Neuropsychiatry Updates December 2025

The newsletter of the Neuropsychiatry Faculty

Editorial: Welcome back!

Dr Maytal Wolfe and Dr Killian Welch

Welcome to the 2nd edition of "Neuropsychiatry Updates", the newsletter of the neuropsychiatry faculty. After our relaunch of the newsletter last year, we heard a lot of feedback that our readers are looking for academic content, but with more focus on clinical updates and interesting case discussions. Our second newsletter attempts to be true to this vision, and we're excited to bring you an excellent update on the Role of Daridorexant in Insomnia, an interesting case of deterioration in mental state in a patient with ring chromosome 20, and a brand new "Ask the Expert" section, this edition focusing on epilepsy and dissociative seizures. We rely on great quality submissions we get from faculty members to bring you a high-quality newsletter, so please keep sending your submissions to neuropsychiatry@rcpsych.ac.uk !

Contents

Interested in contributing to a future newsletter? Contact us at neuropsychiatry@rcpsych.ac.uk

Meet the Faculty: Academic Secretary, Dr Killian Welch

Last year, with Dr George El-Nimr assuming the role of Faculty Chair, the Academic Secretary position became vacant. I was delighted to take this on, particularly when Dr Maytal Wolfe offered to act as Deputy Chair (and share the workload!). George had done a fantastic job over the preceding 10 years. Most visibly, at least from a faculty perspective, he consistently scheduled a stimulating programme for our annual conference. The quality of his programming is illustrated by the fact our annual conference consistently yielded profit, this revenue facilitating the funding of bursaries and other faculty activities.

The historic success of our annual conference inevitably brings some pressures for those taking on the organisational role, but I hope that our September meeting was a worthy successor. Programming was of course greatly assisted by other executive members and I thought every session was really strong. Thanks particularly to Dr Jonathan Bird who managed to get both Prof Mark Solms and Prof Karl Friston in a single session. That was quite a coup, and I challenge anyone to devise a session that more embodies the spirit and ambitions of neuropsychiatry than a consideration of psychodynamic thinking from a Baysian perspective. Notable mention also to Uzma Naseem and her heartbreaking description of a mother's determined efforts to seek a diagnosis and support for her child. This highlighted, in memorable fashion, the unusual cognitive profile of foetal alcohol spectrum disorder.

In terms of my background, I have been a consultant neuropsychiatrist for nearly 15 years, all of them based in Edinburgh. A little atypically, perhaps, my training included an endorsement in addictions psychiatry. I had been inspired by the addictions psychiatrist Prof Jonathan Chick as an SHO, mesmerised by the effectiveness of motivational interviewing in expert hands. I then combined a period of imaging research in schizophrenia (benefitting from the imperious but also rather nurturing supervision of Prof Eve Johnstone) with further training in psychological therapies, ultimately completing a diploma in cognitive behavioural therapy. It was special interest sessions with Prof Alan Carson that led me to neuropsychiatry. Though not by design, a combination of some familiarity with imaging and experience of delivering psychological therapies actually transpires to provide a reasonable grounding for a novice neuropsychiatrist. As I suspect is the experience of many however, even now I struggle to feel fully competent in the field. Neuropsychiatry isn't easy and there is always lots I feel I just don't know. On reflection, it is maybe a rather selfish desire to plug these lacunae that drove a desire to be Academic Secretary!

In terms of how I see this role, of course organising the annual conference is a substantial component. Together with Maytal however, we also want to re-establish the faculty newsletter as an anticipated publication, with high quality content. We welcome (importune?) faculty members to submit to it. It can, of course, only be as good as the content we are provided with and particular thanks to this issue's contributors! We also hope trainees will submit, and we will willingly provide any feedback and assistance we can to maximise

the quality of their submissions. In terms of other endeavours, we hope to launch a neuropsychiatry series in collaboration with ACNR (one of my favourite publications, having as it does a proudly clinical focus), again hoping to fill those knowledge gaps and expand the visibility of neuropsychiatry. We also plan a joint one-day conference with the Faculty of Old Age Psychiatry, provisionally scheduled for 21.5.27. Our two faculties have much potential for cross-over, so this is an exciting prospect. We would also greatly welcome proposals for our next Neuropsychiatry conference in September 2026; please just email Maytal or me. To finish, I would just like to emphasise how grateful I am to be given the chance to fulfil this role, and I hope I do it justice.

Clinical Update

The role of Daridorexant in the Management of Insomnia

Dr Ruta Kontautaite, Dr Sakh Khalsa and Dr Manny Bagary

Regional Complex Sleep Clinic

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Sleep is a critical determinant of health and well-being. Insomnia involves difficulties with initiating or maintaining sleep coupled with significant daytime consequences which compromise optimum daytime functioning. Insomnia is the most common sleep disorder in the adult population and is a major public health concern world-wide (1) with a global prevalence of insomnia symptoms of 30%-35% and insomnia disorder 3.9% - 22.1% (2) noting the heterogeneity between diagnostic systems. Insomnia markedly disrupts quality of life (3). Difficulties with initiating sleep and non-restorative sleep have been associated with an increased risk of death from all-cause mortality and in particular cardiovascular disease (4). However, other meta-analysis have not found an association between insomnia and mortality (5).

Historically, insomnia has been conceptualised as a symptom of an underlying condition such as depression, with the assumption that treatment should focus on the underlying condition to resolve insomnia which has been regarded as secondary. However, contemporary diagnostic frameworks including ICSD III, DSM V, ICD 11 use operational criteria for insomnia as a primary clinical disorder not merely a secondary symptom of another disorder. Insomnia subtypes with different phenotypes (eg hyperarousal, sleep state misperception) are subsumed into the overarching insomnia diagnosis.

Diagnostic frameworks (ICSD III, DSM V, ICD 11) have considerable overlap typically requiring subjective complaints of poor sleep despite adequate opportunity for sleep which are not explained by another sleep, mental health or medical condition. Poor sleep may include difficulties initiating or maintaining sleep or early morning waking. Disruption of daytime functioning is necessary for diagnosis and may include fatigue, depressed mood or irritability, daytime sleepiness, general malaise, cognitive impairment affecting attention, memory and concentration, impaired social, academic, occupational performance.

