Special issue on Movement Disorders
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Graphic Design: Jamie Paton – jamie@twhe.co.uk
Like the image inspired by photographer Anton Giulio Bragaglia on this issue’s front cover, the Newsletter captures a subject in energetic movement. Photodynamism, Bragaglia’s Futurist inspired approach to photography, transformed the photographic image from the trapping of a moment in time to the revelation of essence in motion and possibility. Who we are is revealed in action. As such, Rafey’s editorial should leave no doubt that Neuropsychiatry is a dynamic speciality. We have grown from a Special Interest Group to a Faculty in just over a decade; our membership continues to grow; publications sprout; and expert Working Groups on various themes are flourishing.

The current issue of the Newsletter focuses on movement disorders, as suggested to me some time ago by Elvina Chu, the Movement Disorders Working Group’s excellent Chair. Elvina coordinated the articles and corralled her colleagues in the field to produce a snapshot of the current state of their art. The articles range from the basic science of Huntington’s disease to the delivery of a service for those so afflicted; from the tics of Tourette’s to Parkinsonian impulsivity; and, any discussion of movement disorders would be incomplete without moving from the problem of failed action to the puzzle of failed volition. Each one of these thoughtful articles reveals both current knowledge and future possibility. A fitting tribute to Bagaglia’s passion for the dynamic.

Many psychiatrists will have little experience in the diagnosis and treatment of movement disorders and may see the subject as more properly the preserve of neurology. Indeed, two of the contributing authors are neurologists by training. One need not go so far as Wernicke – he claimed that all psychiatric illness would eventually be seen as disorders of motor function – to appreciate that disordered movement pervades psychiatry, from depression, through schizophrenia to dementia. Psychiatry should have a special skill here, interested as it is with both meaningful and causal processes as suggested by Jaspers in his General Psychopathology. So what could be more core to psychiatry than failure of meaningful action? And who better to manage movement disorders than psychiatrists (and eclectic neurologists such as Andrea Cavanna and Mark Edwards.)

Sermon over, I’d like to thank all those who took part in the recent elections for the Exec. I’d particularly like to thank all those who voted for me. I’m excited to join the cohort of new members and look forward to my four years with the movers and shakers of neuropsychiatry. Pun intended.
Neuropsychiatry is a fast developing specialty. We have formally achieved a Faculty status at the Royal college of Psychiatrists on 24th June 2014. Some colleagues say that we have waited for almost 15 years to achieve this professional recognition, the time that was required to establish the Special Interest Group, progressing to a Section status in the College and finally as a Faculty of the Royal College. The others would argue that psychiatry was practiced as neuropsychiatry to begin with and now with this new era of specialization neuropsychiatry is now again recognized as an area of highly specialist practice. I’ll argue that neuropsychiatry is for all, it is a bridge specialty that has its roots in all branches of psychiatry and as a specialist area of practice it does act as a vital bridge between clinical neurosciences and general medicine.

Special Interest Group in Neuropsychiatry (SIGN) was led wisely by a succession of colleagues including Howard Ring and Simon Fleminger. SIGN became a Section of the College at the 2008 AGM. This allowed us much wider opportunities for participation in the Royal College life; we established important working groups and promoted interfaculty working in order to bring cohesiveness in policy planning and training development. We established financial viability in a fairly short time-frame, and we submitted our proposed curriculum to the General Medical Council. We organized important academic events, networked widely with other organizations, participated in developing public policy agenda, engaged with service users, established international links to help influence revision of classification of diseases.

Contribution of a number of colleagues is worth mentioning. Jonathan Bird established the Section as the Chair in 2008, with able assistance from Niruj Agrawal as Vice Chair and my own input as the Finance Officer. Eileen Joyce and subsequently Howard Ring and Mike Kerr provided valuable input as Academic Secretaries of the Section. We were well placed with a number of senior colleagues as members of our Executive, Tony David, Alan Carson, Michael Kopelman, Saumitra Deb, Mayur Bodani, Malcolm Weller, Kenneth Wilson, and Peter Trimble to name a few. Alex Mitchell and Norman Poole served as the Editor of Neuropsychiatry News and all through this period Kitti Kottasz provided continuity and valuable administrative input as the Committee Manager.

We have recently published our guideline on Alcohol Related Brain Damage, a joint initiative that we
led with the help of other College faculties and in partnership with Royal College of Physicians, Royal College of General Practitioners, and Association of British Neurologists. Our Network on Childhood Onset Neuropsychiatric Conditions and Early Life Brain Injury is flourishing well. We organized a joint meeting with the Childrens Commissioner of England in March this year where we invited key policy makers and other stakeholders from healthcare and the Criminal Justice system to influence health policy and legislative decision making relevant to care of children and adolescents with neuropsychiatric conditions.

We are aware of training development and mentoring needs of our new consultants and psychiatric trainees. We have recently conducted a UK wide training survey alongside our Child & Adolescent Faculty on neuropsychiatric training. We will be sharing key results of this survey at the forthcoming neuropsychiatry residential conference at Oxford in September 2014. I am currently working with Jonathan Bird, Howard Ring, and Czarina Kirk in order to launch our Neuropsychiatry Mentoring Network, the first network of its kind by Royal College of Psychiatrists. We are consulting Pierre Taub College’s new mentoring lead to help build this network as a key membership relations and future workforce development initiative.

We are developing further initiatives; Mayur Bodani is leading on a business case for a trainee focussed Seminar Series in Neuropsychiatry and Monica Mahon will be assisting the Faculty to progress the work on development of a Neuropsychiatry Service Quality Monitoring Network.

I am actively engaged in consulting senior academic leaders in the UK and USA in order to have a wider understanding of training needs of future psychiatric workforce and most importantly how neuropsychiatry can influence competency development framework of our core psychiatric trainees. We have recently co-opted Hugh Rickards to the Neuropsychiatry Executive to benefit from his training development experience. We will be actively engaging with College Divisions and are currently in the process of finalizing structure of our new Education and Training Committee.

We have achieved another important milestone as a developing specialty; there will be more to come.
Huntington’s Disease: From basic science to clinical treatment

Elvina M. Chu

In the practice of clinical neuropsychiatry, we typically find ourselves treating patients with behavioural disturbance and psychiatric symptoms arising from an organic brain lesion caused by neurological disease, which adversely affects normal, brain function. This article attempts to summarise our knowledge of the science underpinning Huntington’s Disease (HD) and how this knowledge has been used to investigate potential treatments and search for possible treatment targets. This piece has been written in order to bridge the gap between science and the psychiatrist, as this disorder classically present with a triad of motor, cognitive and psychiatric symptoms.

George Huntington was a young family doctor following in his father’s footsteps, when he published his one and only scientific paper. He documented his observations that entire families across several generations were affected by a movement disorder that ran the same course of illness and was associated with insanity. Importantly he also noticed that “If the thread is broken then the grandchildren of the original shakers may rest assured that they are free from the disease”.

This observation described the genetic heredity of the disease in 1872, well before the concept of Mendelian inheritance had become widely accepted and recognized. In 1993 the precise genetic mechanism responsible for causing Huntington’s Disease was finally uncovered by a large international research effort. The Huntingtin gene (HTT) on the short arm of chromosome 4 codes for the protein huntingtin (Htt) which is expressed in all mammalian cells with a preponderance in the brain but a CAG triplet expansion on the HTT gene is responsible for production of mutant Htt. With the identification of a genetic mutation and the responsible gene identified, scientific research towards a cure for the disease could finally begin.

HD is an autosomal dominant genetic disorder, which means there is a 50% risk of disease transmission to offspring from an affected parent. Prevalence is 7 to 10 per 100,000 in most of the Western Hemisphere. Testing for the HD gene has been clinically available since 1994 but scientific advances now allow for identification of the exact number of triplet repeats within the expansion. A CAG repeat greater than 28 gives rise to unstable replication, usually
resulting in further expansion of this triplet (in 73% of cases); instability is greater in spermatogenesis hence the occurrence of ‘anticipation’ especially through paternal inheritance whereby successive generations inherit an increasing number of triplet repeats 4. Those with an intermediate repeat number between 35 and 39 are at increased risk of developing HD due to variable ‘penetrance’ referring to the proportion of carriers of a genetic mutation who will manifest the disease; whereas those with 40 or more CAG repeats have a 100% chance of developing HD.

Individuals who decide on genetic testing are possibly not representative of the HD population, they are typically more highly educated than the general population, more likely to be female, with a less depressed and more optimistic psychological profile than those who decline testing 5. Genetic testing is best offered with comprehensive specialist counselling pre-testing as a significant proportion of people will decide not to go ahead once they fully understand the implications and post-testing to monitor for psychological sequelae. Uptake of genetic testing is therefore surprisingly low (<5%) and uptake for prenatal diagnosis even rarer 6. Although anxiety and stress levels increase immediately after a positive genetic test result they soon return to baseline levels and at 2 years, distress is lower and well-being higher irrespective of the genetic test result 7. Individuals who have a negative genetic test result may also experience serious difficulties coping with their genetic status and a small number suffer from survivor guilt 5. In general, the concerns of an increased incidence of completed suicide, attempted suicide or psychiatric hospital admission following a positive test result are unfounded, although recent psychiatric history (>5 years) and unemployment are risk factors 8.

On a cellular level there is a gradual accumulation of mutant Huntingtin protein (Htt) but the normal function of this protein is still not completely understood. Key cellular features in affected individuals are the presence of mutant Htt aggregations (due to the expanded polyglutamine tail that the CAG triplet codes for), impaired cellular mechanisms for handling the abnormal proteins and presence of toxic protein fragments from mutant Htt, which appear to cause neuronal cell death. Selective loss of up to 95% of the GABAergic medium spiny neurons occur in the caudate and putamen 9 resulting in loss of inhibition to the globus pallidus and substantia nigra, while loss of large neurons occur in the cortical layers III, IV and V 10. The presence of intranuclear inclusion bodies, consisting of amyloid–like fibrils that contain mutant huntingtin, ubiquitin, synuclein, and other proteins also suggest that attempts are made to clear the affected neurons but the mutant Htt cannot be effectively cleared and is toxic 11.

Longitudinal MRI studies have demonstrated gross structural changes occurring in–vivo which can even be detected in premanifest individuals who are carriers of the HD gene but do not yet display motor signs required for a formal diagnosis of the disease 12. Specific basal ganglia atrophy is readily visible by magnetic resonance imaging scan and progresses over time 13. Gross cortical atrophy is also readily detectable on magnetic resonance imaging, and increasingly sophisticated volumetric analysis has demonstrated early and progressive changes in the cortex 14.

As HD is primarily a genetic disorder, the obvious treatment target is through blocking the affected gene by “gene silencing” so further production of mutant Htt is diminished. This is the only potential mechanism for curing HD but the difficulty has been finding a suitable vehicle that can pass the blood–brain barrier carrying antisense oligonucleotides (ASOs) to catalyze degradation of huntingtin...
mRNA in the brain. Gene silencing treatment has already been trialled in motor neurone disease (where the aetiology is mostly unknown and only 2% of cases are due to a genetic mutation), clinical trials are due to be extended to include HD patients next 15.

Amongst the current selection of medications used to treat motor symptoms of HD, tetrabenazine is the only licensed dopamine depletor for the treatment of chorea but depression can be a problematic side effect (16). Antipsychotics are a second-line treatment of choice especially for chorea with comorbid aggression or psychosis but no single drug has been demonstrated to be more efficacious and patients appear to be surprisingly responsive to even very small doses e.g. 2.5mg of olanzapine.

Depression, irritability and obsessive-compulsive symptoms are common neuropsychiatric presentations which are often seen in HD but these are usually responsive to treatment with appropriate psychotropic medications. Anecdotal evidence supports the use of paroxetine, sertraline, fluoxetine and mirtazapine but to date there have been no RCTs carried out to inform us if any one drug is more efficacious than the other 17. The efficacy of cognitive behavioural therapy (CBT) in the treatment of anxiety in early stage HD patients has been demonstrated 18 but in the case of apathy (which is a common feature of HD) there are no treatments either pharmacological or psychological which are of proven benefit 19.

Antipsychotic medications are useful in HD for treating chorea and psychosis. A review by Adam and Jankovic 17 described evidence from studies of typical antipsychotics including haloperidol as equivocal but these studies contain only small patient numbers. There was however, clear evidence to support using the atypical antipsychotic olanzapine to help relieve both motor and behavioural features of HD with reductions noted in chorea, depression, anxiety, irritability and obsessions. Olanzapine is both well tolerated and effective, even at a low treatment dose; with risperidone, quetiapine and ziprazidone also of proven efficacy in HD treatment.

