ISSN 1473-9348 VOLUME 16 ISSUE 5 MAY-JULY 2017

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DATE OF PREPARATION: July 2015 JOB NO: REB15-0054

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Date of Prep: May 2017 | UK&IE/REB/0517/0031

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MAY-JULY 2017

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REGULARS

ACNR
Published by Whitehouse Publishing,
1 The Lynch, Mere, Wiltshire, BA12 6DQ.
Publisher. Rachael Hansford
E. rachael@acnr.co.uk

PUBLISHER AND ADVERTISING
Rachael Hansford,
T. 01747 860168
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PRINTED BY Warners Midland PLC T. 01778 391000

ACNR's paper copy is published quarterly, with Online First content and additional email updates.
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Cover image **An artist participating in the 'vs.ms interpreted project'**

Shift.ms – support for people with MS

Shift.ms is a charity and online social network for people with Multiple Sclerosis. The organisation equips people to proactively manage their MS, and aims to do this as soon as possible after diagnosis.

Alongside the social network, which offers support from people who understand what it's like to live with MS, Shift.ms produce films and creative projects – by, for and about people with MS.

'vs.MS' is one such project – Shift.ms worked with Sanofi Genzyme to interpret data gathered from the MS community. Shift.ms commissioned nine artists, all of whom have MS, to reflect on these statistics and offer a human insight to the data collected.

The resulting artworks are inspirational without focusing solely on the positive; they show the whole journey of MS and are relatable because they deal with everyday topics such as work and relationships.

These artworks are a reflection of the Shift.ms community's belief that MS doesn't mean giving up on ambitions, just rethinking how to achieve them. – *www.shift.ms*

Todd Hardy, Co-Editor.

Welcome to the latest issue of ACNR. In a time when there is increasing research and media interest in the consequences of traumatic brain injury among amateur and professional sports people, Clare Fraser and Adrian Cohen from Sydney write about the effect of concussion on the visual pathways, concentrating on sideline testing and laboratory measures to identify deficits when assessing a concussed patient.

Also in this issue, Iulia Monica Bogdan, Mara Cercignani and Waqar Rashid from Brighton discuss the mechanisms which may underlie the important and debilitating problem of multiple sclerosis-related fatigue, and cover current treatment options.

Kim van Dun, Florian Bodranghien, Peter Marien and Mario Manto from Brussels explore whether the technique of transcranial magnetic stimulation of the cerebellum may have potential benefits in rehabilitation, and about how this could be potentially achieved, citing relevant cerebello-cerebral anatomical pathways.

Nick Losseff from London dissects the problems by which patients with neurological conditions access and receive care on a strategic level in the UK, and importantly looks ahead to how solutions might be implemented.

John Pearce from Hull writes an historical piece outlining how our modern recognition of the anatomy of the cranial nerves has evolved since ancient times, while Andrew Larner from Liverpool shows, in his continuing series, how neurophysiological techniques have been portrayed by various writers with selections from their fiction and poetry.

Online readers are encouraged to view a short film of ACNR co-editor Mike Zandi talking to Andrew Lees from London about his new book "Mentored by a Madman" at http://www.acnr.co.uk/2017/04/documentary-andrew-lees-mentored-by-a-madman/. This wide-ranging interview, conducted at Queen Square, covers Andrew Lees' life and career, the influence of writers William Burroughs and Arthur Conan Doyle on Lees, psychotropic drugs, various luminaries of British neurology, and the history of Parkinson's disease.

Conference reports are from Boyd Ghosh on the BNPA, Stella Hughes on "An unexpected evening with Roald Dahl's doctor", Oliver Cousins on the 11th Cambridge Dementia Course, and Joanna Pleming on the UCL Stroke Advanced Neuroimaging day. Our book review is from Rhys Davies.

Finally, I would like to formally thank ACNR Co-Editor Sian Alexander on behalf of the ACNR Editorial team for her great work on ACNR over the last two-and-a-half years. Sian is stepping down as Co-Editor, but the good news is that Ann Donnelly will be stepping in to take her place. Ann is a Senior Neurology SpR in London with prior editorial experience in roles at the ABN, BMJ and BioMed Central.

We hope you enjoy this edition of ACNR.

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Visual testing in concussion

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Conflict of Interest statement: None declared.

Provenance and peer review: Commisioned and externally reviewed.

Date first submitted: 5/7/16
Date resubmitted after peer review: 8/3/17
Acceptance date: 9/3/17

To cite: Fraser CL, Cohen A. ACNR 2017;16(5);5-7.

Key Points

- There is no single diagnostic test or diagnostic criteria for concussion
- Afferent and efferent visual pathways account for over 50% of brain circuitry and are particularly vulnerable to shear injury from head trauma
- A head injury is likely to result in changes within the visual pathways providing a potential target for objective reproducible tests of concussion
- The King-Devick test of ocular saccades is the best studied of all visual tests and has been shown to be valuable in assessment of concussion across many age groups and sports

Abstract
Concussion is a common neurological injury in both amateur and professional sporting codes. Given that the visual pathways traverse a large proportion of the brain, tests of visual function can be useful means of detecting concussion. Some tests can be performed on the sporting sidelines, and others require a dedicated clinical setting. Visual symptoms of concussion are amenable to rehabilitation therapy.

Introduction

Mild traumatic brain injuries (mTBI) are very common, with an estimated 3.8 million cases occurring each year in the United States.[1] However, there are no set criteria for the diagnosis of concussion or post-concussion syndrome. The 2012 Consensus Statement of Concussion in Sport[2] defines concussion as a complex pathophysiological process affecting the brain, induced by biomechanical forces. In particular, the statement separates mTBI from concussion, though the terms are often used interchangeably in the literature. In traumatic brain injury, even if mild, there is some form of intracranial trauma, which can be demonstrated on neuro-imaging.[3] However, several common features can be used to define a concussive head injury; an "impulsive" force transmitted to the head, rapid onset of short-lived impairment of neurological function, acute clinical symptoms largely reflect a functional disturbance rather than structural injury and concussion results in a graded set of clinical symptoms with or without loss of consciousness.[2] By definition, standard neuro-imaging studies are normal in the setting of simple concussion.[3]

Concussion causes a functional disruption of the brain resulting in cognitive, somatic and emotional symptoms, with significant variability between patients. The visual system (afferent and efferent) pathways account for over 50% of the brain's circuits, and are in areas particularly vulnerable to shear-injuries from a head blow.[4] Therefore, in a diffuse brain injury like concussion, there is a strong possibility that there will be some disturbance in these pathways. Vestibulo-ocular symptoms of dizziness, blurred vision and trouble focusing are a well-recognised subset of concussion symptoms.[4,5]

Much research has focused on visual testing as a key part of the paradigm for concussion assessment and treatment. Broadly speaking, there are tests which can be performed at the sideline, and tests that require evaluation in a clinic setting. Vision testing falls into both categories.

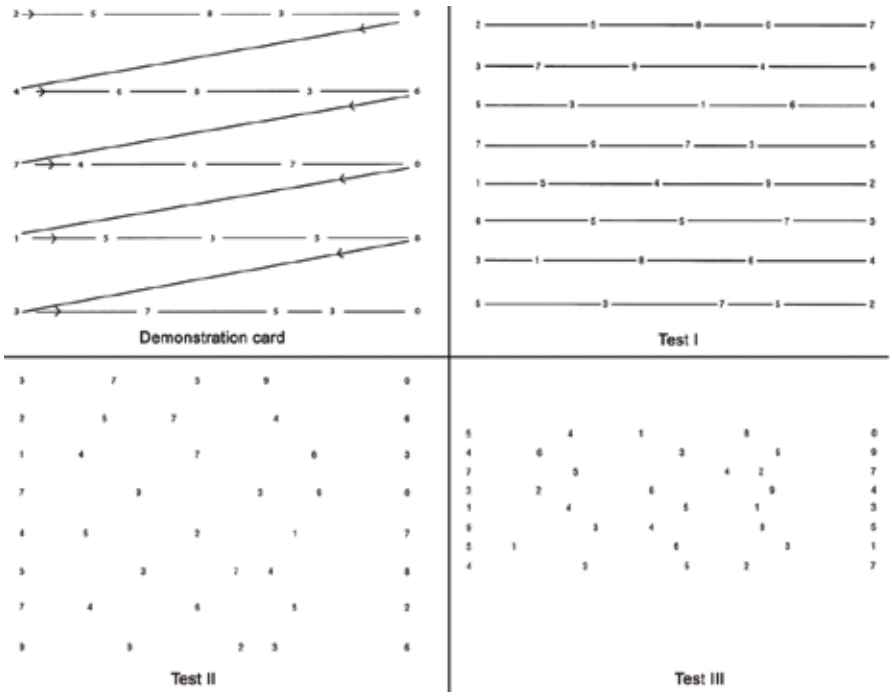
Sideline assessment

Symptom checklists

There is no single gold-standard test for sideline assessment. Symptom checklists include the Rivermead Post Concussion Symptoms questionnaire (RPQ).[6] Among the 16 questions, the RPQ assesses for headache, dizziness, nausea, sleep disturbance, mood and memory changes. From a visual perspective patients are asked about blurred vision, double vision and sensitivity to light. Patients rate the severity of each symptom over 24 hours on a scale of 0-4 compared to pre-injury symptoms. Other tests including Post Concussion Symptom Scale and the Acute Concussion Evaluation have one question for "vision problems" and another for "light sensitivity" scored via a numerical scale.[4] The Sport Concussion Assessment Tool number 3 (SCAT3), recommended as part of the Consensus Statement of Concussion in Sport also contains a symptom evaluation for blurred vision and light sensitivity.[2] However, these are subjective, and studies have shown that athletes who do not report any symptoms (for a variety of reasons), may show objective cognitive changes.[7]

King-Devick testing

The King–Devick test (KDT) was developed as an indicator of saccadic performance as it relates to reading ability.[4] The KDT requires a player to read a series of single digit numbers displayed in the form of a "card" on a 10.1inch android tablet, on a full-sized iPad or from a standardised cardboard flipchart, taking 1-2 minutes on average. The test cards become progressively more difficult to read due to variability of spacing between the numbers. The time to read each card is recorded, and a "total time" is calculated. The total errors in reading the numbers is also recorded. A baseline test score for "total time" is recorded as the fastest time without errors



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that a player can read all three cards. After a potential injury either an increase in the errors made while reading or a slowing of reading speed are included in deciding if a player fails the test. Therefore, this is not just a test of saccadic eye movements, but also attention and language. The downside of the KDT is that it relies on the players having a baseline test score documented.

The KDT has been validated for use as a concussion screening tool. In MMA fighters post-fight KDT scores were significantly worse (higher) than pre-fight scores for participants who had sustained head trauma during the match.[8] Other studies have shown the ability of the KDT to detect concussion in hockey, lacrosse, football, basketball and rugby, with a sensitivity of 86% and a specificity of 90%. On average the time to perform the saccadic tests increased by 4.8 seconds from baseline in concussion.[9]

In our own practice we have found the KDT to be a useful test to detect functional change in rugby players with documented head injuries. Furthermore, we found that stunt performers showed a significant change in KDT scores after falls from heights of 4 meters (in press).

Eye movement abnormalities

Cranial nerve palsies would not be expected in the setting of concussion or mTBI. An acute third, fourth or sixth nerve palsy would suggest either an intracranial haemorrhage or a pre-existing space occupying lesion. However, convergence insufficiency is well recognised following mTBI, with one study reporting 42% of athletes showing abnormal convergence one month after concussion.[10] The convergence or near triad is triggered by retinal disparity, and produces bilateral adduction of the eyes, miosis and accommodation of the crystalline lens. Therefore, it involves both afferent and efferent pathways. Convergence is tested, by measuring the "near-point" or the closest point to the face at which the patient can maintain binocular fusion. A change in the near point of convergence can be tested at the sidelines and compared to a baseline measure, but is not typically done until the player is back in a clinic setting.

Laboratory based tests

Visual tracking tests

Predictive visual tracking requires cerebellar coordination based on retinal inputs as well as higher visual processing, attention and working memory. Changes in visual tracking have been reported in patients with mild TBI.[11] Mild TBI patients displayed impaired target prediction with increases in eye position error. A circular tracking test, such as that used in the Sync Think (Boston, USA), has been shown to be a robust test to distinguish mild TBI from control patients.[12] However, other available devices such as RightEye (Maryland, USA) use a combination of circular, vertical and horizontal smooth pursuits and saccades in the testing protocols.

Visual evoked potentials

Visual evoked potential (VEP) are derived from changes in the electroencephalogram (EEG) measured over the occipital lobes in response to a visual stimulus. The waveform of the signal recorded can be quantified in terms of the size of the signal (amplitude) and the timing of the signal conduction along the visual pathways relative to the visual stimulus (latency). Latency changes have been shown to be significantly increased in those with mTBI compared with controls.[13] Further studies on VEPs have shown change in alpha rhythm attenuation in patients with mTBI and subjective attention deficits.[14] Further work is required to see if intra-subject changes in latency can be used to objectively detect concussion.

Optical coherence tomography

Mouse models of repetitive mTBI have shown a detectable thinning of the inner retina on spectral domain optical coherence tomography (OCT).[15] There are no human studies published to date replicating these changes, but OCT may prove to be an in-vivo measure of cumulative concussive damage.

Treatment

Photophobia and visual discomfort when reading have been reported by mTBI patients. Using the Intuitive Colorimeter System, subjective improvements in visual comfort levels were found in 11/12 patients while wearing tinted lenses, however objective tests of reading parameters and VEP latency were not significantly altered.[16] Authors suggest

these lenses could be used as an adjunct in managing post-concussion photosensitivity.

Convergence insufficiency has been shown to be more common in athletes with higher scores on symptom scales.[10] Treatment aimed at retraining convergence efforts and specialised reading exercises may therefore improve symptomatic issues (blurred vision, double vision) after concussion. The King-Devick Reading Acceleration Program is one example of a commercially available treatment protocol. Vestibular rehabilitation may also assist in improving visual function, by improving the vestibulo-ocular reflex and depth perception.[4,5]

Conclusion

Concussion events occur with contact sports and may lead to subtle but cumulative damage in brain morphology and function.[3] Visually-based concussion tests are of value on the sidelines and in the clinic setting. However further research is still needed. Vision-based tests may prove to be the most reliable, portable and easiest to implement in our schools and in our amateur sporting teams. In the long-term these tests may be able to guide rehabilitation programmes, and allow us to assess when it is safe for a player to return to school, training, work and to the competition.

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Fatigue in multiple sclerosis: Why is it so difficult to manage?

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Conflict of interest statement: None declared.

Provenance and peer review: Submitted and externally reviewed.

Date first submitted: 15/8/16
Date submitted after peer review: 28/2/17
Acceptance date: 1/3/17

Acknowledgements: The authors would like to thank Mr Mihai Robert Danciut FRCS(SN) for contributing the drawing in Figure 2.

To cite: Bogdan IM, Cercignani M, Rashid W. ACNR 2017;16(5);8-11.

Key Points

- Fatigue is a common and significantly disabling symptom in MS and its aetiology is poorly understood
- Pharmaceutical trials so far have not had a strong hypothesis in terms of underlying mechanisms. Consequently, the results are disappointing, Amantadine being the only medication currently recommended by the NICE guidelines
- Emerging theoretical research based on homeostatic and interoceptive circuits and potentially supported by neuroimaging may improve our understanding and help develop therapies

Introduction

This review aims to inform clinicians about the current strategies in managing fatigue in multiple sclerosis (MS) and the challenges which arise from the insufficient understanding of the underlying mechanisms. It gives an overview of the research undergone to find symptomatic treatment, the non-pharmacological approaches and the scarce knowledge about the impact of disease modifying treatment on fatigue in MS. Finally, it presents a few recent hypotheses and underlines the importance of finding a framework which brings together knowledge in the field.

Background

Fatigue is a common symptom for people with multiple sclerosis (MS), affecting between 65 and 97%,[1] independent of physical disability.[2] It has a high impact on the quality of life (QoL) affecting productivity and employment.[3] However, the causes remain elusive. Issues include the difficulty in fully defining the term and the varied additional factors that can influence it. A consistent measure of fatigue in MS is difficult because there are many types of fatigue, many confounding factors and some of the disease modifying treatments might influence fatigue levels.

Fatigue is a subjective feeling; therefore, its self-reported nature makes any conclusion based on such results subjective and possibly less reliable. Patients, physicians and researchers use distinct terminologies. There is no clear definition in the literature. To serve accuracy, different types of fatigue in MS have been proposed (Figure 1).

The search for symptomatic medications in MS fatigue

Starting in the 1980s, there have been several trials[4] trying to find an efficient treatment for MS fatigue. As the mechanisms underlying it are so poorly understood, there was no clear hypothesis. Only one drug, Amantadine (found to have low to moderate benefit) is recommended by the NICE guidelines.[5,6,7] The mechanism by which it alleviates MS fatigue is not entirely clear, but might involve its dopaminergic action.[8]

After conflicting results in research studies,[9,10,11,12,13] Modafinil is not currently recommended for the treatment of MS fatigue.

