnosis (but this now forms part of basic concepts) and vitamin deficiencies (which now forms part of addictive and toxic disorders). Of course there are many new conditions that were not adequately described in 1978 such HIV/AIDS, Dementia with Lewy Bodies, Mild Cognitive Impairment, as well as many rare neurological and vascular syndromes. Most therapeutic aspects are new. It is fascinating to see the gradual evolution of concepts and terminology through the 1978, 1987, 1998 and 2009 editions. For example the decreasing emphasis on the “abnormal personalities” of people with neurological disorders especially parkinson’s disease, epilepsy and head injury. Or the gradual shift away from simplistic accounts of malingering and compensation issues. Many areas of medicine relavent to neuropsychiatry have flourished since 1978. For example
the fields of neuroimaging and medical genetics were in their infancy in 1978. Take for example the following sections on Huntington’s disease. Lishman wrote in the first edition “many lines have been pursued in the attempt to identify carriers of the gene before the disease becomes manifest, but so far with little definitive useful results. A close resemblance in appearance and personality between carries an the affect parent has been claimed but it not well substantiated”. In 2009 Simon Lovestone wrote “Huntington’s disease results from an expansion of the CAG repeat with exon 1 of the gene encoding huntingtin (htt). As CAG is the codon for glutamine, this results in a string of glutamines being incorporated into the protein and Huntington’s disease is one of a series of polyglutamate disorders....” This underlines that many important areas have been significantly developed since the first edition and these are mostly reflected in expanded sections such as those on sleep disorders, neuropsychological testing, prion disorders and addictions. Perhaps surprisingly there is also a new section on schizophrenia, a trend set by the American textbooks of neuropsychiatry which really in my opinion cover “biological psychiatry”. Taken as a whole the coverage here is still very much organic psychiatry (rather than biological psychiatry or tightly defined neuropsychiatry in the sense of psychiatric aspects of neurological disease). This volume could therefore quite appropriately be considered to be “a textbook of liaison psychiatry” or at least have broad appeal to liaison psychiatrists and old age psychiatrists as well as neuropsychiatrists.

My overall impression of Lishman 4/e is that the combined authors have done a remarkable job of bringing this classic text uptodate. Actually many sections of original text are still in place but admixed with new portions. The new case examples in particular are a nice addition that appeal to the descriptive and very clinical style of Lishman’s own work. As such the style is quite different to say Moore’s brilliant Textbook of Clinical Neuropsychiatry (ISBN 0340939532), perhaps less encyclopedic but more familiar, rather like seeing a old friend in a new suit! I personally think that the early editions of Organic Psychiatry will remain valuable as historical documents of the neuropsychiatric approach from 1950 – 1998. Many of the 2000 or so key citations from 1978 have never been digitized so Lishman’s summary of early 1950s and 1960s studies is like a timeless fossilized record of scientific progress! Yes there are inevitably going to be limitations and in general these are fairly minor. In terms of emphasis, I would have liked to see more on impact on families and caregivers, more on medically unexplained symptoms, more on screening instruments, more on disease specific assessments, more on assessing global function and definitely more tables of evidence (although those that do appear are very informative). I do have a gripe about the illustrations as these continue to be rather sparse and plain (there are 13 or so colour images). More seriously the illustrations do not always give the correct credit to the original authors/copyright holders. There are also several indexing errors for example readers looking up myasthenia gravis or alcohol-related dementia will be directed to the incorrect pages. That said we have to look on this new publication as a marker of continued interest in this field and also have to congratulate the new editors and authors for preserving the essence of the original whilst bringing in much that is new. Given the rapidity of progress in the field I hope it is not another ten years before we see Lishman 5/e.

Alex Mitchell (Newsletter editor)
University of Leicester
9c. STANDING ORDERS - 2010 Meetings & Conferences

Cambridge Dementia Course 2009
Organizer: Jeremy Brown & John Hodges
10-11th December 2009
Contact penny.pearl@btinternet.com

10th Annual UK Movement disorders meeting
4th-5th December 2010
London UK
Contact neurology@boehringer-ingelheim.com

12th National Dementias conferences
18-19 February 2010
Contact: conferences@markallengroup.co.uk
(see backpage advert)

1st European Traumatic Brain Injury conference
24-27 February 2010
Vienna Austria
Contact nikolas.steinhoff@hochegg.iknoe.at

Royal College of Psychiatry 2010 BNPA-SoN Joint Conference *
10th February 2010
Topic: Memory disorders
Contact: Dela Goka
College Conference Office
Tel: 020 7235 2351 ext 142 Email: dgoka@rcpsych.ac.uk or Contact admin@bnpa.org.uk