A potential new medication for the treatment of movement disorder in HD is pridopidine (ACR-16), a dopamine (DA) stabiliser that has now reached phase III clinical trials. Results from the large HART study involving 277 subjects from 27 sites found no significant effects in secondary outcome measures with any of the active dosages 20, 45 or 90mg daily. Pridopidine was generally well tolerated but the primary analysis did not demonstrate a statistically significant treatment effect 20. Some benefit in motor symptoms have subsequently been reported using a higher 90mg dose 21. Importantly this drug may also be of interest in the treatment of psychotic illness due to the main mechanism of action on regulating dopamine levels. (−)-OSU6162 and the novel compound NS30678 are the other promising DA stabilizing drugs currently under testing in HD 22. In contrast to the high-affinity typical antipsychotics such as haloperidol and raclopride, the dopaminergic stabilizers ACR16 and (−)-OSU6162 both display fast dopamine D2 receptor dissociation, a feature that has previously been suggested as a feature of atypical antipsychotics and attributed to low receptor affinity. NS30678 (which is equipotent to haloperidol and raclopride) also displays fast receptor dissociation,
suggesting that the agonist-like structural motif of these dopaminergic stabilizers is a critical dissociation rate determinant.

Dopaminergic stabilizers exhibit fast competitive D2 receptor antagonism, possibly allowing for temporally variable and activity-dependent receptor occupancy that may partly account for their unique stabilisation of DA dependent behaviors in-vivo.

In the search for treatments to delay symptom onset, food supplements have stirred recent interest. Creatine is typically used by body builders who are looking to increase muscle mass and has caught the interest of HD researchers looking to prevent body tissue from wasting away. In a recent longitudinal study, premanifest HD gene carriers taking high dose creatine demonstrated slowing of cortical and striatal atrophy at 6 and 18 months; however, no change in the level of cognitive deficits were noted. As the substance was well tolerated by most participants and treatment effects were equivocal, a much larger phase III clinical trial (CREST-E) is now in progress. Evidence suggests that HD causes excessive oxidative damage to cells and tissues due to the mitochondria producing abnormally large numbers of free radicals, hence anti-oxidants might be a preventative treatment. In an attempt to reverse the energy metabolism deficits in HD, co-enzyme Q10 a co-factor of the electron transport chain and another readily available supplement has been trialled. Although the first large clinical trial CARE-100% did not show any benefits it is possible that the blood-brain barrier blocks co-enzyme Q10 from entering the brain and a subsequent large scale study is currently underway.

During the 1950s, serendipity led to chemists finding that phenothiazines were useful antipsychotic drugs when they were in fact trying to develop an antihistamine. In a similar vein, another significant HD treatment trial has included latrepidine (Dimebon) a retired Russian antihistamine. This drug appears to have some cognitive enhancing activity and is currently under evaluation in phase III clinical trials.

In summary, treatment of HD is by no means curative at present but because the disease mechanism is understood, attempts can be made to target prevention of mutant Htt protein production. Neuropsychiatric symptoms of HD can usually be effectively treated with psychotropic medications but of course these do not reverse disease progression and serve only to provide symptom relief. Medication developments currently include investigation of substances that can alter cellular mechanisms involved in metabolism and neurochemical transmission. Such developments are also of potential interest to psychiatrists as some of these mechanisms are also implicated in psychosis. It may be through knowledge gained by studying two different disease models in parallel (HD and schizophrenia) that will help us to develop ideas for future treatment strategies of psychosis.
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Huntington’s disease (HD) bears the name of George Huntington who described the disease in his talk, and subsequent only publication, in 1872 (Huntington 1872) although others had also described essentially the same disease in earlier years.

HD is a complex autosomal dominant neurodegenerative condition that prominently affects the basal ganglia. The basis of HD is a cytosine–adenosine–guanine (CAG) repeat expansion in a gene that codes for a ubiquitous protein known as huntingtin. The size of the expansion is correlated with the disease onset, with increasing CAG accelerating the age of onset. Each child of an affected parent has 50% chance of inheriting the disease. People with HD develop symptoms at any age from infancy to old age, but they usually begin between 30 and 40 years of age. It often presents with psychiatric disorders (including personality changes), cognitive impairment along with motor and other physical problems. About 10% of cases start before the age of 20 years (Juvenile HD) with an akinetic–rigid syndrome and 10% after the age of 60.

It is difficult to obtain accurate prevalence data on HD, not least because of the stigma attached to the condition. Nonetheless, recent studies have indicated that the prevalence in England and Wales is much more than what has always been quoted. This is estimated to be 12.4 per 100 000 or even higher (Rawlins 2010).

**Need for Specialist Services**

The complex and familial nature of the disease raises a number of ethical and clinical issues that would make its management in a less specialist setting, much less favourable. Patients with this disorder have historically fallen between generic neurology and psychiatry services. Understandably, neurological services tend to focus on the motor aspect of the condition, not having the required skills or resources to manage the other aspects. It has long been argued that neuropsychiatric manifestations of HD are much more disabling than the more obvious motor symptoms. It is often the effectiveness of managing neuropsychiatric symptoms that would determine the viability of community treatment as opposed to having to resort to nursing care in a residential setting.

General psychiatric services, on the other hand, will feel less equipped to look after patients with “neurological” problems that include noticeable movement disorders.
Patients themselves may feel vulnerable if they had to receive psychiatric treatment in a “functional” setting, where patients with severe mental disorders tend to be looked after (El-Nimr and Tams, 2011).

This is particularly important for clinicians to appreciate that the needs for HD patients tends to cross boundaries between various disciplines of health and social care.

Specialist services are therefore required to meet the complex needs of patients with HD and to ensure that they are not being “discriminated against” or deprived of essential psychiatric care on grounds related to their physical disability. This perceived discrimination can be partly related to professionals’ reluctance to adopt psychiatric clinical and medico-legal principles in “neurological patients”. One example would be the frequently voiced uncertainty in relation to the appropriateness of utilising the Mental Health Act in certain situations.

The heterogeneity of the clinical presentation among different family members together with the different facets of needs within the same individual throughout the course of the illness do indeed call for specialist skills and resources. Treatment algorithms and care pathways have been developed to address the complexity of managing this disorder especially for primary care colleagues who may have very little encounter with HD (El-Nimr and Barrett, 2006).

The newly developed standard of care guidance that was published by the Standard of Care Working Group of the European Huntington’s Disease Network (EHDN) does highlight the need for input from various disciplines (Simpson and Rae, 2012). This work highlighted the lack of internationally recognised standards, having only a very few peer reviewed papers on this topic. It was also acknowledged that this could be at least partly related to the fact that many countries still lack specialist services for HD patients. Where services do exist, the care that is available for HD families varies quite considerably across various countries. Furthermore, which discipline coordinates care for this patients group does also vary between and within countries. In related work, Simpson presented data collected from 28 clinicians involved in specialist care of HD in 9 European countries. This also highlighted the heterogeneity of care provided to this patient group. Similarly, methods used for clinical assessments of HD features were not uniform.

**Patients’ involvement**

HD provides an excellent model of how effective family engagement can lead to a significant impact on scientific development and care provision. When Woody Guthrie, the American folk singer developed HD around 1952 and died in 1967, his widow Marjorie Guthrie devoted her life to promoting all aspects of HD. In 1967 the Committee to Combat Huntington’s Disease which evolved into the Huntington’s Disease Association, was formed with objectives to provide service to families and to promote education and research. Based on Marjorie Guthrie’s work, in 1978 the International Huntington’s Disease Association (IHA) was established. IHA is a federation of national voluntary health agencies that share common concern for patients and families who are affected by HD. Similarly, the Hereditary Disease Foundation was initiated by Dr Milton Wexler after his wife developed HD and continued by his daughter Nancy Wexler. This organisation was instrumental in the collection and analysis of the large Venezuelan kindreds that were important in cloning of the HD gene (Harper et al 2002).

In 2012, “Project AWARE” (Awareness, Willingness, Ability for Research and Evolvement) in the USA presented a survey conducted by the Huntington’s Study Group defining the issues the HD community faced. This project highlights that without greater family participation, there can be no new knowledge or treatment for HD.

In Europe, the European Huntington’s Disease Network (EHDN) has created an excellent platform for clinicians, scientists and family members where recommendations for good quality care and research work can flourish. A number of working groups have taken on an international lead role in clinical, scientific and other areas related to HD. With regards to service provision, specific working groups have highlighted specific evidence-based interventions that existing and newly developing services should adopt. The value of physical activities and exercise programmes is a particularly good example of this. The essence of many pieces of work is the fact that despite HD is perceived as an incurable condition, it does have a number of treatable consequences.
It has always been highlighted that one important key to success in delivering effective treatment and support to patients with HD is to create integrated interdisciplinary teams with effective family involvement.

The Huntington’s Disease Association (HDA) of England and Wales has been very active in supporting families and clinicians. The association also plays an instrumental role in enhancing the public awareness and raising the HD profile on the political agenda. The work of the association in the context of All Party Parliamentary Group is one example.

Over the recent years, it is certainly inspiring to witness the developments of a number of further initiatives that support and are supported by HD families. HDBuzz provides an excellent service for patients and families, communicating recent research findings and scientific discoveries in an easy-to-understand language. Similarly, Huntington’s Disease Youth Organisation, HDYO offers a great service and support for young people affected by HD (either directly or through living in a family with an HD member) from around the world. It also works with the youth to raise awareness and facilitate access to research activities.

**HD Services in North Staffordshire**

This service was established by a general psychiatrist who developed neuropsychiatry services over 20 years ago virtually by identifying the need and supporting individual patients with psychiatric manifestations in the context of brain diseases or damage. Since then, patients with HD are being looked after in the context of generic neuropsychiatry services. Our service focuses on developing a holistic inter-disciplinary care approach for this patients group and their families in different clinical settings (outpatient clinics, inpatients, day therapy and community treatment). The ethos of the service is in keeping with the National Service Framework for long-term conditions which highlights the need for a comprehensive service for such disorders.

Our service caters for a population of approximately half a million people (over an area of 40 by 20 miles). Out of area referrals are also accepted from different departments and disciplines. Patients with HD and carriers of the mutated gene are looked after from the time of referral (when they could still be in the pre-manifest phase of the illness, having had a positive predictive genetic test or in the context of a pre-test assessment) up until the end of their lives, with no age restrictions. The neurological, psychiatric and social consequences of the illness are managed in the context of the service. The team also work quite closely with our regional clinical genetics services supporting predictive and diagnostic testing.

**Inter-disciplinary clinic**

Our award winning inter-disciplinary HD specialist clinic is one aspect of the service. This takes place every 3 months. It offers services for people across North Staffordshire and surrounding districts who are diagnosed with HD as well as carriers of the expanded Huntington’s gene who could still be in the pre-manifest phase of the illness. A detailed but simple leaflet has been developed outlining what patients should expect during the course of the clinic. Patients are informed of the clinic in the context of other settings of care delivery and it is also promoted by our local branch of the HDA. Our staff attend the local branch meetings to explain the purpose and rationale of the clinic as well as providing educational materials some of which have been developed by our department.

All patients are invited to 1.30 pm appointments where they will be assessed by various professionals including consultant Neuropsychiatrist, Consultant Neuropsychologist, physiotherapist, occupational therapist, speech and language therapist, genetic counsellor and HDA Regional Care Advisor. The clinic is co-ordinated by a community psychiatric nurse who will also be identifying any ongoing needs for community support. At the waiting area, the Chairperson of the local HDA branch will be meeting and greeting patients and families. As a carer, she is also able to have informal discussions with family members, answering questions and providing relevant information leaflets and other information about HD and available support groups. Many carers take the opportunity to discuss their problems and concerns with her, often raising matters with ‘a fellow carer’ they could be reluctant to share with other members of the team. She also spends time with junior doctors and students allocated to the clinic. In the waiting area, there will be refreshments that will be appropriate for this patient group, who are likely to have some swallowing difficulties. This also helps promote appropriate eating and drinking habit.
The consultant Neuropsychiatrist assesses patients’ neurological and psychiatric conditions, reviews medications and provides overall leadership to the clinic. He subsequently communicate the main findings of the team and agreed actions to the GP and other colleagues who may be involved in the patient’s care. Assessing patients’ capacity in relation to making specific decisions, such as accepting / declining life maintaining measures, is also considered by the Neuropsychiatrist.

Speech and language therapist assess and give relevant advice on swallowing and communication. Our occupational therapist looks into everyday functionality and safety issues while our physiotherapist will offer advice on mobility, breathing and physical exercises which tend to be followed up at our day services. Our Neuropsychologist explores cognitive difficulties and looks into ways of how the outcome of subsequent formal assessments can be utilised therapeutically. Equally other non-pharmacological approaches are considered especially in patients with mood or behavioural difficulties. Patients and families do also benefit quite considerably from having a discussion with our genetic counsellor especially with regards to issues related to genetic status within the family.