Modafinil is licenced in the UK for treatment of narcolepsy with or without cataplexy. In 2011, the European Medicines Agency's (EMA) Committee for Medicinal Products for Human Use (CHMP) evaluated benefits and risks of using Modafinil. In addition, risks associated with Modafinil (including psychiatric disorders, cardiovascular symptoms, and serious skin and multi-organ hypersensitivity reactions) led NICE to conclude that it was not recommended for the treatment of fatigue associated with MS.[5]

Other medications were studied, with negative results, including: Pemoline, 4-aminopyridine (not effective in cognitive fatigue), L-carnitine and paroxetine.[4] Further, the NICE guidelines specifically advise not to give medications such as vitamin B12 intramuscular injections, which have been used in the past, as it was not felt to have a sufficient evidence base.[5]

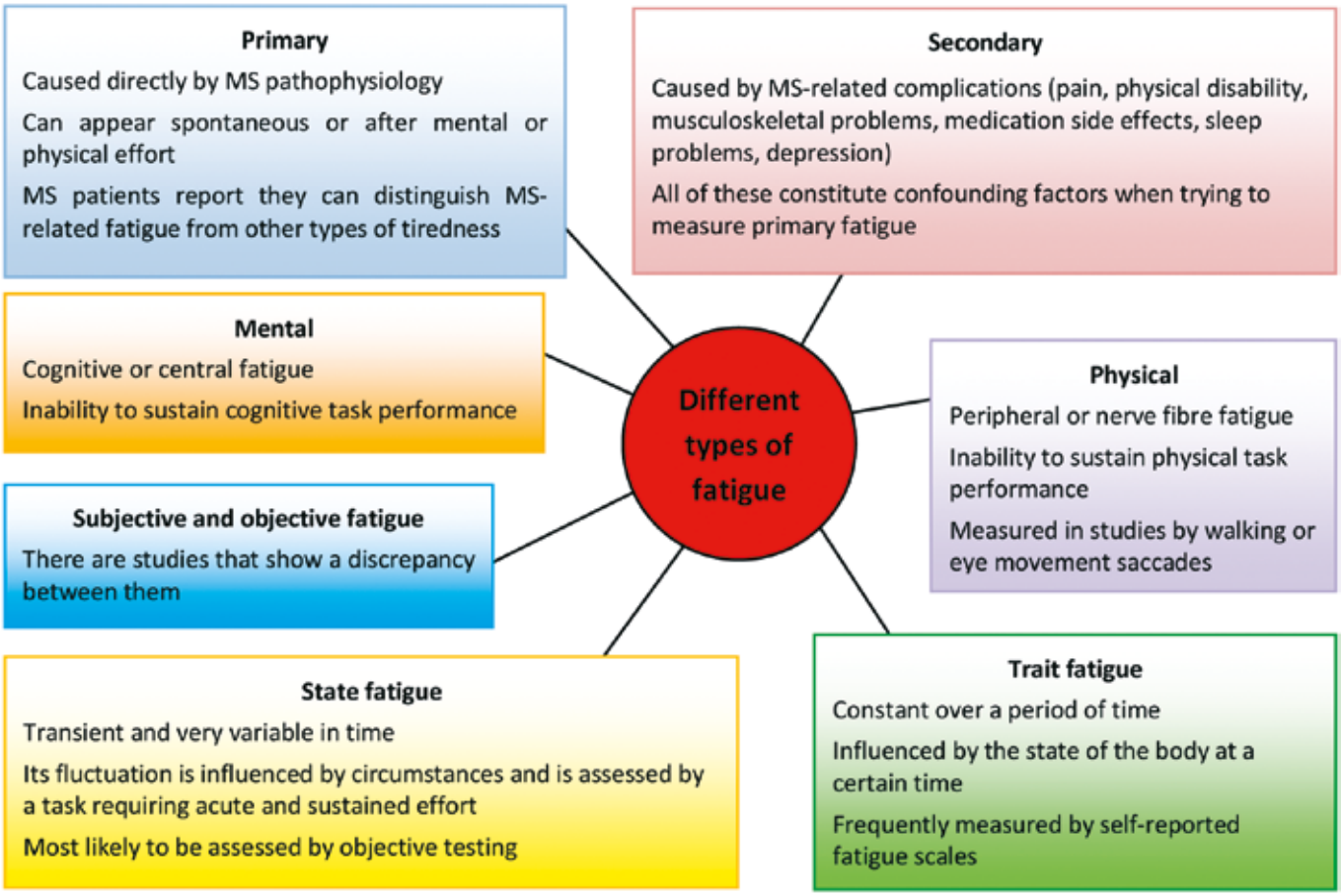
The search for non-pharmacological interventions for MS fatigue

A few non-pharmacological approaches have been shown to be effective and are recommended by the current NICE guidelines, such as mindfulness-based training, cognitive-behavioural therapy, fatigue management interventions and physical exercise (aerobic, balance, stretching, including yoga).[5]

The study with the best methodological quality on mindfulness-based training for MS fatigue was done by Grossman and colleagues.[14] Fatigue was found to be significantly reduced post-intervention.[14]

Probably the most well-known community intervention is the FACETS (Fatigue: Applying Cognitive Behavioural and Energy effectiveness

Figure 1



Techniques to lifeStyle) intervention.[15] It has been tested in a randomised controlled trial comparing with standard care and it encompassed: education about fatigue in MS, structured resting, pacing, breathing exercises, setting goals and challenging negative thoughts.[16] 164 patients were randomised and primary outcome data was available for 146. A significant benefit was found after six weekly sessions of 90 minutes. The benefit was still present at one and four months and one year post-intervention.

The role of exercise in alleviating fatigue in MS has been studied, with conflicting results. A recent Cochrane review concluded that exercise has some benefit in MS fatigue[17] although we should be aware of the methodological flaws of the existent studies so far.

Although the results of these interventions are encouraging, the main concern is the lack of an active control group and the difficulty in blinding for these sorts of trials. Therefore, the benefit might be related to the attention and input received from professionals, rather than attributed to the interventions *per se*.

The influence of disease modifying treatment on fatigue

As there have not been randomised controlled trials looking into fatigue as a primary outcome, there is no clear consensus. The subjectivity of measurement is also a possible limitation.

It is well known that MS patients treated with interferon-beta can experience fatigue as a side effect.[18] However, interferon-beta has also been reported to have slow to moderate efficacy in alleviating fatigue in MS.[19,20] Glatiramer acetate was also reported to reduce fatigue in MS.[21] There have been some reports on the superiority of glatiramer acetate over interferon-beta in improving MS fatigue.[22,23]

There has been some evidence of natalizumab benefit on fatigue.[24,25,26] A cross-sectional case-control study found natalizumab more efficient in reducing fatigue comparing to interferon-beta and glatiramer acetate.[27] There is however a small study which found no changes with natalizumab in fatigue levels in MS patients.[28]

There is no evidence for teriflunomide[29] or dimethyl-fumarate to improve MS fatigue.[30] There is some limited evidence for effectiveness of fingolimod in lowering MS fatigue.[31] Also, switching from interferon-beta (but not glatiramer acetate) to fingolimod might reduce fatigue.[32] A Cochrane review of alemtuzumab in MS did not find any studies looking into its effects on fatigue.[33] There is a need for the available disease modifying treatments to be assessed, from the fatigue point of view.

Additional studies are needed, particularly as the number of disease modifying therapies used in MS has increased, to assess if fatigue can improve with immunotherapies used in the disease. In addition, the apparent difficulty in understanding of the mechanisms underlying MS fatigue has contributed to the conflicting results seen. Future research needs to propose clear and testable hypotheses. It is likely that only with a robust hypothesis, will we be able to develop more effective treatment.

Approaches to understand MS fatigue

These challenges have encouraged research about fatigue in MS from different perspectives. Firstly, the molecular aspects were studied. A relationship was found between elevated blood pro-inflammatory cytokines and increased feelings of fatigue in MS. These results suggested that fatigue might be a form of sickness behaviour.[1] However, the studies of inflammatory markers from the blood provided limited information because, due to the existence of the blood-brain barrier, markers in the blood do not necessarily reflect the presence of cytokines in the brain.

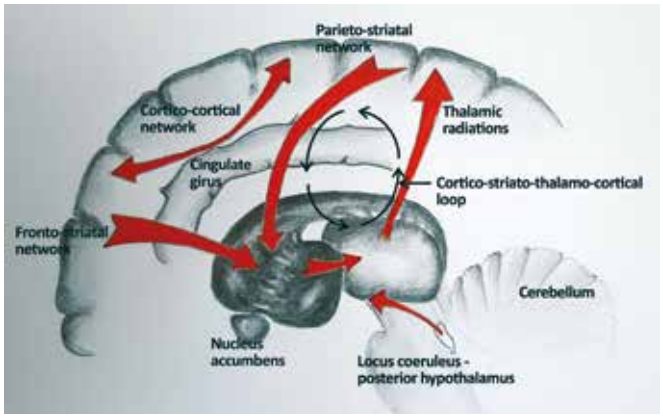
In order to get a better understanding of the exact processes, structural and functional neuroimaging studies were performed. A series of brain areas were found to be statistically correlated to MS fatigue:[34] fronto-striatal network, parieto-striatal network, deep grey matter, cortico-cortical networks, cortico-striato-thalamo-cortical loop, fibres connecting locus coeruleus and posterior hypothalamus, cerebellum, cingulate cortex, right anterior thalamic radiations (Figure 2).

Although the areas identified with imaging studies give a potential anatomical rationale for fatigue, the lack of a strong a priori hypothesis to reflect the areas of the brain involved in MS fatigue makes the findings difficult to interpret.

Unifying model for fatigue

A new model is necessary to explain how information is transmitted in the brain in people with MS experiencing fatigue. In order to build

Figure 2



such a model, we need to remember that fatigue is a subjective feeling (present in other diseases also). Understanding the way that feelings arise is key to understanding fatigue.[35] Hence a framework on how the information related to multiple sclerosis fatigue is processed within the brain is important.

Stephan et al[36] propose fatigue to be a feeling arisen from a state of chronic dyshomeostasis. In order for the body to function normally, it needs to inform the brain about its inner state. The information goes up (via interoceptive afferent pathways) to the brain areas that have set points for normal function (hypothalamus, brainstem, spinal cord). These areas receive information about the inner body and check if the values are in the required range. They send further information (viscero-sensory input) to the brain areas that integrate information about the inner body (the viscero-sensory areas).

However, the brain does not only react to the input it receives (homeostasis). It anticipates homeostatic perturbation by making predictions. It gives commands and then changes its prior beliefs according to what input it receives back (allostasis). This means that the brain has certain expectations about what is going to happen.[36] Although it involves peripheral fatigue, the information on fatigue from sports medicine is within the same line of evidence: the central Governor Model states that human exercise performance is not limited by a failure of homeostasis but is rather regulated in advance in order to avoid it.[37]

The brain evaluates the accuracy of its own predictions by measuring the degree of surprise present when input comes back to inform it. In an acute setting (for example an acute feeling of fatigue), the brain is surprised about what information it is receiving. This results in an adaptive reaction-seeking resting behaviour, therefore consuming less energy. However, persistent interoceptive surprise represents a warning that the brain cannot control perturbations (chronic dyshomeostasis). Moreover, the brain has a metacognitive layer, represented by its beliefs about its own capacity and performance. In the case of chronic dyshomeostasis, the metacognitive layer receives the message that there is a low capacity of the brain to control the inner body states (higher order beliefs about lack of control).[36] Stephan et al raised the hypothesis that the feeling of discomfort related to fatigue could be related to metacognitive dysfunction induced by chronic dyshomeostasis.[36]

Fatigue in multiple sclerosis

Several groups have found structures involved in the dopaminergic networks to be related to MS fatigue. The findings supported the model proposed by Chaudhuri and Behan, who view fatigue as a failure of the non-motor functions of the basal ganglia.[38] The findings of dysfunction in areas in the brain related to the reward system have led to the development of a dopamine imbalance theory.[8] This can be interpreted in light of what we know about the brain: its goal is to be informed if homeostasis is maintained or not. Behaviourally, this is done by seeking pleasure and avoiding pain. Even very simple organisms have seeking and avoidance behaviours (dictated by rules encrypted in their genome), in order to keep the homeostatic levels. In more complex organisms, this developed into the reward and punishment system (the motivation system).

Focusing on fatigue as a feeling, Hanken and colleagues[1] proposed the hypothesis that fatigue is a subjective feeling related to inflammation. They described fatigue as a type of inflammation-induced sickness behaviour induced by cytokine-mediated activity in the brain areas involved in interoception and homeostasis. Their theory is that inflammation may redirect the focus to the interoceptive pathway, away from vigilance and attention.

The idea that the interoceptive system is involved in the feeling of fatigue is not restricted to multiple sclerosis. Kadota et al studied patients with post-infectious fatigue and found a sensitisation in the neuro-visceral regulatory circuits, which led to abnormally heightened perceptions of sensations from the body (referred to as physiological hyper-vigilance).[39]

Conclusions

There is a definite need to find effective medication for MS fatigue. Recent research has linked knowledge from immunology, neuroimaging and consciousness neuroscience, showing that inflammation leads to psychological, emotional and behavioural disturbances. The inflammatory state represents a condition out of the normal homeostatic parameters which is signalled by the afferent interoceptive pathways from which the brain makes hierarchic representations of this bodily state, the highest level being the metacognitive level, which may have a role in the feeling of MS fatigue. Dopaminergic pathways are also involved in MS fatigue and seem to have an important role. Finding a framework that integrates the findings so far and guides further research is crucial to understanding MS fatigue and therefore developing therapeutic targets for it.

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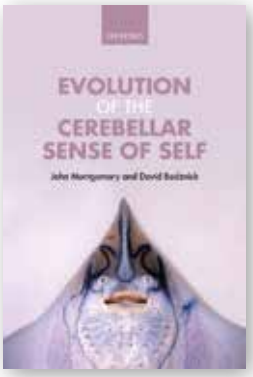
Evolution of the Cerebellar Sense of Self



Authors: John Montgomery and David Bodznick
ISBN: 978-0198758860
Published by: Oxford University Press
Price: £40.00
Pages: 240

Reviewed by: Rhys Davies, Consultant Neurologist, Liverpool.

Could the cerebellum be just about the most charismatic part of the brain? Its name, 'little brain' in Latin, has an appeal and its compact structure must endear it to undergraduate students of Neuroscience and Neurology used to contending with so many tortuous pathways! And more than this, it is the cerebellum's elegant and consistent functional circuitry that we usually get to know as students, ahead of any other brain circuit. And we learn to correlate models of cerebellar function with clinical impairments: this analysis is more sophisticated than the analysis of plain weakness or numbness, without being too abstruse. It is no accident that drawing the circuitry of the cerebellum was pivotal to Ramon y Cahal's realisation that neurons are not continuous but contiguous, which is the foundation of the Neuron Doctrine.

So, we can all agree that the cerebellum is great. How about this book?

Well, it's clearly not a must for the exit exam, or for the busy clinician in need of an update on the SCAs. But I liked it. And that's no small praise from a clinician for a Basic Science tome. In fairness, as tomes go, the book is quite light. I would recommend it particularly for clinicians feeling nostalgic for their preclinical Neuroscience studies!

First of all, it has an engaging picture of a ray on its cover; rays and sharks are big as animal models of cerebellar function, it seems. Although I did not find any mention of a deliberate link, the image reminded me of that iconic 18th century still life painting in the Louvre by Chardin 'The Ray', a painting imbued with existential angst (more than a century before Sartre et al) and far from irrelevant to this volume's philosophical and scientific content.

Montgomery and Bodznick give us a sequence of chapters on aspects of cerebellar function – the cerebellum and 'sense of agency', the cerebellum as a neuronal machine, the workings of specific cerebellar reflexes and so on. Perhaps unsurprisingly for the semi-detached neuroscientist (which condition, as clinicians, we must resign ourselves to be in), the introductory and concluding chapters were the most appealing. The more 'involved' middle sections, I'm sure, would have given greater satisfaction on repeated reading!

For me, the key message of the text is that the neural processing in the cerebellum permits an animal to distinguish between self-generated movements (and self-generated electrical field changes), on the one hand, and those that are externally generated on the other. Such processing is obviously linked to vestibular and somatosensory proprioceptive projections to the cerebellum (the vestibulocerebellum and spinocerebellum). In turn, this might allow a 'sense of agency', an idea of one's own actions or intent. Do you see where the sense of self might come in? And it is not too big a jump to consider impairment of that sense of agency contributing to disordered thoughts, and indeed fragmentation of the self, in mental health disorder.

My one criticism of the flow of their argument is that very little attention is given to the huge cerebrocerebellar projections of the human brain (as distinct from the vestibulocerebellar or spinocerebellar functions). I understand that such discussion would have been highly conjectural and 'unburdened by fact', but it would have added to the 'story', at least to the self-confessed semi-detached neuroscientist clinician-reader.

The chapter on the history of cerebellar research was an excellent read for the generalist. Cerebellar science makes a good case study! I enjoyed especially the authors' treatment of philosophical-scientific progress made by the giants John Eccles and Karl Popper, and others. I had not previously heard of Science being distinguished from other domains of study by its 'epistemological efficiency'. That is, that science builds up new knowledge more efficiently (problems > theories > criticisms > new problems) than other scholarly disciplines – a robust and measured analysis of what science can and cannot offer. In the same chapter, I found their treatment of the anelectrotonus concept (vaguely corresponding with hyperpolarisation) a bit laboured.

In the final chapter, it is suggested that some form of future robotic circuitry might be inspired by the circuitry of the cerebellum. For a clinician, it was a thrill to perceive for the first time the parallels between robotic movements and some forms of ataxia. I trust, however, that we are some way off robots whose moves are cerebellum-smooth, and some way off robots with a cerebellar 'sense of self'.

Naming the Cranial Nerves: a historical note

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Conflict of Interest statement: None declared.

Date first submitted: 4/5/17
Acceptance date: 5/5/17

To cite: Pearce JMS, ACNR 2017;16(5);12-13.

Abstract
This summary relates the history of the Galenic system of ordinal numbering and the later naming of the cranial nerves. It emphasises the original classification by Samuel Thomas Soemmerring's naming of 12 pairs, now universally accepted.

Physicians recognise that the cranial nerves are an important part of anatomy whose precise identity is essential to the understanding of their clinical and pathological disorders and potential remedies. Cranial nerves are derived from embryonic neural crest cells and their anterior extension the cranial placodes. Their history[1] reaches back to ancient Greek Medicine.

Herophilus of Chalcedon (325-255 BC) and his contemporary Erasistratus (325-250 BC),[2] were leading Ptolemaic Alexandrian scholars. From cadaveric dissections and probably vivisection, Herophilus identified at least seven pairs of cranial nerves. He distinguished motor from sensory nerves, and importantly observed that the nerves of the spinal cord were directly linked to the brain.

Similarly, Galen of Pergamum (129-c. 216 AD) described seven pairs of cranial nerves, recorded in *On Anatomical Procedures*.[3] He did not name them, relying on an ordinal numeric system of classification,[4] Galen insisted that descriptions of anatomy were based only on actual observations made at dissection, an opinion strictly respected; this proved a crucial factor in the almost universal acceptance of his dictates over 1500 years. Galen thus dominated medical theory and practice in Europe until the mid-17th century. His authority embraced the Byzantine world and the Muslim Middle East.

