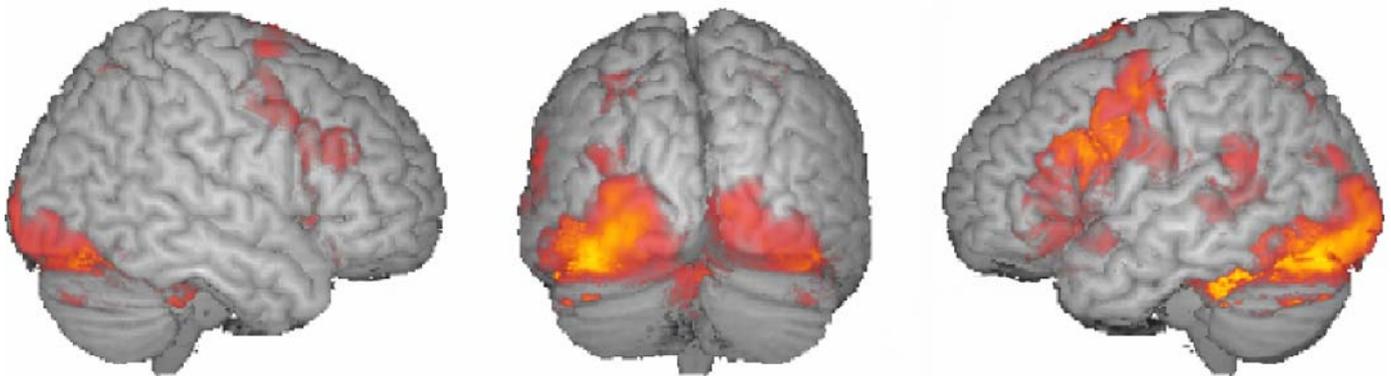


SECTION OF NEUROPSYCHIATRY NEWS

Newsletter of the Royal College of Psychiatry SoN



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1. Introduction to the New Section of Neuropsychiatry!

The Section of Neuropsychiatry is off to a flying start. We have already had two successful meetings in conjunction with other bodies, firstly the Division of Neuropsychology and, secondly, a full day of the joint meeting with the British Neuropsychiatry Association. Both were well attended and stimulating meetings. The Section of Neuropsychiatry meeting with the BNPA was very successful, with about 185 people attending in spite of the snow.

Neuropsychiatry is a sub-speciality which, whilst it clearly has a very definite identity, can be somewhat misunderstood by those outside the field. This must partly be because neuropsychiatry deals, in the clinical sphere, with such a wide range of conditions and uses such a wide range of therapeutic techniques, that it may be difficult to identify the work which one Neuropsychiatrist does as being similar to that which another Neuropsychiatrist does. Everything from the management of very difficult epilepsy with medication through to the management of conversion disorders with psychotherapy is included. Some Neuropsychiatrists will be dealing solely with the effects of severe traumatic brain injury and others with conditions in which there is no evidence of any kind of physical disorder whatsoever. Whilst this does make neuropsychiatry the single most fascinating area of medicine to be engaged in, on the other hand it might leave neuropsychiatry vulnerable to being seen as either a rather nebulous concoction of concepts or as a hyperspecialised clinical area with very little reference to real clinical practice. An important aim of the Section of Neuropsychiatry is to demonstrate that neither is true.

Neuropsychiatry is both a conceptual approach to the human condition and an area of psychiatry with particular clinical skills applicable to a number of specific disorders. Neuropsychiatrists are delighted that the Royal College of Psychiatrists has recognised this. The Section of Neuropsychiatry is, therefore, setting about taking a full part in the life of the Royal College. A programme of meetings is being arranged, firstly as part of the AGM and then with our own Section day on 25 September. Further collaborative meetings and courses are planned.

The Section of Neuropsychiatry has been active in liaising with other sections and with divisions and is maintaining a strong presence in the College. The neuropsychiatric viewpoint is being put forward in educational and training forums as well as in the Policy and Central Committees of the College. Our aim, as a Section, is to expand understanding of and training in neuropsychiatry, to support and expand services for patients with neuropsychiatric problems and to develop training posts in neuropsychiatry. One of our goals is to become a Faculty of the College.

Welcome, therefore, to this, the first edition of the Newsletter of the Section of Neuropsychiatry. Do join in the debate about where neuropsychiatry might go and, indeed, how it might get there. Neuropsychiatrists are a relatively small group of people, which should make communication between us easier. The Section of Neuropsychiatry and all its members need your support and enthusiasm.

Become part of this exciting venture for the further development of neuropsychiatry in the United Kingdom, make your thoughts known.

Jonathan Bird, Chair, Section of Neuropsychiatry



2. SoN Working Group: The Sleep Working Group

A couple of years ago I attended the American Psychiatric Association conference in Toronto. I was struck by how many sessions there were on various aspects of Sleep Medicine, and even more impressive was that these sessions were often so popular they had to turn people away. Apparently, psychiatrists are interested in sleep. This shouldn't be surprising – sleep problems are almost ubiquitous in psychiatric patients, and as sleep is primarily a mental function it seems reasonable that sleep problems should be the province of psychiatry.