Acute insomnia is common, typically lasting a few days or weeks and often resolving once the triggering factor is removed (6). The duration criteria of diagnostic frameworks typically require 3 months of insomnia symptoms to establish chronicity averaging ≥ 3 days week. About 20% of short-term insomnia cases progress to chronic insomnia (7).

Table 1

Diagnostic criteria for chronic insomnia disorder according to ICSD-3 (AASM, 2014)
A. The patient reports, or the patient's parent or caregiver observes, one or more of the following:
1. Difficulty initiating sleep
2. Difficulty maintaining sleep
3. Waking up earlier than desired
4. Resistance to going to bed on appropriate schedule
5. Difficulty sleeping without parent or caregiver intervention
B. The patient reports, or the patient's parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty:
1. Fatigue/malaise
2. Attention, concentration or memory impairment
3. Impaired social, family, occupational or academic performance
4. Mood disturbance/irritability
5. Daytime sleepiness
6. Behavioural problems (e.g. hyperactivity, impulsivity, aggression)
7. Reduced motivation/energy/initiative
8. Proneness for errors/accidents
9. Concerns about or dissatisfaction with sleep
C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e. enough time is allotted for sleep) or inadequate circumstances (i.e. the environment is safe, dark, quiet and comfortable) for sleep
D. The sleep disturbance and associated daytime symptoms occur at least three times per week
E. The sleep disturbance and associated daytime symptoms have been present for at least 3 months
F. The sleep/wake difficulty is not better explained by another sleep disorder

The optimal goal of therapy in chronic insomnia (≥ 3 months) is to improve sleep and daytime functioning. For British (NICE), European and American Academy of Sleep Medicine (AASM) guidelines , CBT for insomnia (CBTi) remains the first-line treatment for chronic insomnia. CBTi has a 70% - 80% response rate and approximately 50% of people whose condition responds to CBTi experience long-term remission. Attempts have been made to improve access to CBTI using digital approaches to CBTi such as Sleepio and Sleepstation (8,9,10,11,12,13). There is insufficient evidence to recommend third wave therapies (mindfulness, acceptance and commitment therapy) as standalone interventions for insomnia. However, elements of exercise and light therapy integrated with CBT-I and may deliver additional benefits (12).

For those that do not respond to CBTi or cannot access CBTi pharmacotherapy needs to be considered. The evidence base for pharmacotherapy in Insomnia is limited. It is recommended that pharmacotherapy is restricted to short periods (≤4 weeks) to avoid tolerance and dependence (12).

Table 2

Pharmacotherapy options for insomnia (mainly without evidence base)	
Antihistamines	diphenhydramine, hydroxyzine, promethazine
Benzodiazepines	diazepam, flunitrazepam, flurazepam, lormetazepam, nitrazepam, oxazepam, temazepam, triazolam
Benzodiazepine receptor agonists	zaleplon, zolpidem, zopiclone, eszopiclone
Melatonin receptor agonists	Melatonin (immediate release and prolonged release) , ramelteon
Orexin receptor antagonists	daridorexant
Phytotherapeutics	kava-kava, valerian, lavender
Sedating antidepressants	agomelatine, amitriptyline, doxepin, mianserin, mirtazapine, trazodone, trimipramine
Sedating second generation antipsychotics	olanzapine, quetiapine

Benzodiazepines and benzodiazepine receptor agonists can have positive effects on sleep when taken up to a maximum of 4 weeks (12). Side-effects particularly with longer term use can include tolerance, dependence, nocturnal confusion and falls, impaired cognitive functioning, hangover effects with impairments in driving capability and rebound insomnia after withdrawal (14,15,16). Longer-term treatment (mostly off-label use) is not recommended but may be considered in some exceptional cases resistant to other treatment strategies preferably using intermittent dosing schedules (12).

Meta analysis datasets report sedating antidepressants are less effective than benzodiazepines and benzodiazepine receptor agonists. However, tricyclics, doxepin and

trazadone have all been shown to be helpful in the short term (12). There are no RCTs for the use of antipsychotics in insomnia either with or without comorbidities.

Whilst hypnotics such as non-benzodiazepine z-drugs (Zopiclone, Zolpidem) or prolonged release melatonin (circadin in >55yrs) can be given for management of short-term insomnia, these medications are not recommended in the management of long-term insomnia.

Hence, there has been an unmet need for pharmacological options in managing long-term insomnia. Ideal hypnotics should have rapid sleep induction, retain the normal distribution of sleep stages and architecture, promote sleep maintenance and have no residual effects in the morning. They should have no detrimental effects on memory or respiratory drive and should not be associated with tolerance, dependence, falls or interact with other drugs or alcohol. They should be safe if the patient overdoses. In search for the ''perfect hypnotic'' the orexin/hypocretin system has been of interest as it plays a key role in stabilising wakefulness and promoting arousal. Dual orexin receptor antagonists (DORAs) block the binding of wake promoting neuropeptides Orexin A and orexin B to receptors OXR1 and OXR2 which is thought to suppress wake drive. DORAs promote sleep by reducing arousing effects of the orexin system without altering the proportion of sleep stages. Suvorexant was the first DORA approved by US Food and Drug administration (FDA) in 2014. The FDA approved Lemborexant in 2019 and Daridorexant in 2022. Daridorexant is the only DORA approved for use in Europe (2022) and the UK (2023).

DORAs have been the most significant development in the pharmacological treatment of insomnia. Daridorexant may provide an efficacious option for patients as a second-line treatment option when there has been a suboptimal response to digital or face-to-face CBTi or as a first-line treatment option when CBTi is not available or unsuitable (17).

