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Graphic Design Jamie Paton – jamie@twhe.co.uk
Speak, Memory, the title of Vladimir Nabokov’s autobiography could just as well be the title for this edition of the Newsletter. The title alludes to the ambiguity of memory. It seems as though memory is a perfect record of our lives that can be “read off” at will. Yet the very act of recollecting, of speaking, distorts each memory anew. There is a paradox here: do we form our memories, or do they form us? The Greeks considered memory a gift from Mnemosyne, the mother of the Muses: without it one could not hear a melody or follow the rhythm of a poem, so there would be no beauty and no joy in life. It is perhaps no surprise therefore, that the only Nobel Prize won for psychiatry was by Eric Kandel, who followed a trail from the study of history, through psychoanalysis to the biological underpinnings of memory in long term potentiation.

I had the privilege of hearing Professor Michael Kopelman’s own memory speak a couple of months back, the results of which can be found in the back pages of this edition. Hear of a lifetime spent in the neuropsychiatry of memory and Professor Kopelman speak his mind on the future for the NHS. The Trans-Atlantic disagreement he describes certainly had the makings of a Greek drama, though it’s uncertain whether this was Comedy or Tragedy.

The same could be said for the story of Drako Zarhazar, an eccentric from Brighton who had lived an extraordinary life but could not recall having done so. Traumatic brain injury caused an anterograde amnesia (at least), though this could not diminish his voracity for life. I assumed the title The Man Whose Mind Exploded was a subtle reference to Luria, but I now understand The Falls’ The Man Whose Head Expanded is the real referent. You’ll either get it or not. Dr Greg Neate reviews this fascinating film and interviews its director for Neuropsychiatry Newsletter in a new Reviews section. Please do contact me with more neuropsychiatically related art and reviews.

The Executive Committee’s Patient Representative, Shelly Harper, has kindly contributed a piece on her many years of experience as a patient. This makes sobering reading and is another reminder of why neuropsychiatric expertise is so valuable yet remains poorly provided for in the NHS. I know that these memories were painful to recall and I thank Shelley for her honest critique and suggestions.

Some may recall the Faculty of Neuropsychiatry held another annual meeting in Lady Margaret Hall, Oxford in September 2014. For those unable to attend and for those whose memory is fading, the Neuropsychiatry Newsletter is proud to present the winners of the Poster and Essay prizes. The well-deserved winners produced original research ranging from mitochondria in Alzheimer’s disease to psychiatric outcomes for epilepsy surgery via the neuropsychological management of autoimmune encephalitis and causation of death in epilepsy. The breadth and depth of neuropsychiatry seems limitless.

Speak, Memory, you will be delighted to hear, has one last pun to give this editorial. It appears from Sahan Mendis’s extensive review that being able to speak more than one language confers some protection against dementia. Or at least delays it till one dies of something else. What isn’t so clear is whether one needs to learn the language in childhood, or whether by the time you get to your age you should just forget it.
Neuropsychiatry – a Service User Perspective

Shelley Harper
Patient Representative, Executive Committee of the Faculty of Neuropsychiatry

‘This poor girl will never achieve an independent life’

Quote from Neurological Unit, Southampton. Neuropsychiatry is an area of psychiatry that I never knew existed until five years ago when I became aware of it through my work as a R.A.B.E. (Research Advisor by Experience) at South West London and St George’s Mental Health Trust. Initially I had been involved with physical disability but on a purely voluntary basis. I turned my attention to mental health as my own mental health deteriorated but became increasingly disillusioned as time and time again I realised my own input was far greater and more effective than those paid to do so.

Thirty-eight years ago I was working with what were then termed ‘maladjusted children’ as a teacher and had a very active lifestyle. I loved all forms of sport and ironically it was while I was out sailing that I had the first indications that something was amiss. It turned out to be a brain stem infarct which left me unable to talk, walk or indeed live my life in the way I had before. I ended up in Intensive Care at Southampton Neurological Unit where it took several months before I had a definitive diagnosis and four months of intensive therapy for speech, physio, hydro and occupational therapy before I was allowed to return home.

But underneath it all I was still me and I will always be grateful to my family for treating me so and encouraging me to become independent as many other people (including those that one would assume should know better) treated me as

Il really hope that neuropsychiatry gains the prominence it deserves because it has the potential to be life changing for some people with relevant neuropsychiatric conditions. I am a good example of this.”.
Fortunately through my work as a Research Advisor by Experience I became aware of the neuropsychiatrist Dr Niruj Agrawal, and knowing that brain damage can cause later depression (ironic that it was myself and not the psychiatrists) I asked if I could be referred to his service. At last there was someone who had an explanation to this reoccurring depression and overwhelming tiredness that seemed to come from nowhere. The medication he prescribed appears to be effective and side effects minimal but once you have had recurrent bouts of mental ill health there is a great tendency to be wary of it happening again. I would suggest that the side effects of some medication encourages non-compliance as a person would rather experience the negativities of a poor mental health as opposed to the extremely debilitating effects of some medications.

One of the really frightening things is that during a period of mental ill health my ability to deal with my physical disability is badly affected and it seems so much worse – not only is your mental ability negatively affected, so is the physical. Poor mental outlook narrows and radically distorts the ability to rationalise perspective on anything and affects all areas of your life. My relationship with neuropsychiatry has radically changed my life and I really hope that in the future people with neuropsychiatric conditions have an easier access to the service than I had. It was so refreshing to speak to someone who after the initial consultation sent me for a CT scan and then with a model of the brain demonstrated where the brain damage was and then went on to explain how this could result in my reoccurring episodes of depression. I cannot tell you how liberating that was. At last I had a diagnosis that made sense to me, and the prospect of medication that might prove to be far more effective than the variety I had previously been prescribed. I know that the Section of Neuropsychiatry at the Royal College of Psychiatrists has recently been granted Faculty status. I hope that it gains prominence and respect amongst psychiatric circles and people with relevant neuropsychiatric conditions find it easier to access than I did. I really hope that neuropsychiatry gains the prominence it deserves because it has the potential to be life changing for some people with relevant neuropsychiatric conditions. I am a good example of this.

Fortunately the passing of time has enabled me to learn how to ‘turn the tables’ on those that are so arrogant to assume they have the right to loudly comment on my disability either expecting me not to hear or maybe not understand what is being said! I have been known to make a tongue in cheek comment and then they really do not know what to make of me!

I have always been forthright about my disability, apart from initial problems coming to terms with such a restricted life (which I have since left far behind) and was heavily involved with disability issues. Surprisingly general psychiatry used this involvement to suggest that exposure to some of the difficult issues I dealt with could be a reason for my recurrent lapses with depression.

something less than human and someone to be pitied. I can well remember a group of teenagers, only a few years younger than me describing my gait as someone who had s..t between her legs. Just as insulting was the fact they assumed I was unable to hear or indeed understand their ignorant comments. It is a sad fact of human nature that you are often judged on external features so you can imagine how I was viewed by those who did not know me – alcoholic, drug addict, mentally incapable of understanding and definitely deaf if the intensely personal and extremely hurtful and humiliating comments which were aimed at me were anything to go by. Even now I still receive comments and sometimes from the most unlikely sources and those whom you would assume should have more insight – for example at this year’s International Conference of Psychiatrists when an overseas delegate assumed she had the right to question a colleague as to what was wrong with me even though I was sat there.

After the initial trauma of a major life changing event and its associated depression I learnt to live with my disability and went on to live my life as an independent person. Unfortunately a few years later I developed severe depression that general psychiatry tried very hard to find a psychological cause for, not making the connection between brain damage and later life depression. This resulted in hospitalisation and which then kept recurring despite the numerous and different antidepressants I was prescribed. It was suggested by several psychiatrists that I had not ‘come to terms’ with my disability which I and those that know me well found very difficult to believe.
The Bilingual Paradigm: A Case for Healthy Ageing

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Dementia: A global and national threat to ageing societies
Dementia has become a global threat to healthy ageing. It has been declared a global public health emergency\(^1\) with 36 million people now living with dementia worldwide\(^2\). This figure is projected to increase to 115 million by 2050\(^2\). It is estimated that 7.7 million people are diagnosed with dementia annually\(^2\). In 2010, $604 billion was spent on dementia, with these costs incurred on informal costs, costs of social care, and direct medical costs\(^3\).

In the UK, there were 815,827 people diagnosed with dementia in 2013, of which 62% is attributable to Alzheimer’s disease\(^3\). This prevalence is projected to increase to 2.09 million by 2051\(^3\). This will cost the UK economy £26 billion per year\(^3\).

Putting bilingualism into context
With no universal panacea on the horizons with failing therapeutic drug trials, and limited current pharmaco-therapeutic options, escalating financial costs\(^4\), attention has recently focussed on potential factors that may underlie healthy ageing and in order to modify the risk of developing cognitive impairment\(^4\).

Cognitive reserve is a concept that proposes factors that promote healthy ageing and other that place individuals at greater risk of developing dementia\(^4,5\). Furthermore, it relates to the brain’s potential to compensate for structural neuronal deterioration through a range of
protective factors\(^5\). It highlights why although some individuals have structural degenerative changes in the brain, they may be less susceptible to the functional changes that are observed in patients with the same degree of structural damage.

Furthermore, cognitive reserve may be associated with greater neuronal efficiency, neuronal capacity, and greater ability for the brain to recruit additional areas for certain functions\(^5\). Factors that may modify cognitive reserve include the level of education, socioeconomic circumstances, premorbid IQ, presence of physical comorbidities, occupational attainment, and the level of physical exercise\(^6\).

These findings suggest a protective influence of bilingualism against cognitive decline that is independent of childhood intelligence, and secondly that bilingualism acquired in adulthood could have some protective benefits.

Bilingualism exists in over 50% of the global population\(^7\) and has been postulated as a cognitive reserve factor\(^6\). This article reviews the emerging evidence for bilingualism and the relationship it has to cognitive processes, highlighting the controversies associated with bilingualism, and addressing the major clinical implications associated to bilingualism in the ageing brain. It will provide a compelling argument for bilingualism being a paradigm for healthy ageing.

**Bilingualism and cognition: the story so far**

Bilingualism emerged at the forefront of cognitive ageing research in 2007 when Canadian Neuroscientist Ellen Bialystock decided to test bilingualism as a potential cognitive reserve factor\(^8\). Her study reviewed hospital records of monolinguals and bilinguals who had been diagnosed with a variety of dementias\(^8\). Her study revealed that on average bilinguals presented with clinical features of dementia, or were diagnosed with dementia 3 to 4 years after monolinguals, with the mean age of diagnosis in monolinguals being 75.4 years versus 78.6 years in bilinguals\(^8\).

Another Canadian study by Chertkow et al which attempted to replicate the Bialystock study revealed less concrete results\(^9\). This study showed that bilingualism appears to delay the onset of dementia symptomology only in immigrant bilinguals, and in multilinguals (only those who spoke 4 languages) within the autochthonous population\(^9\).