Asking how well a person sleeps is a standard component of a psychiatric history. We are often particularly interested in early morning wakening as a symptom of depression. Yet other than one or two very broad questions we rarely explore the patient's sleep much further. We therefore miss out on a large chunk of our patient's lives, one which is often very important to them. That omission may well be more important than we previously thought. In addition to being disorders in their own right, subjective sleep complaints may provide important clues to the patient's diagnosis and can have important prognostic implications.

Insomnia is a good example. Even without associated depression, we are starting to realise that insomnia is a serious disease in its own right. A study on 3445 patients showed that insomnia had a similar impact on quality of life, as measured by the SF-36, to Congestive Cardiac Failure and Clinical Depression [1]. Furthermore, numerous studies have shown that the presence of insomnia significantly raises the risk of depression [2-6], as well as anxiety disorders and substance abuse [3]. As insomnia is one of the diagnostic criteria for depression, we often presume that it is secondary to the depression and will resolve when the depression lifts. But the evidence is mounting that insomnia precedes depression and is a particular risk factor for relapse in recurrent depression [6]. For this reason the National Institutes of Health in the United States have recommended that insomnia be described as comorbid with, rather than secondary to, depression [7]. This implies that, rather than being a symptom of depression, it is a disorder that psychiatrists should be treating assertively.

An understanding of sleep medicine may benefit psychiatrists in other, more unexpected ways. For example Obstructive Sleep Apnoea (OSA) would, at first glance, appear to be very much the business of respiratory physicians and to have little relevance to psychiatrists. But numerous studies have shown a significant overlap between the symptoms of OSA and Major Depression [8]. Many OSA patients are unaware of how disturbed their sleep is, but will complain of daytime symptoms such as tiredness, irritability, low mood and cognitive difficulties. This raises the question: how many patients with OSA are actually presenting with depression to psychiatrists, particularly as initial treatment with antidepressants by the GP is unlikely to resolve their symptoms?

Though we may not always be aware of it, all psychiatrists have an impact on our patients' sleep via the medications we prescribe. Most psychiatric medications alter sleep architecture, not always for the better, and many of them cause sedation or insomnia. They may cause other sleep-related side effects too. It is not surprising that antipsychotics can cause nocturnal Restless Legs Syndrome (RLS) and Periodic Limb Movement Disorder (PLMD) [9] as they cause movement disorders in waking patients. But how often do we warn patients that SSRI's can cause RLS and PLMD [10] or enquire about these side effects when patients are taking antidepressants? And of course we prescribe a great many sleeping pills, both licensed hypnotics and other sedative drugs. These medications all differ in terms of their effects on sleep architecture, daytime sedation, dreaming, and dependence.



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Furthermore, the science of Sleep Medicine has a great deal to offer in terms of understanding the mechanisms of psychiatric illness and its treatment. A case in point is the common finding of increased REM sleep in psychiatric disorders such as depression (and schizophrenia), and the mood elevating effects of REM deprivation in depressed patients. Most antidepressants suppress REM [11] and it is possible that this is the underlying mechanism of action of these medicines. Another example is the theory that Delayed Sleep Phase Syndrome may be the underlying pathology in Seasonal Affective Disorder [12]. And a better understanding of the neurobiological mechanisms of nightmares has opened up new treatment options for this symptom of PTSD [13].

So why aren't psychiatrists paying more attention to the sleep of our patients? Or for that matter why aren't there more psychiatrists working in sleep clinics, bringing our expertise to the field of sleep medicine? I believe this is largely due to failures in our specialist (and medical school) training. I received only two hours of teaching on sleep in medical school and a further two hours in my MRCPsych training program. My impression is that this is more than most. While we are drilled in the skills of taking a psychiatric history and eliciting symptoms which occur in the waking hours, we are not taught how to take a proper sleep history. Nor are we given the tools to interpret the results of that history or any subsequent investigations.



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It was for this reason that we have established the Sleep Group. Our primary aims are to foster a greater awareness of Sleep Medicine amongst UK Psychiatrists, to emphasise the importance of Sleep Medicine in psychiatric practice, and to encourage psychiatrists to contribute to the field of Sleep Medicine. To this end we have developed a curriculum, suggesting which areas of sleep science and medicine

psychiatrists should be familiar with. The curriculum specifies which competencies and areas of knowledge are relevant to each subspecialty and at what point in their training a psychiatrist should be familiar with them. Hopefully, the curriculum will be published on the Section of Neuropsychiatry website in the near future. We are also developing online CPD modules which will be available on the College website, as well as educational sessions and conferences.

The group is a good forum for discussing interesting clinical cases, sharing opinions and getting advice on sleep related issues, both at our 6 monthly meetings and via our moderated email list. Although we are part of the Section of Neuropsychiatry, we hope our activities will be of interest to all psychiatrists. Anyone wishing to join can do so by sending me an email at: sleepgroup@ymail.com

Hugh Selsick (Chair of the Sleep Working Group)

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This is the first of a series describing the SoN working groups. If you have any proposals for articles; case reports or similar for future newsletters.....please email Alex Mitchell ajm80@je.ac.uk

The snow that brought London to a standstill still lay on the ground as this year's Joint Conference with the British Neuropsychiatry Association got underway. But it did not seem to deter the attendees. Professor Trimble was greeted by a large attentive audience as he charted the evolution in our understanding of Broca's limbic lobe: from the localising of emotional response suggested by Papez to Nuata's extended limbic system. Given its rich connections with neocortical and evolutionarily ancient structures, the limbic system is now considered central to emotional and behavioural expression, hence fundamental to psychiatry. The case for making the limbic system the theme of the morning had now been won. Paul Johns, a specialist registrar in neuropathology, used many of his own dissections to provide structure for all this theorizing. Etymological deviations added depth to the most assured and absorbing neuroanatomy lecture this spectator has ever witnessed. Proof that neuropsychiatry addresses important socio-political events was provided by Professor Ray Dolan's presentation on his group's functional neuroimaging research on amygdala activation during financial reward and punishment. The audience was then eased towards lunch by Dr Caroline Brown, who spoke on the limbic system from a neuropsychological perspective.