Following the clinic, the team meet up to agree an integrated and holistic management plan which is communicated to various relevant professionals and the patient’s general practitioner.

This kind of clinics has proven to be quite popular with patients who now do not have to travel on several occasions for hospital appointments. However, follow-up assessments can be arranged by various professionals (including medical outpatient reviews), based on the outcome of the clinic assessments. The clinic has also proven to be an excellent training resource for students and trainees from different disciplines. No specific funding was sought for this clinic. Instead, funding was established through channelling certain resources within our generic Neuropsychiatry service.

No specific funding was sought for this clinic. Instead, funding was established through channelling certain resources within our generic Neuropsychiatry service. It is, however, worth highlighting that the most effective ‘active ingredients’ were patient/carer involvement and motivating staff from different disciplines to take part. Most professionals have found the clinic an excellent forum for them to be able to work holistically with this patient group.

**Day Services Centre**

This service provides a dedicated 2 days/week to HD patients. Patients also have access to various professionals including speech and language therapist, physiotherapist, occupational therapist, and community nurses. Input from dietician is also arranged. Speech and language therapist provides assessment during meal times and offers advice on swallowing and communication. A physiotherapist helps with mobility and balance by using a range of treatments, including manipulation, massage and exercise. Deep breathing exercises are also taught which appears to be not only useful for relaxation purposes but also in maintain lung capacity. This kind of therapy encourages people with HD to remain as active as possible; maintaining cardiovascular fitness. An occupational therapist helps with day-to-day activities and implement safety measures to prevent accidents and promote quality of life. A range of activities are organised to keep patients active and stimulated. Examples include conducting quiz, Dominos, art and crafts. A referral to dietician helps work out an appropriate diet plan and explores the need for a high-calorie diet. Our Neuropsychiatric nurses monitor mental health and relevant aspects of physical health such as involuntary movements and weight monitoring. Other professionals are also alerted should any concerns be identified. The centre also offers therapies like anger and anxiety management and provides general physical health check. This service has proved to be beneficial to the patients both in terms of monitoring their HD symptoms and socialisation. The
centre also provides support to carers and other family members.

**Inpatients Service**

A 15 beded inpatient Neuropsychiatry service available for patients over the age of eighteen. Besides HD, the ward also caters for patients with other brain conditions who are presenting with cognitive, behavioural and other psychiatric conditions. While there are benefits from having an HD-only unit, it has been argued that care should be offered based on need as opposed to condition. Furthermore, it is not uncommon for patients in early stages of the illness to find it quite stressful when nursed alongside others who could be in advanced stages.

Majority of these patients are admitted informally. However, there is a small percentage of patients who have to be treated under Mental Health Act (1983). Individualised assessment, treatment, rehabilitation and behavioural programmes for all stages of the disease are offered.

Patients are either discharged to their homes with a wide range of community support or to a more supported accommodation; depending on their needs. Patients are often followed up in the community by various team members, including regular medical reviews.

**Community Neuropsychiatric Team**

Our Community Psychiatric Nurses offer regular reviews and monitoring for patients in their own homes and in residential settings. Amongst various other aspects, mental health, weight, response to medication, specific risks are monitored. The community team work closely with family members and care homes to support and educate carers and families which in turn will have a positive impact on the quality of life of patients. Inpatients admissions are organised when appropriate, either to address immediate crisis or to undertake a piece of clinical work that could be unachievable or less safe to carry out in the community. Our community nurses also provide help in social needs like processing applications for benefits, continuing health care funding applications and arranging respite care.

Community Support Workers organise weekly support groups and organise trips according to patients’ needs and interests. Examples include gardening, visits to museums and sport activities. They also undertake monitoring of wellbeing and support to access community activities as well as helping with issues such as benefits.

Other members of the team from various disciplines are involved and offer community assessments and support based on the need.

It is not uncommon for patients with HD to be admitted to general hospital due to physical health complications like chest infections, mal nutrition, weight loss as well as unrelated physical problems. Quite understandably that colleagues from general physicians will have limited expertise and resources to manage such patients with significant communication, motor and behavioural difficulties. The team provide advice and support to professionals, patients and families by attending the general words and liaising with the hospital staff.

**End of life**

In the late stages of HD, patients have difficulty thinking and communicating clearly, so decisions about their end-of-life medical care often fall to doctors and other clinicians. Patients are usually encouraged to make advance decisions about end of life issues, while they are fully capable. For example, issues such as possible future PEG feeding is discussed with patients early on in the disease. Information is given and other team members as well as other teams are often involved to ensure that patients are able to make informed decisions.

Accessing support from other teams such as district nurses, orthotics, chiropodist, and dental care are usually facilitated when appropriate.

Similarly, advice on pain control and other specialist palliative care issues are sought from the non-cancer palliative care team (See Department of Health. End of Life Care Strategy, 2008)

**Service evaluation**

Based on a Service Evaluation Project that was conducted in 2009, most referrals came from GPs and clinical genetics (at 40% and 29%, respectively). Motor symptoms formed the initial presentation in 57%. Slightly over half of the patients whose genetic data
were available (73%) had a paternal gene transmission. Twelve per cent of individuals were asymptomatic gene carriers. Virtually all HD clients received outpatient reviews and 74% received CPN follow-up.

About half of the patients had a period of inpatient assessment, 13% of which was under the MHA. Respite admissions and day services were offered to 22% and 29%, respectively. Approximately 75% received input from neuropsychology and SALT. Physiotherapy and occupational therapy offered input to 56% and 61%, respectively (Valanciute & El-Nimr 2009).

**Other activities**

Having won our Trust’s Chairman’s Award back in 2009, the team started to further expand their work in clinical and non-clinical domains. Three UK national conferences were organised by the team in subsequent years. Those events had readily earned an international status, having been supported by international speakers, sponsors and delegates from a number of European countries. Similarly, the team became more involved in research activities, especially in the context of the European Huntington’s Disease Network. More recently, the team has been making arrangements to join the Enrol-HD project which also includes centres from North and South America as well as Australia. Members of the team have also developed and contributed to the development of training programmes and educational materials including national DVDs, CPD module and other online resources. The first author has completed, with a colleague, and online CPD module for the Royal College of Psychiatrists that is currently available for educational purposes.

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**Useful websites:**

www.euro-hd.net
en.hdbuzz.net
en.hdyo.org
www.huntington-assoc.com
www.hdsa.org
www.huntingtonsociety.ca
www.hdfoundation.org
www.huntington-studygroup.org
www.wemove.org
www.kumc.edu/hospital/huntingtons
www.hdac.org
www.nhs.uk (search ‘Huntingtons’).
Gilles de la Tourette syndrome

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Tics and Gilles de la Tourette syndrome

Gilles de la Tourette syndrome (GTS), also referred to as “Tourette syndrome” or “TS” in the UK and USA literature, is a neurodevelopmental condition characterised by multiple motor and vocal/phonic tics, which persist throughout life and tend to be associated with specific behavioural symptoms. This fascinating syndrome was described by French physician Georges Gilles de la Tourette in 1885, and was considered a rare medical curiosity for about a century. Epidemiological studies conducted over the last few decades using standardised diagnostic criteria have revealed that up to 1% of school-age children can be affected and it has been estimated that 200,000 to 330,000 individuals in UK can fulfil diagnostic criteria for GTS, with different degrees of severity. Moreover, GTS has been shown to significantly affect health-related quality of life of patients, thus highlighting the need for a better understanding of its complex clinical presentation and the available treatment options, which range from behavioural interventions to pharmacotherapy and neurosurgery.

The core symptoms of GTS are tics, defined as involuntary, sudden, rapid, recurrent, non-rhythmic movements (motor tics) and vocalisations (vocal or phonic tics, depending on the involvement of the vocal cords). Of note, the adjective “stereotypical” was removed from the definition of tics in the new edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM–5), in order to avoid confusion with...
stereotypies, which are repetitive behaviours of a different nature. The presence of at least two motor tics and one vocal/phonetic tic is required to fulfil current diagnostic criteria for GTS, whereas patients who have motor tics only or vocal tics only are diagnosed with chronic motor tic disorder and chronic vocal tic disorder, respectively. The diagnosis of GTS is based on clinical observation and anamnesis: specific investigations, including laboratory tests and neuroimaging, can be requested to rule out other possible underlying causes for tics in patients with atypical presentations (e.g. late onset etc.).

The average age at tic onset is around 6 years and GTS is about four times more common in males than in females. Simple motor tics like eye blinking, neck stretching, shoulder shrugging usually precede the development of vocal tics (e.g. grunting, throat clearing, sniffing). Tics are characteristically preceded by premonitory urges, i.e. subjective feelings of mounting inner tension, which is temporarily relieved by tic expression. The presence of premonitory urges is an important feature for the differential diagnosis between tics and other involuntary movements, such as tremor and myoclonus. Moreover, patients can be able to resist these urges for a variable period of time (usually seconds to minutes). However voluntary tic suppression results in subsequent rebounds in tic severity, reflecting progressively mounting inner tension.

Each patient with GTS has an individual repertoire of tics, which is not stable over time, as tics tend to vary in frequency, severity and distribution throughout life. They are characterised by a waxing and waning course, usually with a severity peak in early adolescence. Environmental factors such as stress, anxiety or boredom often exacerbate tics, whereas focused mental and physical tasks requiring concentration (e.g. playing sports and music) can alleviate them.

Despite their extensive portrayal in the media, coprophenomena (coprolalia, i.e. swearing as a tic, and copropraxia, i.e. making rude gestures as a tic) are thought to affect only about 10% of patients with GTS (with a slightly higher prevalence in specialist clinics because of referral bias). Importantly, patients who present with these symptoms tend to mask them and are apologetic after they failed to suppress them in public. Of note, Georges Gilles de la Tourette’s original case series described nine patients who also presented with coprolalia and another complex tic, namely echolalia (repeating other people’s words). From the point of view of clinical phenomenology, complex tics tend to occur on top of simple tics rather than in isolation. Moreover, the expression of socially inappropriate behaviours seems to be modulated by the external environment and life situations, as showed by recent studies on social cognition in GTS.

Although the exact pathophysiology of GTS is still largely unknown, there is evidence from neurochemical and neuroimaging investigations suggesting a primary role for dysfunctional dopaminergic pathways within the cortico–striato–thalamo–cortical pathways. Genetic vulnerability initially shown by early family studies has long been investigated. Studies involving segregation analysis on large kindreds with multiple generations affected initially suggested an autosomal dominant transmission model, however subsequent investigations revealed that the hereditary pattern is more complex and GTS is a genetically heterogeneous disorder. The results of both epidemiological and laboratory studies have also suggested a role for environmental factors, ranging post-infections autoimmune processes to pre- and peri-natal problems. The hypothesis that GTS can belong to a group of conditions called Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections (PANDAS) does not seem to
apply to all cases and requires further investigations. Clinical studies conducted over the last decade using principal component factor analysis and hierarchical cluster analysis have suggested the existence of multiple phenotypes within the GTS spectrum, which is in line with the concepts of genetic and aetiological heterogeneity.

The neuropsychiatry of Gilles de la Tourette syndrome

GTS is a quintessentially neuropsychiatric disorder, as the vast majority of patients present with specific behavioural symptoms in association with their tics. Importantly, this co-morbidity complicates the clinical picture considerably. Converging evidence from large clinical studies conducted both in the community and in specialist clinics indicates that only about 10% of patients have no associated psychiatric co-morbidity (‘pure GTS’). Consequently, the behavioural spectrum of GTS is multifaceted and the management of patients with psychiatric co-morbidities (‘GTS-plus’) can pose considerable challenges even to experienced clinicians (See table 1).

The most common psychiatric co-morbidities are obsessive–compulsive disorder (OCD) and attention-deficit and hyperactivity disorder (ADHD), with an estimated prevalence of about 60%. Interestingly, the obsessive–compulsive symptoms associated with tics overlap only partially with the clinical presentation of patients with primary OCD: for instance, concerns for symmetry, evening-up behaviours, arithmomania (obsessional counting) and ‘just-right’ perceptions seem to be more common in patients with GTS, whilst patients with pure OCD report a significantly higher prevalence of compulsive washing and other cleaning rituals, often associated with fears of contamination. It appears that only certain types of obsessive–compulsive symptoms are intrinsic to the pathophysiology of GTS.