Sceptics questioned whether the delay in symptom presentation and diagnosis of dementia was an artefact to other confounding factors such as bilinguals perhaps being more educated, or more intelligent at baseline\(^4\). Was bilingualism truly a cognitive reserve factor, or merely a confounder for other factors such as occupational or educational attainment? Others asserted that if this finding was only observed in immigrant bilinguals that there may be some selection bias that makes the immigrant bilinguals inherently different to the indigenous bilinguals\(^4\). Finally, others criticised that much of the emerging data was restricted to Canada and focussed only on Alzheimer’s disease.

However, in response to the criticism robust studies were designed. A neuroimaging study which examined age-matched cognitively matched monolingual and bilingual participants with Alzheimer’s disease revealed that bilinguals had more AD pathology in their CT heads compared to the monolinguals\(^10\). This provided credence for bilingualism facilitating cognitive reserve and enabling the brain to continue functioning without showing symptoms\(^10\).

A Hyderabad based study compared the onset of dementia symptoms and diagnosis amongst autochthonous monolingual and bilinguals diagnosed with a variety of dementias, which included Alzheimer’s disease, vascular dementia, and front temporal dementia\(^11\). This study recruited 648 participants, and revealed a consistent delay in age of diagnosis by 4 years in bilinguals presenting with different types of dementia, including Frontotemporal dementia, Alzheimer’s disease and Vascular Dementia\(^11\). Furthermore, by recruiting indigenous participants, the findings were not associated with immigration status\(^11\). The age of diagnosis of illiterate bilinguals was strikingly delayed by up to 6 years compared to illiterate monolinguals \(^11\). This suggests that the observed benefit of bilingualism may not be due to
to educational status. Similar findings were observed in a Belgium study\(^{(12)}\) that showed delayed progression of single domain amnesic Mild Cognitive Impairment to Alzheimer’s disease amongst bilinguals\(^{(13)}\).

Bak and colleagues designed a cohort study to examine whether more intelligent people are more likely to become bilingual because of greater cognitive capacity, or whether by becoming bilingual individuals develop greater cognitive capacity\(^{(14)}\). This study sampled participants from the Lothian birth cohort 1936 which tested intellectual attainment amongst 11 year olds living in Scotland in 1947 using the Scottish Mental Survey\(^{(14)}\). Of the 1091 original participants, 853 participants were retested on their cognitive abilities between the years 2008 until 2010 and also completed a bilingualism questionnaire\(^{(14)}\). This methodology enabled Bak to control for childhood intelligence as a confounding factor. This study revealed that the bilingual participants including those who acquired their second language in adulthood performed significantly better on baseline cognitive abilities compared to the monolingual participants on repeat testing, with the greatest effect observed in general intelligence and reading\(^{(14)}\). These findings were independent of other variables such as socioeconomic or immigration status. The study observed that bilingual individuals with higher childhood intelligence appeared to benefit more from bilingualism acquired in early life, where as those with lower intelligence benefit more from later acquisition of the second language\(^{(14)}\).

These findings suggest a protective influence of bilingualism against cognitive decline that is independent of childhood intelligence, and secondly that bilingualism acquired in adulthood could have some protective benefits.

Another Canadian study which explored bilingualism and the role in the progression in Alzheimer’s disease revealed that there were comparable rates of cognitive decline in both monolinguals and bilinguals with dementia\(^{(15)}\).

Figure 1 provides a graphical representation of the differences in the age of diagnosis of Dementia in monolingual and bilingual cohorts, and subsequent projected cognitive decline in both groups. Figure 2 outlines some of the key developments in bilingualism and cognition research.
A neuroimaging study has shown that elderly bilinguals have greater anterior temporal lobe integrity compared to monolinguals. This suggests that bilingualism may induce neuroplasticity. The changes observed in the bilingual brain may promote enhancement grey matter density in areas such as the anterior cingulate cortex. Other neural changes may be observed in relation to learning new vocabulary in the second language by making links with existing vocabulary and semantic knowledge in their first language.

Bilingualism: potential mechanisms against cognitive decline

Although the mechanism by which bilingualism may enhance cognition has not been fully elucidated, it has been hypothesised that the constant need in a bilingual individual to selectively inactivate one language, and activate the second language can contribute to enhanced executive function, with cognitive benefit observed in attentional control, inhibition, and conflict resolution. This may contribute to increased cognitive reserve in bilinguals.

Being bilingual has been linked to the complex cognitive process required to play a musical instrument, to solve complex crosswords or coordinate certain physical exercises. This in turn may enhance neural reorganisation and strengthen neural networks, which may contribute to the delay in cognitive decline that has been observed in bilinguals.

Bilingualism, the media, misconceptions, and clinical benefits

When the initial studies showed delayed onset of dementia in bilinguals compared to monolinguals, there was an outpouring of media attention from a variety of sources, from the BBC to redtop newspapers. Learning a second language was viewed as a potential elixir for healthy ageing. Whilst the bilingual paradigm gained widespread approval, the sceptics drew blood.
depicted earlier, critics’ denounced bilingualism as merely an artefact of other confounding factors and that there were insufficient studies from different cultural contexts. Much of the scepticism about bilingualism and dementia relates to a misunderstanding regarding bilingualism. Principally, bilingualism is viewed as a rare phenomenon, which is of little significance to the global population, and acquired only through immigration, schooling, or being a member of a special linguistic group in Western settings\(^4\). However, bilingualism is common worldwide, such as in places like Hyderabad\(^4\).

Bilingualism can be viewed as holistic in nature\(^4\). This surmises that bilinguals juggle between different sounds, semantic connections, grammatical structures, and social norms related to the languages that they speak\(^4\). This may contribute to significant mental training. There is a need to go beyond traditional ideas of executive function to fully understand the role of bilingualism in cognition.

Also, bilingualism may be associated with changing circumstances, such as marriage, immigration, education, and within different religions\(^4\). It may be linked to both higher and lower socioeconomic status. The proficiency in each language can vary significantly, and either language may be utilised in very different social contexts. Thus these factors may impact how bilingualism influences mental processes.

So where does bilingualism sit within the clinical domain? It is unclear whether linguistic status is documented routinely in clinicians’ consultations for memory problems. Perhaps this should be integrated into routine screening. Conceivably with further evidence bilingualism could be used as a prognostic indicator, and clinicians may be able communicate outcomes to patients newly diagnosed with dementia depending on their pre-existing linguistic status. However, the greatest clinical benefits may be in promoting healthy aging. Organisations such as the Alzheimer’s Society and Alzheimer’s Disease International are incorporating
learning a second language into their public health messages. In the future we may see bilingualism being included in health policy documentation such as the National Framework for Health and Care Excellence.

Research on the role of bilingualism for onset and progression of dementia and in healthy ageing is still in its relative infancy and there are many unanswered questions for exploration. There are few comparative epidemiological studies that have investigated the progression of dementia in bilingual and monolingual cohorts. Will the rate of progression of cognitive deterioration be similar for bilinguals? Will it be steeper? Or more gentler? Does it matter what languages an individual speaks, and when an individual acquired both languages? Is the bilingual advantage observed consistently in different cultural contexts? Are there any differences observed between the elderly Chinese bilingual versus the Chilean, versus the Indian from Hyderabad? How does the pattern of language use affect cognitive ability? Does the linguistic distance between the 2 languages affect cognitive decline? Is the postulated cognitive advantage conveyed to bilinguals affected by immigration status? Are there other neuroimaging correlates to the cognition of bilingual individual? And finally, if there is advantage for the bilingual will there be further benefits to the trilinguals, or multi-linguals?

Even if we come closer to answering these complex research questions, we should be vigilant about how this could be incorporated into clinical practice, and health promotion. These are exciting times in the cognitive neuroscience of ageing and understanding bilingualism phenomenology may provide further invaluable insights into this.

References

Acquired Brain Injury and Violent Criminal Activities

Dr George El-Nimr

Consultant Neuropsychiatrist, Clinical Tutor and Clinical Lead for Neuropsychiatry services in North Staffordshire.

The link between brain injury and criminality has been long studied. The distinction in presentation, pathogenesis and effective management would call for service delivery that is distinctive from main stream Forensic Psychiatry. There seems to be a form of discrepancy between the available evidence and specialist service arrangements and availability.

Three times as many head injured patients show significant aggression during the first 6 months post–injury compared with control group with multiple traumas without head injury, independent to alcohol or depression (Tateno et al 2003). There are also higher rates of Acquired Brain Injury (ABI) in violent offenders compared with controls. Moderate to severe head injury was found in 6 of 20 men convicted of domestic violence compared with only 1 of 20 controls (Turkstra et al 2003). While it is difficult to determine the direction of causality, multiple studies report higher lifetime history of head injury in prison or offender populations (Faruqui 2011). There is also an over–representation of head injury in convicted felons, predating their criminal history (Sarapata et al 1998). Furthermore, approximately 30% of juvenile offenders have sustained a previous brain injury (Farrer et al 2013). This means that juvenile offenders are significantly more likely to have a Traumatic Brain Injury (TBI) and also the rate of TBI within the juvenile offender population is significant.

Research also tells us that men suffering ABI before the age of 15 are more likely to be on national register for criminal offences committed after that age (Timonen, Miettunen et al. 2002); however, in about 30% of cases this was associated with

Various brain areas have been implicated in post ABI aggressive behaviour.
alcohol abuse. Similarly, Males who have committed murder before the age of 18 are very likely to have suffered a serious head injury in childhood (Lewis et al 2004).

Post ABI aggressive behaviour could actually last for years after the injury. About one quarter of patients are found to display aggressive behaviour at 6, 24 and 60 months after discharge from an inpatient rehabilitation unit (Vaishnavi et al 2009).

Various brain areas have been implicated in post ABI aggressive behaviour. The limbic system, especially the amygdala, radiating impulses from the prefrontal cortex and hypothalamus, have been also thought to add emotional content to cognition. Damage to the Hypothalamus with associated sympathetic arousal has also been studied. Damage of the fronto-temporal region has been highlighted as well (Van der Naalt 2000). Frontal lesions were evidenced by some researchers (Tateno et al 2003) but not by others. In any case, there is a recognised association between frontal lobe dysfunction and increased anti-social behaviour (Brower and Price 2001). Focal orbito-frontal injury is associated with increased aggression. Expectedly, deficits in frontal executive function may increase the likelihood of future aggression. There is also evidence that clinically significant focal frontal lobe dysfunction is associated with aggressive dyscontrol. Evidence is strongest for an association between focal pre-frontal damage and an impulse subtype of aggressive behaviour.