However, his work was based not on humans but on dissection of mice, pigs, many domestic animals and the Barbary macaque. He identified the olfactory nerve but considered it an extension of the brain. His optic nerves were therefore the first pair of cranial nerves. He also showed the optic chiasm. Galen demonstrated the oculomotor nerve terminating in the "muscles which move the eye," but he did not identify the trochlear or abducens nerves. The sensory root of the trigeminal was Galen's third pair, and its motor root the fourth. The facial and vestibulo-cochlear nerves he unified as constituting the fifth pair, though he separated their functions: the vestibulo-cochlear served hearing, the facial nerve "arrives on the face." He combined the glossopharyngeal, vagus, and spinal accessory nerve as the sixth pair. The vagus, "lying next to the artery of stupor (carotid artery) and the hypoglossal innervating the tongue was his seventh pair.[3,5]

When Galen's writings in Greek were translated into Arabic, Avicenna (980-1037 AD), Rhazes (864-930 AD), and other celebrated Arabian physicians adhered to his ordinal system. Galen's doctrines became available to other European physicians only in the 13th century when translated into Latin.

Medieval anatomists such as Achillini, Berengario da Carpi, and Massa investigated these nerves but like the ancestors of Graeco-Roman times were seriously restricted in dissection by the contemporary prohibitive laws until after the 15th-16th centuries.

Like Galen, Andreas Vesalius (1514-1564 AD)[6] in his famous *De humani corporis fabrica* 1543,[7] described seven pairs of cranial nerves (Figure 1), but he did identify the trochlear nerve. Vesalius was succeeded by several respected Italian anatomists who published conflicting accounts and variants of the Galenic system with little in the way of additional detail or clarification.

Thomas Willis (1621-1675) in 1664, in his *Cerebri anatome* identified nine cranial nerves but combined the facial and vestibulo-cochlear nerve into his seventh pair, and the glossopharyngeal-vagus-accessory nerves into his eighth pair although he recognised a separate accessory nerve (Figure 2).[8]

All these early anatomists had to rely on naked eye observations, assisted only by low magnification hand-held lenses. Not until the 18th century did our present classification of 12 cranial nerves arise. This would challenge and replace the stupefying *status quo* that had succeeded Galen.

It originated in the work of the German anatomist, inventor and polymath, Samuel Thomas Soemmerring (1755-1830), who in 1778 classified the twelve cranial nerves as we recognise them today (Figure 3).[5,9] His work was part of his student's doctoral thesis: *Anatomica de basi encephali et originibus nervorum cranio egredientium libri quinqe*[10] (on the Base of the Brain and the Origin of the Nerves Exiting the Skull. Five Chapters). It is astonishing that such an important discovery was the work of a student, and it bears testimony to his precocious skills of dissection and observation, not least because it plainly contradicted a long established 'fact'.

Heinrich August Wrisberg (1739-1808), who was Soemmerring's teacher, first named two separate roots of the fifth nerve, naming them *portio major* and *portio minor*. Soemmerring was the first to use the term *nervus abducens* in 1778. Before Soemmerring, the facial and vestibulo-cochlear nerves were classed as a single nerve. Soemmerring named the facial nerve branch—*the nervus intermedius of Wrisberg* in deference to his teacher.[4] Although Haller in 1762 had described the eighth cranial nerves as comprising: the glossopharyngeal, vagus, and the spinal accessory nerves, it was Soemmerring who separated the three components but retained Haller's nomenclature.

His description brought him instant, widespread recognition. It remains valid today.[11] However,

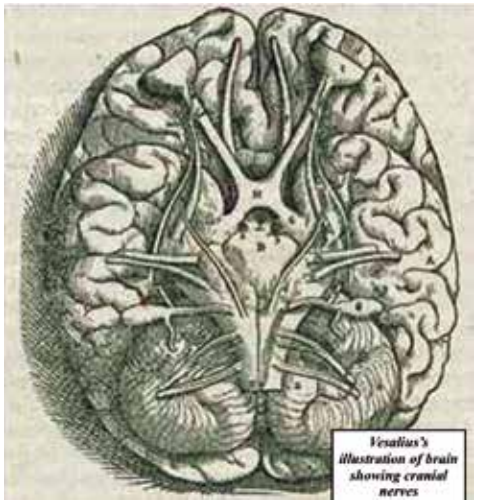

Figure 1. Vesalius's illustration of cranial nerves. De humani corporis fabrica, libri septem.

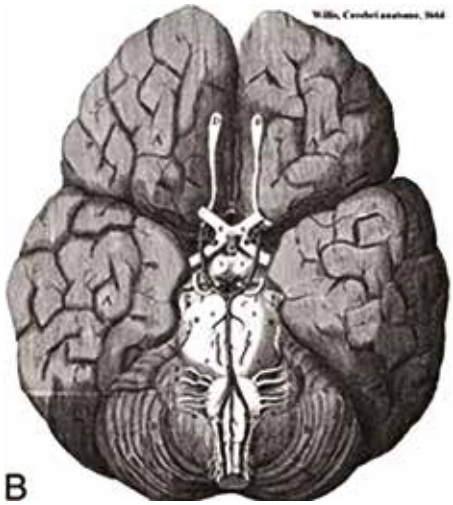

Figure 2. Thomas Willis: Cerebri Anatome, page 25. The brain with cranial nerves and the circle of Willis,

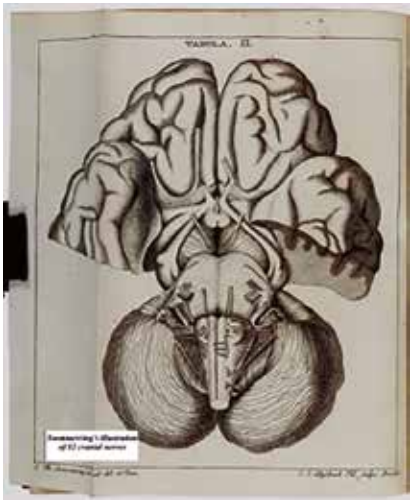

Figure 3. Soemmerring's illustration of 12 cranial nerves, In: Anatomica de basi encephali et originibus nervorum cranio egredientium libri quinque.(Göttingen, 1778).

Soemmerring mistakenly thought that the cranial nerves emerged from the ventricles. In the medieval tradition, he further believed that the cerebrospinal fluid could be animated and was the immediate organ of the soul, the *sensorium commune*.[12,13] The philosophers Goethe and Kant disputed his attempt to localise the soul. Like Descartes, Soemmerring believed the nerves ended in the walls of the ventricle and were stimulated by the flow of ventricular fluid.[14]

Soemmerring's system of cranial nerves was rapidly adopted across continental Europe, although it was only slowly accepted in Britain, not appearing until the 11th edition of *Gray's Anatomy*[15] in 1887.

Nomenclature was diverse and usually in Latin.[16] The reasoning and basis for the naming appears to have been: localise nerves and their anatomy, and to imply their function, and appearance.[1] While the ordinal numbering was unchanged after hundreds of years, Soemmerring's naming of the 12 individual nerve pairs was finally established in the *Basle Nomina Anatomica* in 1895 renamed *Terminologia Anatomica* in 1998.

Current anatomy mirrors the account of Henry Gray's (1821–1865) *Anatomy of the Human Body*, 1918, that relates:

> The fibres of the nerve can be traced into the substance of the brain to a special *nucleus* of gray substance. The motor or efferent cranial nerves arise within the brain from groups of nerve cells which constitute their nuclei of origin. The sensory or afferent cranial nerves arise from groups of nerve cells outside the brain; these nerve cells may be grouped to form ganglia on the trunks of the nerves or may be situated in peripheral sensory organs such as the nose and the eye.

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Neurological Literature: Neurophysiology

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To cite:
Larner AJ. ACNR 2017;16(5):14-15.

Previous articles in this series have focused on literary accounts or narratives of various neurological disorders, including headache, epilepsy, cognitive disorders, and sleep-related disorders. Since these conditions are the very stuff of human experience, and likely to be encountered at either first or second hand by the majority of the population, it is perhaps unsurprising that novelists have on occasion taken such conditions as source material for elaboration in their narratives. Neurological investigations, on the other hand, are perhaps less familiar to the general populace. This brief article looks at some literary references to neurophysiological investigations.

Electroencephalography (EEG)

It is perhaps unsurprising that authors within the genre broadly described as "science fiction" have been attracted by the technological implications of EEG for recording and/or monitoring the human nervous system.

The prolific sci-fi author Philip K Dick (1928-1982) explored the possibilities of EEG in his 1974 novel *Flow, my tears, the policeman said*.[1] (Musicophiles may know that "Flow, my tears" is taken from the title of a lute song composed by John Dowland in the late 16th century; the phrase was also used, almost three centuries later, by Gary Numan, based on his reading of Dick, in the first line of *Listen to the sirens*, the first track on Tubeway Army's eponymous album of 1978, re-issued 1979.) In the dystopian world of Dick's novel (possibly set in 1988), the "pols" (police) want a "fingerprint, voiceprint, footprint, EEG wave pattern" from the protagonist, Jason Taverner:

> ... seated, he allowed terminals to be placed here and there on his head; the machine cranked out three feet of scribbled-on paper, and that was that. That was the electro-cardiogram [*sic*! Checked in two separate editions of the book].

Despite having the EEG print Taverner suspects that the pols will not be able to find his information in their extensive data pool. Later when Taverner is being sought by the pols, they suggest that "we may be able to catch him with an EEG-gram projection from a copter", to get "a match of patterns". Clearly the view here is that EEGs are sufficiently individualised as to permit identification, even if recorded with leads placed "here and there" on the head.

Through its incarnation as the film *Blade Runner* (1982), probably the best known of Dick's novels is *Do androids dream of electric sheep*? (1968).[2] There may be EEG references here too, specifically in the allusions to the "Penfield mood organ", a method of "artificial brain stimulation" which features in both the first and last sections of the book. For example, Rick Deckard, squabbling with his wife, "at his console ... hesitated between dialling for a thalamic suppressant (which would abolish his mood of rage) or a thalamic stimulant (which would make him irked enough to win the argument)." Different dialling codes on the organ permit the selection of different moods, such as 888 for "the desire to watch TV, no matter what's on it" or 670 for "long deserved peace". (Music aficionados will know that this dialling trope also appears in Gary Numan's *I dream of wires* from the *Telekon* album of 1980.) As a neurologist reading this book (en route to, and in the interstices of, an international neurology conference!), I immediately thought the Penfield mood organ must be a reference to Wilder Penfield (1891-1976), whose work (with Herbert Jasper) stimulating the cortex of awake epilepsy patients undergoing surgery allowed him to map the functions of various regions of the brain.[3] The possible influence of Penfield on Philip Dick is acknowledged in a psychology textbook.[4] I do not know whether Dick ever underwent an EEG. His biographer Lawrence Sutin speculates a possible diagnosis of temporal lobe epilepsy to explain some of Dick's experiences, in particular a series of "visions and auditions" experienced in February-March 1974 which influenced his later writing.[5]

Ursula Le Guin (born 1929) is another author categorised as within the sci-fi genre who has explored the narrative possibilities of EEG. The plot of *The lathe of heaven* (1971)[6] revolves around EEG recordings. Dr William Haber of the Oregon Oneirological Institute records EEGs during the dreams of George Orr:

> As soon as the cap was in place he switched on the EEG ... Eight of the cap's electrodes went to the EEG; inside the machine, eight pens scored a permanent record of the brain's electrical activity (20),

Somehow, Orr's dreams affect outward reality ("effective dreaming"), a faculty which Haber seeks to take control of, using his Augmentor which operates by "instigating and then reinforcing ... d-state activity" (56), for his own advancement, with catastrophic results.

In *The word for world is forest* (1976),[7] sometimes regarded as Le Guin's indictment of the Vietnam War, colonists from Earth have enslaved the peaceful Athshean people. Raj Lyubov, the colony anthropologist, has studied the Athsheans:

> He had wired countless electrodes onto countless furry green skulls and failed to make any sense at all out of the familiar patterns, the spindles and jags, the alphas and deltas and thetas, that appeared on the graph.

However,

> It was with Selver [an Athshean] as EEG subject that he had first seen with comprehension the extraordinary impulse-patterns of a brain entering a dreamstate neither sleeping nor awake.

Suffering a migraine headache, Lyubov wonders what Selver would do:

> Although knowing nothing of electricity he could not really grasp the principle of the EEG, as soon as he heard about alpha waves and when they appear ... there appeared the unmistakable alpha-squiggles on the graph recording what went on inside his small green head; and he had taught Lyubov how to turn on and off the alpha-rhythms in one half-hour lesson.

Electromyography and nerve conduction studies (EMG/NCS)

Literary accounts of EMG/NCS might be anticipated in patient accounts and fictional narratives featuring individuals with neuromuscular disorders.

In his account of the episode of Guillain-Barré syndrome (GBS) he suffered in 1981-2, the author Joseph Heller (1923-1999), best known for his 1961 novel *Catch-22*, reported two EMG examinations.[8] The first, performed at the Mount Sinai Hospital in New York, lasted less than fifteen minutes, whereas the second, performed at a rehabilitation facility, the Rusk Institute at the New York University Medical Centre, lasted more than two hours. "The worst both times ... was the needle plunged into the palm of the hand near the base of the thumb." At Rusk, all four limbs were examined as well as the face.

> None of the pain from the individual electric shocks or from the needle punctures was so intense as to make one wish to cry out. It was the repetitions of the electric shocks that rapidly wore me down, and which gradually proved more and more terrible ...

Although "[m]y F-wave responses were not too good" and "the doctor muttered to himself that there was definite facial involvement", Heller was subsequently informed that the "results of the EMG test were inconclusive, neither confirming nor eliminating Guillain-Barré".

In *Solomon's Porch: the story of Ben and Rose*, a college Professor in his 50s develops a neurological illness which is labelled as GBS[9] (I have critiqued this diagnosis elsewhere[10]). It does not appear that EMG/NCS is ever performed, which may account for some of the diagnostic confusion.

Incredible as it may seem, EMG/NCS has been the stimulus for a poem, "The Nerve Conduction Studies" by Simon Armitage (born 1963),[11] from which these selected lines are quoted:

> We loop conductive strips over the toes
>
> and fingers, press conductive strips and pads
> into the calves and wrists, ..
> ...
>
> the trace comes up on the screen and we ask
>
> for a second or third flick of the switch
> if the jolt doesn't travel the distance the first time.
> ...
>
> These tests are well known to hold true; we trust
> they prove nothing less than you dared hope for.

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Transcranial magnetic and electric stimulation of the cerebellum – A potential aid in enhancing rehabilitation of cerebral functions

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Conflict of interest statement:
The authors declare no conflict of interest. Kim van Dun is a doctoral researcher involved in project G035714N granted by the Fund for Scientifc Research Flanders (FWO). This research was also funded by a Strategic Research Program (SRP15) awarded by the Vrije Universiteit Brussel, Belgium.

Provenance and peer review: Submitted and externally reviewed.

Date submitted: 22/11/16
Acceptance date after peer review: 23/2/17

To cite: van Dun K, Bodranghien F, Mariën P, Manto M, ACNR 2017;16(5):16-18.

Abstract
Subserved by a dense network of neuroanatomical connections within the cerebrum, the cerebellum fulfills a crucial role in various motor, cognitive, and affective functions, and is located immediately below the skull. As a result, the cerebellum appears an interesting target for modern noninvasive stimulation techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). This literature survey intends to give a short overview of what is currently known about noninvasive stimulation techniques applied to the cerebellum.

Introduction
Improving rehabilitation of brain functions by modulating the excitability of neurons with noninvasive techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) is an exciting new research domain. Since (a) the cerebellothalamocortical pathways connect the cerebellum with the supratentorial regions subserving motor, associative, and affective functions and (b) the cerebellar circuitry is located immediately below the skull, the cerebellum might be a very promising target for noninvasive stimulation.[1] We conducted a literature search to determine the potential value of cerebellar stimulation in different domains.

Working mechanisms of TMS and tDCS
TMS is a non-invasive brain stimulation technique that offers the possibility of modulating the excitability and activity of specific brain areas.[2] TMS uses a coil to produce a pulsed magnetic field inducing an electric field inside the brain (eddy currents). If the intensity of the eddy current exceeds a particular threshold, action potentials are generated in the stimulated neurons.[3] TMS can be administered in several different ways (Figure 1). A single pulse of TMS can be used (single pulse TMS) or it can be administered in a repetitive manner at different frequencies (rTMS), and in different patterns (such as theta burst stimulation, TBS). When a single pulse is used, it is usually given at an intensity that can generate action potentials, which can temporarily interfere with the function of the targeted brain region. The different TMS paradigms all exert a specific effect on cerebellar (and cortical) excitability, exciting or inhibiting brain function.[4]

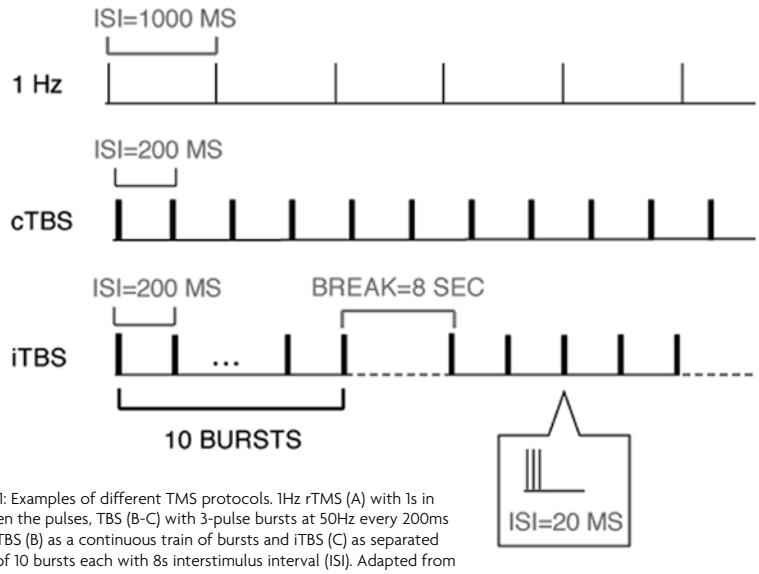

Figure 1: Examples of different TMS protocols. 1Hz rTMS (A) with 1s in between the pulses, TBS (B-C) with 3-pulse bursts at 50Hz every 200ms with cTBS (B) as a continuous train of bursts and iTBS (C) as separated trains of 10 bursts each with 8s interstimulus interval (ISI). Adapted from Müller, Lorenz, Langguth, and Weisz (2013).[5]

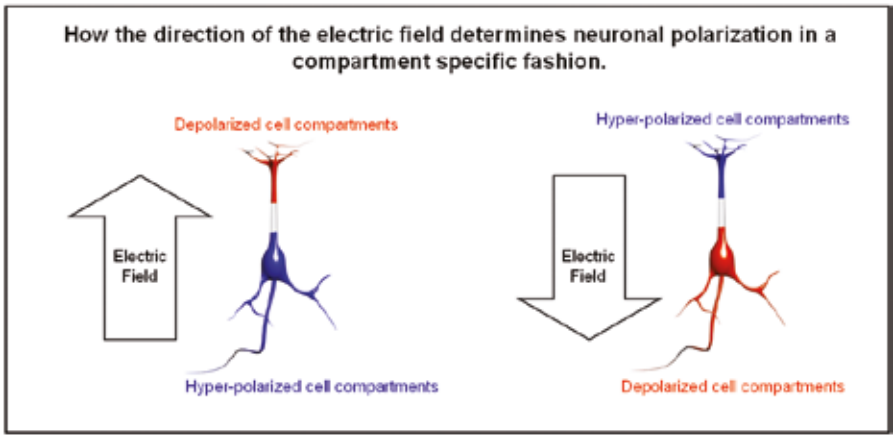

Figure 2: Hyper- and depolarisation of a single neuron, depending on the direction of the current. Courtesy of Neural Engineering Group (http://neuralengr.com/old/research).