The clinical effectiveness for Daridorexant is from two phase 3 double blind RCTS study 301 and 302 and the 301 extension study 303 (18. 19) . Study 301 (n= 930) with DSM-V insomnia disorder (long -term insomnia) with an insomnia severity index (ISI) score of ≥ 15, randomly assigned to one of three groups, daridorexant 25 mg (n=310), daridorexant 50 mg (n=310) or placebo (n=310) for 12 weeks. For the 301 study, waking after sleep onset (WASO), at month 1 and month 3, daridorexant 50 mg was associated with less wake time after sleep onset from baseline compared with placebo (least squares mean [LSM] difference 22.78 minutes [p<0.0001] and 18.30 minutes [p<0.0001], respectively). Similarly, for latency to persistent sleep (LPS), at month 1 and month 3, daridorexant 50 mg was associated with a shorter delay to persistent sleep from baseline compared with placebo (LSM difference 11.35 minutes (p<0.0001) and 11.67 minutes (p<0.0001), respectively. Treatment-emergent serious adverse events were reported in 1.0% (3 out of 308) and 2.3% (7 out of 309) of people in the daridorexant 50 mg arm and placebo arm, respectively. The ethnic representation of the study group is not reflective of UK population with insomnia. Study 301 also excluded people with 'acute and unstable' mental health conditions defined in the trial as any mental health condition needing psychoactive medicine. However, in clinical practice insomnia disorder is often comorbid with mental health disorders resulting in some uncertainty about the generalisability of treatment effect in the 301 study to anticipated treatment populations (18).

Study 301 established the clinical effectiveness of the Daridorexant 50 mg dose. However, a lower 25 mg dose is available for a subjects with moderate liver hepatic impairment or those taking CYP3A4 inhibitors (18). There is no available data in patients with severe hepatic impairment, therefore, it is not recommended.

In study 302 (n= 924) people with DSM-V insomnia disorder (long -term insomnia) were randomly assigned to have daridorexant 10 mg (n=307), daridorexant 25 mg (n=309) or placebo (n=308) for 12 weeks (18).

The 303 study (19) included placebo-controlled subjective outcomes to assess the long-term safety profile and efficacy of daridorexant over a 12-month timeframe. Subjects who had daridorexant 50 mg in study 301 or study 302 continued having the same dose in study 303 (n=137). Those assigned to placebo in study 301 or study 302 were re-randomised to have either

placebo (n=128) or daridorexant 25 mg in study 303. The treatment period lasted 40 weeks with the total follow-up time from study 301 and study 303 being 12 months. Daridorexant did not induce next-morning sleepiness and no withdrawal-related symptoms or rebound were observed after treatment discontinuation. Improvements in sleep and daytime functioning were maintained through to the end of the study and were most pronounced with daridorexant 50 mg. Over a total of 52 weeks of nightly treatment (including the pivotal 12-week trials), daridorexant demonstrated a favourable long-term safety and tolerability profile consistent with the 12-week study findings. No new safety signals were reported and there was no evidence of physical dependence, tolerance or rebound. Daridorexant 50 mg showed the most favourable improvements that were sustained from the baseline in patient-reported total sleep time and daytime functioning compared with placebo.

There is a lack of evidence about longer-term treatment effects and benefit beyond 12 months. A review should be carried out at 3 months to determine benefit and periodically thereafter.

Using post hoc analysis of the global Phase 3 trials (18) to examine the treatment effect of daridorexant, on a large, randomized, dose–response, and placebo-controlled cohort of 1466 chronic insomnia disorder patients (20) it was demonstrated that daridorexant reduces features associated with hyperarousal with significant increases in probabilities to transition from wake to sleep, in addition to significant modifications of EEG spectral power in beta, alpha, and delta bands particularly at the higher 50 mg dose, both after 1 and 3 months of treatment. Taken together, these treatment-related changes underlie a possible mechanism for how DORAs, such as daridorexant, improve insomnia symptoms by reducing hyperarousal. The effects with 50 mg were consistent between months 1 and 3 and less pronounced with 25 mg.

Data from the 301 study (18) of 930 patients with insomnia disorder randomized to daridorexant 50 mg (n = 310), 25 mg (n = 310) or placebo (n = 310) for 3 months was further analysed (21) to demonstrate Daridorexant reduces wakefulness throughout the entire night whilst independently decreasing morning sleepiness and improving daytime functioning and alertness. The effects were greater at the dosage of 50mg.

A recent Phase 1 and Phase 2 placebo-controlled studies in Japanese subjects (22) were consistent with previously reported observations. Daridorexant was quickly absorbed reaching a peak plasma concentration within 1.0 h across every dose (10,25,50mg) and age group (younger men aged ~ 27yrs and older men aged ~70yrs) and cleared from plasma with a t 1/2 of ~8 h and resulted in a significant dose–response (i.e., improvement) of objective and subjective sleep onset and sleep maintenance variables in Japanese subjects with insomnia disorder, without any dose-limiting safety issues. The dose-dependent improvement in sleep maintenance (WASO) was observed across the full 8 hr night, with the effect increasing as the night progressed.

There are no reported major safety concerns, however daridorexant is contra-indicated in patients with narcolepsy and concomitant usage with CYP3A4 inhibitors. Therefore, special warnings and considerations are required for patients with sleep paralysis, cataplexy-like symptoms, hallucinations, compromised respiratory functions, worsening depression and suicidal ideation.

Summary:

Daridorexant is the most significant recent development in pharmacological management of long-term insomnia. Daridorexant is the first dual orexin receptor antagonist (DORA) to be approved in the UK and Europe for the treatment of chronic insomnia. It is an evidence-based treatment with established efficacy and safety for up to 12 months. Based on current evidence it is well-tolerated and effective. Patients treated with daridorexant have not shown any signs of rebound insomnia or withdrawal symptoms upon treatment discontinuation. However, most studies have stringent inclusion criteria, therefore, the study populations and outcomes might not represent real-world populations. Further research is needed to assess efficacy of daridorexant in

patients suffering from co-morbidities such as mental health problems, other neurological and medical conditions. The effects on different ethnic groups also need to be considered.

Please see page 15 of the newsletter for references.