Dorsolateral frontal lobes dysfunction will have an expected impact on organizational strategies, problem solving, planning, shifting and maintaining sets, verbal working memory and verbal fluency which can all secondarily lead to poor frustration tolerance and resorting to aggression in order for one to express emotions or general dissatisfaction. On the other hand, the anterior cingulate frontal lobes have been implicated in apathy, little display of emotions, decreased motivated behaviour and creative thought and failure of response initiation / suppression. Damage of the orbitofrontal lobes can lead to a number of problems. These include Personality change, irritability, lability, tactlessness, fatuous euphoria, obsessive compulsive disorder, depressive disorders and decreased empathic, civil and socially appropriate behaviour. Clearly, some of these difficulties can be easily linked with aggression as the brain injured individual can experience difficulty in understanding other people's feelings, difficulty adhering to societal rules and failure to learn from previous experience.

The exact relationship between ABI-related aggression and underlying pathology is not clear. However, various theories have been suggested. These include the following possibilities:

- **Epileptic phenomenon**
- **Damage to temporal / peri-amygdaloid / frontal areas**
- **Underlying pre-ABI aggressive tendencies**
- **Underlying psychopathology**
- **Underlying personality traits / risk factors for both ABI and aggression or criminal behaviour**

It may be worth noting that a distinct set of risk factors are identified for agitation that tends to take place in the early stages of the brain injury. These include Fronto-temporal injury, disorientation, hypoxia, medications (eg: anticholinergic effects, paradoxical disinhibition, sedation), alcohol or substance withdrawal, pain and comorbid medical complications. This is in contrast to a different group of risk factors that have been implicated in late aggression which include Pre-morbid antisocial behaviour, substance and alcohol use, early agitation, fronto-temporal injury, epilepsy, depression, anxiety, and psychosis.

Exploring the role of post ABI mental health problems would certainly enhance our insight into understanding ABI-related violence. ‘Why does ABI increase the risk of Psychiatric disorders?’ would be a reasonable starting point to explore this area. A number of factors have been implicated (Barrett 1999). Increasing constitutional vulnerability to mental illness together with compromised ability to adjust to life change are obvious possibilities. Equally, loss of role, employment, relationships and finances would expectedly play an important role together with possible physical disability and impaired body image.

Altered ability of appraisal of the world is thought to be an important factor that could, at least partly, explain the higher likelihood of mental distress and subsequently violent behaviour. For example, poor autobiographic memory can lead to frustration and
anger when an injured individual does not recall that he had been through divorce or took early retirement. Perceptual and sensory problems such as hemi neglect can make patients react quite violently when individuals approaching them from the ‘bad side’, ‘suddenly appear’ in an uncomfortable or even perceived threatening manner presenting with an unwelcome invasion of their personal space. Agnosia can create similar problems. Impaired self-awareness can make it difficult for patients to comply with social rules. Likewise, impaired social emotions such as sympathy, empathy and embarrassment can easily make patients behave in an apparently self-centred, inconsiderate and possibly hostile way especially when demands are not satisfactorily met. Frustration can easily develop when patients can no longer understand others or struggle to express their views and wishes in an effective way. Right hemisphere lesions can lead to impaired ability to accurately interpret irony, humour and sarcasm. Taking the literal meaning of a joke can be quite upsetting, offensive or even threatening. Moreover, patients may lose their ability to understand and/or express emotions in body language, facial expressions or changing voice intonation. The world suddenly becomes unanimated and hostile! Patients feel ‘obliged’ to respond in an equally unfriendly manner.

The term ‘organic personality disorder’ has been used rather unsatisfactorily to express many of these difficulties such as poor insight and judgement, compromised social adaptation and impaired empathy. A number of specific problem areas have been studied as a potential explanation of the personality changes that take place after ABI. These include loss of sense of self, childish behaviour, irritability and agitation, impaired judgement, problems with attention, impaired social awareness, language difficulties and perceptual abnormalities.

A number of predictors of aggression after ABI have been identified. These include alcohol, younger age at injury, depression, frontal injury and pre-injury history of antisocial behaviour.

The characteristics of organic aggression syndrome (Silver et al 2005) appear to be fairly distinctive. This kind of aggression tends to be precipitated by minor triggers without prior planning. Aggression in this context has a tendency to be explosive, periodic and non-purposeful. It has also been described as ‘ego dystonic’, causing distress to patients who have been involved in such behaviour.

Post ABI violence require a specific set of resources to address its clinical, ethical and medico-legal aspects. Nonetheless, this is beyond the scope of this article. should know better) treated me as

References:
Psychiatric and psychosocial morbidity before and one year after epilepsy surgery

Dr Maurice Clancy

I undertook the following research study while working as a Clinical Research Fellow in Neuropsychiatry in the National Centre for Neurology and Neurosurgery, Beaumont Hospital, Dublin from 2010 until 2012. This was a post membership post which is accredited for the Irish Senior Registrar Psychiatric Training scheme. My MD thesis which was based on the findings of this research has recently been awarded. This research piece also won the College of Psychiatry of Ireland Trainee Research Oral Prize Presentation in Winter 2013 and the East London Foundation Trust Trainee Oral Research Prize in January 2014.

I wish to thank and acknowledge my colleague Dr Helen Barry who collected the data from 2008 to 2010 which was utilized in this study. I am also indebted to my research and clinical supervisors, Professor David Cotter, Professor Kieran Murphy and Professor Mary Cannon for all their help and support while undertaking the study. I plan to publish my research findings in the future in Epilepsia or a related Neuropsychiatry Journal.

Epilepsy is a common disease with a prevalence of 0.5–1% of the population. Approximately 50% of patients with epilepsy will achieve good seizure control on one anticonvulsant, 20–30% of non-responders will achieve control with addition of a further medication but up to 30% will fail to respond to medication and in this subgroup, surgery for refractory seizures should be considered. In patients with refractory epilepsy, advances in surgical procedures have offered considerable hope for improved outcomes giving seizure freedom in 50–80% of patients (Engel et al., 2003).

There are methodological limitations in the literature on surgery and psychopathology as most reports have been cross sectional studies in small samples or heterogeneous groups of patients and are usually for a limited follow up. It is difficult to compare the studies due to different methods used in identifying or measuring psychopathology. In addition, the standard psychiatric measures have not been validated in patients with epilepsy. Few prospective studies involve large samples or long term follow up which are essential in providing further insights into the psychiatric sequelae following epilepsy surgery. Up
to 70% of patients with refractory epilepsy can suffer from mental illness (Devinsky, 2003, Gaitatzis et al., 2004). Psychiatric comorbidity has been associated with a worse surgical outcome after temporal lobectomy (Kanner et al., 2009, Anhoury et al., 2000, Guarnieri et al., 2009). A more recent study found no significant associations between post-surgery seizure outcome and a current or lifetime history of any psychiatric disorder (Adams et al., 2012). The literature is unclear about improved psychiatric outcome following epilepsy surgery with earlier studies linking surgery to increased rates of depression, anxiety and psychosis (Blumer et al., 1998, Jensen and Larsen, 1979, Glosser et al., 2000, Taylor, 1972, Trimble, 1992). Later studies have shown conflicting results (Devinsky et al., 2005, Pintor et al., 2007).

One aspect of the literature which I found striking while writing my introduction on psychiatric illness in epilepsy was the large discrepancy in the number of patients with epilepsy diagnosed with psychosis. This ranged from less than 1% in some community studies to 35% in some tertiary referral centres (Jensen et al., 1979). This led me to undertake a systematic review and meta–analysis on the prevalence of psychosis in epilepsy (Clancy et al. 2014). I found a prevalence rate of 5.2% (95% CI: 4.8–6.4) of psychosis in epilepsy. There was a high level of heterogeneity. The prevalence of psychosis in temporal lobe epilepsy was 7% (95% CI: 4.9–9.1). The prevalence of interictal psychosis in epilepsy was 5.2% (95% CI: 3.3–7.2). The prevalence of postictal psychosis in epilepsy was 2% (95% CI: 1.2–2.8).

There has been a systematic review on psychiatric outcomes in epilepsy surgery (Macrodimitris et al., 2011). Macrodimitris et al found either improvements in psychiatric outcomes post-surgery or no changes in psychiatric outcome. The two main predictors of psychiatric outcome were seizure freedom and pre-surgical psychiatric history.

De novo psychiatric disorders occurred post-operatively at a rate of 1.1–18.2% with a milder psychiatric illnesses being more common. An important finding was that studies that included a structured clinical interview (eg the SCID as I utilized in my study) produced more definitive positive results compared to studies that employed rating scales which generally demonstrated more mixed or equivocal results. Psychiatric complications presenting as exacerbation or recurrence of pre-surgical psychiatric comorbidities are most frequent in the first postsurgical year as are psychosocial adjustment difficulties (Kerr et al., 2011).

I specifically sought to address the shortcomings in the studies mentioned above by ensuring that my study was of prospective design, used gold standard tools and had an adequate period of follow up.

I formulated the following hypotheses at the outset of my study:

– The prevalence of psychiatric diagnoses will be higher in the pre-operative group compared to the post-operative group
– The severity of psychiatric symptoms will be higher in the pre-operative compared to the post-operative group
– Surgical treatment of refractory epilepsy is associated with an improvement in quality of life.

The study was carried out in Beaumont Hospital, a large 820 bed academic teaching hospital in Dublin, Ireland. It is the Irish national referral centre for neurology, neurosurgery and neuropsychiatry with a national catchment area of 4.6 million people according to the last Irish census in 2011.
The study comprised a prospective cohort study of the patients who proceeded to surgery and who had one year follow up. The study involved baseline pre-operative assessment of a sample of medically refractory epilepsy patients and follow-up of patients who proceeded to surgery at one year post-operatively to determine impact of epilepsy surgery on psychiatric diagnosis and subjective quality of life outcomes. Ethical approval was sought and obtained prior to this study being commenced.

All consenting patients underwent baseline assessments pre-operatively while they were in-patients on the Epilepsy Monitoring Unit. The following items/scales were used: SCID I and SCID II (SCID Structured Clinical Interview for DSM IV), HADS (Hospital Anxiety and Depression Scale) and the QOLIE–89 (Quality of Life in Epilepsy 89). Data was analysed using SPSS version 18.

One of the biggest difficulties involved in the study was trying to recruit a sufficient number of patients and follow them up successfully. Some patients were reluctant to be seen for post-operative follow up SCIDs and required encouragement to partake. I spent quite a lot of time waiting outside Neurology and Neurosurgery outpatient clinics waiting to see patients. The help of the neurology and neurosurgery secretaries in locating patients and their charts was invaluable and definitely contributed to my overall numbers. One of my tips for trainees planning to become involved in clinical research in any medical discipline is to make friends with the administration staff of the patients who you need to recruit.

The demographic results of my study were as follows. Forty eight of the baseline sample of participants with medically refractory epilepsy proceeded to surgery within the timeframe of the study. Twenty male (41.7%) and 28 (58.3%) female patients had surgery. The average age of the patients participating was 34.95 (standard deviation 10.67) and the median age was 33. The range was 17 to 65.