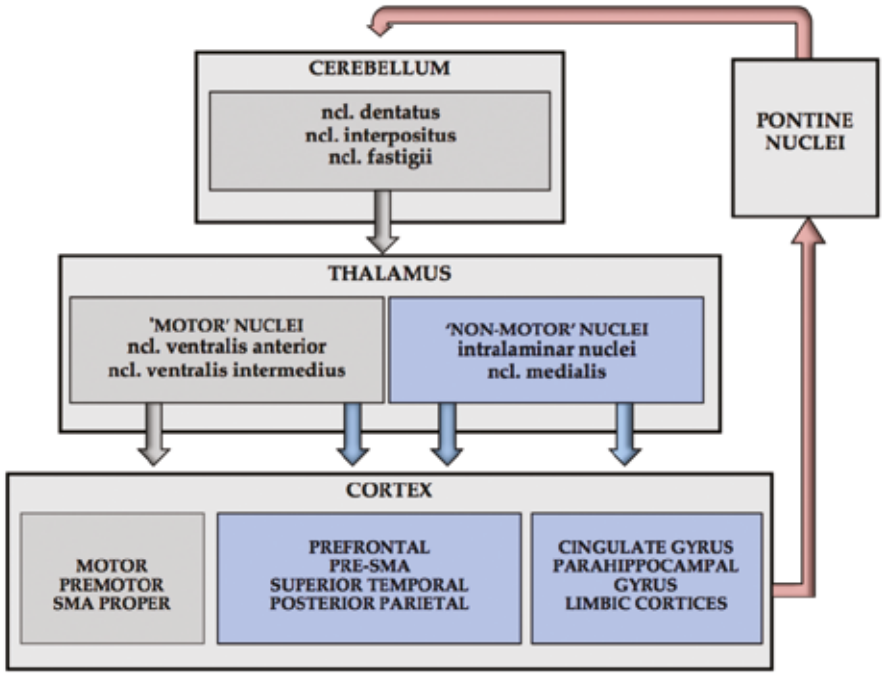

Figure 3: (A) Diagram depicting the cerebello-cerebral connectivity network underlying cognitive and affective processes. The feedback or efferent loop originates from the deep nuclei of the cerebellum that project to the motor (grey arrows) and nonmotor (blue arrows) nuclei of the thalamus. In turn, the motor nuclei of the thalamus project to motor and premotor cortices (grey arrows) but also to nonmotor association cortices (blue arrows). The nonmotor nuclei of the thalamus project only to association cortices (blue arrows). After Schmahmann and Pandya (1997).[16] Adapted from Mariën et al. (2013).[17]

tDCS is another novel brain stimulation technique that uses two electrodes to induce a small electric current in the brain.[6] Anodal and cathodal tDCS are associated with opposite directions in currents. As compared to TMS, tDCS does not generate action potentials in neurons but acts on the polarisation of cellular membranes (Figure 2). This means that tDCS modulates activity in active neurons but has probably little impact on purely resting neuronal population.[7] The general rule is that anodal tDCS increases the excitability of neurons, whereas cathodal tDCS exerts the opposite effect. However, this is a simplification of the physiological mechanisms subserving the effects of tDCS. tDCS creates a difference of electric potential between two (or more) electrodes, which induces a shift in the membrane potential and therefore modifies the excitability of the neurons within the created electric field.[7,8] Electric brain stimulation can also be achieved using alternating current and is called transcranial Alternating Current Stimulation (tACS). At low frequency, this will lead to alternating membrane potential changes following the current wave.[9]

However, little is known about the exact impact of electric/magnetic stimulation of the cerebellum since the cerebellum differs from cortical brain tissue in many respects, especially the cytoarchitecture. In addition, complex cerebellar folding greatly affects the changes in excitability which makes a prediction of the outcome very difficult.[10] It is also challenging that the effects of cerebellar stimulation are difficult to measure in the cerebellum itself. The impact on cerebello-cortical mechanisms (such as cerebellar brain inhibition (CBI)) has to be monitored to evaluate the effectiveness of cerebellar stimulation.[11,12]

Anatomical cerebello-cerebral connections

Neuroanatomical evidence has shown multiple crossed cerebello-cerebral connections, not only with the contralateral motor areas, but also with the associative cortices responsible for cognition and affect (Figure 3). Many functional imaging studies confirmed involvement of the cerebellum in a variety of motor, cognitive, and affective functions.[13] However, several recent studies indicate that there are also non-crossing cerebello-cerebral pathways,[14] and direct connections between both cerebellar hemispheres.[15] Future studies should bear in mind that the functional connectivity of the cerebellum to the supratentorial regions is built on a complex network consisting of crossed and non-crossed pathways,[14] supplemented with parallel connections between both cerebellar hemispheres.[15]

Findings of the literature survey

A literature survey (Electronic online databases: Web of Knowledge, ScienceDirect, PubMed, Medline; keywords: cerebell* AND tDCS OR transcranial direct current stimulation; cerebell* AND TMS OR transcranial magnetic stimulation) yielded 111 original studies using cerebellar TMS and 49 studies using cerebellar tDCS, covering a wide and extensive range of topics. Most studies applied stimulation in healthy subjects (TMS: n=81; tDCS: n=35) with a focus on probing functional connectivity with cerebellar TMS (n=28) and motor function with cerebellar tDCS (n=16). Other areas included cognition and affect, and some studies explored the effects of cerebellar stimulation in a clinical population (TMS: n=30; tDCS: n=14).

GENERAL

In general, the timing of the administration (together with therapy/assessment or not) and the type of TMS (single pulse TMS, low frequency rTMS, high frequency rTMS, intermittent TBS, continuous TBS) or tDCS (anodal, cathodal) stimulation is very important. These parameters determine which process will be affected, and in what way. In addition, the intensity of the stimulation may also be crucial, especially for TMS in which the intensities applied are frequently determined by the resting or the active motor threshold of the contralateral motor cortex. These intensities may vary greatly and probably have a differential impact on neuronal firing and functional connectivity. Moreover, it is important that the stimulation only affects the cerebellum and

does not spread to the adjacent brainstem or to the visual cortex. With regard to cerebellar tDCS, the effect of the intensity of the current on neuronal excitability has not been systematically investigated. Due to the various cerebello-cerebral connections, the lateralisation of functions in the cerebral hemispheres, as well as the cerebello-spinal pathways, it is also important to carefully select the place of stimulation. With TMS the localisation is dependent on the positioning of the coil, the coil orientation, and the type of coil used. Several studies have indicated that the standard figure-of-eight coil may not be optimal for cerebellar stimulation due to the distance between the scalp and the cerebellar cortex. It is therefore important in future studies to use a double cone or a batwing coil, which are specifically designed for stimulating deeper brain regions and which eliminate the stimulation of the peripheral nerves that is observed in cerebellar stimulation with a figure-of-eight coil.[3,4,11] For tDCS, the positioning of the electrodes and their size primarily determines which areas will be excited. The exact electrical pathways taken during tDCS are still unclear and more studies should be made to better characterise them.

MOTOR

The literature on motor function shows that the cerebellum is involved in movement, motor learning, motor adaptation, and even motor imagery. The cerebellum seems to be responsible for monitoring ongoing movements, and predicting future states, but also for detecting and correcting errors (state estimations).[18] Interestingly, however, the complexity of the task at hand has a significant impact on the effect of cerebellar stimulation. The nature of the task also affects the outcome differently (e.g. cerebellar stimulation has a different impact on motor movement than on motor adaptation), and outcome also depends on whether implicit or explicit strategies are needed.

COGNITION

The most important factor that should be taken into account while studying the involvement of the cerebellum in cognition is that cerebellar stimulation usually interferes with cognitive functioning in a very subtle manner. Specific methods to measure potential effects and timing are crucial to accurately observe the impact of cerebellar stimulation. A lot of parallels can be drawn with the findings in the motor literature, with a differential impact on explicit and implicit processes depending on the nature of the task, a role for the complexity of the task, and a role of the cerebellum in perception/processing, error correction, learning, and accuracy.

AFFECT

Not much can be said about the impact of cerebellar stimulation on affective processing. There are too few studies to substantiate any conclusions, although there are indications from experimental and clinical studies that the cerebellum is involved in affective and somatosensory processing.[19]

CLINICAL PRACTICE

In clinical populations, TMS and tDCS seem to have a lot of potential as substituting or adjuvant therapeutic tools. It seems that not only in motor deficiencies, but also in a variety of non-motor and psychiatric conditions, cerebello-cerebral functional connectivity is disrupted, which might be restored employing cerebellar stimulation. Several isolated studies have shown that repeated sessions of cerebellar stimulation may exert a long-lasting positive effect on certain deficits. However, in order to establish TMS and tDCS as standard clinical practice techniques, it is crucial to learn more about the working mechanisms and impact of the different stimulation protocols.

Conclusion

Cerebellar TMS and tDCS are both promising and novel techniques to probe and modulate cerebellar excitability. However, there is a great need for systematic and methodological research to clarify the underlying pathophysiological mechanisms, and the specific impact of the different paradigms and parameters on cerebellar excitability/activity as well as on remote plasticity of the motor cortex.[20] In addition, more research has to be directed to the specific working mechanisms of TMS and tDCS and how these techniques might differ and be differentially used in specific settings. When applying cerebellar stimulation, it has to be kept in mind that the cerebellum has a divergent cytoarchitecture and functioning, and is connected to the cerebrum/spinal cord in different ways, which might result in a very unique response to magnetic and/or electrical stimulation that is not comparable to cerebral stimulation.

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The Neuro Network Programme

The Neuro Network is a programme led by The Walton Centre NHS Foundation Trust to enhance services for neurology patients. Programme Director Julie Riley explains how the neurology model benefits patients, clinicians and the wider health system.

Introduction

The Walton Centre has been working within and strengthening its hub and spoke neurology model for over ten years. This model sees The Walton Centre as the hub, supporting spokes or satellite sites in acute hospitals. Neurologists from The Walton Centre are present in the satellites for typically four days a week (normally two days per Consultant), holding outpatient clinics and undertaking ward consultations; the aspiration would be to continue to increase this over time. All have sub-specialisms and for the rest of the week are based at The Walton Centre.

The hub and spoke model is not a new concept and has been challenged for taking away from the acute hospitals the responsibility to ensure a fully functioning local neurology service. However, with a shortage of Neurologists, and the desire and clinical case for subspecialisation, it is increasingly difficult for local hospitals to provide a full neurology service. The hub and spoke model provides equitable local access with direct links into subspecialty care, enables common clinical governance, and has been successful for recruitment. With funding from NHS England, under the New Care Models Programme, The Walton Centre has a chance to prove how a networked model can enable clinically robust and sustainable neurology services to be provided throughout the country. What NHS England have asked for in return is a model that is replicable in other geographical areas – and potentially adaptable for other specialties – and that offers good outcomes for patients wherever they live, whilst being cost effective.

A whole system approach to neurology services – problem solving

There are around 10 million patients in the UK with long term neurology conditions and only around 600 Neurologists* (Source: Local adult neurology services for the next decade; Royal College of Physicians/ Association of British Neurologists; June 2011). The Walton Centre has 36 Consultant Neurologists who serve a population of 3 million, in North West England and North Wales. Greater awareness, better tests, combined with an ageing population means creating a sustainable service for the next generation is a challenge.

Julie Riley, Programme Director

The Neuro Network model

The Walton Centre's hub and spoke model, currently in place across all 12 acute hospitals within the area it serves in Cheshire and Merseyside, is being strengthened ensuring the patient remains at the centre of care. The idea is to build services around the patient, encourage self-management and provide care closer to home but, further to this, support the wider health system. Hospitals, community services, commissioners, patients, carers, third sector groups have all being working hard, but mostly in isolation. The solutions have to be found together, through co-production and working in partnership.

The Neuro Network has enlisted the support of all these stakeholder groups from the first day of the programme. There has been a step change away from a paternalistic 'we know best' approach to one of listening to patients, carers and colleagues in primary and secondary care, and of being honest enough to discover what works and what doesn't, being willing to stop, review and pivot, if need be.

The Neuro Network is building on The Walton Centre's existing hub and spoke model to achieve an integrated service for neurology patients, linking all those involved in their care:

- Providing a formal advice line for patients to connect them to their specialist nurse quickly and reliably;
- Offering extra support to GPs through clinical pathways (starting with headache), educational support via protected teaching sessions, and a Consultant advice line;
- Supporting acute hospitals on a seven day basis through clinical pathways (starting with post seizure in A&E), teleneurology and the Consultant advice line.

All of these developments have now been introduced and will be fully implemented by September 2017. Teleneurology is currently being piloted with the Countess of Chester Hospital, enabling Consultant Neurologists to examine patients with doctors in the acute hospital facility. This means the patient can be seen quicker, discharged or referred to the Centre, if appropriate – rather than waiting for the next ward consultation to take place.

The key development bringing services together is the team of locally based Integrated Neurology Nurse Specialists, experienced neurology nurses who have received academically accredited training across a range of neurological conditions to Master's level under The Walton Centre's Neurologists. They support patients and connect clinical staff in primary care, acute hospitals and the neurology centre – facilitated by IT - helping patients to self-manage and facilitating continuity of care. They have already been shown to prevent avoidable primary care, A&E and outpatient attendances.

Conclusion

The Neuro Network is not a completely new idea. It is about strengthening partnership working and shared decision making. It acknowledges that one model does not fit all but elements of it can be replicated to make services more sustainable. The success of the model is down to the flexibility of the workforce not working in isolation but in partnership. While The Walton Centre will continue to provide clinical neurology and be responsible for its governance, each partner in the Neuro Network has a responsibility for neurological care and has to take ownership. By working in a new way, with an agenda of collaboration, we are supporting each other to sustain a neurology service and not only maintain it but strengthen and grow it, so that patients are seen quicker, and get the best possible treatment and outcomes. This way not only gives the patient a better experience but also has the by-product of efficiency by seeing the patient in the most appropriate setting and according to their level of need. This networked approach has already been proven to work in major trauma and specialised rehabilitation services at The Walton Centre. As we enter the evaluation phase of the Neuro Network, it is exciting to think about the tangible change this programme of work can deliver.

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Paper of the Month: May

Kermer P, Eschenfelder CC, Diener H-C, et al. Antagonizing dabigatran by idarucizumab in cases of ischemic stroke or intracranial hemorrhage in Germany – A national case collection. International Journal of Stroke 2017; doi: 10.1177/1747493017701944.

The use of non-vitamin-K-antagonist oral anticoagulants (NOACs) to prevent stroke in patients with atrial fibrillation is increasing worldwide. Compared to warfarin, NOACs seem to have less hemorrhagic complications, including intracranial hemorrhage. However, specific rapid reversal agents for NOACS are still lacking. Between the four NOACs available, dabigatran has shown to be superior to warfarin for stroke prevention, and it has the advantage to be quickly antagonized by idarucizumab in case of uncontrolled bleeding or other emergencies. Idarucizumab is a humanized Fab fragment of a monoclonal antibody able to bind dabigatran with high affinity. However, available data on the use of this antagonist in patients with acute stroke who need thrombolysis are still anecdotal.