"All proceeded convivially enough until someone mentioned Irving Kirsch's paper suggesting there is no evidence for antidepressant use even in depression."

One of the reasons for the importance

of the limbic system to psychiatry is that it is the site of action for so many psychopharmacological agents. It seemed natural therefore to organize the afternoon around this theme. Dr Mike Dille provided a comprehensive literature review of antidepressant use in neurological conditions, which included evidence that treatment of depression can have a greater impact on patient reported outcomes than treatment of the underlying condition. This was followed by an exhaustive survey of efficacy for antiepileptic drugs in psychiatric conditions. The audience benefited from Dr Jonathan Bird broadening the scope of his talk beyond the usual suspects to include newer agents. Both presentations also served to underline the dearth of robust evidence for pharmacological treatment in many common clinical situations. Professor Nutt shifted the emphasis in a presentation that demonstrated how biological models of schizophrenia have benefited from the development of antipsychotics and in particular the receptor affinity profile of atypicals. However, as many questions are posed as answered by these intriguing agents. The day ended with a discussion panel formed of the afternoon's speakers. All proceeded convivially enough until someone mentioned Irving Kirsch's paper suggesting there is no evidence for antidepressant use even in depression. Nutt's excoriating response drew a round of applause and left this delegate suspecting it would be safer to horse ride on ecstasy than get on the wrong side of this Professor.

***Norman Poole Locum Consultant Neuropsychiatrist
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When I saw the programme for this year's AGM of the British Neuropsychiatry Association (BNPA), I was immediately attracted to the timetable. I saw a unique opportunity to hear the latest theories from distinguished academics in the field. Unfortunately I was only able to make the final day. With train ticket in hand I braved the Nordic conditions and tested the limits of my aerobic capacity, ascending the 175 steps of Russell Square tube station en route to the meeting venue.

The opening lecture by Dr. Paul Allen focused on neuroimaging studies used to look at the hallucinating brain and what this has taught us so far. Dr. Allen has previously published a review on the same subject¹ and this formed the background of his presentation with a focus on the current progress on the topic. Dr. Allen covered the better known findings based on analytic and cognitive studies and then focused on current ideas around disconnectivity between various neural pathways being implicated in hallucinations. The lecture concluded with a discussion on gaps in our knowledge such as a lack of research from an affective neuroscience point of view and the role of dopamine transmission in the hallucinating brain.

Moving onwards from hallucinations, Professor Chris Frith discussed some of his work on understanding the neurobiology of passivity phenomena. This fascinating talk took us through how the self monitoring of motor actions can be described using a probabilistic Bayesian model and how dysfunction can lead to passivity. These complex models were nicely demonstrated by Prof. Frith raising and explaining the question of 'why can't we tickle ourselves?' Prof. Frith concluded by briefly covering the role of dopamine in prediction-error related belief formation². As was pointed out by the audience, symptoms of thought alienation and non-motor passivity phenomena can not be wholly explained by this model, and researching it would be a difficult task. The gauntlet has therefore been thrown down for any dedicated academics to take up this challenge.

After a short break, Professor Shitij Kapur presented his popular lecture on dopamine and psychosis. Professor Kapur rightly identified a gap in our understanding of psychosis between the neurobiological and clinical views. After setting the scene he explained the concept of motivational salience and how this bridges the divide between brain and mind^{3,4}. Kapur was quick to point out that this is a framework to focus future research into making the link between theory and what we see. As an added bonus, Kapur also briefly reviewed his own work on D2 receptor occupancy and fast dissociation rates. I personally feel this needs wider acknowledgement as it has a significant impact on our current clinical practice.

To conclude the theme Professor Paul Burgess explored the role of the rostral prefrontal cortex (PFC) in discriminating real from imaginary events. The rostral PFC is a mysterious part of the brain, with real insight into its function only being discovered in the last 10 years. In schizophrenia, a wide variety of abnormalities have been noted, however, the lecture limited itself to dysfunction in source memory: the ability to discriminate the real from the imaginary. It was proposed that the rostral PFC acts as a gateway for this function and convincing evidence was presented⁵.



After an extended lunch, the afternoon session picked up with the thought provoking theme of 'Neuroscience and Society.' Professor Barbara Sahakian discussed the neuroethics of cognitive enhancers to improve performance in healthy individuals. Anyone following media coverage of this topic will understand the controversy of advocating performance enhancing medications. However, remove the sensationalism, and Professor Sahakian's contention was that with the right ethical basis, why not⁶? Professor Sahakian did inject a modicum of common sense into the proceedings by reminding us that the best form of cognitive enhancers were physical and mental exercise and good education.