Localisation related epilepsy is also known as partial or focal epilepsy ie arising from a specific focus. As would be expected in a cohort assessed as suitable for surgical intervention, my entire group of patients had localisation related epilepsy. Of these, 42 (87.5%) had temporal lobe localisation epilepsy, 5 patients (10.4%) had a frontal focus and 1 patient had an occipital focus.

Data on the number of seizures of any type per month was gathered. The mean number of seizures per month in the surgical group was measured pre-operatively and post-operatively. The frequency of seizure episodes per month was also subdivided as a measure of both the severity of epilepsy and improved impact on quality of life into groups: one or less seizures per month, between 2–4 seizures a month, between 5–15 seizures a month, between 16 to 30 seizures a month and greater than 30 seizures per month. The table below shows the number of seizures pre-operatively and post-operatively in the cohort.

Comparing seizure frequency groups pre-operatively and post-operatively
Ten patients had the same frequency of seizures pre-operatively and post-operatively, (relatively rare, except for patients who were already in the lowest frequency group) Thirty-five patients had a lower number of seizures post-operatively. Three patients had an increase in seizure frequency post-operatively.

<table>
<thead>
<tr>
<th>Number of seizures per month</th>
<th>Frequency pre-operatively</th>
<th>Percentage pre-operatively</th>
<th>Frequency post-operatively</th>
<th>Percentage post-operatively</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 or less</td>
<td>10</td>
<td>20.8%</td>
<td>34</td>
<td>70.8%</td>
</tr>
<tr>
<td>2–4</td>
<td>13</td>
<td>27.1%</td>
<td>9</td>
<td>18.8%</td>
</tr>
<tr>
<td>5–15</td>
<td>12</td>
<td>25%</td>
<td>3</td>
<td>6.3%</td>
</tr>
<tr>
<td>16–30</td>
<td>7</td>
<td>14.6%</td>
<td>2</td>
<td>4.1%</td>
</tr>
<tr>
<td>30+</td>
<td>6</td>
<td>12.5%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>48</td>
<td>100%</td>
<td>48</td>
<td>100%</td>
</tr>
</tbody>
</table>
GMC saw their symptoms remit and one patient with brief psychotic disorder had their symptoms remit. See table 3

In order to clarify what changes had occurred, ie whether SCID diagnosis had resolved post-operatively or whether a de novo (new onset) diagnosis had developed, the cases were examined in more detail. A total of 4 patients (8.3%) developed a de novo psychiatric illness post-operatively.

With regard to affective disorders, one patient developed a new onset major depressive episode post-operatively. Two patients with pre-operative SCID diagnoses had changes in their SCID diagnoses post-operatively. One patient who had a diagnosis of psychosis NOS had this resolve post-operatively but had a diagnosis of an adjustment disorder post-operatively which was unrelated to epilepsy or physical illness. The second patient who had a diagnosis of a major depressive episode with agoraphobia continued to have a major depressive illness post-operatively and they also developed panic attacks as well as agoraphobia.

There were no new cases of de novo (new onset) psychosis post-operatively. Two patients developed de novo anxiety disorders post-operatively; both patients were diagnosed with generalised anxiety disorder.

Hospital Anxiety and Depression Scale (HADS)
The pre and post-operative HADS scores were analysed using paired t tests. There were no significant differences pre-operatively versus post-operatively in the overall HADS score or for the anxiety or depression subscales. The mean pre-operative HADS score was 8.1 (sd 6.7) and the mean post-operative HADS score was 8.7 (sd 7.3).

### All Psychiatric diagnoses
Pre-operatively 24 patients (50%) of patients were diagnosed with a psychiatric disorder, 24 (50%) had no psychiatric diagnosis. Post operatively, 18 patients (37.5%) were diagnosed with a SCID I disorder. Thirty four patients (70.8%) did not meet criteria for a psychiatric disorder. This result was statistically significant (p < 0.021). See table 2

### Pre and post-operative changes in psychotic disorders status
High numbers of patients were diagnosed with psychosis pre and post operatively. This appeared to be a spurious finding due to SCID I being over-rigorous in diagnosing psychotic symptoms which over-lapped with seizure auras, I will elaborate on this later. Pre-operatively 18 patients were diagnosed with a psychotic illness of some type. Sixteen patients were diagnosed with psychosis due to a general medical condition (GMC), one was diagnosed with psychosis not otherwise specified (NOS) and one was diagnosed with a brief psychotic disorder. Eight patients post operatively had a diagnosis of psychosis due to a GMC and one had diagnosis of psychosis NOS. Eight patients with psychosis due to a GMC saw their symptoms remit and one patient with brief psychotic disorder had their symptoms remit.

See table 3

In order to clarify what changes had occurred, ie whether SCID diagnosis had resolved post-operatively or whether a de novo (new onset) diagnosis had developed, the cases were examined in more detail. A total of 4 patients (8.3%) developed a de novo psychiatric illness post-operatively.

### Pre and post-operatively

<table>
<thead>
<tr>
<th>Psychiatric diagnosis</th>
<th>Pre-operatively N (%)</th>
<th>Post-operatively N (%)</th>
<th>Exact Significance (2 sided) McNemar test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood diagnosis</td>
<td>8 (16.7%)</td>
<td>5 (10.4%)</td>
<td>0.5</td>
</tr>
<tr>
<td>Psychosis diagnosis</td>
<td>18 (37.5%)</td>
<td>9 (18.8%)</td>
<td>0.004</td>
</tr>
<tr>
<td>Anxiety diagnosis</td>
<td>4 (8.3%)</td>
<td>4 (8.3%)</td>
<td>0.625</td>
</tr>
<tr>
<td>No diagnosis</td>
<td>24 (50%)</td>
<td>24 (70.1%)</td>
<td>0.021</td>
</tr>
</tbody>
</table>

### De novo psychiatric illness post-operatively

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalised anxiety disorder</td>
<td>2 (4.2%)</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Adjustment disorder</td>
<td>1 (2.1%)</td>
</tr>
<tr>
<td>Psychosis (any type)</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>4 (8.3%)</td>
</tr>
</tbody>
</table>
Quality of life in Epilepsy 89 (QOLIE–89)
A paired t test was used to examine the change between the pre-operative and post-operative group in the Quality of Life in Epilepsy 89 score (QOLIE–89). Thirty patients (62.5%) completed a pre-operative QOLIE–89 and 33 patients (68.8%) completed a post-operative QOLIE–89. The mean pre-operative QOLIE–89 score was 69.9 (sd 12.2) and the post-operative QOLIE–89 score was 74.8 (sd 14.1). This result was statistically significant (p<0.02).

In conclusion, my study was a prospective cohort study using structured clinical instruments which examined the relationship between psychopathology, medically refractory epilepsy and quality of life.

The main findings of my study were:
There was a significant reduction in psychopathology following surgery from 50% to 29.2%

There was a statistically significant improvement in quality of life scores following surgery (p<0.02)

There was a statistically significant reduction in the number of patients who had a diagnosis of a psychotic disorder post-operatively (p<0.004)

There was a slight increase in the severity of self-reported psychiatric symptoms experienced post-operatively (8.8–overall HADS score) compared to pre-operatively (8.1–overall HADS score). This result was not statistically significant.

There was a statistically significant reduction in the rate of psychiatric diagnosis following surgery for epilepsy compared to the pre-operative psychiatric diagnosis rate. Of the 48 participants who proceeded to surgery, pre-operatively 50% were diagnosed with a psychiatric disorder while one year post-operatively, 29% were diagnosed with a disorder. (p<0.021) Therefore one of the key hypotheses of my study, that the prevalence of psychiatric diagnosis will be higher in the preoperative group compared to the post-operative group was proven. My findings are an addition to the literature which contradict older studies which linked surgery to serious psychiatric sequelae including increased rates of suicide, psychosis and depressive disorders (Jensen and Larsen, 1979, Taylor 1972, Trimble 1992, Blumer et al 1995). More recent studies have found conflicting results, some found an increase in rates of depressive and anxiety symptoms but no change in rates of suicide or psychosis (Anhoury et al 2000, Bladin 1992) whereas other studies with greater numbers or using structured clinical instruments found reductions in rates of depression and anxiety (Macrodimitris et al 2011, Devinsky et al 2005, Pintor et al 2007).

There was a very significant reduction in the number of patients who pre-operatively had a diagnosis of a psychotic disorder. This was due to auras which commonly comprise mainly hallucinatory phenomenon which ceased post-operatively. The most common type of auras my patient group experienced were olfactory, gustatory and somatic phenomenon. I find it particularly fascinating that auras lend credence clinically to the association between schizophrenia and epilepsy which has already been confirmed from neuropathological, neuroimaging and genetic perspectives. Unfortunately there is dearth of material in the literature of epilepsy and psychosis about this interesting clinical overlap.

It has been hypothesised in the older literature that studies which show a decrease in the number of patients with post-operative psychopathology are complicated by the presumed rejection for surgery of patients with pre-operative psychopathology. However, no patients in this study were rejected for surgery based upon the presence of psychiatric pathology. This study therefore lends weight to the body of research establishing the potential benefit of surgery for patients with a comorbid psychiatric disorder (Fenwich, 1994, Reutens et al, 1997).

Implications for clinical practice
Patients with epilepsy have significantly higher rates of psychiatric disorder than the general population. Therefore identification and treatment of psychiatric illness especially depression is needed to improve quality of life in patients with epilepsy. I feel that there is a high level of need for specialist neuropsychiatry services in order to screen for psychosis and depression in epilepsy. This study demonstrated a positive effect of surgery on medically refractory epilepsy in terms of mental health. The presence of a psychiatric disorder should not be considered a contraindication for surgical treatment for epilepsy when surgery is warranted. This is in contrast to the view in the older literature that mental illness is a contradiction for surgery. Careful attention to the provision of effective psychiatric treatment of pre-surgical psychiatric comorbidities...
can help reduce the risk of postsurgical psychiatric complications. Eleven of the fourteen patients with a post-operative SCID diagnosis had a pre-operative SCID diagnoses. It has been argued that a detailed psychiatric assessment of patients should be as integral to the epilepsy surgery work-up as neuropsychological testing or the recording of seizures by video EEG.

Undertaking this study has stimulated my interest in pursuing a career in neuropsychiatry in the future. I find the overlap between the neurological and psychological aspects of patients’ presentations fascinating. I have learnt that there is no such thing as a ‘normal’ patient in neuropsychiatry – the impact of a patient’s illness on their quality of life, the family dynamics of somebody with a chronic sick role, personality factors which may be maladaptive, as well as the physical and psychiatric aspects add up to a complex patient group.