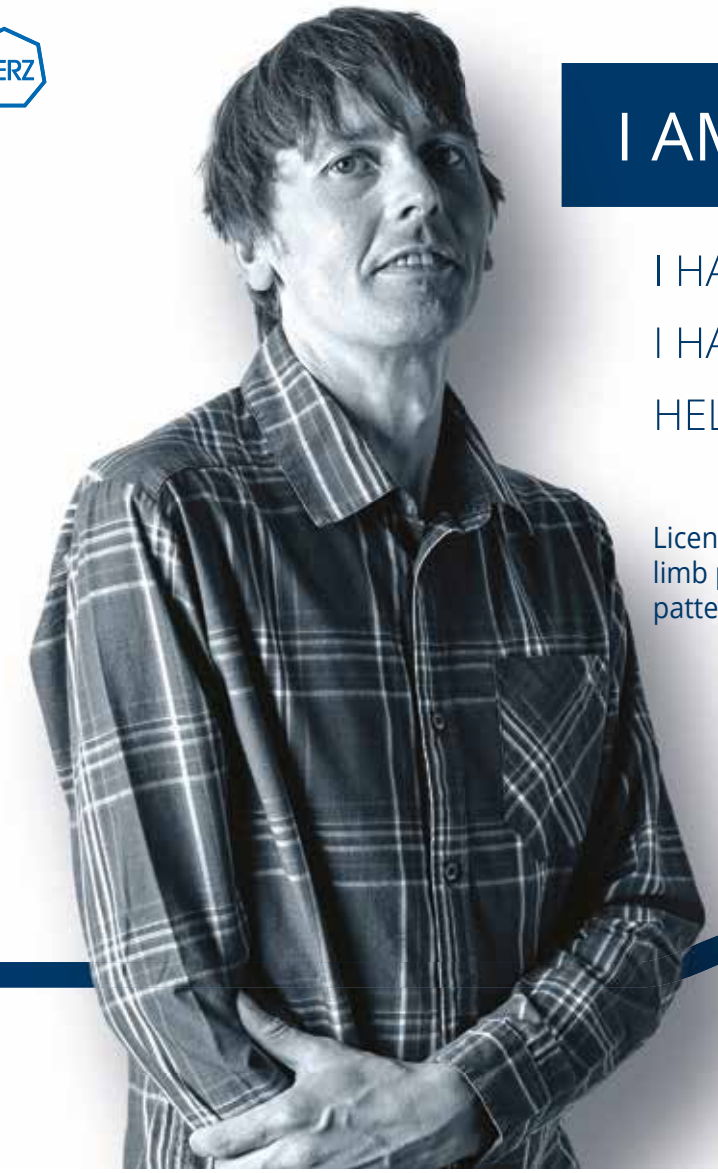
In this retrospective German multicenter study, the authors report on the national experience with idarucizumab in dabigatran-treated patients with acute stroke or intracranial bleeding since the availability of the drug, January 2016, to August 2016. Collected data included baseline clinical characteristics, clinical findings, coagulation and other laboratory parameters, imaging, clinical course, etcetera. For the hemorrhagic group of patients, further data about size and hematoma growth, idarucizumab adverse events, and modified Rankin score (mRS) outcomes were obtained. For the ischemic stroke group of patients, information about the NIHSS, mRS, bleedings and thrombotic complications were included. Full data were available in 31 patients, 19 with ischemic stroke, and 12 with intracranial bleeding.

Between the ischemic stroke patents, 18 were eligible to systemic thrombolysis within the 4.5-hour window. Most patients were receiving 110 mg bid dabigatran for atrial fibrillation. The partial thromboplastin time (aPTT) was normal in 13 patients, whereas the thrombin time (TT) was abnormal in 11 patients at admission. All patients received idarucizumab, which normalized the coagulation parameters, and allowed rt-PA administration. One patient had also additional thrombectomy. Improvement after thrombolysis was observed in 15 patients, who gained 5 points in the NIHSS as median. Anticoagulant therapy was restarted between 10 days after stroke in 74% of patients. Two patients did not recover: one had signs of acute severe vertebrobasilar stroke, and the other died five days after treatment due to pneumonia and pulmonary embolism.

Between the patients with intracranial bleeding, 8 had intracranial hemorrhage, 3 had subdural hematoma, and 1 subarachnoidal bleeding. All had atrial fibrillation. TT was elevated at admission in 9 patients. After idarucizimab administration, no hematoma grow was observed in 10 patients. The median NIHSS improved of 5.5 points. One patient presenting with massive bleeding at admission died.

"Although the study has obvious limitations such as the retrospective nature and the small number of patients, its findings are relevant to clinical practice, especially in emergency settings when intravenous thrombolysis is indicated. Idarucizimab has shown to be safe and efficacious in reversing dabigatran anticoagulant effects", says Prof Thierry Moulin, Division of Neurology, Besancon, France.

"Idarucizimab seems to be effective also in limiting the expansion of intracranial bleedings in patients taking dabigatran. This effect might have an important role in reducing mortality and improve the outcome. Therefore, idarucizimab should be used in all patients with hemorrhagic stroke or intracranial hemorrhages", says Prof. Hans-Christoph Diener, Department of Neurology, Essen, Germany.



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Should not be used during pregnancy unless clearly necessary. Should not be used during breast-feeding. ***Blepharospasm:*** Careful testing of corneal sensation should be performed in patients with previous eye operations. Due to its anticholinergic effects, it should be used with caution in patients at risk of developing narrow angle glaucoma. ***Spasmodic Torticollis:*** Patients should be informed that injections of Xeomin for the management of spasmodic torticollis may cause mild to severe dysphagia with the risk of aspiration and dyspnoea. Limiting the dose injected into the sternocleidomastoid muscle to less than 100 units may decrease the occurrence of dysphagia. 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Frequency by indication defined as: *very common* (≥ 1/10); *common* (≥ 1/100 to < 1/10); *uncommon* (≥ 1/1,000 to < 1/100); *rare* (≥ 1/10,000 to < 1/1,000); *very rare* (< 1/10,000); *not known* (cannot be estimated from the available data). ***Blepharospasm:*** *Very Common:* Eyelid Ptosis, dry eyes. *Common:* Headache, facial paresis, blurred vision, visual impairment, diplopia, increased lacrimation, dry mouth, dysphagia, rash, injection site pain, fatigue, muscular weakness. ***Spasmodic torticollis:*** *Very common:* Dysphagia. *Common:* Headache, presyncope, dizziness, dry mouth, nausea, hyperhidrosis, neck pain, muscular weakness, myalgia, muscle spasm, musculoskeletal stiffness, injection site pain, asthenia, upper respiratory tract infection. ***Post-stroke spasticity:*** *Common:* Headache, dysaesthesia, hypoaesthesia, dysphagia, muscular weakness, pain in extremity, feeling hot, and injection site pain. 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Date of preparation: October 2016 M-XEO-UK1-0063

Lessons from running a neurology strategic clinical network

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Conflict of Interest statement: None declared.

Date first submitted: 8/5/17
Acceptance date: 9/5/17

To cite: Losseff N, ACNR 2017;16(5);22-24.

Acknowledgement: Many people gave their time for free to deliver our work programme. It is not possible to list them all but particular thanks are owed by the NHS to Michael Oates (our amazing project manager), Bernadette Porter, Jacqueline Wakefield, Jo Jarosz, Nassif Mansour, Davina Richardson, Lionel Ginsberg, Alexa Coombes, Bal Athwal and Dominic Heaney.

Despite a considerable number of reviews, from high level political statements to local initiatives, the pace of service development for people with neurologic conditions remains slow. The Neurological Alliance recent 2017 review[1] of patient experience makes a disheartening read, showing a worsening of many metrics, including an increased proportion of patients needing to see their GP five times or more before a specialised referral was made and a decreasing number who feel involved in making choices, or that their health care professionals work together well "at least some of the time". Unfortunately, the system does not seem to learn from its mistakes. In 2012, The House of Commons Public Accounts Committee[2] released an important report on services for patients with neurological conditions. It concluded that the implementation of the National Service Framework[3] for Long Term Conditions had failed, causation including a lack of leadership at a national and local level, poor data, huge postcode variation in expertise, poor integration of health and care and a paucity of quality standards.

Part of the NHS response to this was to appoint a National Clinical Director (NCD) for neurological conditions and establish Strategic Clinical Networks (SCNs) in 2013 to drive local developments. The NCD catalysed many important developments, including the development of the much-needed Neurology Intelligence Network whose "fingertips" publications have been considerably illuminating. However, was it all just a sop to the politicians? In a further 2016 review NHS England reported to the same committee that the NCD would not be reappointed, but instead neurology would be led in a collaborative way "based around strategic clinical networks". So, it's noteworthy that the strategic clinical networks were dissolved shortly after this statement.

However, were the SCNs cutting the mustard? In some areas yes, but unlike all the other priority area SCNs such as mental health, cancer, dementia*, stroke* (neurological conditions largely managed by other specialists*), there has been no alignment with clinical commissioning groups (CCGs) operational frameworks. Hence it is not incumbent on CCGs to improve services for patients with neurological conditions, whereas they have to report on specific actions, for example in patients with dementia. It is important to realise that the rate limiting step is usually business capacity rather than financial restriction. So, running a programme of change in a sustainable way is incredibly hard when the only driver is local enthusiasm.

So, what is the problem? After all there are 2 million people in the UK with a neurological condition (excluding migraine) and we are spending over £2 billion in un-coordinated health services and the same amount again in care. Neurological conditions account for 10% of all hospital admissions, 17% of all emergency admissions and 10% of consultations in primary care. A snapshot of information from four local authorities suggests that 50% of people aged 18–65 receiving social services support have a neurological condition. If this were extrapolated for England, it would equate to about 63,000 people aged 18–65 with a neurological condition needing such help.[4]

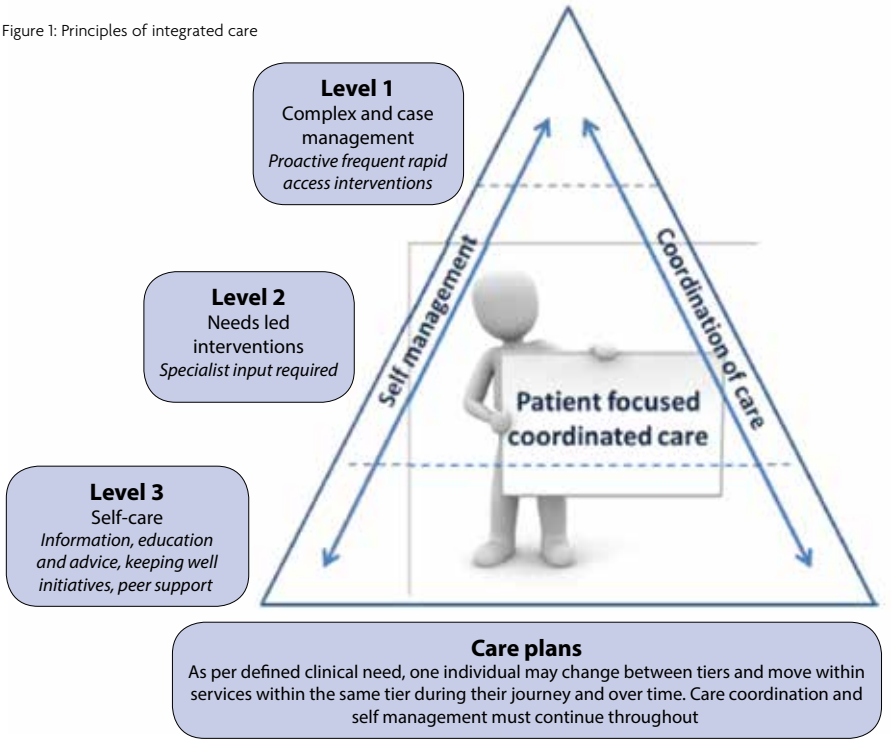
Why neurology has not been able to articulate a national message and remained invisible is only speculative. If I was a cynic I would suggest it's rooted in a cultural indifference to people with neurologic disabilities. One of my patients recently asked me why if you have cancer and are trying to get back to work everyone is bending over backwards to help, but his experience (following a stroke leaving him only with mild dysarthria) was to be shown the way to the car park.

Credible proposals to modernise the way neurology is delivered are simple including that:

- The management of common neurologic conditions in primary care could be stronger (in many areas).
- Systematic ownership could be taken at a secondary care level by Neurologists for emergency and urgent care.
- Variation in access and services offered by neuroscience centres and local hospitals could be reduced.
- Outpatient neurological services models need rethinking as they are often unresponsive to need, clogged with unnecessary referrals, and operate on a top heavy one in one out model.
- Management of neurological crises in the community could be strengthened – lack of knowledge/confidence, unresponsiveness all result in waiting for an outpatient appointment or reliance on A&E with subsequent unplanned admission.

This involves people working in a different way, such that more of the precious resource is shifted from outpatients to acute and community settings, interfacing with integrated care systems in the community and building important relationships.

Figure 1: Principles of integrated care



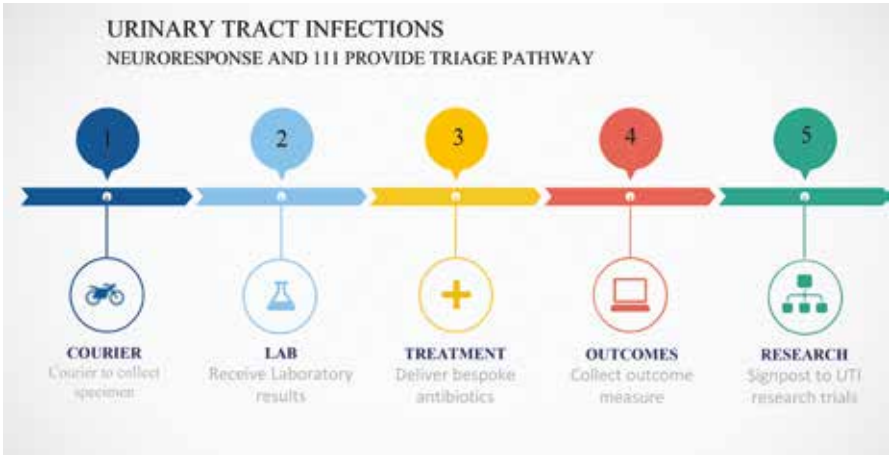


Figure 2: Neuroresponse triage pathway

Acute neurology

In London, we undertook an audit examining the delivery of neurology at a secondary care level,[5] finding no hospital offering first line assessment and admission of patients with neurologic conditions by Neurologists. Considering the mass of Neurologists at some regional centres this is notable. UCL Partners undertook an evaluation for us of "hyperacute neurology services" based on the concept of hyperacute stroke units but evaluating different models whose commonality was a reorganised front end with earlier senior input. This was trialled in four teaching hospitals, though DGH models exist.

We found that:

- A&E admissions of patients managed by the service were reduced.
- Early diagnosis improved with more appropriate use of diagnostics.
- Readmissions of patients managed by the service was reduced, partly through appropriate signposting. (e.g. patients with epilepsy were organised to attend seizure clinics)
- Inpatient transfers to tertiary neuroscience centres were reduced.

The increase in breadth of diagnosis was considerable (30 fold), but perhaps not surprising if the generalists' differential diagnosis for severe headache is subarachnoid haemorrhage or subarachnoid haemorrhage. It turned out there was no need for a hyperacute neurology unit as the actual numbers requiring admission were very small, and the concept of the next day acute medical unit round became redundant (there were few neurology patients on it).

Integrating care

Outpatient referral rates for adult neurology published by Public Health England reveal staggering variation. In Camden CCG, the rate is 2470 per 100 000 per annum and in Doncaster CCG its 147.[6] Despite this 17-fold variation the rates for unplanned admission are much the same, so you could argue that having considerable outpatient access does not prevent unplanned admission.

This is a key issue for patients with long term conditions where services have traditionally been organised around the secondary and tertiary sector. Other services e.g. therapists, social services often require a separate referral and delayed access to expert advice, particularly at times of crisis.

Explicit coordination and integration improves movement through care pathways by reducing duplication, avoiding suboptimal pathways, and minimising risk. It can also enhance prevention activity and rehabilitation. Better co-ordination reduces emergency admissions to hospital or unsafe discharge, and improves the provision of information for self-management.

The principles of integration are simple and include:

- Case ascertainment
- Care planning
- Promotion of self-management
- Risk stratification for crisis avoidance
- Community MDT working

(Figure 1).

Most of this is generic and more than deliverable for patients with neurological conditions, requiring minimal, highly specialised support. Usually the most important person in the MDT is the psychologist. An exemplar has been developed by the Thames Valley SCN who have launched a new commissioning brief[7] to support local commissioners to improve the services provided in community settings to people diagnosed with a long-term neurological condition. Ground breaking work has also come from Bernadette Porter, a Nurse Consultant who has developed a unique telephone based system "Neuroresponse" to guide patients in crisis into appropriate care settings from the outset and avoid the pinball effect where no one in multiple agencies can / will take responsibility. She has also identified urinary tract infection as a major cause of unplanned admission for patients with LTCs and is trialling community intervention (man on a bike) for early diagnosis and prevention of systemic complications (Figure 2).

Common conditions

It's easy to say that more common conditions could be managed in primary care but being a GP at present must be a great challenge with a huge raft of conditions being pushed out of the secondary care sector. Education is laudable and we and others have produced a series of video casts to guide management of common conditions. These have had thousands of views but I doubt they impact on referral rates in isolation. Referral management alone certainly delivers restriction but is a blunt tool compared to improving communication between primary and secondary care. Talking

to GPs never imbues me with confidence that we (Neurologists) as a generalisation are great at this. London has a high rate of neurology referrals to hospital outpatient departments (OPD) compared to England; 30 of 32 London CCGs have referral rates greater than the England average. We estimated that 50 to 60 percent of referrals are for common conditions, and 30 percent of these could have been managed in the community. However more appropriate models could be within the multispecialty community provider model which could:

- Improve response time and diagnosis averting the development of chronic problems.
- Reduce outpatient appointments for common conditions by 17 percent.
- Encourage rational prescribing, as specialist reviews are likely to standardise drug usage, cost effective use of available drugs (generics over branded), counsel on lifestyle impacts on the condition (e.g. migraine, manage medication overuse headache).
- Reduce ambulance callouts.
- Provide active referral management both into the integrated system and onward to secondary care.
- Allow dissemination of skills across the primary/secondary care interface.
- Co-ordinated care between primary and secondary care; improved collaboration and communication.

The near future

There remain several ongoing mechanisms for improvement. NHS England have established a National Advisory Group on Neurologic Conditions. With its leadership aligned with the neuroscience CRG this looks exciting. The "Right Care" concept is providing a significant window into local conversations with commissioners, though its output poses another round of questions. As its core principle is variability it will not produce a sting where provision is universally poor. The "Getting it Right First Time" concept also is seeking to establish a neurological conditions programme, principally working at a secondary care level. However, the key requirements for commissioners at a local level to deliver for neuroscience remain absent. We previously lobbied NHS England to get some development of this but it wasn't going to happen. The issue was prioritisation, which is a fair enough principle though still makes little sense to me on a public health basis.