Royal College of Psychiatry 2010 Faculty of Liaison Psychiatry Annual Meeting
3-5th March 2010
Cardiff, Wales
Contact: Dela Goka
College Conference Office
Tel: 020 7235 2351 ext 142 Email: dgoka@rcpsych.ac.uk

8th World Congress on Brain Injury
March 10-14th 2010
Washington DC
Contact congress@internationalbrain.org

21st Annual American Neuropsychiatric association Meeting
March 17 - 20, 2010
Marriott Tampa Waterside Hotel & Marina; Tampa, Florida;
Contact http://www.anpaonline.org/contact.cfm

62nd Annual meeting of American Academy of neurology
10-17 April 2010
Toronto Canada
Contact memberservices@aan.com

Association of British Neurologists Annual Meeting
11-14 May 2010
Bournemouth UK
Contact Karen.reeves@theabn.org

Royal College of Psychiatry 2010 Annual Meeting (featuring neuropsychiatry programme) *
21-24 June 2010
Edinburgh UK
Contact: Dela Goka
College Conference Office
Tel: 020 7235 2351 ext 142 Email: dgoka@rcpsych.ac.uk

Note : Call for new research papers and posters: deadline 11th January 2010

* of special interest
### 9d. STANDING ORDERS - Top 10 Web Resources in Neuropsychiatry

*Accessed December 2009*

#### Focus on Epilepsy

<table>
<thead>
<tr>
<th>Ranking</th>
<th>Link</th>
</tr>
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| #1      | Epilepsy Action  
http://www.epilepsy.org.uk/ |
| #2      | Epilepsy (Information, community, empowerment  
http://www.epilepsy.com/ |
| #3      | NHS clinical Knowledge Summary on Epilepsy  
http://www.cks.nhs.uk/epilepsy |
| #4      | Living Beyond epilepsy  
http://www.livebeyondepilepsy.com/home |
| #5      | Patients guide to functional Neurological Symptoms  
www.neurosymptoms.org |
| #6      | Fleminger - The neuropsychiatry of epilepsy JNNP 2003 [free full text paper]  
| #7      | ILAE - Evidence-Based Guidelines for the Treatment of Epileptic Seizures with AEDs [50 slides]  
www.ilae-epilepsy.org/Visitors/.../Guidelines-pptpresentation_001.ppt |
| #8      | AAN - Practice Parameter Update: Management issues for women with epilepsy [105 slides]  
| #9      | WHO Primary Care Guide to epilepsy  
www.whoguidemhpcuk.org/downloads/primary_care/Epilepsy.rtf |
| #10     | Emotional Concomitants of Epilepsy [145 slides]  
www.pnns.org/Emotion_Epilepsy.ppt |

*Rankings reflect opinion of the editor alone!*
MEETING OF THE SECTION OF NEUROPSYCHIATRY, ROYAL COLLEGE OF PSYCHIATRISTS & THE BRITISH NEUROPSYCHIATRIC ASSOCIATION.

10 February 2010, Institute of Child Health, London

Symposium on Disorders of Memory

(convened by Prof Michael Kopelman)

0920-1000 Concepts of memory
Alan Baddeley (University of York)

1000-1040 Clinical varieties of memory disorders
Michael Kopelman (St Thomas's Hospital, London)

1040-1120 Theories of hippocampal function in anterograde amnesia
Andrew Mayes (The University of Manchester)

1120-1140 Refreshments

1140-1220 A theory of hippocampal function in retrograde amnesia
Morris Moscovitch (Department of Psychology at the University of Toronto)

1220-1300 A theory of confabulation
Armin Schnider (University Hospital of Geneva)

1300-1400 Lunch

1400-1440 Clinical advances in our understanding and classification of the dementias
Nick Fox (Dementia Research Centre, Institute of Neurology)

1440-1520 Clinical, neuropsychological and neuroimaging studies of MCI
Peter Nestor (University of Cambridge)

1520-1540 Refreshments

1540-1620 Neuropsychological advances in our understanding and classification of the dementias
Julie Snowden (Greater Manchester Neuroscience Centre)

1620-1700 Rehabilitation of memory disorders
Barbara Wilson (Director of Research, Founder of the Oliver Zangwill Centre, Cambridge)

PLEASE SUPPORT YOUR NEWSLETTER AND SoN!

Please send me ideas and contributions for the next newsletter; especially articles relating to head injury to ajm80@le.ac.uk