Neuropsychiatry Faculty Research Prize 2024

This research prize was awarded to Dr Andrew McWilliams, Consultant Child and Adolescent psychiatrist at Royal Free London; Honorary Clinical Lecturer at King's College London. The winning paper is freely available as has been published with open access at:

McWilliams A, Bibby H, Steinbeis N, David AS, Fleming SM. Age-related decreases in global metacognition are independent of local metacognition and task performance. Cognition. 2023 Jun;235:105389. doi: 10.1016/j.cognition.2023.105389. Epub 2023 Feb 9. PMID: 36764048; PMCID: PMC10632679.

This work arose out of my academic clinical fellowship in core training, followed by my PhD project as a specialist registrar in child and adolescent psychiatry, although the research in fact deals with questions more relevant to older adulthood than to children, having a participant age range of 18 to 83 years.

I first became interested in metacognition (the ability to reflect on and control other cognitive processes, sometimes described as "thinking about thinking"), when I heard Prof Steve Fleming (Wellcome Centre for Human Neuroimaging, 12 Queen Square, UCL) speaking about his work at an RCPsych trainees' conference. I was initially struck by the challenges inherent in using self-reporting on self-confidence as a window on metacognitive ability, given my reflections from clinical work around how to interpret patients self-reporting on their experiences.

Steve led a vibrant and stimulating lab and offered me the opportunity to develop a novel, gamified measure of metacognition. We met with a number of technology firms, looking for one interested in the science rather than merely wanting to focus on the design and tech-y aspects. Ultimately, we engaged *DamnFine Ltd*, a small firm based at that time in the coffee-drinking technology hub that is Hoxton, London. We brainstormed ideas for an engaging task format, settling on a web-based task which asked the participant to navigate an alien planet, completing 2 missions to find rewards, and gave it the hopefully catchy title of *Metacogmission*. The 2 missions corresponded with the 2 cognitive domains for metacognition which we wished to research, namely metacognition of vision and metacognition of short-term memory. Studying more than one cognitive domain would allow us to explore questions around whether metacognitive judgements are best understood as operating only within individual cognitive systems, or whether metacognition should be better viewed as a domain-general ability, operating across multiple cognitive domains. The task underwent iterative development via test-driving and feedback with more than 400 people at science public engagement events, for which I won a UCL Public Engagement award in 2019, and – importantly – we involved close feedback from a mental health service user advisory group (The McPin Foundation).

Shortly after starting my higher training, I went out-of-programme to undertake a PhD on mental capacity, working with Prof Gareth Owen

as part of the Wellcome-funded Mental Health and Justice Project at the Institute of Psychiatry, Psychology and Neuroscience, King's College London, with Prof Tony David also supervising me at UCL. My PhD work sought to explore associations between mental capacity and metacognition, and a major part of this was built on using *Metacogmission* to study metacognition across the adult lifespan.

The study which was awarded this research prize involved a general population sample of 304 adults, aged 18 to 83, recruited via a scientific crowd-sourcing website. The experimental design and planned analyses were pre-registered (see Open Science Framework; https://osf.io/6t7fn/) before data collection took place. Previous research has produced conflicting evidence about how "local" metacognition (monitoring of individual judgments) and "global" metacognition (estimates of self-performance) change across the lifespan, or whether the degree to which metacognition operates in a domain-general manner changes. Our participants carried out the task remotely over the web, with real-time support offered to handle issues with task completion and other participant concerns. Our procedures used trial-by-trial staircasing of task difficulty to control subjects' task performance within a narrow range, and participants then gave confidence ratings about the quality of their own decisions after each trial. This allowed effective isolation of individual differences in local metacognitive efficiency (the accuracy with which trial-by-trial confidence ratings track performance). I analysed the data with the hierarchical Bayesian models developed by Steve Fleming, to characterise metacognition across the age range. We also asked for self-report on performance over whole tasks, a kind of "global" task confidence and hence allowing us to measure global metacognition. All analysis code is publicly available, and links to the data (on the Open Science Framework) can be found at https://github.com/metacoglab/McWilliamsBibbySteinbeisDavidFleming_AgeingMetacogmission2022

We obtained several key findings. Firstly, we found that local metacognitive efficiency was stable across the lifespan, consistent with other work showing that local metacognitive capacity is relatively preserved into older age. Notably, however, we found that metacognitive bias (participants' average confidence judgments about their performance, irrespective of accuracy) became more negative with age, as did self-performance estimates about one's ability to perform entire tasks (an aspect of global metacognition). These age-related decreases in self-confidence were not accompanied by objective changes in performance, which remained stable across the lifespan. Finally, both local and global aspects of metacognition tended to be correlated across domains, and this correlation was stable into older age. These finding are reported in full and discussed in the published paper.

Through this interesting project, I gained experience in running largescale studies with healthy participants, developed skills in programming via the mathematical coding language *MatLab,* and acquired an initial understanding of the issues surrounding entering into contracts with business partners. Being a child and adolescent psychiatrist, I was particularly pleased to find that the adolescents and even younger children who we met at public engagement events found the task fun, so I am currently working with a group In France who are using the task in school children. I would be pleased to discuss the work further with any interested readers.

The Quality Network for Neuropsychiatry Services (QN-Neuro)

Josh Coelho and Jem Jethwa, QN-Neuro Project Team

The Quality Network for Neuropsychiatry Services (QN-Neuro) launched in 2024 and is now one of nearly 30 networks within the College Centre for Quality Improvement (CCQI). QN-Neuro is a collaboration between the CCQI, clinical experts from member services, and people with lived experience of neurological conditions.

Our key objectives are to:

- Support neuropsychiatry services in evaluating themselves against agreed standards.
- Promote local clinical and service improvement aligned with those standards.
- Enable the sharing of good practice across services.

The network provides assurance to service users, carers, staff, commissioners, managers, and regulators that a service is of high quality. It also demonstrates staff are committed to improving care and raises morale and the profile of the team within the wider organisation.

Our standards

The standards used to assess services are developed with reference to current literature, best practice guidance, and in consultation with key stakeholders including service users, clinicians, service leads, and relevant professional bodies.