My experiences in assessing, diagnosing and managing this particular patient cohort have helped me to offer overall better care to all patients that I have seen subsequently, be it in a General Adult, Liaison or Neuropsychiatric ward round or out–patient clinic. I would finally like to thank all the patients who kindly participated in this study.

No financial disclosure to declare

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Long term rehabilitation management and outcome of NMDAR encephalitis: Case reports

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Aims
- To describe long term community rehabilitation management of patients with NMDAR encephalitis.
- To highlight the need for holistic intervention and integrated joined up working in the community.
- To highlight specific issues of working with NMDAR patients in the community.
- To highlight and address specific ethical considerations in working with NMDAR patients in the community.

Methodology
Retrospective study describing three cases. Inclusion Criteria – selection of three patients with NMDAR encephalitis treated by the ABI Specialist Outreach team. All three cases are young women, two of whom presented with ovarian teratoma. Case KH is the most impaired and was resident in a slow stream rehabilitation care home and presented with challenging behaviour. Cases RM and OA both lived in the community and presented with similar anxieties but diverse levels of cognition and motivation.

Introduction
Identification of NMDAR encephalitis is fairly new thus the extant literature on long-term rehabilitation treatments and outcome is limited. Even in cases of full recovery or those with only mild persistent deficits more than 85% of patients show significant psychiatric symptoms at time of discharge with persistent cognitive deficits of memory loss, impairments in executive function, including inattention, poor organisation and planning difficulties, disinhibition and lack of impulse control.

This study is the first to describe the long term outcome of NMDAR encephalitis receiving rehabilitation from a specialised ABI community team. The behavioural and psychological management of this relatively rare syndrome is presented with a view to highlighting the long term challenges and support required for this condition.

Measures
Key Outcome Measures
(1) Brain Injury Community Rehabilitation Outcome Scales (BICRO-39) (Powell, 1999) assess level of social functioning and participation in the community following brain injury. 39 item self-rating scale.

Patients completed two BICRO–39 scales; one rating their current functioning and a second retrospectively rating functioning prior encephalitis. A significant other (e.g. spouse, parent carer) independently completed a BICRO–39 scale providing their perception of the current functioning of the patient.
**BICRO-39 Scales scoring system**

BICRO-39 Scales scoring system:

- Personal care: independence in basic self-care activities e.g. using the toilet.
- Mobility: independence in more physically demanding tasks e.g. laundry, shopping.
- Self-organisation: independence with structuring personal and domestic activities e.g. managing appointments.
- Socialising: frequency of contact with people other than immediate family e.g., friends, colleagues.
- Productive employment: frequency of engagement in education, work, or childcare.
- Psychological wellbeing: frequency of feeling impatient with self, bored, lonely, worn out, hopeless about the future, and angry with others.

Rated on 5 point scale: 0=with no help to 5=can’t do at all. 0=never to 5=daily, 0=not at all to 5=several hours a day, 0=never to 5=almost always.

(2) Quality of Life After Brain Injury (QOLIBRI) (von Steinbuechel et al, 2012) provides a profile of health-related quality of life (HRQoL) in domains typically affected by brain injury

- Overall how satisfied are you with your physical condition cognition, memory and thinking skills?
- Emotional condition?
- Ability to carry out day-to-day activities?
- Personal and social life?
- Current situation and future prospects?

Scored on a 4 point scale: Not at all satisfied = 0, Slightly satisfied = 1, Moderately satisfied = 2, Quite satisfied = 3, Very satisfied = 4

(3) Hospital Anxiety and Depression Scale (HADS)

**Secondary Measures**

(1) Cognitive Measures

- REASONING: Wechsler Adult Intelligence Scale IV (WAIS-IV) Similarities, Matrix Reasoning, Digit Span
- MEMORY: Wechsler Memory Scales IV (WMS-IV) Story Recall Immediate and Delayed
- ATTENTION AND COGNITIVE FLEXIBILITY: Trail Making Test (TMT) Parts A and B
- VERBAL FLUENCY: phonological (FAS), semantic (animals, boys’ names)
- EXECUTIVE : INHIBITION, PLANNING, ORGANISATION – Behavioural Assessment of Dysexecutive Syndrome (BADS)

(2) Social Cognitive Measures

Theory of Mind Questionnaire (TOMQ) patient and informant version.

**Example questions:**

- How much of a difficulty do you have in recognising when something you say or do has upset someone else?
- How much of a problem do you have in being tactful?

Rated on a 5 point Lickert scale: Can’t do, Very difficult to do, Can do with some difficulty, Fairly easy to do, Can do with ease

**Case KH**

- 28 year old, right handed, young woman who suffered antibody NMDAR encephalitis and bilateral frontal infarcts.
- She had an oophorectomy and EEG established the presence of tonic clonic seizures.
- Seizures were on-going.
- Discharged after 6 months to a Care Home, for slow stream rehabilitation.
- At 2 years post diagnosis seen by RNRU Outreach team for assessment and management of challenging behaviour.
- Current medication included anti-seizure medication, steroids, warfarin and antipsychotics.
- Cognition: Impaired for attention, episodic memory and executive.
- Emotional memories strong.
- Comprehension, reasoning limited.
- Rigid thinking, unable to link concepts such as the need for rehabilitation.
- Insight into cognitive deficits impaired.
- Communication: Impaired – severe dysarthria. Able to follow single stage commands and give some limited verbal responses.
  - Baseline – refusing to wear splints, kicking, refusing physiotherapy.
  - Therapists’ goal – KH to wear splints and engage in walking in the parallel bars during physiotherapy session.
  - Motivational goal – KH to be able to walk her dogs independently.
  - Intervention – Graded behavioural program employing KH’s dogs as positive reinforcements and to enhance
positive emotional memories. KH’s two dogs were brought into the session and KH was given the lead to hold along with the physiotherapist for safety. Verbal encouragement provided.

- Outcome: Overreached target of 10 lengths of the parallel bars within a few weeks and this was consistently maintained in the long term.
- At 12 months – further improvements. Now living back at home and supported by her husband. Able to walk with crutches up inside her home. Concentration and memory continue to improve although executive difficulties remain for planning and organisational tasks. Mood is much improved. KH describes herself as positive and happy. Outbursts of frustration are minimal.

**Case OA**

- 23 year old, right handed, young woman NMDAR encephalitis and ovarian mass.
- Presented to hospital with seizures and lowered CGS.
- Discharged to home after 6 months.
- Ongoing complaints: lack of motivation, fatigue, noise sensitivity, hyper sensitive to taste, daily headaches, pain throughout body.
- Social Cognition: she reported difficulties in recognising that she said things that upset others, understanding jokes, showing sympathy and being tactful.
- Mood: low mood, loss of confidence, fears that illness may reoccur.
- Anxiety – did not go out on her own because she was fearful she may ‘pass out’.
- Quality of Life: marked reduction for physical condition, ability to carry out day-to-day activities, current situation and future prospects.

**Goal** — OA had just completed an undergraduate degree and had been about to enrol in a Masters degree. Her ambition was to return to university to achieve her Masters degree.

- Intervention: Cognitive strategies to support attention, memory and executive function. Fatigue management. CBT.
- Outcome: Returned to university and studying part time for a Masters degree in English literature. Able to complete journey to and from university independently and was using appropriate strategies to manage her studies at university.
- Mood was positive. Hopeful about her future prospects. Unfortunately OA did not maintain this gain over the next two months as she found the commute to university fatiguing.
- In summary, OA demonstrated modest, positive outcomes on measures of community integration, mood and quality of life. These gains were maintained at 6 month follow–up despite her setback in educational plans due to physical fatigue.

**Case RM**

- 24 year old, right handed, young woman, two week history of worsening confusions dysphasia, dyspraxia and emotional lability with personality change.
- Following hospital discharge a deterioration in function was noted with ongoing cognitive difficulties including reduced concentration, poor memory and reduced speed of information processing.
- The most significant deterioration is that she had become doubly incontinent.
- Everyday functional activities: Independent in personal care but required constant help/assistance to organise herself.

- Cognition: Impaired in verbal reasoning ability, sustained attention, memory, speed of information processing and executive difficulties on planning tasks.
- Social Cognition: Difficulties with empathising, tactfulness, ability to sympathise and some reduced insight into these problems.
- Mood: Low mood, low self-esteem and reduced confidence in abilities to do things.
- Anxiety: Significant anxiety issues and low mood. Highly anxious about going outside on her own, worried she may suffer an episode of confusion.
- Quality of Life: Reduced for current situation and future prospects, mood, ability to carry out daily activities, social life and level of cognitive function.
- Behaviour: Fatigue, lack of motivation, avoidance to engage in specific tasks. Found activities effortful and overwhelming. Her low mood and anxiety acted as a barrier to her engaging in tasks.
- Intervention: Graded behavioural exposure program to reduce anxiety. She required much encouragement to engage fully due to her anxiety. Fatigue management.

### Table 1 Brain Injury Community Rehabilitation Outcome (BICRO–39*) scores

<table>
<thead>
<tr>
<th>BICRO–39 Scale*</th>
<th>RM Baseline average score</th>
<th>RM Discharge average score</th>
<th>RM 6 month follow-up average score</th>
<th>RM Baseline Informant average score</th>
<th>RM Discharge Informant average score</th>
<th>RM 6 month Informant follow-up average Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal Care</td>
<td>0.0</td>
<td>0.0</td>
<td>0.7</td>
<td>0.0</td>
<td>0.0</td>
<td>1.5</td>
</tr>
<tr>
<td>Mobility</td>
<td>4.0</td>
<td>3.0**</td>
<td>2.3**</td>
<td>4.0</td>
<td>2.8**</td>
<td>2.8**</td>
</tr>
<tr>
<td>Self–Organisation</td>
<td>3.0</td>
<td>2.0**</td>
<td>2.5**</td>
<td>4.0</td>
<td>3.0**</td>
<td>2.5**</td>
</tr>
<tr>
<td>Socialising</td>
<td>4.0</td>
<td>–</td>
<td>2.7**</td>
<td>4.0</td>
<td>–</td>
<td>3.0**</td>
</tr>
<tr>
<td>Productive Employment</td>
<td>5.0</td>
<td>5.0</td>
<td>4.5**</td>
<td>5.0</td>
<td>5.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Psychological Wellbeing</td>
<td>5.0</td>
<td>3.0**</td>
<td>2.3**</td>
<td>5.0</td>
<td>5.0</td>
<td>3.7**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BICRO–39 Scale*</th>
<th>OA Baseline average score</th>
<th>OA Discharge average score</th>
<th>OA 6 month follow-up average score</th>
<th>OA Baseline Informant average score</th>
<th>OA Discharge average score</th>
<th>OA 6 month follow-up Informant average score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personal Care</td>
<td>0.2</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Mobility</td>
<td>3.8</td>
<td>0.4**</td>
<td>0.0**</td>
<td>3.6</td>
<td>0.6**</td>
<td>0.0**</td>
</tr>
<tr>
<td>Self–Organisation</td>
<td>3.8</td>
<td>2.0**</td>
<td>1.3**</td>
<td>3.5</td>
<td>1.7**</td>
<td>2.0**</td>
</tr>
<tr>
<td>Socialising</td>
<td>3.2</td>
<td>2.7**</td>
<td>3.0**</td>
<td>2.7</td>
<td>2.3**</td>
<td>2.5**</td>
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<tr>
<td>Productive Employment</td>
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<td>3.5**</td>
<td>4.0**</td>
<td>4.7</td>
<td>3.2**</td>
<td>4.2**</td>
</tr>
<tr>
<td>Psychological Wellbeing</td>
<td>2.8</td>
<td>2.2**</td>
<td>2.2**</td>
<td>3.2</td>
<td>3.2</td>
<td>3.7</td>
</tr>
</tbody>
</table>

*BICRO–39 scoring system: scoring ranges from 0 (doesn’t do at all) to 5 (no help). Lower scores indicate greater independence. See Appendix for further details of scoring system. ** indicates improvement in BICRO–39 average scores from baseline score.