Professor Sergio Della Sala concluded the conference and session with his talk on the 'use and misuse of neuroscience in education.' He was well aware of the perils of providing the concluding lecture of a three day conference but did so with the showmanship of a circus ringmaster. Despite the riotously good humour, Professor Della Sala was making an important point. Neuroscientists, individually and collectively, have a duty to challenge the public perception of the brain. Otherwise tall tales will be accepted as fact and in extremis, quakery can take hold. As a taste of what was missed, a similarly themed and slightly more sober lecture of his can be viewed online⁷.

The atmosphere of the whole day was relaxed and the superb organisation was commendable. All in all, I returned to the hubris of the London Underground with a suitably stimulated Nucleus accumbens!

**185 delegates attended
the first SoN meeting!**

Raj Mann ST in Psychiatry, Leicester

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4. Service Report: The Birmingham Neuropsychiatry Service



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The Birmingham Neuropsychiatry service has been in existence for many years and was led by Dr Tim Betts through the 80's and 90's. Originally the service focussed on seizures and sleep disorders and developed a small video telemetry unit at that time.

More recently, the service has grown and now has three full time Consultants, Hugh Rickards, Manny Bagary (neuropsychiatrists) and Andrea Cavanna (a behavioural neurologist.). We have a visiting consultant in neurophysiology, Dr Alison Blake. The broad strategic aims of the service are to provide a service to people with the full range of neuropsychiatric disorders and to be at the cutting edge of research and treatment for these disorders.

The "bread and butter" disorders of the service are seizures, sleep disorders, tic disorders, Huntington's disease, chronic fatigue syndrome and somatisation disorders. We also see people with the psychiatric consequences of the full range of central nervous system illnesses.

The team consists of around 20 people including doctors, nurses, OT's, EEG technicians and an administrative team. There have been a number of new developments within the service recently. We have started to provide a service for people with CFS and related disorders following a grant from the Department of Health. This service has sat surprisingly well within neuropsychiatry, partly because of our experience of dealing with problems between the mental and physical domains. We offer diagnosis and a rehabilitation programme, mainly delivered by Occupational therapists and nurses.

Our telemetry equipment has recently been upgraded, which gives us a much higher quality of video than we were used to and the ability to navigate through hours of recordings very quickly. The technology allows us also to perform full polysomnography (including respiratory and posture measurement). This means that we are able to see and diagnose people who have the full range of sleep disorders.

As a result of high demand for the service (we have around 20 referrals to the service each week) we have recently introduced a triage system. People with seizures, CFS or sleep disorders are seen initially by an experienced clinician and routine investigations ordered at that time. In some cases, patients can be fast-tracked to therapy without seeing a doctor (particularly in cases of CFS). In other cases, the triage allows the relevant information to be collated before the appointment with the consultant.

Our specialist Huntington's disease clinic operates a "one-stop" approach with a full interdisciplinary team present in the clinic alongside a Regional Care Advisor from the Huntington's disease Association.

We have also pioneered a joint clinic with a consultant neurologist which takes referrals of people who have combined motor and psychiatric disorders. Commonly in this clinic, patients with complex diagnostic problems are seen including people with psychogenic motor disorders and the psychiatric consequences of motor disorders such as Parkinson's disease.



Research is an integral part of the service and research projects are often based in the clinics, which are commonly disorder-based. Part of our ethos is to develop young researchers into "clinical scientists" and give them the experience of collecting and presenting data as well as interacting with Journals and their editors. The department also runs a successful MSc in clinical neuropsychiatry with Birmingham University.

The main barriers to service development in the last few years have been related to the low profile of neuropsychiatry in major policy documents such as the NSF for mental disorders and to the relative importance placed by Mental Health Trusts on the development of functionalised teams within general psychiatry. Sometimes, neuropsychiatry has been viewed as an area with a dominant "medical model" and, therefore, out of step with modern and "new" ways of working. However, the patient group is still large and the need largely unmet so we are starting to make progress again with our Trust. Our main selling points to our Trust are that we can generate income and increase the profile of the Trust; both important factors to a Foundation Trust.

Our plans for the future are mainly to do with filling in the gaps to make a comprehensive neuropsychiatry service. This means developing better treatment options for people with somatoform disorders (particularly non-epileptic seizures), expanding the sleep diagnostic service and developing services for people with neurodevelopmental disorders in adulthood. So far the Trust have prevented us from developing this latter area as the amount of demand it would create would lead to a waiting list breach.

Advances in cognitive neuroscience have heralded a renaissance in neuropsychiatry and behavioural neurology. Clinicians of all disciplines are now starting to conceptualise more disorders as "neuropsychiatric" (a good example being Parkinson's disease) and, at last, Trusts are beginning to value neuropsychiatric service provision as a way of generating income, attracting good staff and improving their corporate image. Birmingham is a good example of how an excellent general neuropsychiatry service can develop and thrive.



The Birmingham Neuropsychiatry Team

Hugh Rickards
Consultant Neuropsychiatrist
Birmingham

Mr. DW is a 55 year's old divorced man referred to the Mental Health Services in March 2008. Initial assessment revealed several months' history of low mood accompanied with biological functions disturbance and experience of multiple somatic symptoms. There is past four year's history of seizures (grandmal tonic clonic epilepsy). The development of epilepsy was sudden on one morning when going to work on his push bike. No recollection of the incident but woke up in hospital. He was thoroughly investigated by neurologists with no specific findings. Epilepsy responded to Gabapentin 400mgs daily.