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6. https://fingertips.phe.org.uk/profile-group/mental-health/profile/neurology
7. http://www.londonscn.nhs.uk/networks/mental-health-dementia-neuroscience/neuroscience/integration-and-coordination/

To list your event in this diary email Rachael@acnr.co.uk by 7th July, 2017

JUNE

Trigeminal Neuralgia Study Day for Healthcare Professionals
3 June, 2017; London, UK
www.tna.org.uk – T. 01883 370214

Non Specialist Multiple Sclerosis Masterclass – MS Academy
7-9 June, 2017; Sheffield, UK
info@neurologyacademy.org – T. 0845 338 1726 – Module 2: 12 January 2018

Overcoming Personality Disorders in Brain Injury Rehabilitation
16 June, 2017; Ely, Cambridge, UK
Rachel Everett – E. courses@ozc.nhs.uk – T. 01353 652165

British Neuro-Oncology Society (BNOS) 2017 – Engaging Science, Enhancing Survival
21-23 June, 2017; Edinburgh UK
http://www.bnos2017.efconference.co.uk

British Neuro-Oncology Society
21-23 June, 2017; Edinburgh, UK
http://www.bnos.org.uk/events/bnos-conference/

JULY

Frontiers in Traumatic Brain Injury
13-14 July, 2017, Imperial College, London
www.frontiersintbi.org

Alzheimer's Association International Conference
16-20, July 2017; London, UK
www.aaic2017.com

RehabWeek 2017
17-20 July, 2017; London, UK
www.rehabweek.org

Functional Symptoms in Neurology & Psychiatry
20–21 July, 2017; Royal Society of Medicine, London, UK
2 day meeting. www.rsm.ac.uk/events/cnh06

September

FND 2017-The 3rd International Conference on Functional (Psychogenic) Neurological Disorders
September 6-8, 2017; Edinburgh, Scotland
www.fnd2017.org

Community Brain Injury – Developing a treatment plan for cognitive, communication and emotional changes
22 September, 2017; Ely, Cambridge, UK
Rachel Everett – E. courses@ozc.nhs.uk – T. 01353 652165.

Brain Injury Rehabilitation Trust Conference
September 27-28, 2017, Glasgow, UK
www.birt.co.uk/conference

October

ILAE British Chapter Annual Scientific Meeting
4–6 October, 2017; Leeds, UK
http://www.ilaebritishconference.org.uk – E. members@ilaebritish.org.uk

10th Practical Cognition Course
October 12-13, 2017; Liverpool, UK
E. sam.pickup@lmi.org.uk – T. 0151 709 9125 ex 103) – www.lmi.org.uk/pcc

NOVEMBER

Brain Injury and Alcohol
10 November, 2017; Ely, Cambridge, UK
Rachel Everett, E. courses@ozc.nhs.uk T. 01353 652165.

Specialist Multiple Sclerosis Masterclass – MS Academy
22-24 November, 2017; Sheffield, UK
info@neurologyacademy.org – T. 0845 338 1726 – Module 2: 15 June 2018

Palliative Care MasterClass
27 November, 2017; Manchester, UK
www.neurologyacademy.org

2018

FEBRUARY

10th World Congress for NeuroRehabilitation – WCNR2018
7-10 February 2018; Mumbai, India
E: traceymole@wfnr.co.uk
www.wcnr2018.com

British Neuro-Psychiatry Association Conference

Conference details: February 22-24, 2017, London, UK. ***Report by:*** Dr Boyd Ghosh, Consultant Neurologist, Wessex Neurological Centre, Southampton and Treasurer of the BNPA. ***Conflict of interest statement:*** None declared.

The BNPA conference this year was a special one, celebrating 30 years since a small band of Neuropsychiatrists, Neurologists and Psychologists banded together to have an academic meeting. To mark the occasion, there was a special day looking at Neuropsychiatry – past, present and future.

Jonathan Bird, the founding member of the BNPA, started us off by detailing the split between Neurology and Psychiatry which he proposed started because of the opposing philosophies of clinicians like Charcot and Maudsley. This resulted in the Neurologists having a steady stream of well to do patients asking for private consultations. The separation was consolidated with Freudian views, which finally enabled the Psychiatrists to obtain some private work! In typical style Jonathan was frank and contentious in his views and referred to the aristocratic Neurologist of the time – a theme that was continued throughout the day, although no Neurologist sported a bow tie as resplendent as Jonathan's! Laura Goldstein followed him to give a moving account of Maria Wyke, a Neuropsychologist and founding member of the BNPA who died recently. She was one of the first on the executive committee for the BNPA.

Andrew Lees was invited back to talk, having last been invited in 1988. He talked about the link between anxiety and Parkinson's disease (PD) and the phenomena described by Gowers where a sudden shock could seemingly unmask PD and the possibility of chronic stress triggering PD. As usual there were lots of interesting snippets of information, but perhaps his most interesting, from an introspective point of view, is the assertion made by William Burroughs, that "Doctors are limited in their outlook…they have read all there is to read on a subject and that is that".

Due to a software hitch on Jon Stone's computer (luckily not the hardware which would be bad news for a functional Neurologist!) Michael Trimble came next, taking us from Hippocrates, through the development of EEG by Berger, to Meduna and the development of electro-convulsive therapy as a treatment for schizophrenia.

Jon Stone did eventually get his software working and treated us to a masterful account of the history of functional disorders. As he stated, they were very popular disorders in the time of Charcot and before the 1930s and then apparently disappeared from view after that until the 1990s. This was not because they had all got better but because they were all going to Neurology clinics and not to the psychiatrists! He treated us to a range of films showing us the similarities in gait between the patients in the early part of the 20th Century and now.

Anyone who has heard Ray Dolan speak will know what a tour de force he is. He managed to explain neuro-economics to us and show that his computational analysis explained the change in gambling strategies over different age groups when correlated with decline in dopamine levels, which we all suffer after the age of 20. He did this by showing us a range of mathematical equations, quelling our fears by telling us that "It is all very simple really". Despite the maths he also managed to explain why happiness does not depend on winning money – a lucky thing really if you work for the NHS.

Brian Simpson told us about neurosurgery in psychiatry and the frontal lobe operations which were done widely with no good evaluation of the side effects in the 1930s. He also postulated on the possibility of using deep brain stimulation for Alzheimer's disease, which is apparently in trial as we speak.

Modern psychopharmacology was started with Jean Delay as recounted by David Healy. He described the discovery of the benefits of chlorpromazine and went on to explain the pitfalls of psychopharmacology in relation to side effects and the rise of rating scales and the possible demise of the role of the doctor! David Linden finished off the section by detailing the various methods by which patients can communicate or control devices with their mind.

The last part of the day was explaining the training regimes for Neurology, Neuropsychiatry and Neuropsychology. It appears that everyone takes 12 years to train apart from the Neurologists who take 14 years at the moment. However Tom Hughes, chair of the Neurology Specialist Advisory Committee for Neurology, was very descriptive with 5 glasses, a tea cup and 5 bottles of water in explaining that shape of training would mean that we would all have less water…..and trainees, in the new neurology system.

The next day started with a giant in the field, Trevor Robbins. He took us through the neurobiology of addiction relating to impulsivity and compulsivity in rats and humans. A learning point for me was that addicts may well have a predisposition to impulsivity. Importantly changes in the brain occur in addicts to suggest that they have more habitual responses than goal directed behaviours therefore compounding the problem as they are cognitively less able to come off the drug.

Valerie Voon continued the theme, talking about impulsivity in Parkinson's disease. She postulated that the lack of dopamine made

patients more susceptible to the reward effects of the dopamine or dopamine agonists that they are given, therefore leading to impulsivity. Importantly, dopamine agonists can turn off autoreceptors, leading to reduced reuptake of dopamine from the synaptic cleft, therefore further potentiating the reinforcing effect of the drugs.

Killian Welsh discussed alcohol dependence and the effects of withdrawal. He showed evidence that patients were more likely to develop seizures if they had withdrawn from alcohol on more than 5 occasions. On the positive side, he stated that improvement in cognition and brain volumes do occur after abstinence and can occur even up to 7 years after giving up.

Sanjay Manohar used saccadic eye movements to explain apathy, discovering that eye movements are not confined to the main sequence and can move faster than expected and with more accuracy if suitably motivated. Apparently noradrenaline is related to the effort of an action and this was used as the explanation why dopamine agonists and cholinesterase inhibitors can be used to help patients with apathy.

Irene Tracey from Oxford was the JNNP plenary lecturer talking about Magnetic Resonance imaging in pain. She was my favourite speaker of the conference and showed clearly how a peripheral source of pain, for example osteoarthritis of a joint, can be magnified by central processes and lack of inhibition of the pain signals by the dorsal horns and midbrain. Crucially she stated that chronic pain is not all in the patient's mind but is enhanced pain. She intimated that there are drugs on the way to effectively block peripheral pain which will therefore open up the path for those central processes, divorced from a peripheral stimulus, to die away.

Three junior presentations showed us what great researchers we have in the making with subjects as diverse as: phenotypes of different types of organic psychosis; deep brain stimulation in obsessive compulsive disorder and cognitive loss in limbic encephalitis. Sarosh Irani from Oxford expanded on testing of limbic encephalitis and explained that there are a number of assays that are not clinically relevant when testing for voltage gated potassium channel antibody. Some assays detect patient immunoglobulin binding to the snake venom used as a vector rather than the channel! He therefore urged us to request LGI1 and CASPR2 antibodies only. Lastly Niels Detert, also from Oxford, discussed the widespread advantage of mindfulness training in depression, fibromyalgia, chronic pain and many other conditions. Most importantly it stands alone as a treatment which can provide cognitive benefits, although the patient may need to attend a 3 month retreat!

It is a tradition of the BNPA to have a dinner somewhere special. Previous years have been in the magic circle and the museum of comedy. This year was in the headquarters of the Order of St John in Clerkenwell, an ancient building full of history. There we were given tours and regaled by a choir while we ate and drank.

Our last day started with David Sharp discussing brain imaging and traumatic brain injury (TBI). He discussed, among other things, the finding of Tau protein deposition in sulci after injuries and the model he developed showing that, in head injury, the majority of the damaging forces acting on the brain are in the sulci. This contrasted with our third talk by Alan Carson who asserted that pathologists cannot always reliably determine chronic traumatic encephalopathy and that American football players, who have many head injuries, are on the whole healthier and have a lower risk of Alzheimer's disease than the general population. He coped admirably as first the projector stopped working and then the technicians took away his computer thereby removing his prompts for his speech. Peter Hutchinson continued the theme of TBI, albeit in patients with more severe injuries, by discussing trials looking at the early decompression of bleeds (not helpful) with late decompression, after extensive medical therapy to reduce rising intracerebral pressure, which did seem beneficial.

Nick Ward talked about stroke and the importance of starting rehabilitation as soon as possible after the onset in order to capitalise on the best chance of plasticity and recovery. Perhaps we will soon see physiotherapists called urgently to the ward to treat our stroke patients.

Martin Rossor gave the BNPA medal lecture detailing his long involvement in dementia. He described his attendance at the first BNPA meeting in 1987 when he apparently stood up and suggested it should be called the "Association of Behavioural Neurology". This did not seem to go down well for some reason! He presented his involvement in the discovery of presenilin and the surprises that he has encountered over the years: the tau mutation which presented with hippocampal atrophy and the man with posterior cortical atrophy who couldn't see static objects but could play badminton due to the preservation of visual networks for moving objects. A fascinating talk.

After lunch we were treated to a talk by Lord David Owen who trained as a doctor with rotations in Neurology and Psychiatry. He discussed the Hubris syndrome, the tendency for people with power to be corrupted. He warned us to be on the lookout for leaders who treated those who worked for them with contempt and described the previous prime ministers who he felt had Hubris Syndrome including Margaret Thatcher. His insights into the working of the Government in his role as foreign secretary were even more revealing and compelling.

The BNPA conference is always varied and stimulating and this year was no exception. The introduction of guided poster presentations really helped showcase the many posters received.

The next BNPA event is the teaching weekend on the 8-10th December 2017 aimed at registrars who wish to understand neurology and psychiatry at the points they overlap. The next conference will be held on the 1st and 2nd of March – see http://bnpa.org.uk for details.

A Dahl-icious evening: fireside with Professor Tom Solomon

Details: 'An Unexpected Evening with Roald Dahl's Doctor', Black Box, Belfast, February 17, 2017. ***Report by:*** Dr Stella Hughes, Consultant Neurologist, Belfast HSC Trust, UK. ***Conflict of interest statement:*** None declared.

Neurologist, encephalitis guru, marathon runner and now launching a new live show and book about Roald Dahl; is there anything Tom Solomon can't do? A bit like Dahl himself, known for writing best-selling books, such as *Matilda* and the *BFG*, but with a list of interests and achievements beyond that of children's fiction. As part of the Northern Ireland Science Festival 2017, Tom took to the stage to enlighten us about Dahl's extra-literary interests. And a fine stage it was, with Tom seated in a leather armchair by the fireplace, with just a few mod cons to help illustrate the salient points – a laptop, big screen and large glass of red wine. To set the scene, Tom explained how he developed a friendship with "the great author" after meeting him whilst working as a junior doctor in 1990. It was Dahl's curiosity about Tom's research that initially brought the two together and the friendship quickly blossomed. As the evening unfolded, we listened intently to the tales Tom had heard from Dahl through their regular evening encounters on the ward.

We heard how Dahl's life was marred by several significant medical encounters and loss of loved ones. Tom transported us to a world in the pre-antibiotic era, where Dahl had vividly described looking on as tragedy unfolded. Rather than crumble, Dahl developed his love of medicine, using his experiences, not only to enhance his writing but also to help advance stroke rehabilitation and medical inventions (he co-created the Wade-Dahl-Till valve for hydrocephalus). We were not subjected to a didactic lecture as Tom invited audience participation; sometimes taking on a professorial role (demonstrating the 'Stroop Effect'), other times as quizmaster with occasional retorts to silence the fellow Dahl experts in the crowd! We were by no means restless when he suggested an interval with a complimentary drink, but this is always welcomed in Belfast! Of course this was no ordinary tipple, but a Dahl-themed 'William and Mary' passion fruit cocktail to drink to his memory.

So there we were, feeling relaxed in the intimate company of our convivial host, hearing about a fascinating life and a heartening friendship between two kindred spirits. From a medical perspective, it was interesting to see how intrigued Dahl was with every aspect of medicine, curiously seeking the "sights smells sounds" of Tom's clinical experiences. It struck me afterwards how fateful it was that their worlds collided by chance and how they seemed to complement each other perfectly. As Tom quietly admitted in his book '*Roald Dahl's Marvellous Medicine*', "Just as he, a writer, has always wanted to be a doctor, so have I, a doctor, always wanted to be a writer". The book is a great read but I highly recommend seeing Tom bring Dahl to life in this unique and entertaining live show; I think his fellow writer Dahl would be very proud.

11th Cambridge Dementia Course

Conference details: December 7-9, 2016, Cambridge, UK. ***Report by:*** Oliver Cousins MBBS BSc MRCP, Neurology Registrar, Kent, UK.
Conflict of interest statement: None declared.

With the students away on Christmas holiday, Homerton College in Cambridge opened its doors to a host of Neurology Trainees and Consultants, as well as Psychiatric and Neuropsychology colleagues and a few Care of the Elderly specialists, for the 11th Cambridge Dementia Course. Whilst three long days makes this an intense course, the approximately 80 delegates were certainly well looked after. Having table service at lunch in the great hall across from world cognitive experts and candlelit dinners beside the Christmas tree, were in themselves an experience.

The bulk of the teaching comprised a formal lecture programme. However, this was broken up with a variety of DVD sessions and interactive workshops. A quiz involving matching clinical vignettes with neuroimaging and neuropsychology ran in the background of the course. Every day I would ponder and change my choices prior to the results being revealed on the final day.

The clinical assessment of the patient with cognitive impairment was covered comprehensively and a structured approach provided. The basics of matching patient symptoms to different cognitive domains and then to a suggested pathology was covered during the first day. Furthermore, the session on bedside cognitive tests improved my understanding of test interpretation and limitations, although perhaps there was a slight bias to those tests that had been developed in Cambridge. The associated neuropsychology sessions were interactive and provided a flavour for the vast range of additional assessments available.

The two DVD sessions demonstrating cognitive assessment helped to consolidate what was learnt in the lectures. The examination of speech for patients with primary progressive aphasia and vision with posterior cortical atrophy, including the use of plastic model animals, was particularly memorable and would be challenging to understand without such video aids.

Further to the general approach taught, each of the key neurodegenerative conditions were discussed separately in depth and the 'unusual dementias' and autoimmune encephalitis were not missed. Particular highlights included the talks on progressive supranuclear palsy and corticobasal degeneration by Professor James Rowe who used multiple videos to demonstrate features such as early gaze paresis and automatic grasping. The talks on frontotemporal dementia by Professor John Hodges were comprehensive, covering the different phenotypes and current advances in predicating underlying brain pathology from neurolinguistics and neuroimaging.

Neuroimaging was covered over several sessions ranging from the basics to the latest advances, such as tau imaging. The lecturer's objective to convince the audience that the role of imaging in dementia was beyond 'excluding other cerebral pathologies' was certainly met.

The management of patients with cognitive impairment was discussed throughout the course. There were lectures covering depression in dementia, behavioural symptoms and driving with dementia, as well as detailed management discussions in the individual condition lectures. On the other hand, the ethical issues that arise such as enteral feeding and end of life care in patients with dementia were not mentioned. However, hearing practical management advice from experienced clinicians will certainly be useful for my ongoing practice, especially in those conditions with a more limited evidence base.

This course was certainly a comprehensive review of the clinical assessment and management of patients with dementia and it succeeded in linking together the underlying pathology, neuroimaging and neuropsychology. I feel this course was both fascinating and relevant. I would certainly recommend it as one of the 'must attend' courses for neurology trainees.

The UCL Institute of Neurology Stroke Advanced Neuroimaging day

Conference details: November 9, 2016, London, UK. ***Report by:*** Dr Joanna Pleming, MBBS BSc, SpR ST5 Geriatrics/GIM/Stroke Medicine.
Conflict of interest statement: Dr Joanna Pleming is currently a Stroke Fellow at UCLH and employed by the Trust.