The prevalence of co-morbid ADHD is particularly high in children and adolescents with GTS, resulting in challenging clinical pictures from both the diagnostic and therapeutic points of view. The hyperactivity and restlessness caused by the tics and the constant efforts to actively suppress tics often interfere with ability to sustain concentration which is required in school settings. The diagnosis of co-morbid ADHD should be established by experienced child and adolescent specialists on the basis of a comprehensive clinical assessment that takes into account the symptomatology overlaps between GTS and ADHD. The decision whether to prioritise treatment of ADHD or tic symptoms is crucial for the optimal management of young patients and is complicated by the observations that central nervous system stimulants used to treat ADHD have the potential to increase tic severity. The consensus view reached by experts in recent years is that it is appropriate to provide treatment with psychostimulants to patients with tics where the ADHD symptoms cause a significant impairment to their health-related quality of life, provided that particular attention is paid to dosages and titration protocols.

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Definition</th>
<th>Reference</th>
<th>Relative frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure GTS</td>
<td>Multiple tics (simple)</td>
<td>DSM/ICD criteria</td>
<td>Rare</td>
</tr>
<tr>
<td>Full-blown GTS</td>
<td>Multiple tics (simple + complex: e.g. coprolalia, echolalia)</td>
<td>George Gilles de la Tourette’s original description</td>
<td>Rare</td>
</tr>
<tr>
<td>GTS plus</td>
<td>Multiple tics + psychiatric co-morbidities (e.g. OCD, ADHD)</td>
<td>Clinical practice</td>
<td>Common</td>
</tr>
</tbody>
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Abbreviations: GTS, Gilles de la Tourette Syndrome; OCD, Obsessive–Compulsive Disorder; ADHD, Attention–Deficit and Hyperactivity Disorder; DSM, Diagnostic and Statistical Manual for Mental Disorders; ICD, International Classification of Diseases.
A consistent finding is that GTS is not associated with learning disability and patients with tics can be talented people who excel in different areas of life despite their condition.

GTS has also been associated with the development of affective disorders through different mechanisms. It is acknowledged that depression can be an understandable psychological reaction to living with a potentially disabling condition and its significant social stigma. Moreover, it has been suggested that the multiple neurotransmitter abnormalities in the cortico–striatal pathways responsible for the tics may also account for the impairment in affective tone. Finally, the aetiology of affective disorders in GTS can be iatrogenic, as certain medications commonly used for tic management (especially antidopaminergic agents) are known to cause depression as a side effect in a significant proportion of patients. Importantly, both tics and GTS are reported with increased frequency in patients with autistic spectrum disorders and it is common for patients with GTS to report temper tantrums and impulse control disorders with a higher prevalence than the general population, with relevant medico–legal implications. A relatively under–investigated area in the behavioural spectrum of GTS is the association with personality disorders, which have been shown to be over–represented in GTS populations, especially in patients with other psychiatric co–morbidities.

The cognitive performances of patients with GTS has been investigated in several neuropsychological studies. A consistent finding is that GTS is not associated with learning disability and patients with tics can be talented people who excel in different areas of life despite their condition. The results of neuropsychological studies assessing executive functions have been controversial and over the last few years research has focused on more subtle, yet clinically relevant, alteration in the domain of social cognition, which could be related to underlying fronto–striatal dysfunction. Both behavioural and cognitive problems have been shown to have the potential to affect health–related quality of life of patients with GTS, sometimes to a greater extent than tic severity. This is reflected in the multidimensional structure of the Gilles de la Tourette Syndrome–Quality of Life scale, the only disease–specific quality of life instrument for GTS to date, which covers four domains: physical, psychological, obsessional and cognitive.

The management of Gilles de la Tourette syndrome

GTS is a chronic condition with far–reaching implications. Patients with GTS present with a wide range of tic severity, from mild tics that do not cause significant impairment and often go unnoticed, to forceful movements and loud noises that can sometimes result in self injury. The presence of co–morbid psychiatric disorders adds a considerable burden in terms of distress and impairment. Due to the potentially disabling nature of the multiple symptoms, patients can face significant problems with their daily life activities. The European Society for the Study of Tourette Syndrome (ESSTS) recently published the first European set of guidelines for the assessment and management of GTS, which encompass behavioural interventions, pharmacotherapy and more invasive options for severe and refractory cases.

A number of behavioural techniques have been developed or adapted to enhance tic control in patients with GTS, however the number of large therapeutic trials in this field is still limited and it is difficult to draw firm conclusions about their efficacy. Habit Reversal Therapy or Exposure and Response Prevention stand out as the most promising behavioural approaches for tic management. Both techniques aim to enhance the patient’s ability to recognise the premonitory urges and to delay and eventually abolish tic expression. Two recent randomised controlled trials in paediatric and adult GTS populations have shown that a comprehensive behavioral intervention for
tics incorporating the habit reversal training method led to significant reduction in tic severity in about half of the patients.

The European Society for the Study of Tourette Syndrome (ESSTS) has recently published guidelines based on expert consensus about the indications to pharmacological intervention in GTS. Pharmacotherapy should be considered in addition to psychoeducation and as an alternative/add-on to behavioural therapy for patients with clear impairment associated with their tics. Impairment has been defined as follows: tics causing subjective discomfort, sustained social problems, emotional problems, or functional interference. Unfortunately the number of double-blind randomised controlled trials conducted to test the efficacy of pharmacological agents for tic management is limited, especially with regard to newer medications. The first pharmacological agents to show effectiveness in tic control were neuroleptics, especially haloperidol, pimozide and fluphenazine, which are still considered among the most effective anti-tic treatments.

However, their poor tolerability profile, mainly due to extrapyramidal and metabolic side effects, restricts their use to selected patients, usually as second- or third-line options. These medications have been largely replaced by the atypical antipsychotics, which are overall better tolerated and have demonstrated similar efficacy for tic control. Within this group, there are positive data from randomised controlled studies for risperidone and promising findings from open-label studies for aripiprazole, which seems to be associated with the best tolerability profile thanks to its partial dopamine agonist action. Substituted benzamides such as sulpiride and presynaptic dopamine depletors such as tetrabenazine can offer valuable alternatives, although both these classes and the newer antipsychotics can still cause relevant metabolic side effects, including hyperprolactinaemia and weight gain. Importantly, antidopaminergic agents can also be useful as augmentation therapy in patients treated with serotonergic agents for severe co-morbid OCD. Alpha-2 agonists (clonidine and guanfacine) can be considered as first-line pharmacological options for young patients, as these medications have a better tolerability profile compared to other classes and their anti-noradrenergic action can improve control over co-morbid ADHD symptoms. Overall, these medications have the advantage of being more manageable, however the evidence for their efficacy against tics is less robust and mild side-effects related to their hypotensive action should be monitored. Other pharmacological options which have shown promising results in terms of tic management include GABA-ergic agents, such as the antiepileptic drug topiramate, and cannabinoi ds.

Finally, a few selected patients with severe tics who failed to respond to conventional treatment interventions might be considered for more invasive approaches, including functional neurosurgery. The first patient with GTS who successfully underwent the procedure of deep brain simulation was reported in the Lancet in 1999. Since then, over 100 cases have been published in the scientific literature, with heterogeneous interventional paradigms and overall mixed results. Open questions in this field are the criteria for determining the suitability of a candidate and the optimal brain target for the deep brain stimulation, since pallidal and thalamic stimulation have often yielded similar results. It is hoped that future research sheds more light on the brain mechanisms of this fascinating disorders, thereby informing treatment strategies and unravelling the multiple links between motion and e-motion which are encapsulated in GTS.
References


Parkinson’s disease: a quintessential neuropsychiatric disorder

How we think about Parkinson’s disease (PD) has shifted dramatically in the last decade from that of a disorder of movement to that of a neuropsychiatric disorder. PD is classically thought of as a disorder of movement expressed by tremor, rigidity, bradykinesia and postural abnormalities. We now understand PD to be intrinsically associated with a myriad of autonomic and neuropsychiatric symptoms including but not limited to, mood, anxiety, psychosis, impulsivity, cognitive deficits and dementia. The behaviours have significant clinical impact on quality of life, functional impairment and caregiver burden: comorbid depression in PD is a more important predictor of quality of life than the movement symptoms; PD psychosis and dementia are important predictors of nursing home placement. Depression and anxiety can predate the onset of motor symptoms thus possibly acting as biomarkers of disease onset.

To optimize the management of the neuropsychiatric aspects of PD requires us to understand and consider the complex mechanistic interplay of the pathophysiology of conventional psychiatric disorders (e.g. family history, biological susceptibility and psychological factors), the neurobiology of PD and the How we think about Parkinson’s disease (PD) has shifted dramatically in the last decade from that of a disorder of movement to that of a neuropsychiatric disorder. PD is classically thought of as a disorder of movement expressed by tremor, rigidity, bradykinesia and postural abnormalities. We now understand PD to be intrinsically associated with a myriad of autonomic and neuropsychiatric symptoms including but not limited to, mood, anxiety, psychosis, impulsivity, cognitive deficits and dementia. The behaviours have significant clinical impact on quality of life, functional impairment and caregiver burden: comorbid depression in PD is a more important predictor of quality of life than the movement symptoms; PD psychosis and dementia are important predictors of nursing home placement. Depression and anxiety can predate the onset of motor symptoms thus possibly acting as biomarkers of disease onset.
important predictor of quality of life than the movement symptoms; PD psychosis and dementia are important predictors of nursing home placement. Depression and anxiety can predate the onset of motor symptoms thus possibly acting as biomarkers of disease onset.

To optimize the management of the neuropsychiatric aspects of PD requires us to understand and consider the complex mechanistic interplay of the pathophysiology of conventional psychiatric disorders (e.g. family history, biological susceptibility and psychological factors), the neurobiology of PD and the influences of dopaminergic medications and surgical interventions. Here I consider a general overview of the relationship between neuropsychiatric symptoms in PD and their underlying mechanisms and provide a few specific examples.

What are these behaviours?
Depression occurs in 30 to 40% of patients with PD. It can occur several years before the onset of the motor symptoms and is one of the most important predictors of quality of life in PD. Apathy, which can be distinguished from depression, occurs in 17 to 46% of patients and is defined as decreased motivation with decreased initiative, interest, and emotion. Anxiety disorders affect up to 60%, and can precede the onset of motor symptoms by up to 20 years. Impulse control behaviors, which include pathological gambling, compulsive shopping, hypersexuality, binge eating, punding and compulsive medication use related to dopaminergic medications can occur in up to 13.6%. Psychosis, which predominantly presents as visual hallucinations but can include other sensory modalities, minor phenomena and delusional states, commonly occurs in 30%. Cognitive deficits, particularly executive deficits are common in PD. Dementia occurs in 20% and in late onset PD, 80% develop dementia over 8 years followup.

Apathy, which can be distinguished from depression, occurs in 17 to 46% of patients

The role of individual susceptibility
Both the individual biological and psychological predisposition may play a role in underlying pathophysiology. Understanding their role may help guide treatment beyond evidence based approaches. For instance, depression in PD can present with different phenomenology from the general population. A patient presenting with excessive anhedonia (or lack of pleasure) and apathy might suggest a role for the neurobiology PD as compared to one with significant guilt and a family history of depression which might suggest a role for an individual susceptibility. Therapeutic interventions may in part depend on the influence of individual biological susceptibility (e.g. an extensive family history), psychological coping with PD (e.g. coping with early retirement, changes in identity and losses), or the influence of the pathology of PD as discussed below. Impulse control behaviours in PD similarly are likely related to an individual predisposition (e.g. a family history or personal history) interacting with dopaminergic medications, possibly influenced by the pathology of PD.

The role of neurobiology
The neuropathology of PD is characterized by Lewy Body deposition and neurodegeneration affecting the midbrain dopaminergic system. The neurodegenerative process of PD is now understood to prominently affect serotonergic, noradrenergic and cholinergic cell bodies all of which have influences on neuropsychiatric behaviours.

The motor symptoms of PD are not expressed until up to 80% of dopaminergic cell bodies have degenerated. The degeneration first affects lateral dopaminergic cell bodies projecting to the dorsal striatum (e.g. putaminal motor and caudate cognitive) and over time and more variably affects medial cell bodies affecting projections to the ventral striatum
behaviours. Dopamine agonists can also be effective for the management of depression and apathy in PD. Other interventions such as deep brain stimulation (DBS) targeting the subthalamic nucleus (STN), an effective intervention for control of motor symptoms, has its own set of influences. The STN is a small nucleus within the indirect pathway of the frontostriatal circuitry and is believed to play a crucial role particularly in inhibitory processes of the parallel motor, cognitive and limbic frontostriatal circuitry. For instance stimulation of the STN interacts with the dose of dopaminergic medications and can be associated with post–operative new onset mania, which responds to either a decrease in stimulation parameters or dopaminergic dose. STN DBS is also associated with various forms of cognitive impulsivity. That STN DBS allows a marked decrease in dopaminergic medication dose and discontinuation of dopamine agonists also underlies the increasing tendency to consider STN DBS as a possible therapeutic intervention for treatment refractory medication–related impulse control behaviours in PD.