Currently, standards are available for inpatient neuropsychiatry services, with development underway for community/outpatient services. If you are interested in being part of the development of community/outpatient standards please get in touch with us.

Types of QN-Neuro membership

QN-Neuro offers three types of membership. All services begin with Developmental Membership:

Developmental Membership
This is the entry point for all new services. It supports services as they work towards meeting the standards required for accreditation.

Accreditation Membership
For services that meet the majority of QN-Neuro standards and have completed at least one year as a developmental member. Accreditation is a recognised badge of quality, reflecting a rigorous external assessment and high compliance with standards.

Affiliate Membership
Designed for community/outpatient neuropsychiatry services, affiliate members benefit from access to the network and peer support, with the aim of working towards accreditation in the future.

The peer review process

The process begins with a 12-week self-review, during which services assess themselves against the QN-Neuro standards. This includes online questionnaires completed by staff, patients, and carers.

A peer review team comprising at least two professionals from other QN-Neuro member services and a member of the central QN-Neuro team then visits the service to discuss and validate the self-review findings.

The data from both stages is used to produce a comprehensive local report, highlighting achievements and outlining areas for improvement.

QN-Neuro Advisory Group

The newly established QN-Neuro Advisory Group includes professionals representing key areas of expertise in neuropsychiatry. The group provides strategic direction and guidance on:

- Network development and priorities
- National policy and service context
- Methodologies underpinning the programme
- Engagement with professional bodies and patient/carer organisations
- Involvement of patients and carers
- Development and implementation of national recommendations
- Identifying funding opportunities
- Supporting the network by attending or facilitating reviews

How you can get involved

We welcome interest from any service considering QN-Neuro membership. We also run several free training sessions each year for staff from member services. These sessions prepare participants to become peer reviewers and offer valuable insight into how other neuropsychiatry services operate.

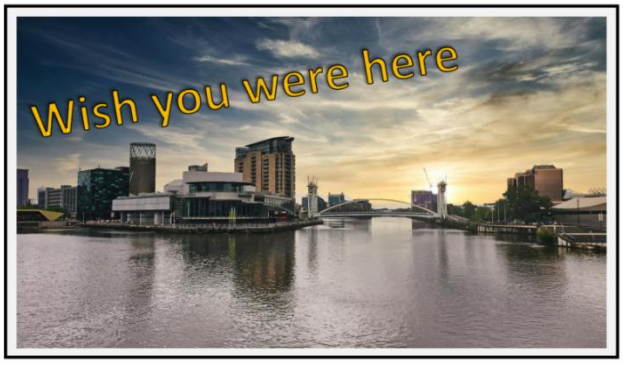
Details of upcoming events and training opportunities are available on our website.

For more information, please contact us:

Neuro@rcpsych.ac.uk
0208 618 4061
The Royal College of Psychiatrists, 21 Prescot Street, London, E1 8BB

Postcard from...Manchester!

Dr Rachel Thomasson



Manchester. The rain receptacle of the North and home to Neurology-Psychiatry collaborations for over 40 years. The 1980's were hallmarked by Neuropsychiatry colleagues Stephen Brown and David Craufurd delivering epilepsy and Huntington's disease (HD) services. Stephen eventually relocated to the Southwest and is keeping busy in retirement as a cellist in a Cornish string quartet. David is still running the HD service as a sprightly seventy-something and we will certainly miss his wise counsel when he retires next year!

Fast forward to 2025. The Neuropsychiatry service is run by Dick Hackett and I at the Manchester Centre for Clinical Neurosciences. It's one of the largest tertiary neuro centres in the world and we live in the neurology department with 50-60 consultant colleagues. They are a fantastic bunch and we are very much part of the neurology family. Sadly, my dreadful taste in fiction got me exiled from the book club but a shared interest in good wine, running and climbing enables a shred of redemption and social currency...

Dick was an epileptologist for many years and now runs general neuropsychiatry and sleep neurology clinics. He's a fantastic mentor and colleague. His interest in inherited metabolic disorders has led to a tight collaboration with the regional metabolic medicine unit and a shared interest in Neuropsychiatric complications of rare disease. I run general neuropsychiatry, sleep neurology, cognitive neurology and HD clinics. I have built a collaboration with the regional medical genetics centre to further the team's interest in the neuropsychiatry of rare diseases and with oncologist colleagues at the Christie Hospital, a regional centre of excellence for cancer care. There is a great psycho-oncology service there and it's great to share thoughts on some of the neuro cases.

The job is very broad based – we see anything and everything and it's imperative to continually read one's way out of trouble. Yesterday afternoon was assessment and treatment of motor and psychiatric complications of HD. We have a team meeting (Neurology, Neuropsychiatry, Medical genetics, Genetic counsellors) to discuss the cases of the day after clinic ends and our

colleagues from the neighbouring service in Liverpool are always welcome. Tomorrow I'm scheduled to see patients with Rosai-Dorfman disease (yes, reading up on it), tics and unexplained blackouts, MS related fatigue, paranoia and raised lead levels, presurgical assessments of patients with Parkinson's disease being considered for DBS surgery. If we need to investigate, we have access to a day case investigation unit for scanning, lumbar puncture, blood and urine tests, and muscle biopsies.

What next? I hope that in time, we can build up our ward liaison sessions and connect to the neuro-trauma unit as I think we have a lot to offer but with just the two of us it's sadly not possible. I read the postcard from Edinburgh with interest as they have clearly trailblazed on neurotrauma. Might pay a visit...

Charity Spotlight: Epilepsy Connections

Epilepsy Connections: 25 Years of Support, Awareness and Community

Epilepsy Connections was established in April 2000 by a group of passionate individuals who recognised the need for dedicated support for people with epilepsy and their families. Founded in Glasgow, we've since expanded our reach across Greater Glasgow & Clyde and Forth Valley, remaining committed to raising awareness of epilepsy and its often-overlooked impact on daily life.