- Outcome: RM adopted cognitive compensatory strategies e.g. diary use, to help support aspects of her poor memory functioning. With structured support, RM was able to engage in a number of activities and was more confident completing activities within home. environment. Ability to access the community or to be on her own remained limited. Her levels of anxiety and depression remained borderline.
Table 2. Neuropsychological Profile for cases RM and OA

<table>
<thead>
<tr>
<th>Test</th>
<th>RM</th>
<th>OA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WAIS-IV</strong></td>
<td>Age Scaled Score</td>
<td>Age Scaled Score</td>
</tr>
<tr>
<td>Similarities</td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td>Matrix reasoning</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Digit Span</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Digits Forwards</td>
<td>5 (unreliably) 3 (reliably)</td>
<td>6 (unreliably) 5 (reliably)</td>
</tr>
<tr>
<td>Digits Backwards</td>
<td>4 (unreliably) 2 (reliably)</td>
<td>4 (reliably)</td>
</tr>
<tr>
<td><strong>WMS-IV</strong></td>
<td>Age Scaled Score</td>
<td>Age Scaled Score</td>
</tr>
<tr>
<td>Logical Stories Immed</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>Logical Stories Delayed</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td><strong>Verbal Fluency</strong></td>
<td>15</td>
<td>Age Scaled Score</td>
</tr>
<tr>
<td>Letter (FAS) Fluency</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Semantic Fluency</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td><strong>Trail Making Test (TMT)</strong></td>
<td>Time (secs)</td>
<td>Centile</td>
</tr>
<tr>
<td>TMT Part A</td>
<td>39</td>
<td>&lt;10</td>
</tr>
<tr>
<td>TMT Part B</td>
<td>74</td>
<td>&lt;10</td>
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<tr>
<td><strong>BADS</strong></td>
<td>Profile score</td>
<td>Profile score</td>
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<tr>
<td>Rule Shift</td>
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<td>3</td>
</tr>
<tr>
<td>Action program</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Key search</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Temporal Judgement</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Zoo map</td>
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<td>2</td>
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<tr>
<td>Modified six elements</td>
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<td>2</td>
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### Table 3. Mood (HADS) and Quality of Life (QOLIBRI-OS*) scores for RM and OA

<table>
<thead>
<tr>
<th>Scale</th>
<th>Baseline</th>
<th>Discharge</th>
<th>6 month follow-up</th>
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<tr>
<td><strong>Patient RM</strong></td>
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<tr>
<td>HADS Anxiety score</td>
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<td>15</td>
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<tr>
<td>HADS Depression score</td>
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<td>QOLIBRI-OS Physical condition</td>
<td>Quite</td>
<td>Moderately</td>
<td>Moderately</td>
</tr>
<tr>
<td>QOLIBRI-OS Cognitive</td>
<td>Moderately</td>
<td>Slightly</td>
<td>Slightly</td>
</tr>
<tr>
<td>QOLIBRI-OS Mood and emotions</td>
<td>Slightly</td>
<td>Slightly</td>
<td>Moderately**</td>
</tr>
<tr>
<td>QOLIBRI-OS ability to carry out day to activities</td>
<td>Slightly</td>
<td>Slightly</td>
<td>Slightly</td>
</tr>
<tr>
<td>QOLIBRI-OS personal and social life</td>
<td>Slightly</td>
<td>Slightly</td>
<td>Moderately**</td>
</tr>
<tr>
<td>QOLIBRI-OS current situation and future prospects</td>
<td>Not all</td>
<td>Moderately**</td>
<td>Slightly**</td>
</tr>
<tr>
<td>QOLIBRI-OS Average score</td>
<td>2.3</td>
<td>2.3</td>
<td>2.5**</td>
</tr>
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<td><strong>Patient OA</strong></td>
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<tr>
<td>HADS Anxiety score</td>
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<td>9</td>
<td>9</td>
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<tr>
<td>HADS Depression score</td>
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<td>4</td>
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<tr>
<td>QOLIBRI-OS Physical condition</td>
<td>Not at all</td>
<td>Slightly**</td>
<td>Moderately**</td>
</tr>
<tr>
<td>QOLIBRI-OS Cognitive</td>
<td>Moderately</td>
<td>Moderately</td>
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<tr>
<td>QOLIBRI-OS Mood and emotions</td>
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<td>Quite**</td>
<td>Moderately</td>
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<tr>
<td>QOLIBRI-OS ability to carry out day to activities</td>
<td>Slightly</td>
<td>Moderately**</td>
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<tr>
<td>QOLIBRI-OS personal and social life</td>
<td>Moderately</td>
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<td>Moderately</td>
</tr>
<tr>
<td>QOLIBRI-OS current situation and future prospects</td>
<td>Slightly</td>
<td>Moderately**</td>
<td>Moderately**</td>
</tr>
<tr>
<td>QOLIBRI-OS Average score</td>
<td>2.3</td>
<td>3</td>
<td>3.2**</td>
</tr>
</tbody>
</table>

* QOLIBRI-OS scoring system: Not at all satisfied = 1, Slightly satisfied = 2, Moderately satisfied = 3, Quite satisfied = 4, Very satisfied = 5. ** indicates improvement in QOLIBRI score from baseline
Conclusions

- NMDAR encephalitis leaves patients with persistent and significant cognitive, behavioural and emotional problems, well after discharge from hospital.
- Long term management and input of varying levels of intensity from multi-disciplinary agencies is required.
- Communication lines between all professional working with these patients is vital as information about NMDAR needs to be consistent across all those involved in the patient’s care.
- Memory and executive deficits are persistent in the long term and severity of impairments show wide variability between patients.
- Each of these patients had markedly differing cognitive profiles suggesting that in the context of long term rehabilitation outcome, cognition may have less valence than emotional and behavioural factors.
- Poor sleep, fatigue and incontinence are persistent and greatly impact on rehabilitation.
- Infertility is a major issue. For cognitively able patients counselling around infertility should be integrated into treatment/rehabilitation pathways.
- Quality of life can be enhanced and maintained in the long despite deterioration in other aspects of community function suggesting that quality of life may be independent of function and overall mood and possibly an aspect of patients’ perception of self-awareness into their condition and recognition of some improvements in their daily life. This is an area we hope to explore further.
- These three cases highlight variability in terms of when and how long for intervention is required.
Why Do People With Epilepsy Die?

Introduction
Epilepsy is the most prevalent neurological condition with a prevalence rate around over 50 million people in the world. The mortality rate is about 2–3 times more when compared to general population. There have been 108 deaths from Epilepsy last year in UK. Mortality rates are more in men than women and in younger age groups. The co-morbid physical health problems, mental illness and learning difficulties also increase the risk of death in epilepsy. The aim of the study was to analyse the multifactorial causes of death in Epilepsy.

Methodology
Both cross sectional and longitudinal cohort studies identified by using the NHS Athens website and simultaneous search done on EMBASE, MEDLINE, PsychINFO and CINAHL to establish the causes of death in epilepsy. Limitation applied to the publication years from 2003–2013 and 158 papers selected and 5 papers with the primary data on causes of death in epilepsy chosen.

Studies
Study 1: MU et al. (2011) (1)
This prospective study took place in rural West China between May 2005 to December 2009. The follow up duration was 54 months and data analysed using various statistical tests.

Results: total deaths: 106 (male 70; female 46); commonest cause of death - accidental death (60%): drowning, fall from heights, suicide, road traffic accident. Commonest death in age group between 16–45 years (57/106 deaths); Highest accidental death by drowning (46/106 deaths: 32 male and 14 female); Next common cause of death is from SUDEP (sudden unexpected death) 15/106; Status epilepticus 7/106.

Study 2: Robert et al. (2013) (2)
This retrospective study in Norway examined 140 patients with chronic refractory epilepsy who died between 1965 and 1996 to examine the risk factors and their possible role in sudden unexpected death in epilepsy (SUDEP). Results: Out of a total 140 deaths, 42 were classified as

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Dr. Basavaraja Papanna1 & Dr Hugh Rickards 2

1 ST6 Registrar (MSc Clinical Neuropsychiatry) North Essex Partnership NHS University Foundation Trust.
Conclusion
From all the population studies it is clearly evident that the risk of premature death is 2–3 times higher in people with epilepsy than in the general population. The mortality risk is highest in the early years following diagnosis. Most common cause of death in epilepsy – accidental death (60%) as a consequence of seizures including drowning, fall from heights, suicide, road traffic accident, aspiration pneumonia. Next common cause of death is from SUDEP (sudden unexpected death in epilepsy). Few deaths were also related to subtherapeutic concentration of AEDs and their adverse reactions.

Hypothesis for further research
1. Need further study/research in deaths associated with AEDs (antiepileptic drugs) as the use of new AEDs in children is on the raise, the risk of mortality needs to be continually reassessed
2. Need long term prospective cohort studies to get most reliable means of examining the risk of premature deaths in epilepsy patients

REFERENCES
3. Gail S Bell. Suicide in people with epilepsy: How great is the risk? Epilepsia, 50(8): 1933-1942, 2009

SUDEP. The leading cause of death was pulmonary oedema (62%) and most cases are preceded by seizures.

Study 3: Gail et al. (2009) 
This study was aimed to estimate the suicide risk in people with epilepsy. This was done by using a meta-analysis of 74 (literature articles) cohorts of people with epilepsy in whom the number of deaths by suicide. Results: 190 observed deaths by suicide in most groups with chronic severe epilepsy, newly diagnosed and temporal lobe epilepsy/surgery.