The Stroke Advanced Neuroimaging day was held in the heart of London at the Institute of Neurology. The Institute is located next to, and associated with, the National Hospital for Neurology and Neurosurgery, the largest dedicated centre for neurology and neurosurgery in the UK. This course aimed to deliver a focused review of the most commonly used imaging modalities in stroke and discuss how they are used to assess and evaluate brain infarct and haemorrhage. Within this context, there was an update on neuroanatomy as applied to neuroimaging, introductions to the application of mechanical thrombectomy and the imaging of stroke recovery.

We began the day with Professor David Werring giving a review of the clinical stroke syndromes in Applied Neuroanatomy for Stroke. Prof Werring's concise presentation made a complex topic simple, as he stressed the importance of stroke syndrome localisation. He emphasised the usefulness of differentiating between the cortical and subcortical syndromes, both to provide clues on the aetiology of the stroke and also, crucially, to help predict progression over the acute period and determine optimal place of care.

Next, Professor Rolf Jager took us on a comprehensive, whistlestop journey through the Anatomy of the Cerebral Circulation. We were whisked up the carotids and through the arterial cortical territories, coursing around the Circle of Willis to the borderzone areas and the perforators before finishing in the venous system. Particularly interesting was a detour into the embryology of the cerebral circulation, including the genetically determined anatomical variants of the vessels. One striking example was that of an absent A1 segment of the Circle of Willis, resulting in an incomplete circuit, prevalent in different populations worldwide and conveying an increased lifetime stroke risk.

Next up, Professor Xavier Golay took us through his Introduction to Imaging for Stroke. We were taken through the inner workings of the stroke imaging modalities (CT and MRI) with interest kept high with memorable and absorbing facts. It was interesting to hear that CT sensors rotate at 100 times per minute, and that MRI measures the "spin" of nuclei but that only those nuclei with an odd number of particles can spin. His comprehensive diagrams helped non-specialists understand this difficult topic. We enjoyed being taken back to basic principles to understand the rationale for differing MRI sequences as applied to stroke medicine, including different types of MRI contrast such as PD, T1, T2, T2*, DWI, MRA, PWI and SWI.

Following this, Prof Jager returned to the lectern to present Neuroimaging 1: Ischaemic Stroke. We were taken through the pathophysiology within the acute timeframe of an ischaemic stroke. Prof Jager used example images at every stage to illustrate the transition from early to late ischaemic changes, later introducing the Alberta Stroke Programme Early CT Score (ASPECTS) used in CT in MCA stroke. We learned about clinical uses of different MRI sequences in stroke and heard about ongoing research using MR in wake-up stroke. Prof Jager also spoke about uses of neuroimaging in subacute stroke, in particular in elucidating aetiology and thereby, appropriate secondary prevention.

Next was Neuroimaging 2: Haemorrhagic stroke, presented by Prof Werring. Prof Werring took a stepwise approach to this topic starting with the aetiology of haemorrhagic stroke, taking us through the most common causes, including hypertension and cerebral amyloid angiopathy through the macrovascular causes including AVM and aneurysm rupture, and into rarer causes such as inflammatory conditions. We were then taken through practical guidelines in the investigation of the cause of haemorrhage, AHA recommendations on imaging and the uses of Digital Subtraction Angiography.

A lively panel discussion followed, chaired by Prof Werring and Dr Sumanjit Gill. The audience included UK-based and international stroke practitioners, and questions ranged from the controversies surrounding use of anticoagulation in patients with haemorrhagic stroke and concurrent atrial fibrillation to access to CT-angiogram reports in district general hospitals.

After lunch we returned to a fascinating lecture by Dr Peter Cowley on Endovascular Treatment. Dr Cowley took us through the landmark trials (Mr CLEAN, EXTEND -1A, ESCAPE, SWIFT-PRIME, REVASCAT) illustrating the different devices in use. A particular highlight was a video reconstruction of the device in action. This part of the day led nicely to the interactive cases chaired by Dr Cowley.

Three case presentations from stroke trainees followed, including cases such as successful thrombectomy in a palliative cancer patient. The cases stimulated further lively discussion from the floor.

This portion of the day was followed by a very interesting lecture on Imaging Stroke Recovery by Dr Tom Hope. He detailed his work using the PLORAS database ("Predicting Language Outcomes and Recovery After Stroke") which has over 750 stroke patients and links their scores in detailed language and cognitive assessment with brain lesions on T1 MRI. He discussed three fascinating studies that used this database looking at predicting longitudinal change in chronic aphasia patients and prognoses in complex populations (including bilingual patients). Dr Hope's work provides some context to taking neuroimaging beyond the acute stroke into prognosis prediction and potentially into treatment response.

The course was extremely informative and well-presented, with engaging and knowledgeable speakers. The course materials were easily accessible and will be a useful reference tool and a comprehensive reading list was provided for further study. I think the lectures would be hard to follow without a medical background but I highly recommend this course to those interested in gaining depth in their understanding about neuroimaging as applied to stroke medicine.

PREVIEW: Advancing Epilepsy: ILAE British Chapter Annual Scientific Meeting

Join us at the 2017 International League Against Epilepsy British Chapter Annual Scientific Meeting in Leeds, England, from October 4th to the 6th. The 3-day conference features a strong scientific programme with poster and oral presentations focusing on cutting edge basic and clinical research, innovative practice techniques and scientific topics including brain maturation and seizure semiology, neurobiology of epilepsy and its clinical relevance, super refractory Status Epilepticus, metabolic disorders in epilepsy and more. In addition, the programme includes the Basic Science Free Communications session, co-hosted by Epilepsy Research UK, and the Yorkshire Research and Epilepsy Surgery Network update sessions. The aim is to provide a high quality teaching experience, with the overall objective of improving epilepsy management in Britain.

We invite submission of abstracts for posters and submissions for two Gower Awards. This is a brilliant occasion to further your professional development by sharing your research ideas, networking with colleagues and building collaborative relationships. We hope you will attend and find it interesting and instructive. 20 CPD accreditation credits from the Royal College of Physicians have been applied for. ILAE British Chapter members receive discounted registration. Join the ILAE today, visit http://ilaebritish.org.uk

To learn more and register for the 2017 meeting, visit www.ilaebritishconference.org.uk

PREVIEW: The 10th Practical Cognition Course

Conference details: October 12-13, 2017, Liverpool, UK.

The highly successful Practical Cognition Course will take place for the tenth year running on Thursday 12th to Friday 13th October 2017 at the Liverpool Medical Institution. This course is for Consultants and trainees in neurology, psychiatry, neuropsychology and rehabilitation medicine who want to develop their practical expertise in cognitive assessment and relate this to clinically relevant neuroscience.

There will be a practical introductory session to cognitive assessment followed by four sessions of case presentations discussing the assessment, diagnosis and management of common cognitive syndromes.

The course begins and ends with the patient. Case presentations will feature video material illustrating disorders that clinicians may encounter in daily practice. Each session will also include a talk from an invited expert, who will provide a framework for understanding the clinically relevant neuroscience.

This year's programme will cover DISORDERS OF LANGUAGE, FRONTAL LOBE DISORDERS and SLEEP & COGNITION. Guest speakers include Jason Warren (UCL), Rhys Davies (Liverpool), and Kirstie Anderson (Newcastle).

The course is organised by Neurologists Tim Griffiths (Newcastle), Chris Butler (Oxford) and Andrew Larner (Liverpool), has been previously sponsored by the Guarantors of Brain, and will be accredited for CME points with the Royal College of Physicians (12 points last year).

Please contact Sam Pickup – sam.pickup@lmi.org.uk or 0151 709 9125 ex 103) for more details. More details to follow on LMI website www.lmi.org.uk/pcc
Early bird registration before 1st Aug: £300 (includes course dinner).
Standard registration £350.

PREVIEW: Brain Injury Rehabilitation Trust Conference

Conference details: September 27-28, 2017, Glasgow, UK.

Be a part of BIRT's world class conference. Leading brain injury rehabilitation researchers, academics and practitioners from around the globe will be examining and sharing pioneering developments, ideas and research about the rehabilitation of people with brain injury at the BIRT Conference 2017 in Glasgow.

Day one includes keynote plenary sessions where new research in brain injury will be presented by experts in the field. On day two, choose four of the 11 workshops/symposia running simultaneously throughout the day, and participate in debate and discussion, learn new techniques and share good practice.

- Hear the latest research about emotion technology from world renowned scientist Dr Rosalind Picard, Massachusetts Institute of Technology, then discover what our teams have been doing in this field in the UK in the 'Thinking Technology' symposium.
- Join a team of 'movers and shakers' in health and rehabilitation research in our Live Research Lab, to learn and have your say about the roadmap to making brain injury rehabilitation ever better.
- Learn the current theory of executive functioning and rehabilitation from internationally recognised expert Professor Brian Levine, University of Toronto, then find out how this can be put into practice in his Goal Management Therapy workshop.
- Be inspired by Professor Jon Evans' keynote presentation about positive psychology, and his ideas for practical interventions in the workshop on Day 2.
- Question and challenge our expert panel members in our (BBC style) 'Question Time on Concussion'. We promise an enlightening and stimulating debate!
- Understand how Professor Russell Bauer, University of Florida, has matched specific memory interventions to individual patients.
- Discover how to embed ABI research project in your practice with our step-by-step guide, including handy hints and tips.
- Network informally with peers and other professionals, and share good practice during formal sessions and debates.

Full programme and online booking www.birt.co.uk/conference
Early booking discounts are still available.

PREVIEW: Frontiers in Traumatic Brain Injury 2017

Conference details: July 13-14, 2017, London, UK.

- A 2-day scientific programme at the forefront of TBI research.
- Wide range of topics
- World-renowned international speakers

We welcome you to the inaugural Frontiers in TBI conference this July, which delivers a 2-day programme of talks and debates, poster sessions and plenty of opportunity to network. Many conferences in TBI focus on a particular area; this conference brings together many topics and scientific disciplines so that delegates can get an insight into all the hot topics in TBI. Given its broad scope, this conference will be of interest to any scientific or clinical discipline working within TBI.

There is a programme of invited talks from early stage researchers as well as a faculty of internationally renowned scientists.

Profs David Sharp, David Brody and Steve Gentleman will be discussing neurodegeneration after TBI. We will have early stage researchers presenting the latest approaches to intervention. The use of big data in clinical research will be presented by CREACTIVE (Guido Bertoloni), CENTER-TBI (David Menon) and TRACK-TBI (Ramon Diaz-Arrastia). The specific issues of TBI in sports and military action will be discussed in talks by Simon Kemp, Mazdak Ghajari, Lee Goldstein, Tony Goldstone and Tony Belli. We will also be exploring the neuropsychiatric consequences of TBI with Simon Fleminger, and the medico-legal aspects of TBI assessment with Daniel Friedland. Alex Leff will be discussing e-therapy approaches to rehabilitation. Tom McMillan, Seena Fazel and Faraneh Vargha-Khadem will be presenting the wider societal impacts from TBI, including in paediatric and prison populations.

We will also be hosting two debates on the contentious issues of whether cycling helmet use should be compulsory and whether the term 'concussion' should be retired. During these debates, the audience will be invited to participate by live-tweeting their questions. This promises to be a dynamic conference, yet is also small enough to facilitate networking and encourage the formation of new collaborations.

Abstract submission for the poster competition and Early Bird registration is still open (£130/£105 students/trainees).

FULL SPEAKER LIST:

- Neurodegeneration after TBI – Prof David Sharp (Imperial College London), Prof Steve Gentleman (Imperial College London), Prof David Brody (Washington University, USA)
- TBI in Sports – Dr Simon Kemp (Head of Sports Medicine Rugby Football Union), Dr Pashtun Shahim (Washington University, USA), Mr Tony Belli (Birmingham University)
- Treatments – Dr Pete Jenkins (Imperial College), Dr Lucia Li (Imperial College), Dr Greg Scott (Imperial College)
- Blast & Biomarkers – Dr Sandra Magnoni (Granda-Ospedale Maggiore Policlinico, Italy), Dr Tony Goldstone (Imperial College), Prof Lee Goldstein (Boston University, USA)
- Big Data in TBI – Prof David Menon (Cambridge University), Guido Bertolini (Mario Negri Institute, Italy), Ramon Diaz-Arrastia (University Pennsylvania, USA)
- Modelling TBI – Dr Cornelius Donat (Imperial College), Prof Marten Risling (Karolinska Institute, Sweden), Dr Mazdak Ghajari (Imperial College)
- Psychiatry/Psychology – Dr Simon Fleminger (Imperial College), Mr Daniel Friedland (Imperial College), Prof Alex Leff (UCL)
- Paediatric/Social – Prof Tom MacMillan (Glasgow University), Prof Seena Fazel (Oxford University), Prof Faraneh Vargha-Khadem (Great Ormond Street Hospital)
- Debates – Prof Mark Wilson (Imperial College), Prof David Sharp, Prof Lee Goldstein.

For more information and how to register, please visit: www.frontiersintbi.org
@FrontiersinTBI

PREVIEW: Neurology Academy launches first Palliative Care MasterClass

After the Neurology Academy's recent successful development of separate Parkinson's, multiple sclerosis and dementia training streams, the Neurology Academy has now taken another step to launch a Palliative Care MasterClass.

Palliative care has long been a part of the Parkinson's MasterClass, and following increasing interest in this area the Academy is now expanding this subject area into a course of its own which blends cutting edge research with established clinical practice.

The MasterClass, which will be held in Manchester this winter, is specifically aimed at health professionals who are involved in the palliative care of patients with Parkinson's and dementia.

A great deal of the treatment offered to patients with Parkinson's and dementia is after all palliative in nature, so it is important that the key principles of palliation are at the heart of care delivery.

The course will highlight how for many newly diagnosed patients at the early stages of disease, some will have important palliative care needs which are important to address, such as concerns around advanced care planning (ACP). Delegates will consider how and when to consider ACP in the context of cognitive impairment.

The MasterClass will be delivered by the Neurology Academy's expert faculty, led by Dr Ed Richfield, a Consultant Geriatrician at Leeds Teaching Hospitals NHS Trust, to help advance skills and knowledge for a range of professionals, including allied health professionals, doctors (grade SpR and above), specialist nurses, community teams and GPs.

The one-day course will focus on the individual conditions and their practical management, taking time to consider commonly encountered medications and their side effects, and the particular palliative issues faced by patients in both conditions, including needs in the terminal stages.

The course will introduce specific palliative assessment tools for each condition, and look at approaches to assessment and management of common areas of unmet need, which for Parkinson's can include pain, urinary symptoms, nausea, constipation, and poor swallow. Patients with dementia also commonly experience problems with pain, as well as behavioural and psychological symptoms and impaired sleep.

The day will also include a look at service delivery, with consideration of integrated service design for neurological conditions, and an evaluation of delegates' own service delivery.

The Neurology Academy's educational model has an emphasis on interactive learning in small groups, and delegates will complete the day with a discussion about the opportunities for improvement and how examples of good practice can be spread and incorporated elsewhere.

Experience tells us however that the conversations will not stop there: as with each MasterClass delegates form an invaluable peer network to draw on for sharing expertise, advice and support long after the course is over.

Palliative Care MasterClass
27 November 2017, Manchester
Cost: £275 plus VAT
Applications to join the course are now open, for more information visit www.neurologyacademy.org

rehabweek
london '17

17 - 21 JULY 2017
LONDON, UK
QUEEN ELIZABETH II
CONVENTION CENTRE

Registration now open!

Join us at RehabWeek 2017!

Are you interested in advancing the field of Rehabilitation Technologies by exchanging your ideas and sharing your experiences with engineers, clinical researchers and clinicians from all around the world?

Then we invite you to join us in making **RehabWeek 2017** an inspiring, innovative and successful event!

The event takes place in **London, United Kingdom, from July 17th - 21st, 2017** and will bring together four conferences:

- International Neurorehabilitation Symposium (INRS), organized by the International Industry Society in Advanced Rehabilitation Technology (IISART)
- International Conference on Rehabilitation Robotics (ICORR), organized by the International Consortium on Rehabilitation Robotics (ICORR)
- Annual Meeting of the International Functional Electrical Stimulation Society (IFESS)
- British Society of Rehabilitation Medicine (BSRM) Meeting

The London RehabWeek congress will target basic science, the translation of technologies across fields and promote clinical delivery. The congress will offer strong user involvement, inspire the younger generations and galvanise experts into tackling the challenges ahead through collaboration.

Special focus on Intensity July 20th, 2017

On July 20th RehabWeek will explicitly target the topic of Intensity with lectures of RehabWeek keynote speakers **Dale Hull** and **Michelle Johnson** and a RehabWeek panel discussion on **How to Make Very Intensive Therapy Effective and Affordable** featuring patients and speakers from the fields of healthcare, insurance, industry, patient associations.

An INRS session with a discussion on **Intensity matters: How rehabilitation technology can help!** further elaborates on the topic.

In the afternoon a clinical visit will complete the day. See how the experts at **Wellington Hospital** integrate Rehabilitation Technology into their everyday clinical practice to provide their patients with highly intensive therapy.

Register now!

For more information, visit:
www.rehabweek.org

PREVIEW: RehabWeek - fostering cross-disciplinary communication

Conference details: July 17-21, 2017, Queen Elizabeth II Centre, London, UK.

What is RehabWeek?

RehabWeek is a week-long event bringing together different conferences in the field of rehabilitation technology at the same time and place, in order to foster cross-disciplinary communication and the development of relationships between different players.

RehabWeek includes common keynote lectures and other mutually organised sessions, such as panel discussions and poster sessions. In addition, each conference also organises its own, conference specific sessions. Visitors can freely choose which conference to attend at any given time.

Why RehabWeek?

As initiators and co-organisers of several conferences in the field of rehabilitation technology, we have been aware for a long time of the lack of (scientific) communication in this field between those designing and building the devices and those using them with their patients.

This lack of communication between device makers and device users is disastrous because successful inventions are always driven by the needs of the user, in our case, the clinicians and their patients.