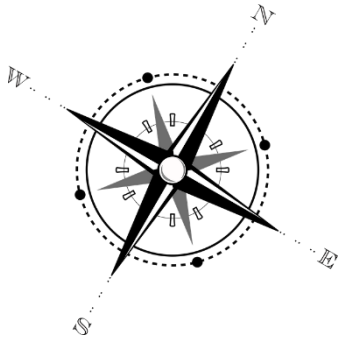
Now in our 25th year, we reflect with pride on the connections we've bult and the difference we've made – many of those we support have been with us since the beginning. Our mission has always been to help people with epilepsy develop resilience, independence, and the confidence to take charge of their condition and live well.

Our services are designed to support people through the many challenges epilepsy can bring – not only seizures, but also isolation, poor mental health, social exclusion and discrimination. We offer a range of services that foster community and connection, including a Befriending service, monthly meetups, support groups, and weekly sessions at our community allotment in Glasgow's West End. We also provide free counselling sessions to support mental wellbeing and develop coping strategies. Our fieldwork team offers one-to-one support on practical matters such as advocacy & welfare rights, employment, housing, and access to healthcare – providing a vital bridge to greater stability and quality of life.

Awareness and education are central to our work. Stigma and misunderstanding remain widespread, especially for children, who are often excluded from activities and miss out on the support they need. Our Seizure Smart awareness sessions – delivered in schools to pupils and staff – aim to build understanding from an early age and create more inclusive environments. We also organise activity trips, including regular visits to Ardentinny Outdoor Centre and, most recently, a family respite trip to Netherurd Garden House – offering fun, connection, and much-needed rest for families affected by epilepsy.

At Epilepsy Connections, we're proud to walk alongside people living with epilepsy, offering practical support, community, and hope. As we look to the future, we remain dedicated to tackling stigma, reducing isolation, and ensuring everyone affected by epilepsy has the opportunity to live a full, empowered, and connected life.

Training Journeys: Submissions from resident doctors



"In a World of Her Own": Challenges of diagnosing and managing neuropsychiatric symptoms in a patient with refractory epilepsy caused by Ring chromosome 20 r(20)

Dr Nida Munawar

This case is published with the consent of the patient's guardian.

K is a 34 old woman with r(20) mosaicism and moderate intellectual disability currently under the care of adult neurology and intellectual disability community psychiatry team. She recently presented with a deterioration in her mental state and was admitted to a tertiary residential epilepsy unit for assessment.

Background

Her mother's pregnancy was uneventful, with a normal delivery at term. She had no morphological features suggestive of an underlying genetic disorder and showed normal development until the age of five. There is no family history of intellectual disability, genetic syndromes, epilepsy or psychiatric disorders.

Seizures and anti-seizure medication

Her seizures started at the age of five and initially they were all focal. There would be posturing of her left arm with some jerking, and usually head turning to the left. She would look fearful, often pale with very sweaty palms, and towards the end there would be some shuffling as if she was sitting on something uncomfortable. The seizures were reasonably well controlled following several trials of anti-seizure medications (ASM) until the age of eleven.

Sodium valproate had to be discontinued due to thrombocytopenia, Vigabatrin gave behavioural side effects, she had an allergic reaction to Lamotrigine, and Topiramate caused worsening of the seizures, and falls, and she reported seeing "snakes" on few occasions. Carbamazepine provided optimal seizure control and was well tolerated. K was able to come off her ASM for a year around the age of eleven until she had her first tonic clonic seizure at the age of 12. At this point Carbamazepine was restarted.

Investigations

MRI brain scan at the age of six showed no abnormalities. EEG at that time showed a few bilateral generalised disturbances and, on photic stimulation, clear generalised spike wave epileptic abnormality with no clinical changes. A further EEG following the onset of tonic clonic seizures showed epileptiform discharges with a fairly generalised distribution but a clear right posterior emphasis. At the age of fourteen, based on clinical suspicion of a genetic

syndrome, karyotyping revealed ring chromosome 20 in 70 per cent of cells. (Peron et al., 2020)

Development

K attended mainstream primary school and no learning difficulties or behavioural issues were reported in her earlier years. Following the onset of seizures, as time went on she was unable to progress cognitively in line with her peers and she required additional supports. She has always lived with her parents and requires support and prompting around activities of daily living. She continues to attend a day centre twice weekly.

Progression of seizures and deterioration in mental state

K's seizures worsened in her teenage years. She would have various episodes of focal seizures, along with around six generalized tonic clonic seizures per month. She would have multiple brief focal seizures during the day and about one event per night where she would kick and shout and then be slow to settle. There are other episodes described that seem most likely to be non-convulsive status epilepticus. They occur infrequently and during this period K would stare, grasp, appear unresponsive and confused for 30-45mins. The seizures were manged with optimisation of carbamazepine and addition of clobazam.

In the recent years, K's behavioural outbursts became more concerning to her parents than the seizures. Her parents describe her to be "chatty", socialising and engaging in family activities in her younger years. She enjoyed going to the cinema, theatre and drawing. She started having difficulties in her 20s where she became quiet, withdrawn, not really coming out of her room much, not really relating or having meaningful conversations with her family and also started talking to herself. She was commenced on Citalopram by her psychiatrist and at this point some improvement was noted by the family.

Recently parents described episodes where she becomes irate and angry, slamming doors and wandering around the house. There does not seem to be a clear trigger for these episodes. There were concerns that these episodes were related to her seizures (either ictal, pre-ictal or post-ictal psychosis) and she was admitted to a tertiary residential epilepsy unit for a period of assessment and optimisation of treatment.

Assessment during admission

During her stay in the epilepsy unit, she was noted at times to be "in a world of her own". She would appear to talk to herself and be in conversation with someone and it would be impossible to have any meaningful exchange with her during this period. Her EEG during these spells was recorded to be non-epileptic in nature and at times showed an alert normal background alpha rhythm. It was noted that if she got anxious, she appeared to talk to herself much more and the exchange seemed to represent a self-soothing behaviour. It was concluded that these episodes had no link to her seizures and are driven by anxiety. At this point, Aripiprazole (which had been started by her community psychiatrist due to concerns about possible psychosis) was withdrawn, and her citalopram was continued. She continued to have frequent, regular seizures and following discussion with her parents she was commenced on Cenobomate. She was discharged after two weeks with the plan to continue to titrate Cenobomate in the community and review in the outpatient clinic.