The initial diagnosis was depressive disorder (S Paradiso et al 2001) and commenced on Citalopram 20mg. He started feeling fearful during the nights with increased agitation, restlessness and distaste for foods, it was taken as further deterioration in depression and the dose of Citalopram increased to 40mgs daily.

The agitation and restlessness became worse and Mr DW reported "hearing a female voice", the voice appeared unpredictable, mainly commenting on his actions and more intense during the night (D Landsborough 1987). He initially thought there was a woman at the door whilst lying in bed and later on realised the voice as separate from his own thoughts. He was reassessed by the Team in August 2008, and Olanzapine was added to treat the voice which was thought as psychotic phenomenon. During the subsequent reviews the dose of Olanzapine was increased to 20mg daily. This generated experience of multiple voices including a male commanding voice ordering him to end his life (EMR Critchley 1998). Mr. DW took an overdose as a result and self presented to A&E asking to be kept safe.

The treatment with antidepressant medication triggered off temporal lobe epileptic phenomenon.

At that stage the condition was thought to be enduring and was referred to the Treatment Team for medium to

long term management. He was reassessed in clinic in February 2009. The link between "voices" and medication (antipsychotic / antidepressant) was established considering no associated psychotic phenomena (Masato Matsuura 1999). The initial clinical impression of Temporal Lobe Epilepsy was formed considering the association with inter-ictal language dysfunction (L Bartha et al 2005).

He was advised to start reducing Olanzapine and Citalopram supported by CPN in the community and prescribed Clonazepam 0.5mg at night for 2 weeks. He was reviewed 2 weeks later whilst on reducing regimen, and the male voice had disappeared by then. At that time Lamotrigine was discussed and commenced as per BNF guidelines. Further review of 4 weeks showed significant improvement with no experience of voices and improvement in mood.

Clinical Impression

Mr. DW developed an episode of depressive disorder due to possible limbic system dysfunction in temporal lobe epilepsy (F. Gilliam 2007) and multiple socioeconomic factors. In my opinion the treatment with antidepressant medication triggered off temporal lobe epileptic phenomenon. This became significantly worse when Olanzapine was added. The patient responded to Lamotrigine (D.

Chadwick 2007) addition which is a first line treatment for complex partial seizures, mood stabilizer and an antidepressant.

Discussion and Implications for Practice

The Wernicke's language understanding area is located on the posterior section of the superior temporal gyrus, encircling the auditory cortex on the Sylvian fissure (S S Shergill 2001). This area has connections with Broca's area, visual cortex and primary auditory cortex. The likelihood is that this area might have been the focus of epileptic activity created the clinical presentation as a solitary auditory hallucinatory experience with no associated features of psychosis. Alternatively, this may be due to reorganisation of motor and cortical language redistribution in human brain (H W Lee 2009).

Further Discussion & Learning Points for Section Members

1. Is there sufficient information to clearly establish epilepsy and or subtype?
2. Is there a robust relationship between biological aspects of seizure activity and depression?
3. Which antidepressants are recommended for those with established epilepsy?
4. Is there RCT evidence supporting specific antidepressants in epilepsy?

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

The Limits of the MMSE as a tool for Primary & Secondary Care Background to Cognitive Tests & Screening

We all know dementia is a clinical syndrome comprising many separate degenerative and acquired brain diseases, each capable of causing severe cognitive problems. Unlike most other psychiatric diagnoses verification is theoretically possible in the form of neuropathology at autopsy. However health professionals can really only diagnose “probable Alzheimer’s disease” *in life* even though they can diagnose definite dementia (as a syndrome) if persistent severe cognitive impairment is present. Actually, the diagnosis of dementia itself is not quite as straight forward as it sounds and this is reflected in the high rate of under-detection of dementia in primary care.¹ A possible solution to under-detection is for health professionals to routinely screen for dementia, ideally using a “*validated*” tool. A slightly less demanding approach is to screen for dementia only when cognitive impairment is suspected. The latter has been endorsed by NICE and also the American Academy of Neurology.² However, surveys show that the proportion of GPs who regularly look for dementia is less than 50%.^{3 4 5} Even for those with memory complaints cognitive testing seems to occur relatively rarely in primary care.⁶ Most people with memory problems ask their GP for help, hence GPs are particularly important in dementia care.⁷ Lawlor’s group found that GPs had difficulty differentiating normal ageing from symptoms of dementia.⁸ As a consequence, when unassisted, the recognition of dementia by GPs is less than ideal, particularly in the early stages.^{9 10 11}

Enter the Mini-Mental Status Examination (MMSE)! The MMSE is the most commonly used cognitive tool (in fact it is the most commonly used psychiatric tool of any type). Its 1975 publication became the most highly cited paper in psychiatry. It is purported to aid in detection of dementia but it is too time consuming for many.^{12 13} If shorter methods were available, surveys suggest they would be acceptable to most patients and PCPs.^{14 15} What is the current thinking about the value of the MMSE itself?