Study 4: Bell et al. (2008) 
This study was aimed to quantify the risk of drowning in people with epilepsy. This was done by using a meta-analysis of 51 articles. Results: 88 deaths by drowning. The increased risk was in people with prevalent epilepsy, comorbid learning disability, in institutions, temporal lobe surgery. This study showed a 15–19 fold increased risk of death by drowning in people with epilepsy when compared with the general population.

Study 5: Ackers et al. (2011) 
In this retrospective cohort study, showed children prescribed a new AED were at increased risk of mortality for example; pancreatitis on sodium valproate, metabolic condition on topiramate in the months prior to death particularly in combination therapy.
Mitochondrial protein expression in white blood cells as a potential biomarker for Alzheimer’s disease

Dr. Manraj Bhamra and Dr. Joanna Riddoch-Contreras
Email: manraj.bhamra@nhs.net

Aim
To determine human mitochondrial protein expression in white blood cells (WBC), as a potential biomarker for Alzheimer’s disease (AD).

Alzheimer’s disease
- Progressive age-related neurodegenerative disorder and leading cause of dementia.
- Features: memory loss and cognitive decline.
- Pathological hallmarks: beta-amyloid plaques and neurofibrillary tau tangles.
- Affects ~34 million people worldwide [1], expected to quadruple by 2050 [2].
- Major public health concern, costing the UK economy £23 billion per year; greater than cancer and heart disease combined [3].
- This highlights the increasingly important need for early diagnosis and the development of disease modifying treatments.

Biomarkers
- Definitive diagnosis of AD still relies on post-mortem [4].
- Neurodegenerative and cognitive changes begin in patients years before clinical manifestations [5].
- The field is therefore in critical need of a reliable biomarker to aid in early diagnosis and identification of preclinical disease.

- Biomarker: a biological parameter that can be measured and used to assess normal biologic or pathogenic processes, or clinical responses to therapeutic interventions [6].
- Current biomarker approaches: neuroimaging and biochemical studies (cerebrospinal fluid, plasma, serum, urine, saliva) [7].
- Recent findings suggest a role for blood-based biomarkers as non-invasive and cost-effective tools [8].

Mitochondrial dysfunction
- Considered detrimental to cellular and metabolic homeostasis [9].
- Early pathological feature, particularly in neurons vulnerable to degeneration [10].
- Similar changes have been found in peripheral blood cells such as platelets and white blood cells [11].

Fig 1 Annual costs of major diseases to UK economy [3]
Gene expression analysis has revealed a significant down regulation of mitochondrial gene expression in white blood cells of mild cognitive impairment subjects (MCI, a prodromal stage of AD and AD patients compared to elderly controls.
- These changes are similar to those described in post mortem AD brain samples.

Methods
- White blood cells were extracted from 17 AD, 11 MCI and 20 healthy age matched elderly controls.
- Bead based multiplex panel assay simultaneously detected changes in mitochondrial protein complexes I–V and NNT in these patients using a 96 well plate format.
- Samples were analysed using the Luminex xMAP detection system.

Results
- We found a significant downregulation of complex I, II and IV protein expression in white blood cells in AD compared to controls.
- Most significant difference: decrease in complex II in MCI and AD (p<0.007) compared to controls.

Discussion
- Alterations in mitochondrial oxidative phosphorylation occur in AD, with evidence to suggest this also occurs early on in MCI.
- White blood cell mitochondrial protein expression is as a potential biomarker of AD, with diagnostic and prognostic value.
- Suggests the utility of a blood–based biomarker of AD.
- Further validation studies are required in a large population to improve the reliability of these results.

Further work
- These results facilitated a collaboration between KCL and Merck, USA.
- We further evaluated these findings and extended our analysis in white blood cells and post–mortem brain samples using a greater number of subjects, with the overall aim of developing a peripheral biomarker for early AD.
- We did not replicate the results in a larger cohort n=80 due to patient heterogeneity and using a multicentre cohort across Europe. The project needs larger sample numbers to overcome the variability in patients samples.
- Further investigations warranted to investigate the involvement of mitochondria as AD biomarkers.
References


The Man Whose Mind Exploded

This affectionate, unflinching gonzo documentary sees arts and travel presenter Toby Amies explore the experience of being with an eccentric, extraordinary man who describes his adaptation to anterograde amnesia as living “completely in the now”. In doing so, Amies is drawn into becoming a carer whilst delivering the film that the self-styled Drako Oho Zarhazar was seemingly destined to star in.

With his shaven head, waxed moustache, tattoos and piercings; this caped and croc wearing septuagenarian clearly has cinematic charisma that fills the screen.

However that only hints at what’s inside his cluttered, council flat in Brighton where much of the filming occurs. Here self-penned notes, old letters, photographs and homoerotic pornography dangle on countless strings creating a hectic, projected installation of his mind. With these hanging threads, ‘Drako’ remains connected to his past while Amies peers in wonder amongst the increasing debris and disorder.

It’s poignant that a younger Drako would have no shortage of material to recount from a colourful life that included associations with Salvador Dali. Unfortunately, following two life-threatening brain injuries that have shattered his memory, daily events...
can’t be recalled and only fragments of his past remain. Filmed interviews and interaction with his family demonstrate that the former hedonistic dancer can relate meaningfully with those from his past, while his sister observes that the ‘damaged’ Drako is even strangely likeable compared to how she recalls. Nevertheless, self-determination and risk remain personal characteristics that are as constant as his larger than life persona.

As interesting as the film makes as a case study of brain injury, Amies goes further by revealing the evolving relationship between the two men, even though one of them can never recall meeting the other. Drako’s inability to provide reliable consent also poses ethical dilemmas for the first-time director over when to challenge his stubborn ‘star’. These exchanges of respectful but exasperated concern will be familiar to many families and professionals who care for those with deteriorating health and faltering cognition.

“Trust. Absolute. Unconditional” declares Drako whilst sat exposed on Brighton’s pebbled, naturist beach. It’s one of many repeated phrases and recollections that preserve his identity and which he emphasises by pointing at the words that are permanently inked on his arm. Moments later that trust is memorably demonstrated when filmmaker leaves his camera and appears cheekily in frame to help his disrobed friend with rising to his feet.

The Man Whose Mind Exploded is available for digital download through its website, www.themanwhosemindexploded.com

Greg Neate – greg.neate@nhs.net
Toby Amies interview

Dr Greg Neate interviews Toby Amies, director of The Man Whose Mind Exploded, about how he documented the experience of being with Drako Oho Zahazar, a man whose anterograde amnesia subsequent to two traumatic brain injuries means he can never remember his personal champion. Here, Amies describes his approach to filming ‘Drako’, the ethical considerations involved and his thoughts following the film’s national release in June.

Following TMWME’s national release, what are your thoughts about the response to the film?
I am very, very happy with the way that the film has been received. We only had a very small budget to make and market the film but the reactions of the media and audiences has made sure that the film’s message has resonated much further than we expected. I very much doubt that there is another film that has received positive reviews in both the Lancet and Bizarre magazine!

Your approach to interacting with Drako reminds me of what’s been described as the ‘mentalizing stance’ (as described in mentalization-based treatment theory), where the interviewer takes a non-judgemental stance while remaining inquisitive about the individual’s thinking. Were you aware of that and how did you adapt your interview approach specifically to Drako?
I wasn’t aware of that approach and I’m not sure that I would describe my stance as non-judgemental! That said I would love and strive to be calmly objective in all the difficult situations I encounter. I think it’s probably fair to say that with Drako, I certainly didn’t think that his brain damage and condition made his point of view or experience any less valid than anyone else’s. Not having had any clinical or ethical training, I tend to enter and approach situations on the basis of what I ascertain to be fair. There were instances in my interactions with Drako where I felt that he was unfair to me and I document one of these in the film.

One of the guiding principles of the film was not to try and impose any kind of theories, narrative or worldview upon Drako but rather let him be the inspiration for the film’s form and structure and also the agent of his care and fate.

In the film his sister describes how she didn’t like Drako when they were younger though this changed after he adapted to his brain injuries. What would your views have been had you met the younger Drako?
The more time I spend (and waste) on social media the more I grow to dislike narcissists. From what I know of Drako before his accidents, he seemed to be very self-involved. Whilst I might have appreciated his beauty and fearless pursuit of physical pleasure, I’m not sure that we would have got on as individuals.
If you had the opportunity, what else would you have liked to have included in the film?

Less! It’s probably more a question of showing less than I did as there are still shots in the film that I think we could have done without and still had the same impact.

When I watch the film now, I see a series of mistakes and compromises and shoddy camera work. But now that it’s out there, all that really matters to me is how it affects the audience and how they have been moved by Drako’s story.

Some people have said that they would’ve liked to have known more about Drako’s biography but one of the things that we noticed during editing was that those historical sequences were a rather flat experience. One of my favourite things about the film is that so much of it is in the moment - that we are experiencing being with Drako - not just looking at flat pictures and hearing a story. Instead and appropriately, we’re “being in the now”, which was Drako’s dominant experience of life.

You said at one of the film screenings that his most creative phase was possibly during his final years. How would you describe what that involved?

I don’t think I investigated Drako’s creative process perhaps as deeply as I could have. However, it seemed to me that without [Drako] being overly self-conscious or aware of it, that he was involved in creating a tangible version of his identity through an intuitive process of montage in two and three dimensions within his flat. Within the context of this work, Drako expressed his obsessions, spoke to himself and through the visitors to his flat, to the outside world.

After his death I archived as much of this work as possible because I think it’s an important piece of British outsider art and one day I hope to rebuild part of it as an installation.

Clearly there were considerable ethical concerns of filming someone with questionable capacity. What has been the most justified criticism?

I suppose the one that’s implicit in your question; should I have been working with someone who, to use your terms, had ‘questionable capacity’? I am not in any sense a medical professional, so in working with Drako I first, and consistently ensured that I had the permission and blessing of the people who were closest to him. In addition to his own point of view, I thought that they were in the best position to decide what was “best for him”.

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After his death I archived as much of this work as possible because I think it’s an important piece of British outsider art and one day I hope to rebuild part of it as an installation. It’s significant that Drako trained as an interior designer and without wanting to be glib, it’s tempting to think of him as an outsider interior designer!
Interview with Michael Koperman

Norman Poole
Consultant Liaison Psychiatrist, Royal London Hospital

On a wintery Friday evening last November, I met Professor Michael Kopelman at his office at St Thomas’ Hospital, with its spectacular view across the Thames to the Palace of Westminster. So close are the two Houses that, hearing Big Ben’s chimes, I surreptitiously scanned the room for a radio, assuming Radio 4’s Six O’Clock News had just come on, only to realise the chimes were live. This setting felt apt, for few psychiatrists have done more to reveal the workings of the palace of memory while simultaneously challenging the lawmakers. A glass of red wine helped to put me at ease in the great man’s company, and then I pressed record...