After a few weeks, K had to be readmitted to the residential epilepsy unit following concerns raised by the parents. Her mental state had deteriorated considerably in the week prior to admission. She was not sleeping well and her behaviour was unpredictable. She was giggling to herself, very angry with her parents and just prior to admission had been trying to cut the electricity wires in the house. The impression upon admission was that K was psychotic and she was commenced on Risperidone and a

short course of regular lorazepam. Risperidone was chosen as trials of Aripiprazole and Quetiapine in the past by her Intellectual Disability psychiatrist had yielded minimal results. As the admission progressed, K appeared more settled, with no behavioural outbursts for several days. Lorazepam was gradually reduced and Risperidone was titrated to 1mg twice a day. It was discussed that as she is on Carbamazepine she will likely require higher than usual dose of Risperidone as carbamazepine induces the metabolism of Risperidone via CYP2D6 activity. Citalopram was reduced to 20mg from 30mg and she was on Cenobomate 200mg at the point. K was discharged home with the plan to titrate Cenobomate to 250 mg in four weeks.

Recent progress

K continued to do well and in her parents' view Cenobomate had greatly reduced seizure frequency. A few months later at her outpatient review parents reported new episodes occurring in the evenings or at night. On review of the video recordings taken by mum K looks dazed during those episodes. There is possible eyelid twitching , she is extremely sweaty, pale and seems frightened and agitated. Mum reports she often asks them to take away the things that are touching her as she feels someone is on her back or touching her legs. She is a bit tremulous, with lots of borborygmi, and there is some burping. The episodes can last 15-20mins. It was decided to increase the Cenobomate to 300mg as these are likely episodes of non-convulsive status epilepticus, during which K is experiencing hallucinations. Mum was also advised about judicious use of Lorazepam. She was on Risperidone 1.5mg BD at this point and no change was made to the dose.

The family had also described episodes where she feels that she needs to move all the time and is shuffling her legs when sitting down. This seemed to be akathisia, a side effect of Risperidone, but due to the significant benefit of the medication reported by the parents it was decided to closely monitor the situation with dose unchanged. K was subsequently re-admitted to the residential epilepsy unit for further assessment and optimisation of her medications as the new episodes resulted in disrupted sleep at night and behavioural disturbance during the day.

Discussion

The case highlights the challenges of discerning and managing affective and psychotic symptoms in patients with co-morbid epilepsy in the context of r(20). Vignoli et al. (2016) have demonstrated that seizures in r(20) are often focal motor seizures associated with frightening hallucinations or frequent nocturnal motor seizures, particularly when epilepsy starts in childhood, and often progress to non-convulsive status epilepticus and epileptic encephalopathy. In the above case EEG during the first admission proved invaluable in diagnostic clarity. However, this was not possible in subsequent admissions due to K's unwillingness to tolerate the EEG electrodes and challenging behaviour. The clinical picture during the second admission was more consistent with psychosis as the delusional beliefs and hallucinatory experiences were pervasive and persistent over days rather than episodic related to seizure activity. The amelioration of hallucinatory experiences, challenging behaviour and distress with Risperidone also validated the clinical diagnosis. There has also been a previous report of persistent psychotic symptoms in r(20) which did not seem related to seizure activity. (Shelton et al., 2017). Interestingly in that case too tactile hallucinations were prominent. In this case the authors speculated if forced normalisation may have been relevant to the emergence of persistent psychotic symptoms.

It can be debated that the addition of Cenobomate may have lowered the carbamazepine levels that led to the emergence of NCSE or that it was the evolving natural course of r(20) (Smith et al., 2022). An early age of epilepsy onset has shown to be a non-

modifiable poor prognostic factor (Tokumoto et al., 2025).

The case illustrates the ways that psychiatric comorbidity can complicate r(20) management. It can worsen psychosocial outcomes even when seizures are well controlled, add to diagnostic complexity and complicate ASM prescription due to concerns about behavioural side effects of medications and drug interactions. Antipsychotics are not recommended to manage hallucinatory experiences and challenging behaviour in this patient group unless they are resulting from a psychotic illness. Collaborative working between neurologists and psychiatrists with expertise in managing epilepsy patients is the key to optimising outcomes in such patient groups.

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Ask the Expert

Dr Maria Oto

Welcome to our new “Ask the Expert” feature – it’s your opportunity to ask someone in the know about the things you always wanted to know!

This edition welcomes Dr Maria Oto, consultant neuropsychiatrist and lead clinician at the Scottish Epilepsy Centre. Since 2014 she has been a part of the multidisciplinary team based at the West of Scotland Regional Epilepsy Programme. She is the chair of the Epilepsy Working Group at the RCPsych Faculty of Neuropsychiatry, and a member of the ILAE council. She has published widely and remains committed to improving access to neuropsychiatry teaching to psychiatry trainees.

Levetiracetam is a fantastic antiepileptic medication, but can be a bit demonised by psychiatrists. How much do you think it deserves its psychiatric reputation?

The potential side effects need to be considered against the fact that Keppra is a highly effective antiseizure medication and that has little interactions with other drugs.

A percentage of patients on levetiracetam will develop neuropsychiatric side effects mostly

irritability ,aggression or depression and these effects tend to be dose related . It is important to stress however that it is a small percentage (8-14%) of people that will develop these side effects

Patients with previous psychiatric history or current psychiatric symptoms are at higher risk therefore screening, informing patients of possible side effects as well as close monitoring after initiating treatment is key

It is not unusual to see that a patient is taken off Keppra because of neuropsychiatric side effects and when going through the history it turns out that they were only on very small dose and for a short time and or the neuropsychiatric symptoms are not new and were there before the start of the medication. Remember that this could be the only effective medicine for this patient.

We often assess prospective epilepsy surgery patients as part of the pre-surgery work up. Data on psychiatric complications are hard to come by however; how do you counsel patients about these risks? What psychiatric sequelae do you tend to see most often?