Value & Limitations of the MMSE

The Mini-Mental State Examination (MMSE) was published more than 30 years ago in 1975¹⁶ and has become the most commonly used cognitive screen. About 9 out of 10 specialists report using the MMSE “often or routinely” compared to 7 out of 10 who use the second most common tool, the clock drawing test.^{17 18} It is also the most extensively studied regarding diagnostic accuracy. In fact over 50 validation studies exist many direct comparisons with other methods (see our meta-analysis in margin). In one of the largest O’Connor et al. (1989)¹⁹ studied the instrument using a cut-off 23vs24

in 2,302 primary care patients, of whom 586 received a CAMDEX interview. Sensitivity was 86% and specificity 92%. This large evidence base is an advantage because any score on the MMSE is fairly well understood by colleagues. The cut-off on the MMSE that Folstein and colleagues recommended was 23vs24 in persons with at least 8 years of education. However this is arbitrary and numerous other cut-offs have been suggested depending on the population under study. The 2006 NICE Dementia guidance still advocated the MMSE but recommended a cut-off of 26v27. The MMSE has a number of limitations as a diagnostic test.²¹ It has a floor effect in advanced dementia, in those with little formal education and those with



Read more about our meta-analysis of the MMSE in *J Psychiatr Res* 2009; 43 (4):

severe language problems.²² More importantly there is a ceiling effect meaning it cannot easily score people with very mild disease.²³ Indeed its sensitivity is not high when diagnosing early dementia and MCI.²⁴ Further, some MMSE items and possibly the MMSE as a whole, may be unduly influenced by age, education, and ethnicity.²⁵ But as mentioned above it is considered too long by many clinicians (especially those working in primary care). It takes about 10 minutes to administer (longer in the case of someone with dementia) whereas most clinicians want to screen in 1-2minutes.

Alternatives to the Classic MMSE

Given these limitations with the MMSE what are the alternatives? Some have taken the approach of trying to improve the MMSE itself. Either by making it into a structured interview²⁶ or refining the discriminating items.²⁷ Fountoulakis and colleagues showed that no single item on the MMSE could act as a substitute for the whole examination in diagnosing dementia.²⁷ Certain items have been criticized because of completion difficulties. For example “naming” the 3 step command (because this is usually done correctly in early and moderate dementia), and the phrase repetition, reading the sentence "Close your eyes."

Competitors to the MMSE

There are many well-studied alternatives to the MMSE and several appear to be briefer but no less accurate than the MMSE. These fall into the following categories. Subjective memory tests (these have the least evidence to date); informant questionnaires, short batteries (mainly aimed at primary care), long cognitive batteries (like the CAMCOG) and specific neuropsychological tests. Taking the last these are typically memory tests but also tests of executive function reveal early changes in Alzheimer’s disease and other dementias, probably preclinically.²⁸

We recently conducted a meta-analysis about approximately 100 studies offering alternatives to the MMSE. In 20 studies authors looked at diagnostic accuracy head-to-head with the MMSE. Short screening methods of no more than 10 minutes had an overall sensitivity of 80.6%, specificity of 86.6%. The most successful battery methods were the original Blessed dementia rating scale (BDRS), the Memory Alteration Test, the DemTect, the Mini-Cog and the 6-item Cognitive Impairment Test (6-CIT). Most were superior to the MMSE itself. The most successful single domain quick screens were delayed recall (memory); verbal fluency and the clock drawing test. However all tests performed best at excluding a diagnosis of dementia (that is identifying healthy people) meaning there is no perfect screening test for dementia. The best tests help to narrow down the likely diagnosis but always with some margin for error.

Implementation Studies Involving the MMSE and Competitors

Given that a large number of screening methods exist, can they actually help clinicians improve detection rates? Khachaturian et al (2000)²⁹ used a sequential screening technique in Cache County, Study of Memory in Aging. 5092 elderly over > 65 were screened for dementia using the 3MS or, when subjects were unable to take this test the IQCODE. 91.2% completed the 3MS while 74 subjects had required a proxy assessment using the IQCODE. At the specified 3MS/IQCODE threshold of 86–87/ > 3.27, the authors were able to identify (and eliminate from further diagnostic consideration) over two-thirds of Cache County’s individuals without dementia, while overlooking fewer than 2% of those with dementia. In this sample of approximately 5000 subjects, a two-stage