The role of therapeutic interventions

The mainstay of therapy are dopaminergic medications which includes Levodopa, a precursor of dopamine that is converted to dopamine, and dopamine agonists, which directly stimulate dopamine receptors. Dopaminergic medications are implicated in psychotic phenomena and dopamine agonists and to a lesser extent Levodopa, implicated in impulse control behaviours. Dopamine agonists can also be effective for the management of depression and apathy in PD. Other interventions such as deep brain stimulation (DBS) targeting the subthalamic nucleus (STN), an effective intervention for control of motor symptoms, has its own set of influences. The STN is a small nucleus within the indirect pathway of the frontostriatal circuitry and is believed to play a crucial role particularly in inhibitory processes of the parallel motor, cognitive and limbic frontostriatal circuitry. For instance stimulation of the STN interacts with the dose of dopaminergic medications and can be associated with post–operative new onset mania, which responds to either a decrease in stimulation parameters or dopaminergic dose. STN DBS is also associated with various forms of cognitive impulsivity. That STN DBS allows a marked decrease in dopaminergic medication dose and discontinuation of dopamine agonists also underlies the increasing tendency to consider STN DBS as a possible therapeutic intervention for treatment refractory medication–related impulse control behaviours in PD.

Summary

PD is a quintessential neuropsychiatric disorder representing the overlap between neurology and psychiatry. Understanding the role of individual susceptibility, the influence of PD and its therapeutic interventions on neuropsychiatric symptoms can help guide management.

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A Perspective on Impulse Control Behaviours in Parkinson’s disease

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Introduction
Over the last 10 years, there has been increasing recognition of a range of Impulse control disorders (ICDs) in Parkinson’s disease, linked by their repetitive reward based behaviours. Their core feature is the failure to resist an impulse, drive, or temptation to perform an act harmful to either self or others. The term has been adopted for use in Parkinson’s disease (PD) for a range of conditions that include pathological gambling; compulsive shopping, compulsive eating, sexual behaviour; complex (hobbyism) and simple (punding) repetitive behaviours, and dopamine medication overuse also known as ‘dopamine dysregulation syndrome’ (Giovannoni et al., 2000; Lim et al. 2008).

Because of difficulties in the application of standard and consistent criteria across the range of problems, the term Impulse Control Behaviour (ICB) is preferred to describe this set of problematic behaviours. ICBs are thought to be drug–related effects of dopamine replacement therapies and occur in 14% of PD patients (Weintraub et al, 2010). They are commonly associated with high levels of neuropsychiatric co-morbidity and carer burden or distress.

The implications of the work around PD–ICBs are wide reaching in that the association of such behaviours with dopaminergic medication acts as a demonstrable link to an underlying biological process to the behavioural addictions. Specifically pharmacological substances that directly affect reward systems can manifest with a similar, if not identical phenomenology to behavioural disorders that only indirectly affect these neurotransmitter systems. They may further serve as a link in the chain for our understanding of a common mechanism for the development and maintenance of these behavioural addictions in the general population and also serve to inform current understanding of substance addictions.

This article does not attempt to provide a full review of the ICBs seen in PD which can be found elsewhere (Callensen et al 2013; Lim et al, 2008), rather we seek a somewhat philosophical consideration of two questions based on the observation of their occurrence. Firstly, are we justified in use of the phenomenon of PD–ICBs for (intelligible) interpretation of ICDs in the non–PD population? Secondly, how far can we extend such interpretation to the spectrum of what is potentially harmful but socially acceptable behaviour in the general
population? In other words, what can the PD–ICBs teach us about the spectrum of human behaviour from the normal to the pathological?

**Parkinson’s disease ICBs compared to ICDs in the general population**

The first of our two questions, regarding how these behaviours parallel those found in the general population is the easier of the two to address, or rather it is the easiest to explain where the limitations in our knowledge lie. Unfortunately, the current level of knowledge of ICDs in the general population is relatively poor, especially in relation to more common conditions like depression and comments that aside from pathological gambling (now relabelled a behavioural addiction, under ‘substance related and addictive disorders’) there is insufficient peer reviewed evidence to establish the diagnostic criteria and course descriptions needed to identify other behaviours as mental disorders.

Despite such a poor starting base, not all hope is lost. Much work has already gone into exploration of how cognitive, behavioural neurobiological and genetic factors are shared in ICB populations (PD and non PD) in comparison to healthy respective controls, and indeed where these conditions share similarities with substance use disorders. From the PD point of view, we know a personal or family history of alcoholism is associated with the conditions (Voon, 2006), as is a history of obesity (Nirenberg and Waters, 2006). Single young(ER) males, are overrepresented in both PD and non PD ICD sufferers (Weintraub, 2010), and there is preliminary data to show that PD–PGers appear just as responsive to talking therapies as their non PD counterparts (Jiménez-Murcia et al., 2012; Okai et al, 2013).

Nonetheless, other factors appear to be less convincing by way of shared investigation. Whilst there is reasonably robust data for positron emission tomography demonstrating reduction in striatal dopamine binding (Cilia et al, 2010; Steeves et al, 2009; van Eimeren et al, 2010) – similarly the case for substance misuse sufferers (Volkow et al., 2007; Fehr et al., 2008), the recent studies in non PD gamblers have all failed to detect such a difference when compared to healthy controls (Clark et al., 2012; Boileau et al, 2013). One study to use a more specific measure of dopamine binding did observe a positive correlation between gambling severity scores and binding in the substantia nigra (Tziortzi et al., 2011).

Clearly this is still a relatively young field of research by any measure, and there is unquestionably a large number of steps to go. There is however no doubt that the occurrence of ICBs in PD has allowed for the acquisition of improved, more ‘malleable’ information about this spectrum of behaviours than observation of the same behaviours in the general population. The known aetiology and pathology related to their occurrence in PD gives rise to understanding of dopamine mediated anxiety. This stems from their historical context where formal instruments for diagnostic assessment were not developed and validated concurrently with those for substance use, depression, anxiety and other psychiatric disorders. Consequently, ICDs were omitted from major epidemiological studies and much basic information, available for other psychiatric conditions is therefore unavailable. In the new era of DSM–III diagnosis led psychiatry, ICDs initially went relatively unrecognised and untreated but of recent have gained greater recognition. This is partly as a result of increased availability to many of these behaviours, for instance the prevalence of gambling behaviour sufficient to meet the threshold for ‘disorder’ has increased with increasing availability of legalised forms of gambling.

Additionally, the media’s intrigue with what they view as bad behaviour, particularly of celebrities has raised a number of the conditions in the public eye. Concomitant moral issues subsequently become of relevance as figures such a Tiger Woods take to the public stage with a rather dubious self-proessed ‘sexual addiction’. Changes in technology have increased access to such behaviours, and internet addiction itself has now been proposed as an ICD along with others such as exercise addiction. As it currently stands DSM–5 specifically

**Concomitant moral issues subsequently become of relevance as figures such a Tiger Woods take to the public stage with a rather dubious self-proessed ‘sexual addiction’**
interaction of pathology and treatment appear central to
the genesis of PD-ICBs, psychological and social factors
predominate in the clinical formulation of PD-ICBs and
have been shown to explain up to 62% of the variance
in their occurrence (Voon et al., 2007). Thus, whilst
variation exists from country to country in the various
prevalence’s of each PD-ICB disorder (Weintaub et al.,
2010; Lim et al., 2011), on the whole, the behaviours seem
to cut across differences in background, personality and
social environment.

On a more global level, the compulsive behaviours
demonstrated in the case of Parkinson’s disease,
potentially have wider reaching implications. Their
aetiological specific range of clinical presentations, with
changes in personality, affective disturbance, neurotic
symptoms and variation in the manifestation of reward
based activity, call into question (and indeed may be
a leading piece of evidence) of a flaw in our disease
classification system. It argues for greater emphasis of
the biomedical model as central to the manifestation
of this (neuro)psychiatric disease process; alongside
perhaps greater inclusion of social conditions and life-
course events. This is especially pertinent in an era
where there is an increasing trend to describe various
types of behaviour as disorder. Such understanding is
especially important to counter an inflationary use of the
concept of behavioural addictions where the behaviours
themselves are dimensional in nature, and variably
associated with distress.

A controversial implication of the ICB phenomenon
would be that research in the area, particularly in relation
to assessment and rating of severity models may benefit
from steering away from over commitment or allegiance to
a specific method of diagnostic criterion, be that DSM
or ICD-10 in keeping with an argument supporting a
conceptual range of ‘disinhibitory psychopathologies’,
which may ultimately prove more valid by way of a
 genetic, neurological and biological influences. In other
words, the greater understanding of nosology may
allow for a shift away from the Kraepelian reliance on
syndromes, to a different means of describing and
organising certain disorders more pertinent to a specific
underlying cause.

Of course it is important to keep these behaviours in
context. Firstly the range of behaviours is not restricted
to PD as an organic condition. Two weeks ago I saw a
lady in my clinical practice with symptoms consistent

The final issue around ICDs in the general population and
in PD is one of comorbidity. High rates of depression
and anxiety are known and associated with ICDs in
the general population and in PD prevalence rates of
depression and anxiety are as high as 50% and 46%
respectively (Okai et al., 2012). This means that a patient
may simultaneously satisfy several diagnoses. The
consistent nature of the overlapping diagnoses found
within the ICB population, along with the suggestion that
some of these diagnoses may fuel such behaviour (we
have suggested dysphoria fuels ongoing engagement in
these behaviours; Okai 2011), makes it at times difficult to
identify the primary illness.

Spectrum of similar harmful behaviours –
excessive through to disorder
The second of our two questions is more difficult a
concept to explore. Of particular interest is that whilst
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types of behaviour as disorder. Such understanding is
especially important to counter an inflationary use of the
concept of behavioural addictions where the behaviours
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Spectrum of similar harmful behaviours –
excessive through to disorder
The second of our two questions is more difficult a
concept to explore. Of particular interest is that whilst
interaction of pathology and treatment appear central to
the genesis of PD-ICBs, psychological and social factors
predominate in the clinical formulation of PD-ICBs and
have been shown to explain up to 62% of the variance
in their occurrence (Voon et al., 2007). Thus, whilst
variation exists from country to country in the various
prevalence’s of each PD-ICB disorder (Weintaub et al.,
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Conclusion

We caution the reader against an interpretation of our arguments, as a reductionist approach to the ICBs and ICDs, that one day the behaviours will simply be understandable purely by way of biochemistry, neuroscience and genetics. Indeed, the only evidence based treatment approach thus far is a CBT intervention that incorporates biological, psychological and social elements to management the full range of disorders despite varying levels of heterogeneity within treating groups (Okai, 2013). Similarly CBT interventions within the general populations for ICDs (gambling in particular) demonstrate larger effect sizes than any single medication (Grant et al., 2008).

Whilst the mechanism of treatment currently remains unclear, it may be of relevance that the hallmark of the most effective evidence based treatment for ICDs in the general population, cognitive behavioural therapy is not to focus too narrowly on overt (e.g. gambling) behaviours or cognitions but to attempt to address underlying factors, such as coping strategies that have now become maladaptive or dysphoria. For instance if a PD sufferer is gambling to escape loneliness, then any treatment strategy that focuses entirely on their gambling behaviour is unlikely to be successful in the longer term. Indeed even if successfully halted, the sufferer may subsequently simply turn to another form of avoidance, such as excessive eating or shopping in order to cope with her ongoing loneliness.

In conclusion, have I answered any of the questions I set out at the beginning of the article? No. However, PD-ICBs have already started a trend of reciprocal understanding between themselves, behavioural and substance addictions. If the current leaps in gambling knowledge that the occurrence in PD-ICBs have brought about are any indication for the future, a note of optimism is surely worth a bet ;-)
Acknowledgements

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References


The Special One

What is so special about functional (psychogenic) movement disorders? They are certainly common, making up about 50% of those with functional neurological symptoms. They are disabling, producing a reduction in quality of life similar to that seen in patients with Parkinson’s disease. They even performed a disappearing act, being said (at least in some manifestations) not to exist anymore during the seismic reclassification of movement disorders in the 1970’s and 80’s led by the late great David Marsden.

However, the special feature that rises above all these for me is the manner of their diagnosis. It is this that opens the door to insights regarding mechanism, challenges orthodoxy regarding aetiology, provides new avenues for treatment. Importantly, functional symptoms very commonly co-occur: a single patient with a functional movement disorder may also have functional sensory loss, fatigue, pain and attacks of apparent unconsciousness. This common co-occurrence of functional symptoms at the same time or the evolution of symptoms from one to another over time suggests a common underlying pathophysiology. Lessons learned regarding pathophysiology of functional movement disorders and discoveries regarding treatment are therefore likely to be transferrable to those with other functional symptoms.