We knew, therefore, that if we wanted to advance rehabilitation technology to the next level, a platform was needed where all players in the field could be brought to the same table, to present and exchange ideas, receive feedback on their work and build long term partnerships. RehabWeek is this platform and it allows attendees to visit several conferences and meet with their peers all in one place at the same time—instead of competing for attendees with individual conferences, RehabWeek brings them all together.

History of RehabWeek

In 2011, the IEEE International Conference on Rehabilitation Robotics (ICORR), the International Neurorehabilitation Symposium (INRS) and the International Conference on Virtual Rehabilitation (ICVR) first joined forces to bring together engineers, clinical researchers, practicing clinicians and industry representatives in one place at the same time to maximise exchange across different disciplines for the benefit of the patients. This first RehabWeek took place in Zurich, Switzerland and attracted over 650 attendees from all over the world.

The feedback from participants across societies was overwhelming and many long-lasting partnerships were formed during this exciting event.

We were therefore encouraged and conducted a second RehabWeek in 2015 in Spain, which included the INRS, the Conference on Recent Advances in Neurorehabilitation (ICRAN) and, again, the ICVR. Over 500 people enjoyed interesting lectures and stimulating debates.

RehabWeek 2017

During RehabWeek 2015, the involved societies agreed to continue the successful tradition of RehabWeek and founded an umbrella society (the International Consortium on Rehabilitation Technology, ICRT), responsible for organising and conducting this event on a bi-yearly basis. London was chosen as the location for RehabWeek 2017 and the International Functional Electrical Stimulation Society (IFESS), the ICORR and the INRS (organised by the International Industry Society in Advanced Rehabilitation Technology, IISART) committed to participate. Furthermore, the organising body decided to invite the British Society of Rehabilitation Medicine (BSRM) to join RehabWeek in 2017 as a local society representing, by and large, clinicians. We are thrilled the BSRM accepted our invitation and will be organising an extraordinary BSRM Meeting during RehabWeek (this will not replace their annual meeting, taking place in Cambridge in the Autumn of 2017).

Delegates can register on www.rehabweek.org and choose either:

- Day passes for Tuesday, Wednesday, and Thursday
- A pass for the workshop day on Monday
- A full RehabWeek pass Tuesday-Thursday.

PREVIEW: The 3rd EAN Congress

Conference details: June 24-27, 2017, Amsterdam, the Netherlands.

The European Academy of Neurology (EAN) is the organisation that unites and supports Neurologists across Europe. Currently, 47 European national neurological societies as well as 800 individuals are registered members of the EAN. Thus, the EAN represents more than 21,000 European Neurologists.

We are pleased to invite you to this great annual event on behalf of the EAN and the Netherlands Society of Neurology. Amsterdam is one of the world's most vibrant capitals, with an impressive cultural, historical, scientific and economic scene. Settled as a small fishing village in the late 12th century, Amsterdam became one of the most important ports in the world during the 17th century, the Dutch Golden Age, a result of its innovative developments in trade. Since then, it has been a leading trading and cultural city, where art, commerce, creativity and tolerance are guiding principles.

The 3rd EAN Congress in Amsterdam will provide the ideal platform for continuing education in all fields of neurology, covering a broad spectrum of topics with state-of-the-art lectures by renowned experts. The EAN strives to provide the highest quality of continuing medical education and to open professional education opportunities. The Overarching Theme of the 2017 Congress is Outcome measures in neurology.

The highlights of the congress are:

Presidential Symposium on Sunday, 25 June 2017 with 3 named lectures:

- Moritz Romberg Lecture – Epilepsy: Where do we stand? Where are we headed? – Professor Christian E. Elger, Bonn, Germany
- Camillo Golgi Lecture – Autoantibodies and the nervous system: Breadth, depth and challenges – Professor Angela Vincent, Oxford, UK
- Charles-Edouard Brown-Sequard Lecture – Why Neurologists should be interested in the human brain project: A change of clinical paradigm – Professor Richard Frackowiak, Lausanne, Switzerland

Saturday, 24 June: Opening of the Congress

Sunday, 25 June: CME Topical Symposium on Spinal Muscular Atrophy

Monday, 26 June: Outcome measures in Clinical Studies

Tuesday, 27 June: Highlight Session: Late Breaking News in neurology

Other Symposia will be:

- MDS-ES/EAN: The natural history of movement disorders
- ESO/EAN: Uncommon cerebrovascular diseases
- DNA repeat syndromes in neuromuscular disorders
- Neuroscience of sleep
- ILAE-CEA/EAN: Recent and upcoming new drugs and devices for the treatment of epilepsy
- Amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD) as a model of interaction between cognition, behaviour and motor impairment
- ECTRIMS/EAN: New developments in multiple sclerosis (MS)

Apart from that, a wide range of lectures in the fields of stroke, epilepsy, Alzheimer's and Parkinson's disease, multiple sclerosis, headache, neuromuscular diseases, and also rare and rarely diagnosed diseases will be covered. Find out about all the sessions you should not miss at our 3rd Congress in our blog: www.eanpages.org.

UCL

The First Queen Square Multidisciplinary Neuro-oncology Teaching Course

Course Directors: Dr Jeremy Rees and Dr Jonathan Martin
The National Hospital for Neurology and Neurosurgery, University College London Hospitals Foundation Trust & UCL Institute of Neurology

The need for multidisciplinary working in neuro-oncology is well established but a common theme that will be addressed is the need for better understanding between core specialities within the Neuro-oncology Multidisciplinary Team. To address this, this course has been designed for Trainees, Consultants and Clinical Nurse Specialists in the core specialities of neuro-oncology – Neurology, Neurosurgery, Clinical Oncology, Neuroradiology, Neuropathology and Palliative Care.

The Aims of the Course are:

- To introduce the basic principles and recent advances in Neuro-oncological treatments
- To develop a standardised approach to the diagnosis and classification of brain and spinal tumours using modern imaging, histopathology and molecular genetic techniques.
- To develop an understanding of the multidisciplinary management of patients with primary and metastatic brain and spine tumours, and other rarer tumours of the nervous system.
- To develop an understanding of the symptomatic management of the brain tumour patient with neurological symptoms, including palliative and end-of-life care and treatment related toxicity.
- To provide an opportunity for multidisciplinary discussion of common and rare cases

The course will be divided into four days throughout 2017-2018 and will be delivered by the consultant staff of the UCLH/UCL/National Hospital Neuro-oncology multidisciplinary team. There will be a series of lectures followed by an opportunity to discuss Case Presentations at the end of the day.

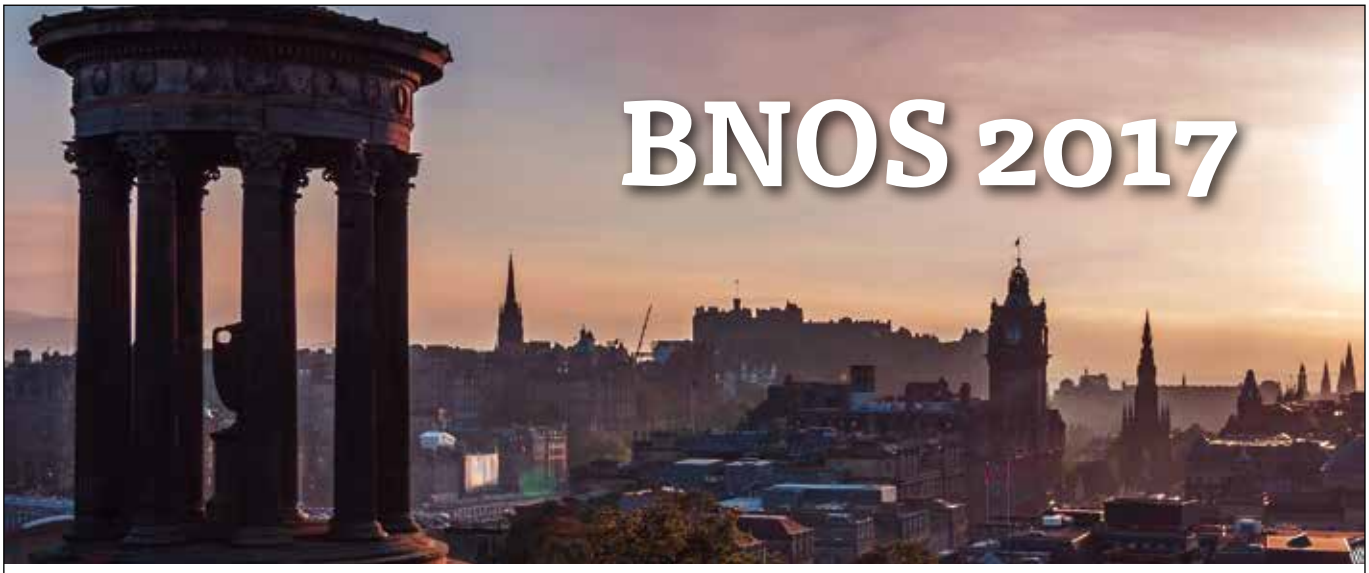
Dates:
30th November 2017 – Basic Principles – Epidemiology, Pathology, Imaging, Surgery, Radiotherapy, Chemotherapy, Biological Agents, Symptomatic treatments, End of Life care
25th January 2018 – Gliomas/Teenage Young Adult Tumours (medulloblastoma, germ cell tumours)
12th April 2018 – Benign and Metastatic Tumours – Skull Base, Pituitary, Spinal tumours, Cord compression
11th July 2018 – Leptomeningeal Metastases, Neurotoxicity, Paraneoplastic Syndromes, Rare Tumours (Primary CNS lymphoma, pineal tumours), Ethical Considerations

Course Fees

Category	Early Bird Day rate	Day rate	Early Bird Full course rate	Full course rate
Consultants	140	150	480	500
Trainees (UK)	100	120	340	400
Trainees (non UK)	120	135	400	450
Allied professionals (physios, nurses etc)	100	120	340	400
CPD points will be available				

Special offer: Free membership of BNOS (British Neuro-oncology Society) for 1 year if you book the whole course rate by November 2017.

Booking at https://www.ucl.ac.uk/ion/education/courses/other/neurooncology
For more information Email: ion.educationunit@ucl.ac.uk



Engaging Science Enhancing Survival

Wednesday 21st – Friday 23rd June 2017

Preliminary Programme and Faculty are now available

British Neuro-Oncology Society

Venue: John McIntyre Conference Centre, University of Edinburgh
Dedicated Parallel Quality of Life Sessions for Nursing staff and Allied Healthcare Professionals
Keynote Speakers: James Perry, Toronto – Late Effects and Anthony Byrne, Cardiff – End of Life Care
Welcome Reception: Dynamic Earth
Gala Dinner: Playfair Library, Old College, University of Edinburgh

Visit **www.bnos2017.efconference.co.uk** for further information – *See you there!*

@BNOSofficial

The 7th Keele Course on Headache Disorders

June 29th – July 1st 2017

University of Keele, Stoke-on-Trent, North Staffordshire, UK
Organised by the Midlands Headache Clinic
In conjunction with
the British Association for the Study of Headache

The course format will include:

- An update on the accurate recognition, diagnosis and evidence based management of common & rarer headache & facial pain disorders
- Lectures & interactive Masterclass challenging cases and "live patient" sessions to illustrate the spectrum of headache disorders
- Speakers based in UK and international specialist headache clinic and research institutions

This intensive teaching course aims to address the practical recognition and modern management of headache disorders highlighting recent advances in an interactive faculty environment. The meeting registration includes KCHD 2017 course lecture materials, 1 or 2 nights' accommodation at Keele University, all meals and course dinner in Keele Hall on the Friday.

Register at
https://www.eventsforce.net/kchd2017
In case of any problems
Email: pauline.eccleston@uhnm.nhs.uk

Keele University

BASH BRITISH ASSOCIATION FOR THE STUDY OF HEADACHE

39th Clinical Neurology Course

2nd-3rd October 2017 – University of Edinburgh

Topics will include:
• Difficult case studies
• Common problems for common neurologists
• CPC

The course is aimed at neurologists in training, but others are very welcome
The course fee and catering for both days is £250 / Monday £130 / Tuesday £120

Further details from:
http://www.ed.ac.uk/clinical-brain-sciences/postgraduate-training/edinburgh-clinical-neurology-course

Or Mrs Judi Clarke
email Judi.Clarke@ed.ac.uk

THE UNIVERSITY OF EDINBURGH

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19th national conference

Parkinson's 2017

America Square Confernce Centre, London
6th July 2017

Highlights will include:

- How far have we come in the treatment of Parkinson's? Summary of success and challenges – Professor Thomas Warner
- Psychosis and Parkinson's: aetiology and clinical significance – Dr Paul Shotbolt
- The role of neuroinflammation in Parkinson's and anti-inflammatory drugs as neuroprotective strategies – Professor David T Dexter
- Palliative and end of life care in Parkinson's – Professor Richard Walker

Our latest addition to the programme is Tom Isaacs, President and Co-Founder of the Cure Parkinson's Trust!

For more information
www.mahealthcareevents.co.uk/parkinsons2017

18th national conference

Autism Today Annual Meeting 2017

Academy of Medical Sciences, London
12th and 13th July 2017

Highlights will include:

- CBT and counselling for anxiety in young people with high functioning Autism Spectrum Disorder (ASD) – Dr Uttom Chowdhury
- Not quite Autism or ASD – Professor Samuel Stein
- Characterising the autism phenotype in females – Dr William Mandy
- Adult ADHD; recognition and management – Dr Rashid Zaman

For more information
www.mahealthcareevents.co.uk/autismtoday2017

For more information and to book your place:
☎ **Call us on +44(0)20 7501 6761**

Organised by
BRITISH JOURNAL OF HOSPITAL MEDICINE
BRITISH JOURNAL OF NEUROSCIENCE NURSING

CPD CERTIFIED

Neurokinex chosen as first UK affiliate by Christopher & Dana Reeve Foundation

A genuine breakthrough in neurological activity-based rehabilitation has arrived in the UK with Neurokinex being chosen by the Christopher & Dana Reeve Foundation as the first international Community Fitness and Wellness Affiliate of its NeuroRecovery Network (NRN).

The NRN was conceived by Christopher Reeve who believed the way forward for rehabilitation from spinal cord injury was to provide activity-based therapies that promote functional recovery. Its ground-breaking Locomotor Training and NeuroMuscular Electrical Stimulation (NMES) protocols are now at Neurokinex, Crawley, near Gatwick airport, within reach of people in the UK and Europe.

Locomotor Training harnesses the understanding that the spinal cord can interpret sensory information below the level of injury and relay signals to generate a motor response. It awakens dormant nerve pathways as participants are suspended in a harness over the treadmill while therapists move their legs to simulate walking. Simultaneously, sensory information from the legs and trunk is repetitively sent to the spinal cord, triggering the nervous system to relearn motor patterns associated with walking.

NMES targets multiple muscles during a useful movement, using parameters that activate the spinal cord. Combining this precise administering of the electrical stimulus to move the muscle or paralysed limb excites the central nervous system to promote neuroplasticity. Electrical stimuli are administered through electrodes while the individual performs an exercise to develop or strengthen pathways in the spinal cord circuitry. The programme is delivered on a specialised electrical stimulation device capable of applying pinpointed parameters for developing upper extremity, trunk and lower extremity function.

See more at neurokinex.org and christopherreeve.org/NRN.

Ready-to-use Epistatus® 10 mg Oromucosal Solution Midazolam - Marketing Authorisation granted

Special Products Ltd has announced that Epistatus® 10 mg Oromucosal Solution, Midazolam, has been granted a UK Marketing Authorisation for use in the treatment of prolonged, acute convulsive seizures in children and adolescents aged 10 to less than 18 years, who have been diagnosed with epilepsy.[1] Buccal midazolam is recommended by NICE[2] for the management of prolonged acute convulsive seizures, and is preferred by most patients and carers compared to the administration of rectal diazepam.[3,4]

Epistatus is presented "ready-to-use" in a novel, pre-filled, single-dose syringe, to provide carers with the confidence that they are administering the correct dose.[1,5] The pre-filled syringe is contained within a specially-designed, secure and tamper-evident protective packaging.

In response to market research and insights, Special Products Ltd has invested heavily in the development of this new bespoke syringe, which is designed for administration into the buccal cavity.[5] The granting of this Marketing Authorisation represents an important milestone for Special Products Ltd, for whom Epistatus is their first licensed product.

The ready-to-use Epistatus pre-filled syringe will be available to prescribe from August 2017.

1 Epistatus 10mg oromucosal solution. Summary of Product Characteristics.

2 National Institute for Health and Care Excellence (2012). *The epilepsies: the diagnosis and management of epilepsies in adults and children in primary care.* NICE clinical guideline CG 137.

3 Nakken K and Lossius M. *Buccal midazolam or rectal diazepam for treatment of residential adult patients with serial seizures or status epilepticus.* Acta Neurol Scand: (2011); 124:99-103.

4 Ashrafi MR et al. *Efficacy and usability of buccal midazolam in controlling acute prolonged convulsive seizures in children.* European Journal of Paediatric Neurology (2010); doi10.1016/j.ejpn.2010.05.009.

5 Data on file – Excerpts from Epistatus Patent Application.

Stroke patients in England set to receive revolutionary new treatment

An estimated 8,000 stroke patients a year are set to benefit from an advanced emergency treatment which can significantly decrease the risk of long-term disability and also save millions of pounds in long term health and social care costs.

NHS England has announced that it will commission mechanical thrombectomy so it can become more widely available for patients who have certain types of acute ischaemic stroke – a severe form of the condition where a blood vessel to the brain becomes blocked, often leading to long-term disability. If used within the first six hours of symptoms beginning to show – alongside other specialist medical treatment and care – the procedure has been shown in clinical trials to significantly improve survival and quality of life by restoring blood flow and therefore limiting brain damage.

Work by NHS England is now underway to assess the readiness of each of the 24 neuroscience centres across the country which are set to introduce the service. It is expected the treatment will start to be phased in later in this year with an estimated 1,000 patients set to benefit across the first year of introduction. NHS England will work with Health Education England and trusts to build on the expertise that is currently available in these specialised centres, developing the workforce and systems to enable an estimated 8,000 to receive this treatment in coming years.

Ask your patients if having children is part of their plan

UK/UKCPX/16/0031a
Date of Preparation: February 2017

TEVA UK
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