First question, why did you chose medicine as a career?
My first-degree was in psychology and I came from a medical family. If I hadn’t, I probably would have ended up in research or clinical psychology but there was an encouragement that the best way of doing it was medicine, and so I went into medicine intending either to be a neurologist or psychiatrist. I knew that. When people used to ask what do you want to do, I would reply “Caputology! Something to do with the head.”

So what was it about Caputology that so interested you?
I had gone into psychology and enjoyed it, and before that — oh I hate to say the cliché — I was interested in people, and enjoyed literature from the psychological angle. So this is what I was curious about. I’d been interested in the neuropsychology of memory, which was just developing at the end of the 60s and 70s. I was reading quite a lot of the amnesia stuff at an early stage and I was interested in the more biological aspects of psychology. I knew I would either do neurology with an interest in cognitive neurology or psychiatry with an interest in neuropsychiatry, and I’ve ended up in the middle discipline.

Who were you reading at that time?
I read quite a lot of Freud as a psychology student. I read Pribram’s book on Freud’s Scientific Project and was interested in ideas about how Freudian concepts relate to contemporary neuropsychological findings and concepts. There’s been a revival of interest in that topic in recent years with Mark Solms and the neuro–psychoanalytic approach. I think that the present move to suggest a lot of neuropsychology can be translated into Freudian concepts, or Freudian concepts translated into neuropsychology, rather overstates the case.
What made you opt for the psychiatry route rather than neurology?
I was an undergraduate student for 10 years and so partly it was that the psychiatry route was quicker to get to my particular goal, and partly because in those days there were not very many cognitive neurologists. It was a substantial risk that, having done a lot of guts and urine medicine, I would then do neurology and find I was with a peripheral neuropathy specialist and I might never get to my chosen area of interest. Whereas I could go to the Maudsley and people interested in neuropsychiatry would be there more or less straightaway. Also, it struck me that schizophrenia and bipolar disorder were a lot more interesting than the bread-and-butter neurology of peripheral neuropathy and muscle diseases.

When I got to the Maudsley I really did think – and it’s sad in a way how much it has changed – that this is the place for me. There were psychiatrists, a neurology contingent under David Marsden, and psychology with Hans Eysenck, who people had mixed feelings about but he was certainly one of the two big psychology names in Britain at the time. There was a little bit of biochemistry but not the huge masses of people doing genetics [that there are now].

What distinguishes Neuropsychiatry from other psychiatric specialities?
It isn’t as if we have some unique particular skill, like being able to put an endoscope in some way into the brain. We perhaps have some skill in seeing things from different perspectives, and trying to take account of different views of the world. I get a bit anxious about what I call naïve reductionism, the kind of approach that thinks that all PTSD and depression are just disorders of the hippocampi or the frontal lobes or whatever. I think that is very simplistic.

Our society, in my view, is becoming more and more authoritarian. It’s very worrying the direction we are moving in

Did you read any neuropsychologists at that time?
I read Luria quite early on, when I should have been reading anatomy textbooks. And there were big debates between Elizabeth Warrington and Larry Weisicrantz on this side of the Atlantic, and Butters and Cermakon on the other. Later, Larry Squire came on the scene and Huppeert & Peircy were another very important team. They had furious rows. They were the only people in the world looking at the neuropsychology of memory, and they all had different theories and got very angry with one another for not agreeing with their own respective theory. But the ideas were fundamental, fascinating, and in fact they’ve never properly been resolved.

Could you summarise the transatlantic disagreement?
Some of some of it was rather pathetic. It was all about the so-called Brown-Peterson test: whether for the first 15 seconds to 1 minute after learning something people with an amnesic disorder forget exceptionally fast, or whether they are actually normal during that duration and the forgetting comes later. That’s what got people very heated. What’s not been resolved yet is: what is the fundamental deficit that gives rise to an amnesic syndrome? That to me is at the heart of amnesia research.

So we are synthesisers of different fields of knowledge?
There are disorders that we deal with — amnesia is one — where you can have a brain lesion that gives rise to a specific pattern of psychological deficit. But we have to remember the brain is operating within a social context and not to underplay the importance of our social context. So I suppose that is saying we are synthesising, but if synthesising means just reducing things to brain function, then I’m a bit wary of it.
Do you see the widespread adoption of dementia diagnostic clinics as vindication of your chosen career?
No, quite the contrary. The memory clinics that are being set up under the Dementia Strategy are not what I would advocate. There is a wide range of memory disorders to be diagnosed, and the early diagnosis of dementia, even by the very experienced, is difficult. There are very sad implications if you get it wrong. The earlier we go for diagnosis the more likely we are to get it wrong, whatever the clinical/ genetic/ biomarker tools that we have.

I think, and I’m not popular for saying this, that nurse-led diagnostic teams making early diagnosis is not the direction to go in. The direction to go is better care for those with established diagnoses of dementia. What we have at present is shameful, and in my view it’s actually now worse than in the 1980s. I think we have gone in the wrong direction in dementia care and resourcing, and the whole idea of paying GPs £55 to make a diagnosis is absolutely disgraceful.

Have you seen the film The Man Whose Mind Exploded, reviewed on pXX in this issue?
I contributed to a discussion at the ICA on that film. I have to say, I had slight misgivings about going but I thoroughly enjoyed the discussion; it was a good intelligent discussion although it did polarise people. The man certainly had frontal lobe damage as well as some memory impairment. So there was a heated debate about whether he was able to give consent, and whether it was ethical to film him. One person in the audience felt very strongly that he wasn’t in a position to give consent. I thought that they had done it is as tastefully as you probably could in the circumstances, but I am a bit ambivalent about that kind of film.

You are renowned for your forensic work. What has been your motivation to become involved in that?
I was interested in forensic psychiatry while I was training. Although I was a neuropsychiatrist working in Alwyn Lishman’s team, when I was a lecturer at the IOP I did clinics with Paul Bowden and also John Gunn, and I was friendly with and wrote a paper with Pamela Taylor. I was also friendly with Jim MacKeith and Paul Mullen, before he went to Australia. So I always had a forensic interest. What I didn’t know at that stage is how much criminal and appeal court work hangs on neuropsychiatric issues. Peter Fenwick was an example of someone who, as a neuropsychiatrist, did a lot of very good forensic work, and he would report these fascinating cases at case conferences. Most neuropsychiatrists just do civil cases, which I find terribly boring by comparison.

It was Jim MacKeith, more than anyone, who encouraged me in forensic work, and I just started to get bigger and bigger cases. I have been extremely lucky because compared with the average forensic psychiatrist I have seen a very high proportion of very high-profile cases. Through Jim and Gisli Gudjonsson, I got into false confession cases, which I see as a form of memory disorder, and was involved in overturning convictions; one from 50 years ago, another after 25 years, and a delusional memory case at 26 years.

Have these cases of wrongful conviction made you interested in jurisprudence more generally?
Through getting to know Gareth Peirce, whom I very much admire, I got involved in human rights issues, which are not necessarily neuropsychiatric at all. I was seeing people who were being held as Belmarsh detainees and was involved in a report, with Ian Robbins and Jim MacKeith as senior authors, that contributed to the House of Lords’ 2004 ruling making indefinite detention without trial illegal.

Then with two others, a therapist and a GP, I wrote a report in 2010 on people who had come back from Guantánamo, including some prominent names, and this resulted in them getting substantial amounts of compensation. Ken Clarke announcing this in Parliament made the somewhat ambiguous statement that “We must never let this happen again.” I feel in some ways I have done more good from this sort of work than anything else, and that’s what I’m going to do in my retirement.

Gareth Peirce is superb at using the legal rules to beat authoritarians and government. She plays within the system, but she does it better than the government lawyers and beats them. That fits my temperament. Not shouting or protesting on the streets, but playing the system to get justice for people.
I think looking for the brain lesion or anomaly in schizophrenia is a bit like looking for a needle in a haystack. But in neurological amnesia you’ve got fairly discrete lesion and a fairly specific disorder of function. But the question of relating one to the other is actually much more complicated than it looks.

Now it’s really the chief executive and his executive team who run things. With no disrespect to any particular individual, some of whom are very good, I can’t see that they have proper accountability. We doctors have accountability. We can be hauled before the GMC. But managers and commissioners, who are changing the healthcare environment, often for the worse, are not held to account in the same way. These are matters I feel strongly about.

Being as you are on the verge of retirement from the NHS, how would you summarise your career?

I have been interested in the range of memory disorders, from the amnesic syndrome to confabulation to false confession to psychogenic amnesia and how these happen to matter in the law courts. In a sense, my career has been working as a clinical neuropsychiatrist and a research neuropsychologist and so in some sense I got the best of both worlds.

I tried to pull together proper clinical neuropsychiatric assessment of patients with proper neuropsychology – to some degree theoretically driven, although I’m not a big theorist – or what some people call ‘cognitive neuropsychology’. That doesn’t always happen. Neuropsychiatrists often do relatively lowbrow neuropsychology, and neuropsychologists are often rather ignorant about the clinical context. I have tried to bring these two disciplines together.

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Having been a bit ambivalent about going into medicine – whether I was setting myself off on an unnecessarily long route – I think it was well worth it in the end because I can see things from the different perspectives. And I’ve always enjoyed dabbling.
The Brain and Behaviour: Forthcoming National Neuropsychiatry Conference

Thursday 12th March 2015 at Stoke on Trent Moat House

Research tells us that behavioural changes, resulting from brain damage, is one of the most significant factors in compromising community treatment.

Following on from our successful national conferences over recent years, our forthcoming event will focus on how various brain disorders can bring about a distinct set of behavioural, cognitive and emotional changes. The conference will explore the challenges around clinical management, ethical and medico-legal dilemmas in relation to brain diseases and injury. How those issues can and perhaps should shape our service developments will also be explored.

Despite the specialist aspects of various brain conditions, this educational event will also aim at targeting generalists who are most in contact with patients with this kind of disorder.

The event will be offering expert, evidence-based management tips in relation to acquired brain injury, degenerative brain conditions, sleep disorders, epilepsy and movement disorders. The interaction between brain disease / injury and the law, and how experts could best support the legal system will also be specifically addressed.

Our host of eminent national speakers will share their academic and clinical experience with the audience in order to ultimately contribute to an ever improving patient’s care. Similarly, patients, carers and support groups will be contributing to this event to ensure that their voice is heard by scientists, researchers and clinicians. This should not only inform future clinical care but also general issues around service development and delivery.

Conference audience will hear from a senior psychiatrist who survived a significant brain injury but still has to live with specific Neuropsychiatric consequences. Similarly, a Mental Health manager will tell her story of looking after her deceased husband who suffered from an aggressive brain disease.

Our patients, who have already designed the Conference Logo, have been particularly supportive of this initiative and their role is highly regarded by the organising team.

For further information on the conference please visit: www.kc-jones.co.uk/brain or contact: info@kc-jones.co.uk or event queries hotline: 01332 224501

Brain and Behaviour

Striving to complete the picture