screening method would save 360 clinical examinations of subjects with false-positive screening results. Barker and colleagues (2005) looked at retrospective data from 1489 consecutive patients with AD who presented to an outpatient memory disorders clinic between 1993 and 2002. Subjects with AD, who were referred by the memory screening program, had milder dementia and a lower reported duration of illness at presentation.³⁰ In a second retrospective study, [Borson and colleagues \(2006\)](#)³¹ looked at a primary care sample of (n =371) of predominantly ethnic minority elderly screening was studied. Clinicians correctly classified 59% of all subjects but identified only 41% of cognitively impaired subjects. 50% were recognized by the Mini-Cog but not by physicians, 32% by both, and 15% by neither. Only 3% were recognized by physicians but not by the Mini-Cog. The same group also reported a quality improvement screening project and quasiexperimental comparison of 2 intervention clinics and 2 control clinics. The Mini-Cog was administered by medical assistants to clinic patients aged 65+ years (n=524). 18% screened positive. Relative to baseline rates and control clinics, Mini-Cog screening was associated with increased dementia diagnoses, specialist referrals, and prescribing of cognitive enhancing medications. However, relevant physician action occurred in only 17% of screen-positive patients. Responses were most related to the lowest Mini- Cog score level (0/5) and advanced age.³² [Boustani and colleagues \(2006\)](#)³³ screened individuals aged 65 and older attending 7 urban and racially diverse primary care practices in Indianapolis using a screening algorithm. This included a first step 6 item screen and second step Community Screening Interview for Dementia (CSI-D). Results were compared against primary care records. Among 3,340 patients screened, 434 scored positive on both tests but 48% refused formal diagnostic assessment. Those refusing dementia assessment were older and had a better screening score. Only 19% of patients with confirmed dementia diagnosis had documentation of dementia in their medical notes. Recently [Jansen and colleagues \(2007\)](#)³⁴ conducted a cross-sectional comparison between usual identification of dementia by GPs and a two-stage screening to identify cognitive impairment. The two methods were implemented on the same older general practice population involving 44 GPs and 2,101 general practice patients aged 75 who lived at home. The two-stage screening yielded 117 patients with cognitive impairment who needed further examination; in most cases (70.1%) their GP was unaware of the symptoms. Among patients identified by the screening, GPs' awareness was associated with co-morbidity of chronic diseases, depressive symptoms and cognitive functioning.

Conclusions

These studies of screening programmes suggest, but do not prove, that screening for dementia works but with the caveats that only half of those initially identified as high risk may actually agree to formal cognitive testing and clinicians only act upon a positive screen in a minority of cases.

Ultimately, cognitive screening has to be acceptable not just to staff but also to patients. The MMSE is a useful bedside tool but it is probably too cumbersome for primary care and too imprecise for specialists. Many shorter, alternatives exist. No patients want to hear they may have dementia and as such any diagnosis must be accompanied to further help, follow-up and support. The MMSE can be a useful tool but should be considered a prelude to further input. Some of the flaws in the MMSE may be addressed in the forthcoming MMSE-II (currently under development and testing in the US).

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Membership of the Neuropsychiatry Executive Committee (cont)

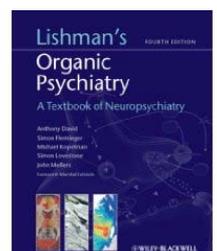
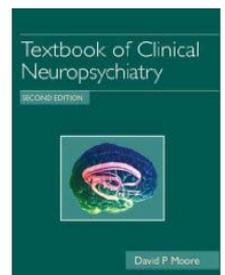
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Book Reviews & Previews

Mini Book Review: Textbook of Clinical Neuropsychiatry, 2nd edn

David P Moore, Hodder Arnold, 2008, 752pp, £115 / \$120

David Moore has written several textbooks on Medical Psychiatry (Mosby 978-0815164845) but now turns his hand to the “Textbook of clinical neuropsychiatry”, surely his most ambitious to date. It comprises 731 pages of detailed descriptions of CNS and systemic conditions that cause psychiatric complications. The book borrows some useful sections from his previous work and as a result is closer to a textbook of organic psychiatry than “pure neuropsychiatry.” The coverage is certainly comprehensive with often overlooked areas such childhood onset conditions, sleep disorders, drug induced disorders and substance use disorders conveyed with generous detail. All told there are almost 200 specific medical conditions discussed, each with a uniform approach: clinical features, course, aetiology and treatment. This makes the book ideal as a reference source for neuropsychiatrists. For me it is discussions on treatment that are the weakest but this is in no small part due to the difficulties in handling rapidly changing information. These sections would benefit from some attempt to convey levels of evidence underlying recommendation. It is notable that the book is very sparsely illustrated but this is compensated to some extent by some useful tables and excellent referencing. This text could be considered as replacement for a mainstream psychiatric textbook or as a competitor to Lishman’s Organic Psychiatry (3/e). However the new edited volume 4/e of Lishman...now “Organic Psychiatry - A Textbook Of Neuropsychiatry” (Eds Antony David, Simon Fleminger, Michael Kopelman, Simon Lovestone and John Mellers) is due for release on 3rd July at £99. It will be fascinating to compare the two.



Alex Mitchell (Newsletter editor)

2009 Meetings & Conferences

Joint BSRM/IARM Spring Meeting
14-15 May, 2009; Dublin, Ireland
T. 01992 638865
E. admin@bsrm.co.uk
Epilepsy: Psychological and Social Wellbeing
14 May, 2009; Edinburgh, UK
T. 0141 427 4911, www.epilepsyscotland.org.uk

12th Multidisciplinary International Conference of Neuroscience and Biological Psychiatry
"Stress and Behavior" - 2nd International Stress and Behavior Society (ISBS) Congress
16-20 May, 2009; St Petersburg, Russian Federation
E. isbs-2008@inbox.ru

Implementing the National Dementia Strategy
18 May, 2009; London, UK
T. 0870 400 1020 Capital Conferences,
E. david.moffat2@capita.co.uk

Royal College of Psychiatry Annual Meeting *
BT Convention Centre, Liverpool, 2-5 June 2009
A Fair Deal for all: mental health in a multicultural society
Contact: Dela Goka
College Conference Office
Tel: 020 7235 2351 ext 142 Email: dgoka@rcpsych.ac.uk

13th International Congress of Parkinson's Disease and Movement Disorders
7-11 June, 2009; Paris, France
Sarah Smith, The Movement Disorders Society,
T. 001 414 276 2145, E. ssmith@movementdisorders.org