From Clinical Signs to Pathophysiology

One of the major shifts in neurological practice with regard to functional movement disorders has been towards positive diagnostic criteria based on clinical features of the movement disorders.
directed attention is an anathema to normal movement; I hope I am not alone in recalling walking on stage in a school play and having my normal gait turn inexplicably into a robotic parody of walking.

Regarding belief, one example is the typical gait pattern of patients whose main complaint is of poor balance. Such patients may walk with a narrow base lurching dramatically from side to side without falling—usually called walking on ice gait. This pattern of movement, shifting the centre of gravity of the body from side to side without falling, represents a major challenge for the balance system, and its successful performance positively demonstrates good balance despite the subjective complaint. However, I think it is the case that if you were to ask someone without medical training to pretend to have poor balance, most would stand on a narrow base, rocking from side to side while flailing their arms around in a parody of a tightrope walker or indeed someone walking on ice.

Thus, this pattern of walking can be interpreted as a reasonable lay belief about how poor balance might be. Extending this idea more broadly, the suggestion is that functional symptoms are moulded by high level beliefs/expectations about physical symptoms and the way the body might fail due to illness. Such beliefs and expectations can certainly be moulded by personal and societal illness beliefs, something seen clearly in the interaction between cultural expectations regarding whiplash injury and the actual prevalence of whiplash injury after minor road traffic accidents.

There is one more component that needs consideration, and this is perhaps the most difficult of all. This component is agency, in other words the correct classification of movements (and sensations) as self-generated or externally generated. This is perhaps partly what Wittgenstein was getting at in a famous question: “What disorder itself, and away from diagnosis based on exclusion of organic disorders, or on the presence of psychological factors thought to be of aetiological importance (e.g. traumatic life events).

Though the precise phenomenology of functional movement disorders is diverse, the two clear clinical features are:

1. **The movements appear voluntary because they require attention to manifest and improve with distraction.**

2. **The movements fit with reasonable lay beliefs about how a movement disorder might manifest, but they break fundamental rules that we know to be true from basic anatomy and physiology.**

For example, patients with functional tremor show clear dual task effects. When asked to tap at a different frequency to the tremor with their unaffected/less affected limb, the tremor may entrain to the tapping frequency, the tremor may pause or stop, or the patient may inexplicably be unable to perform the simple tapping task.

These and other simple clinical tests positively separate patients with functional tremor from those with tremor due to neurological disease with high sensitivity and specificity. This is clearly useful diagnostically, but it is also telling us something about the importance of abnormal self-directed attention in the pathophysiology of functional movement disorders. Patients with unilateral functional weakness may have zero power of hip extension, but this same muscle activates normally when the movement is triggered by flexion of the other leg (Hoover’s sign).

One interpretation of this phenomenon is that movement is impaired when it is being accessed in the presence of self-directed attention, but movement normalises when the movement occurs automatically without this attentional focus. Self-directed attention is an anathema to normal movement; I hope I am not alone in recalling walking on stage in a school play and having my normal gait turn inexplicably into a robotic parody of walking.
remains if I subtract the fact my arm went up from the fact that I raised my arm?” One answer to this question is “Will”, but if we want to avoid the philosophical issues, another answer would be “agency”.

The reason for considering this issue here is that clinically, the only difference between a faked movement disorder and a functional movement disorder is the self-report of agency: the patient says the movement is not under their control. This is one of the most problematic issues in attitudes to patients with functional neurological symptoms, and perhaps accounts for a lot of the ambivalence of neurologists and psychiatrists towards them. However, even this is not an insoluble issue, because if our patients are telling the truth, then there must be a deficit in the system that confers agency to account for their apparent misperception of movements that appear voluntarily generated as involuntary. Though difficult, this is an area that is open to scientific study.

Not so “unexplained” after all...
This discussion of clinical signs/phenomena leads us into quite familiar territory from a neuroscientific perspective. The concepts of attention, beliefs/expectations (in the sense of predictive internal models of the world) and sense of agency are all topics of historic and current neuroscientific study. We can therefore use this knowledge to design experimental paradigms to compare patients with functional movement disorders and controls (both healthy and with typical organic movement disorders).

There is now a small but growing research literature which points to abnormalities in attention, beliefs/expectations and agency in patients with functional symptoms (for a review see 3), and is beginning to provide an evidence base for the underlying mechanism of symptoms.

How could we try an implement this mechanism within the brain? One proposal draws on the concept of a hierarchical structure of the brain where data at one level interacts with predictions about that data at the level above, which build through levels of increasing complexity to perception and movement. The idea here is that the brain is an “inference machine”, sensing information from the environment and building this into a model of the world and how our bodies interact with the world. By acting on the world through movement we can change our environment, detect this change via our sensory system and then compare this with the sensory predictions we made prior to moving. By repetitive experience over time our internal model of the world becomes a more and more precise representation of reality 4.

The crucial point with regard to applying this model to functional movement disorders (and other functional symptoms), is that the probability or “precision” attached to sensory data and predictions regarding sensory data is not fixed. We can think of perception and movement as having a Bayesian quality, in other words resulting from an interaction between probability weighted “bottom up” sensory data and “top down” sensory predictions 4. There is always uncertainty in both the sensory data we are sampling with our sensory system and how confident we are in our predictions regarding that sensory data. The level of this uncertainty differs in different contexts. Imagine navigating around a room in the dark. If it is our own bedroom, then we might stride out across the floor, confident in our internal predictions regarding a safe route. However, if it is an unfamiliar room we would be likely to advance cautiously, feeling our way as we go, trusting the evidence of our incoming sensory data rather than any internal predictive model. It is proposed that attention is an important force in increasing or decreasing precision/weighting of sensory data or predictions regarding that data.

If we apply this model to functional disorders, one hypothesis is that an abnormally strong prediction regarding movement or sensation arises at a high level in the hierarchy 5. There could be a variety of factors relevant to formation of this prediction or “prior”, including physical precipitants that provide novel sensory data about the self, panic responses at the time of symptom onset, affective disorders, personal and cultural illness beliefs, and particular decision making styles. In the presence of abnormal self-directed attention, this prediction can be activated and can overwhelm any sensory data to the contrary, therefore producing abnormal movement or sensation in keeping with the prediction. However, when attention is distracted the prediction loses this activation and normal movement and sensation can occur. With regard to agency, all the patient did was to turn attention onto
Both of these statements are true, but are only true in part. They are laughable because we readily accept the complexity of disorders such as stroke and Parkinson’s disease and the diversity of risk factors and underlying aetiology. This results in a broader and more cautious approach to these disorders in terms of research into their pathophysiology but also in diagnostic explanation and treatment selection.

I have recently become more aware of the reasons why many neurologists and psychiatrists might find this approach to be challenging. We know from fantastic epidemiological work amongst Scottish neurologists that patients with functional symptoms are hugely common in neurology practice, in fact the second commonest disorder neurologists see in outpatients, just behind headache6. Yet when asked about their experience with these patients I’ve often found that neurologists only recall a specific subset of these patients: those who create havoc in their interactions with health care, write long letters of complaint, wear huge dark glasses and clutch teddy bears when in hospital even though they are in their 30’s. This stereotypical patient is recalled with “cognitive ease”, but those of us who spend every week in clinics specifically for patients with functional symptoms rarely see these patients.

This is nonsense. Why should we be arrogant enough to assume that patients with functional symptoms are so much easier than patients with any other disorder, and that we can simply jump from symptom to one-dimensional aetiology without pausing for thought? If this is ok, why can’t I get a paper published that starts: “Parkinson’s disease is a disorder presumed due to getting older” or “Stroke is a disorder presumed due to smoking”?

From “how” to “why”
Some might think that this discussion above regarding mechanism is a bit of a distraction from the main issue: that of getting the patient to understand the psychological nature of their symptoms and to stop bothering their “organic” doctors for more tests. Some might even feel that the discussion of mechanism in the terms used above is directly counter-therapeutic. Does it just allow patients to avoid a difficult confrontation with the true nature of their illness and instead to retreat to the level of pseudo-physical mechanism in a similar way to that provided by terms such as fibromyalgia, myalgic encephalomyelitis, reflex sympathetic dystrophy and the like? Indeed, should neurologists even be doing around in this area at all? Shouldn’t they just do their job of excluding the neurological disease and then punt the patients over the philosophical fence from the side of the brain to the side of the mind?

This is nonsense. Why should we be arrogant enough to assume that patients with functional symptoms are so much easier than patients with any other disorder, and that we can simply jump from symptom to one-dimensional aetiology without pausing for thought? Why is acceptable for a paper published in a high ranking journal on this topic to open with: “Psychogenic movement disorders are movement disorders presumed due to psychological factors”? If this is ok, why can’t I get a paper published that starts: “Parkinson’s disease is a disorder presumed due to getting older” or “Stroke is a disorder presumed due to smoking”? 
stereotype, but instead are much more “ordinary” and fade from memory very quickly. This is especially true for patients with neurological disease who have additional functional symptoms. Having a neurological disease is a stronger risk factor for developing functional symptoms than almost anything else, and yet such patients often pass relatively un-remembered by their neurologists.

For their part psychiatrists are much more likely to be referred patients with functional symptoms in whom psychopathology is overt or where patients are in conflict with medical services (perhaps likely to be an enriched sample for personality disorder). Indeed many psychiatrists would be unwilling to label patients as “conversion disorder” without overt psychopathology: I am sure I am not alone in experiencing neurology/psychiatry ping pong with patients who have clear functional symptoms on the basis of their neurological examination but are told by their psychiatrist that they do not have that disorder as they are psychiatrically well. Many psychiatrists may therefore not see the bulk of patients with functional symptoms where the aetiological model of overt stressors through to physical symptoms is an uncertain fit for the story that patients give. The fundamental point here is that once one starts defining this disorder on the basis of positive clinical signs and not on the presence of psychopathology, an incredible diversity of patients is revealed and along with it the flimsy nature of our common explanatory models.

None of this negates the role of psychiatric and psychological expertise in the management of these patients. In fact it argues for greater development of this expertise and more joint working between neurologists, psychiatrists and psychologists. There is clearly a psychological dimension to functional symptoms and bringing patients to understand this in an open minded and genuine way is often an essential part of treatment. There are some patients where psychopathology is clearly dominant and needs addressing first and foremost. There are patients who absolutely refuse to accept the presence and relevance of their psychopathology even though it is clearly important in the aetiology of their physical symptoms, and such patients are very hard to manage.

Some patients have malingered or factitious symptoms and are also very hard to manage. However, in my opinion we need to embrace the complexity of our patients and not simply dismiss as abnormal illness behaviour a patient’s difficulty in accepting that their leg paralysis or shaking arm is due to “stress” when they neither feel stressed nor have had specific traumatic experiences. In such patients I have found that an explanation of symptoms based on mechanism (this is a functional tremor because it stops when you are distracted) followed by a broad discussion of potential risk factors including the role of physical precipitants (injury, illness) and those related to emotional and psychological health, is often successful and may in itself lead to clinical benefit. This explanation does not negate the value of exploration of psychological issues as part of treatment. It is not about allowing patients a convenient physical way out. It is simply being a bit humble in the face of a disorder which is complex and poorly understood.

This approach also allows us to explore alternative treatments and to understand why treatments that are in current use might work (and from that how they can be improved). For example, selected patients with motor symptoms get great benefit from specialist physiotherapy. Improvement with such physical treatment is sometimes dismissed as “face saving”. However most patients I see who gain benefit from specialist physiotherapy specifically designed for treatment of functional movement symptoms have already had physiotherapy elsewhere, often more than once and with good physiotherapists, and have not got much benefit — why haven’t they chosen to “save face” before? I think an alternative explanation for their improvement is that some patients can learn to control their abnormal movements if they are shown how they are generating them and how to stop them happening.

So — what is so special about functional movement disorders? To me they provide a clear example of how to build a true bio-psycho-social model of functional disorders, where all parts receive the focus they deserve. They reveal the glorious, scientifically interesting and exciting complexity of functional symptoms that in the end is likely to tell us a great deal about brain function in health and disease. They also represent a common cause of neurological symptoms which is often disabling and persistent, but where for a reasonable proportion of patients treatment can make
a real and lasting difference. What is lacking is a service structure that can deliver rapid positive diagnosis and coordinate patient access to specific treatments depending on their needs. Joint working and sharing of expertise between neurology and mental health services at a physician and therapist level is the only way to deliver this service. If this can be achieved, then not only would it benefit care of patients with functional symptoms, but it would be an exemplary model of care for patients with brain disorders in general. Now that would be something special indeed.