Following surgery particularly temporal lobectomy up to a quarter of patients will present with mood changes in particular anxiety but also emotional lability or depressed mood. These symptoms changes present within 3 months of surgery and tend to spontaneously resolve within 6 to 12 months. Within two years of surgery most patients get back to their baseline.

More severe psychiatric disorders are infrequent. About 10 % of patients will develop severe depression and 1% de novo psychosis .

Psychiatric complications are more common after temporal lobectomy and in patients who have a past psychiatric history.

I think we all appreciate depression can present a little atypically in epilepsy patients (e.g. with briefer episodes, more prominent irritability etc). Do you frame this simply as depression or do you think there is something in the concept of inter-ictal dysphoric disorder?

The most common psychiatric comorbidity in people with epilepsy are interictal depression and anxiety. The prevalence of depression in PWE in general is around 22% however the rates are far higher in patient with refractory focal epilepsy (50%) than in patients with well controlled epilepsy with rates of depression closer to the general population (10%).

The interictal dysphoric disorder (IDD) is a mood disorder that affects people with epilepsy characterized by a cluster of symptoms such as depressed mood, irritability and anxiety. The prevalence of interictal dysphoric disorder is higher than clinical depression however there is a significant overlap between both. There is no universal agreement regarding IDD as a disorder specific to epilepsy.

Depression is at times difficult to detect since some of the core symptoms can overlap with symptoms associated with having recurrent seizures or the side effects of anti seizure medication. There is a screening test which has been designed specifically to detect depression amongst patients with epilepsy (NDDIE)

The prevalence of interictal psychosis is rare amongst people with epilepsy but it is higher than in the general population. The presentation it is not dissimilar to a psychotic illness in someone without epilepsy although has a more benign course and patents suffer less negative symptoms.

In TBI treatment we often see patients who had seizures in the first few days post-injury but, though subsequently seizure-free, are continued on antiepileptics (generally Levetiracetam). What is your approach to these patients?

Post traumatic epilepsy (PTE)refers to epilepsy that is the result of a traumatic brain injury , 50 % of patients will present within the first year .

The severity of the brain injury , the presence of an Intracranial haemorrhage and early seizures all increase the risk of developing post traumatic epilepsy

Post traumatic seizures are classified as early seizures if occur within a week of the injury or late seizures if occur after the first week

There is evidence that prophylactic antiseizure medication can protect form early seizures however does not prevent late post traumatic seizures or modifies the chances of developing PTE.

In practical terms someone with a history of a brain injury that has never had seizures and that is on antiseizure medication which was originally started as prophylactic should not be on medication and would benefit from coming off this medication (following consultation with an epileptologist).

Next edition's Expert will be Dr Czarina Kirk, forensic neuropsychiatrist. Please send your questions to neuropsychiatry@rcpsych.ac.uk for next time.

Announcements

2025 Conference Award Winners

Best resident doctor/SAS poster

1st place: Dr Katherine Lynch-Kelly

Runners up: Dr Sanhita Battacharya and Dr Sirous Golchinheydari

Best medical student/FD poster

1st place: Yihui Cheng

Runners up: Michael Graczyk, Anusha Prabnhu

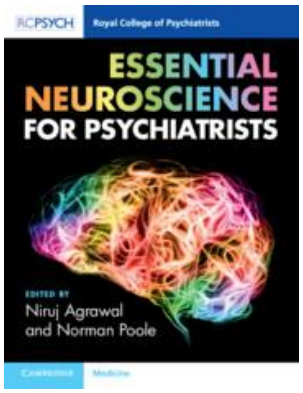
Best oral presentation

Michael Graczyk

Save the Date

The Scottish RCPsych Neuropsychiatry Faculty Conference will be in Edinburgh on 26 March 2026. More details to follow soon.

Free Member Access to Essential Neuroscience for Psychiatrists



The College Library has added Essential Neuroscience for Psychiatrists to its ebook collection, making it available to all members! *Browse the collection*

If you haven't used the online library before, you'll need to set up an RCPsych Athens account, which is also free as part of your membership. You can get in touch with our library team at: infoservices@rcpsych.ac.uk

Notice of Faculty election 2026

In 2026 there will be vacancies on the Neuropsychiatry Faculty Executive Committee for Vice-Chair, Financial Officer, and six elected executive members.
The deadline for nominations is **12 noon on Friday 6 February 2026**.
Faculty elections

Faculty prizes and bursaries

Neuropsychiatry Faculty: Medical Student Systematic Review Prize
2026 title: Huntington's Disease, how close are we to finding a cure? Please summarise and appraise recent advances.
Prize: £500
Deadline: **30 April 2026**
Prize: £250 and subsidised attendance at the faculty conference
Eligible: medical students in the UK

Neuropsychiatry Research Prize
Deadline: **30 April 2026**
Prize: £500 and subsidised attendance at the faculty conference

Eligible: Medical students, Resident doctors (in foundation years, core and higher training including non-psychiatry trainees, SAS doctors or consultants in psychiatry (within three years of their first consultant appointment), based in the UK.

Neuropsychiatry Faculty educational bursary
Deadline: available throughout the year
Bursary: up to £200
Eligible: medical students, Resident doctors (in foundation years, core and higher psychiatry training), SAS doctors, based in the UK

Neuropsychiatry Faculty: Bursary for psychiatrists from LAMI countries
Deadline**: 30 April 2026**
The Neuropsychiatry Faculty has established an annual bursary to enable a psychiatrist from a low- and middle-income (LAMI) country (defined as World Bank classification C and D) to attend the Faculty Annual Residential Meeting (usually held in September) in order to give an oral or poster presentation or deliver a workshop.

Disclaimer:

Please note that the Neuropsychiatry Faculty newsletter is intended for our membership. The content is provided by contributors and does not necessarily reflect the views, position, or policy of the College. The Royal College of Psychiatrists does not provide any endorsements or assume any responsibility for the accuracy, completeness, or suitability of the content provided.

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