5th World Congress of the International Society of Physical and Rehabilitation Medicine
13-17 June, 2009; Istanbul, Turkey
Ms. Sezen Elagoz,
T. 902 123 438 003 E. selagoz@teamcon.com.tr

Association of British Neurologists Annual Meeting
22 - 26 June 2009, Liverpool, UK
www.abn.org.uk/meetings/annual-meeting.php
E. Karen.reeves@theabn.org

International Conference on Alzheimers Disease (ICAD)
11th - 16th July 2009
Vienna
Tel: +43 1 58804-0 icad@alz.org

Royal College of Psychiatry 2009 Neuropsychiatry Section Conference *
25 September 2009
Contact: Dela Goka
College Conference Office
Tel: 020 7235 2351 ext 142 Email: dgoka@rcpsych.ac.uk

Royal College of Psychiatry 2010 BNPA-SoN Conference *
10th February 2010
Topic: Memory disorders
Contact person: Jackie Ashmenall
+ 44 (0) 560 114 1307
Email: admin@bnpa.org.uk
<http://www.bnpa.org.uk/>

Royal College of Psychiatry Annual Meeting 2009 Highlights

Neurology training course on 3rd June will include:

Core clinical skills in neuropsychiatry
What is neurology?
Common problems in brain injury
Conversion disorder Masterclass

Neuropsychiatry Institute on 5th June will include:

Mild cognitive impairment: what clinicians need to know?
How useful are advanced neuroimaging techniques in neuropsychiatry?
Epilepsy and neuropsychiatric aspects: what can a psychiatrist do?
Traumatic brain injury

* of special interest

Top 10 Web Resources in Neuropsychiatry

Accessed May 2009

Focus on Sleep

Ranking	Link
#1	The Sleep Site http://members.tripod.com/~sleephealth/phys1.html
#2	Sleep medicine Site http://www.users.cloud9.net/~thorpy/
#3	Update On The Evaluation & Treatment Of Insomnia (95 slides) <small>FREE POWERPOINT</small> Eben L. McClenahan, Louisiana State Department of Health & Hospitals www.dhh.louisiana.gov/offices/publications/pubs-305/INSOMNIA.ppt
#4	NHLBI (US) Resource site http://www.nhlbi.nih.gov/health/public/sleep/index.htm
#5	Home Sleep Australia http://www.homesleep.com.au/sleep_disorders.html
#6	Understanding Sleep, Part 1 & 2 http://counsellingresource.com/distress/sleep/understanding.html
#7	Dr Gelb / Andrea Knittel's (University of Michigan) Lecture notes on Sleep <small>FREE POWERPOINT</small> www-personal.umich.edu/~aknittel/Neuro%20Notes/Sleep.doc
#8	Dr. Edmondson, Uniformed Services University Sleep disorders Slides (48 slides) <small>FREE POWERPOINT</small> www.usuhs.mil/fap/capcon07/SleepDisorders.ppt
#9	Narcolepsy, Restless Legs Syndrome, and Sleep Apnea http://www.medscape.com/viewarticle/487399
#10	ShutEye (Help for individuals with insomnia) <small>INDUSTRY</small> http://www.shuteye.com/

Rankings reflect opinion of the editor alone!

**Date for
your diary!**

Section of Neuropsychiatry
Academic day

25th September 2009
14/15 Belgrave Square
London SW1X 8PS

Medically Unexplained Neurological Symptoms

9.00 Registration

9.30 – 10.15 Alan Carson, Consultant Neuropsychiatrist, Edinburgh
The nature and prevalence of medically unexplained neurological symptoms

10.15-11.00 Richard Kanaan, Clinical Lecturer in Psychiatry, Institute of Psychiatry
Neurologists' understanding and management of conversion disorder

11.00-11.20 coffee

11.20 – 12.00 Mark Edwards, Clinical Lecturer in Neurology, Institute of Neurology
Psychogenic movement disorder

12.00- 13.00 Sue Humbelstone, Psychiatric Occupational Therapist and colleagues from the National Hospital, Queen Square

The management of medically unexplained neurological symptoms by multidisciplinary therapy teams

13.00 – 14.00 Lunch

14.00 – 14.35 Epilepsy Working Group:
Non-epileptic seizures

14.35-15.10 Memory Working Group:
Psychogenic amnesia

15.10-15.30 Tea

15.30 -16.30 Clinical Forum:
The development of UK management guidelines for medically unexplained neurological symptoms

16.30 Close

Further Queries:

Conference Office
Royal College of Psychiatrists
17 Belgrave Square
London SW1X 8PG
Tel: 020 7235 2351 ext 145
Email: conference@rcpsych.ac.uk

Thirty Years of Neurobehavioural Rehabilitation

Past, Present and Future

8 – 9 June 2009
Hilton Hotel, Northampton

Speakers include:

Dr Peter Eames
Professor Jon Evans
Professor Peter Fenwick
Jakki Livesey van Dorst
Dr Tony Ward
Professor Huw Williams
Professor Barbara Wilson
Professor Rodger Wood
Dr Gavin Tennent
Peter McCabe
Laurence Marshall
Professor Nick Alderman
Dr Neil Rutterford
Laurence Marshall
Dr Peter Fenwick



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