REFERENCES


Conference Report


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The 27th annual British NeuroPsychiatry Association (BNPA) AGM conference was held on 27 and 28th of February 2014 at the Institute of Child Health, London that was attended by delegates from all over UK, other parts of Europe, USA and even as far as Australia. Day one witnessed packed lecture theatre with renowned experienced speakers. The initial focus was on social cognition in autism, followed by neurodevelopment in schizophrenia. Later discussions on primary and secondary prevention measures in Alzheimer’s disease (AD) seemed to gain attention.

The first session began with theme ‘New development in Cognition’ chaired by Peter Halligan. During the first talk by Child Psychiatrist, Professor David Skuse, he emphasised the need for standardised objective measures of social development. He gave a summary of his decade long work on oxytocin hormone and receptors in social cognition. There are reports of increased retention of social cognition in autism after using intravenous or intranasal oxytocin. One of the delegates asked whether there was a role of oxytocin neuropeptide in autism treatment. Dr Roland Zahn (RZ) who is currently working on fMRI biomarker in recurrent depressive disorder, later took us through the neuropsychiatry of social knowledge and moral motivation. He discussed the fMRI findings in Fronto-

Temporal dementia patients and showed that it is possible to map brain areas related to emotions. Professor Trimble in the audience queried to RZ about why he was trying to link ‘morality or moral’ which is related to divine with the neuropsychiatry.

After a coffee break, the delegates had pleasure to listen to three speakers with a varying style and pace of presentations. Professor Sarah-Jayne Blackmore (SJB) presented experimental findings on increased risk taking behaviours in adolescence. It is known that accidents are the leading cause of death in adolescents. Also, 75% of the adult mental illness has its onset before 24 years of age. This was why SJB chose to work on neurodevelopment in adolescence. The important finding was that brain development correlated with level of pubertal hormones than the chronology of age. There is a contracted brain development during the adolescent period. Declining grey matter overall is thought to be due to synaptic pruning. Following on from this, Professor Eileen Joyce (EJ) suggested that cognitive impairment may be the fundamental part of schizophrenia syndrome. She showed data to suggest the process of cognitive decline begins prior to patients presenting with psychotic symptoms. Working memory and planning are generally the worst affected ones. EJ agreed with SJB on the synaptic pruning, but possibly
During the afternoon session, there were three research platform presentations. The topics included gene variant in AD, post-ictal psychosis and association of joint hyper-mobility with autonomic hyperactivity with a possible link to the developmental disorders. The late afternoon session chaired by Marcus Reuber on the theme ‘Neuropsychiatry research update’. David Okai’s talk was on ‘Impulse Control Behaviours’ in Parkinson’s disease (PD) where he discussed findings of their group’s unique CBT trial to treat those behaviours. Impulse Control Behaviours in PD is gaining more focus in the recent years. Notably the phenomenology of impulse control disorders have been recognised to be similar to addictive behaviours. There are reports of lawsuits involving pharmaceutical companies that led to increasing focus on these behaviours which initially were thought, simply an adverse effect of longer term use of dopamine agonist therapy.

Day one ended with presentation by Dr Hugh Rickards (HR) on ‘Do cholinesterase inhibitors work?’ who took us through systematic review evidence, suggested that they may benefit but only ‘little bit’. It was a challenge for HR to keep audience interested as it was the day’s last presentation, which he did well. He presented hypothesis which he justified. The talk made us realise that most of studies are done looking at response on the Neuropsychiatric Inventory scale, which was initially designed to evaluate spectrum and severity of complications of dementia. There is a need for well

Rightly, the discussions on latest developments in schizophrenia will probably continue to be a hot topic in the Neuropsychiatry Conferences.
designed validated instruments in terms of assessing and rating severity in dementia. This talk did generate dilemma whether to recommend cholinesterase inhibitors based on best available evidence. The need for earlier identification of dementia was also emphasised.

But the Psychiatrists in UK have not got much choice apart from cholinesterase inhibitors, which are only disease modifiers and probably memantine in terms of pharmacological treatment. Patients and families may have to wait till dementia has reached moderate degree to have it prescribed. Should these drugs be commenced early, is there going to be any more benefit? However, there was clear general sense of apprehension over growing concern of dementia as a massive public health problem in the future years to come.

On Day two, Professor Eileen Joyce, the chairperson, welcomed the delegates and the programme started by a talk on ‘Recognising and diagnosing inflammatory brain disease’ by Professor Neil Scolding. He informed that inflammatory brain diseases are uncommon, that they may be under–diagnosed or over–diagnosed. He later discussed their clinical features and management of CNS vasculitis. Learning about all the different types of vasculitis and that the treatment regimes have not changes, the delegates were wondering how to treat these conditions. The lecture ended with a positive note that the treatment options are improving.

Dr Jeremy Rees (JR) then spoke on Paraneoplastic neurological disorders. He discussed the definition, their milestones, antigens involved and highlighted importance of recognising paraneoplastic syndromes. It was interesting to note that contrary to belief they present to neurologists, JR explained that patients usually present to psychiatrists. JR emphasised the neurological symptoms may precede malignancy and these symptoms are treatable.

There was lecture on ‘Infectious encephalitis with psychiatric presentations’ by Professor Tom Solomon (TS). He presented a case series of six patients and discussed the symptoms which may have altered the psychiatrist to make a referral to a neurologist at an earlier stage of the disease process. The Institute of Infection and Global Health situated in Liverpool is involved in great work in the UK and around the world. There was a First ever BNPA medal lecture on ‘autoimmune encephalitis’ delivered by Angela Vincent. She spoke on antibody mediated diseases. This is new beginning in the BPNA Conference and hopefully it would encourage the delegates to reflect on their work. In the late afternoon session, the chair Peter White announced prize winners of platform and poster presentations and the administrative staff members were felicitated. The chair then introduced the Wellcome Trust Debate on “This house believes that neurology and psychiatry should be one medical discipline”. Professor Geriant Rees along with a Clinical fellow in Neurology spoke in favour of the motion and Professor Sir Simon Wessely and a Psychiatry trainee argued against the motion. Each speaker had 10 minutes which was followed by question and answers. The meeting concluded with closing remarks by the chair. Interestingly, during the question and answer session with the audience, there was varying opinions from the delegates. Generally Neurology and Psychiatry should be linked as one discipline or under a single Post-Graduate training Board similar to the system in the USA and also the Doctors training in Neurology should be offered some training in Psychiatry and vice-versa. Elderly Medicine Registrars having at least two week placements in Liaison Psychiatry of Old Age is not uncommon. One of the delegates who was in favour of these two disciplines being combined as one discipline gave an example of medical training in Germany where neurology and psychiatry are closely linked. Others wanted clear demarcation between these specialties as the argument was that a neurologist would not be able to treat schizophrenia and related mental health diagnoses in the current system. Finally when voting was conducted, more delegates voted that neurology and psychiatry should be one medical discipline.

The tea and the lunch breaks gave opportunity to view posters on both days and various neuropsychiatric textbooks. Annual General Meeting of BNPA was also held during the lunch break on the second day of the conference. Posters presentations were mainly on Tourette’s syndrome, Huntington’s disease, Parkinson’s disease, traumatic brain injury, mood disorder, ADHD and OCD. Overall the Conference that is attended by neurologists, psychiatrists provides platform where exchange of ideas takes place. Researchers are introduced to new ideas and create enthusiasm in clinicians working closely in the interface of Neurology and Psychiatry specialties.
When in 2013 US President Obama announced the decade of the brain there was already a great foundation on which to build. In April 2014 I had the privilege to explore these foundations when I attend the 16th Neuroscience Winter Conference in Austria. This is an established high quality meeting on contemporary neuroscience.
Year on year the organizers aim to top the previous year’s programs by inviting distinguished neuroscientists as keynote speakers and developing high quality symposia. The program looked promising and as a clinician I was keen to go back to “basic sciences” to help me understand the conundrums in my clinical practice as a child and adolescent psychiatrist.

The speakers hailed from the most renowned universities in the world. References to the abstracts included journals such as Nature, Science and the Lancet.

I work with PTSD and dissociation in minors and it troubles me how long it takes for the minors to experience recovery, if at all. Ideally I also would like to see child abuse be a thing of the past. Unfortunately, I have no answers to these questions so I suppose a reasonable area to start is with the sciences basic to mental health. Direct answers to my questions were not provided but nevertheless I found the conference stimulating and has encouraged me to research various topics and themes further.

The speakers hailed from the most renowned universities in the world. References to the abstracts included journals such as Nature, Science and the Lancet. At one point there was a call for Nobel Laureates to gather for a group photograph. Post Docs were acknowledged for their work. A link to the book of abstracts and the conference program can be found at www.winterneuroscience.org and clicking the final program tab [abstracts are listed alphabetically according to speaker’s last name – download is free of charge, as last accessed on 4 May 2014]. All keynote lectures and symposia were somehow relevant to psychiatry either because of direct clinical relevance or because of the importance of basic sciences findings and possible subsequent testing for future biological therapies.

I found especially intriguing findings presented by Jianfeng Feng on the Brain Wide Association Study (B–WAS). They have access to multicenter data from circa 0.5 Million patients per year. His group takes the most significant findings from studies in an area and synthesizes the results. They discovered the import of thalamo–sensory tract enhancement and thalamo–frontal tract decrease in schizophrenia.

Gunter Schumann shared his finding from monozygotic twins studies focusing on twin discordance, the role for non-heritable differences and the genome wide methylation analysis. He uses skills from epigenetics research where there are suggestions for a marker for alcohol use disorder in adults and a neurobehavioral risk factor for adolescents and impulsivity.

Stem cell growing from astrocytes and rhinencephalon for spinal cord injury found mention by Philip Beart, Eva Sykova and Sarka Kubinova. Robert Schwarcz showed evidence for the role of kynurenic acid in cognitive impairment.

Christine Konradi presented on the markedly vulnerable respiratory chain in hippocampal interneuron mitochondria in schizophrenia (also seen in glucose-deprived lymphocytes in bipolar disorder). Robert Nitsch presented on early neurodevelopment and “just right” membrane lipids in the postnatal period and how this can later influence cortical information processing, relevant to psychiatric disorders.

Importantly, negative findings were also mentioned: Wolfgang Fleischhacker admitted on the so far futile attempts to improve on negative symptoms by translating research from glutamatergic agents and the NMDA receptor.

There were also presentations and display posters on the relevance of melatonin levels as yielded from saliva and clock gene expression in buccal swabs in Smith Magenis syndrome and bipolar disorder. Latest developments in research regarding multiple sclerosis (implicating killer T cells), Huntington’s disease (axon transport) and Parkinson’s disease (explanations for cell death of dopaminergic cells) were presented.
As a practicing clinician I gained an update on basic sciences peripherally or directly relevant to psychiatry. Coming almost 3 decades after my medical studies one might expect that exposure to highly specialized research may leave a clinician at a loss. However, the well-rehearsed expert speakers, the wide-ranging symposia from a variety of sub-specialties, and the presence of fellow non-specialists (at least non-specialist in comparison to the speaker) in the audience, greatly aided my comprehension.

While cytoplasmic processes, endocytosis and kiss and run and vesicle fusion were a focus of the meeting, very topical “temperature” and “altitude” receptors were not discussed at the scientific meeting itself, though engaged post-conference by the delegates. The next Neuroscience Winter Conference adjourns in Sölden, Austria in April 2015. The venue “Central Hotel” with its excellent restaurant is located only 15 minutes by gondola from the ski runs. 2015 will surely be another wonderful opportunity for to reduce complex human experience to stimulation of taste buds and cerebellar dysregulation on the slopes.

Fascinating animal research from lower mammalians (such as mice and rats) was presented too: developments from neural circuits in basal ganglia, spinal tract and higher motor centers were discussed. Moreover research was presented on individual “memory” of single (here visual cortex) neurons, GABA and glutamate receptors, the lysophosphatidic acid synthesizing molecule autotaxin and the role of synapses in prion disease. Zebra fish, I now understand, are great for research on neurological substrates such as response to temperature, light, odor, learning, and brain lateralization. In rodent models neuropsychological substrates to decision making and intent were presented. Reference was made to the neuronal and genetic basis to brain plasticity and then linked especially to the glycoprotein encoding gene NPTN.

These findings may support the potential role of NPTN in regional synaptic dysfunction, which may clinically lead to specific intellectual impairment. Dynamics of cytoskeletal structures, Shank protein and postsynaptic density have been linked to human’s autism, intellectual impairment and also schizophrenia disorders.
New anti-psychotic drug reduction trial at Cardiff University

**ANTI-psychotic Drug REduction in primary care for Adults with Learning Disabilities (ANDREA-LD): A Randomised Double-blind Placebo Controlled Trial**

Prof Mike Kerr and team at Cardiff University are in the process of setting up a double blind, placebo controlled randomised controlled trial to address the question of whether anti-psychotic medication prescribed to adults with learning disabilities for the treatment of challenging behaviour can be withdrawn or reduced without behaviour or mental health deteriorating and treatment costs escalating.

**Why is the trial being run?**
Use of psychoactive medication in adults with LD is high and there is particular concern over the use of anti-psychotic medication that is prescribed for reasons other than the treatment of psychosis. Control of challenging behaviour is the primary reason why such medications are prescribed despite the absence of good evidence for any therapeutic effect for this purpose.

**What will happen?**
We will recruit GP’s to act as Principal Investigators and lead treatment in the trial. They will be supported by a specially designed, trial specific treatment and safety package. Patients at each practice will then be approached and recruited if eligible (adult LD patients without a current diagnosis of psychosis who are being prescribed either risperidone or haloperidol for the treatment of challenging behaviour).

During the trial, those in the intervention arm will proceed through 4 monthly approximately 25% reduction stages within a 6 month period (although blinded, the GP has discretion to delay progression to the next step). The control group will maintain baseline treatment. Treatment achieved at 6 months will be maintained for a further 3 months under blind conditions. At 9 months, the blinding will be broken for clinicians and participants and medication changes monitored over the 12 month period from baseline.

Once a patient confirms they are interested in taking part in the study, we will ask for details of their wider care team to inform them of this. GPs may be advised to refer to secondary services if they have any concerns over the patient.

**When is the trial starting?**
We already have our MHRA and REC approvals so are looking to recruit GP practices in South Wales and the Bristol area now. We will expand to other areas of England later in the trial.

**What can I do?**
This is going to be a large study to get up and running so we are keen to spread the word and keep everyone informed so that there is nothing coming out of the blue! Please don’t hesitate to contact us should you have any thoughts or comments about the study – we would love to hear from you. We very much want to work with psychiatric services to ensure the success of the project.

**Contact details**

**Chief Investigator**  
Prof Mike Kerr, Welsh Centre for Learning Disabilities, Cardiff University,  
KerrMP@cardiff.ac.uk  
02920 687100

**Trial Manager**  
Elizabeth Randell, South East Wales Trials Unit, Cardiff University  
Randelle@cardiff.ac.uk  
02920 687608
Royal College of Psychiatrists
Section of Neuropsychiatry Conference

Thursday 4 – Friday 5 September 2014
Lady Margaret Hall, Oxford
Preliminary Programme

<table>
<thead>
<tr>
<th>Thursday 4 September</th>
<th>09:15–09:50</th>
<th>REGISTRATION AND REFRESHMENTS</th>
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<tbody>
<tr>
<td>09:50–10:00</td>
<td>Welcome and Introduction</td>
<td>Dr Rafey Faruqui</td>
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**Autoimmunity & Neuropsychiatry Chair: Prof Mike Kerral**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>10:00–10:45</td>
<td>Autoimmunity &amp; Psychiatric disorders</td>
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<tr>
<td></td>
<td>Dr Andrea Cavanna, Honorary Reader in Neuropsychiatry, School of Clinical and Experimental Medicine, University of Birmingham</td>
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<tr>
<td>10:45–11:30</td>
<td>Autoimmune limbic encephalitis: recent advances</td>
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<td>Keynote Speaker: Professor Angela Vincent, Professor of Neuroimmunology, University of Oxford</td>
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<tr>
<td>11:30–12:15</td>
<td>Autoimmunity &amp; childhood movement disorders</td>
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<td>Dr Ming Lim, Consultant Paediatric Neurologist, Evelina Hospital London</td>
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<tr>
<th>12:15–13:30</th>
<th>LUNCH AND POSTER VIEWING</th>
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<tr>
<td>Chair: Dr Niruj Agrawal</td>
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<th>Time</th>
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<tr>
<td>13:30–14:30</td>
<td>Interactive case discussions–Autoimmunity in clinical practice</td>
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<td></td>
<td>Dr Jan Coebergh Consultant Neurologist, St George’s Hospital London</td>
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<tr>
<td>14:30–15:10</td>
<td>Trainee award presentations</td>
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<tr>
<td>15:10–15:30</td>
<td>AFTERNOON REFRESHMENTS AND POSTER VIEWING</td>
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**Neurostimulation in Neuropsychiatry Chair: Dr Rafey Faruqui**

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<tr>
<th>Time</th>
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<tr>
<td>15:30–16:00</td>
<td>DBS</td>
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<td></td>
<td>Professor Keith Matthews, Head Of Neuroscience, University of Dundee</td>
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<tr>
<td>16:00–16:30</td>
<td>VNS</td>
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<td></td>
<td>Dr Manny Bagary, Consultant Neuropsychiatrist, Birmingham and Solihull Mental Health NHS Foundation Trust</td>
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<tr>
<td>16:30–17:00</td>
<td>TMS</td>
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<td>Professor Mark Richardson, Director, Institute of Epileptology, Institute of Psychiatry, London</td>
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<tr>
<td>17:10 – 18:00</td>
<td>Section of Neuropsychiatry Business Meeting</td>
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**Friday 5 September**

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<tr>
<th>Time</th>
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<tr>
<td>09:00–09:25</td>
<td><strong>REGISTRATION AND REFRESHMENTS</strong></td>
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<tr>
<td>09:30–10:15</td>
<td><strong>Automatisms &amp; Criminal behaviour</strong> Chair: Professor Michael Koppleman</td>
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<tr>
<td>09:30–10:15</td>
<td><strong>Neuropsychiatric perspective</strong> Dr Jonathan Bird, Bristol</td>
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<td>10:15–11:00</td>
<td><strong>Neurological perspective</strong> Professor Matthew Walker, Department of Clinical and Experimental Epilepsy, UCL</td>
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<tr>
<td>11:00–11:20</td>
<td>Questions and Panel discussion</td>
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<td>11:20–11:40</td>
<td><strong>MORNING REFRESHMENTS</strong></td>
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<tr>
<td>10:45–11:30</td>
<td><strong>Criminality: Neuropsychiatric Perspectives</strong> Chair: Dr Howard Ring</td>
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<tr>
<td>10:45–11:30</td>
<td><strong>Acquired brain injury and crime</strong> Dr Rafey Fauruqi, St Andrew’s Healthcare</td>
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<tr>
<td>11:30–12:15</td>
<td><strong>Diagnosis of abnormal sleep behaviours</strong> Dr Sofia Eriksson, Consultant Neurologist and Honorary Senior Research Associate at National Hospital for Neurology and Neurosurgery and UCLH NHS Hospitals NHS Trust, and the UCL/Institute of Neurology</td>
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<tr>
<td>13:10–14:15</td>
<td><strong>LUNCH AND POSTER VIEWING</strong></td>
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<tr>
<td>14:15–14:45</td>
<td><strong>Criminality: Neuropsychological perspectives</strong> Chair: Dr Hetal Mehta</td>
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<tr>
<td>14:15–14:45</td>
<td><strong>Aggression and sexual behaviours in individuals with acquired brain injury</strong> Dr Caroline Knight, Consultant Clinical Neuropsychologist, St Andrew’s Healthcare</td>
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<tr>
<td>14:45–15:15</td>
<td><strong>Symptom validity – lessons from neuropsychology</strong> Dr Martin Van den Broek, Head of Neuropsychology &amp; Clinical Health Psychology, St George’s Hospital London</td>
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<td>15:15–15:45</td>
<td><strong>Engagement with aggressive individuals who have cognitive impairment</strong> Professor Carol Ireland, University of Central Lancashire</td>
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<tr>
<td>15:45–16:15</td>
<td>Questions and Panel discussion</td>
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<tr>
<td>16:15</td>
<td><strong>Closing comments</strong> Dr Rafey Faruqui</td>
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Call For Abstracts

PRIZES
The Section of Neuropsychiatry (SoN) has established an annual awards for trainees in order to promote the highest standards of critical thinking and communication by psychiatry trainees in the field of neuropsychiatry. The awards are given for the best original research, audit, literature review or clinically focused essay in the field of neuropsychiatry. Winners will be announced at the conference and prizes and certificates sent out at a later date.

1. SoN Trainee Oral Presentation Competition
Eligibility: The Award is open to all Psychiatry trainees (CT1–3 and ST4–6) working in the United Kingdom. Any trainee can win this prize only once during their career. Application procedure: Candidates wishing to be considered for the Trainee Oral Presentation Competition should email rbrake@rcpsych.ac.uk with the subject reference ‘YOUR SURNAME NEURO SoN ORAL PRIZE 2014’. The following must be attached to the email:

   a. A covering letter explaining clearly in a maximum of 300 words the reason for their application and its suitability for this award. They should clearly specify their role in any collaborative projects.
   b. A description of a research or audit project, a review article or an essay. The total length excluding references should not exceed 5000 words.
   c. A letter from their educational / research supervisor supporting their application and highlighting the trainee’s contribution to the project.

Please refer to instructions on the preparation and submission of abstracts on page 2 when preparing your application. N.B Failure to follow these procedures will automatically disqualify the candidate and no resubmissions will be allowed for that year.

Judging: The judging panel will select up to 5 applicants to present orally at the conference on Thursday 04 September, all of whom will have their registration fee for that day waived. The eventual winner will receive £500 and a certificate.

**The submission deadline is 15:00 on Friday 20 June 2014**

2. SoN Trainee Poster Presentation Competition
Eligibility: The Award is open to all Psychiatry trainees (CT1–3 and ST4–6) working in the United Kingdom. Any trainee can win this prize only once during their career. Application procedure: All posters submitted with a trainee as the lead author will automatically be entered into the Trainee Poster Competition. The abstract must be no longer than 300 words. Abstracts should be e–mailed to rbrake@rcpsych.ac.uk with a subject reference of ‘YOUR SURNAME NEURO 2014’. The following information should be included in the covering message:

   a. Name and email address of main presenting author
   b. Membership number (if applicable)
   c. Job category, e.g. med student, CT1–3, ST4–6
   d. Daytime telephone number
   e. Name of author’s work organisation
   f. General subject of presentation
   g. Whether the abstract should be considered for one of the SoN Trainee Support Grants (if applying for a grant a covering letter must also be attached to the email).

Judging: The three best poster presentations displayed at the conference will be selected for publication in Neuropsychiatry News, the Section of Neuropsychiatry Newsletter and will also receive a certificate. The Editor of Neuropsychiatry News and Academic Secretary of the Section will finalise any decisions on short–listing and final selection of best posters. Their decision will be final and not subject to any appeals.

**The submission deadline is 15:00 on Friday 18 July 2014**
Instructions on the preparation and submission of abstracts:

1. Abstracts should be typed single spaced, font size (10), Verdana in Word format using standard text, or text-only format. Apple Mac documents cannot be accepted.

2. The title should be in bold, followed by the full name(s) of the author and all co-authors, including titles (e.g. Dr) and their affiliations, e.g: Assessment of depression in Emergency Unit of DGH Dr Andrew Brown and Dr Beverley Jones, Riverview Mental Health NHS Trust, Southington; Ms Wendy Smith, The Nightingale Clinic, Staunton.

3. Hand-written abstracts will not be accepted.

4. The title must contain no abbreviations.

5. The abstract should present in order: the aims, methods, results and comments or conclusions. If no information is given in the abstract about the results of the study, the authors must include a covering letter of explanation with their submission.

6. Bibliographic references (Vancouver style referencing only), tables and appendices must not be included in the abstract.

7. Page borders or illustrations/graphics must not be included in the abstract.

8. All spellings should be carefully checked.

N.B Normal conference fees will apply to submitted poster presenters.

3. Grants

SoN Trainee Support Grants
The Section will award trainee grants to enable one Junior Trainee (CT1–3) and 1 Higher Trainee (ST4–6) to attend the Section annual meeting. Each grant will cover free registration and one night’s accommodation at the conference. Travel expenses will not be covered.

Applications will be judged competitively on the quality of the abstract of the paper/poster submitted.

Please note that bursaries are only available to those that submit an abstract for presentation at the conference. To apply for a bursary applicants should follow the instructions on the preparation and submission of abstracts on page 2. A copy of the abstract and a covering letter of support from a supervising colleague, who may be a Trainer, mentor, or Director of Medical Education should also be emailed to rbrake@rcpsych.ac.uk.

Successful candidates will be notified and their grants confirmed by the Chair of Section.

**The closing date for grant applications is 15:00 on Friday 18 July 2014**

Contact
Rosanne Brake, Royal College of Psychiatrists, Centre for Advanced Learning and Conference (CALC) E: rbrake@rcpsych.ac.uk | T: +44 (0) 20 3701 2622 | F: 020 3701 2761