GLOBAL NEUROPSYCHIATRY GROUP (GNG)

Monthly Newsletter

May 2026

Connecting minds across borders · Neurology · Psychiatry · Brain Sciences

Welcome to the May 2026 edition, bringing together the month's anonymised case discussions, group survey results, shared papers, recommended books and upcoming events. If you would like to present a case, run a survey, or contribute to a future issue, please get in touch.

In this issue

1. Case Discussions

Cases are shared with permission and anonymised. They reflect group discussion for peer learning; all clinical decisions remain with the treating team.

Case 1 — Catatonia in a young man: balancing treatment and diagnostic workup

A young man with no significant prior history developed hypokinetic catatonia over 1–2 days (some social stressors; premorbid obsessionality; a history suggestive of possible myoclonic jerks). Admitted to psychiatry with neurology input; LP and MRI arranged. Lorazepam was titrated to 16 mg/day — he remained catatonic but significantly improved, able to eat and drink. The group was asked how to balance diagnostic assessment against treatment when high-dose benzodiazepines may be suppressing seizure activity, and whether an EEG is worthwhile at this dose.

Discussion. Practice varies. One approach is to reduce or stop lorazepam 12–24 hours before EEG — highly informative where autoimmune encephalitis is in the differential — with enhanced nursing observation during the window. At 16 mg, the EEG is likely to show generalised beta throughout, limiting yield. Where ECT is anticipated, a coordinated sequence is preferred: stop lorazepam for 24 hours, perform EEG, then proceed to ECT shortly after — serving diagnostic and therapeutic goals together.

Teaching points. New-onset catatonia demands a broad, simultaneous differential (affective, psychotic, autoimmune, neurological). High-dose benzodiazepine treatment and investigation are not mutually exclusive but require deliberate sequencing; brief supervised benzodiazepine reduction before EEG is reasonable with enhanced monitoring; where catatonia is persistent or only partially

responsive, early ECT is evidence-based and time-critical.

Case 2 — Depersonalisation/derealisation disorder

A treatment-resistant DPDR case with comorbid OCD features prompted discussion. The OCD reframe: derealisation may be a downstream consequence of pervasive, undertreated OCD-driven anxiety, so treating the OCD may reduce it. A potent serotonergic strategy was favoured — clomipramine, potentially with fluvoxamine (a CYP450 inhibitor that raises clomipramine levels, allowing lower dosing; fluvoxamine also has sigma-1 activity of possible benefit in DPDR). Sustained DPDR can arise from subclinical epileptogenic activity, so EEG should be considered; an abnormal EEG would strengthen the case for lamotrigine as an add-on (not monotherapy). Psychotherapy remains the mainstay and should not be deferred.

Agreed plan. Explore OCD, migraine and seizure phenomena before any change; administer the Cambridge Depersonalisation Scale; keep olanzapine for now; consider EEG per further history; likely introduce lamotrigine as add-on; prefer fluvoxamine if an SSRI is added; and treat CBT-based psychological intervention as essential (never previously offered).

Further reading: Hunter ECM et al. Understanding and treating depersonalisation disorder (Adv Psychiatr Treat)

Case 3 — FND with shifting symptom clusters: the "whack-a-mole" phenomenon

An adult with mixed functional neurological disorder showed a relapsing, shape-shifting pattern: occupational therapy produced remission, but remission was followed by a new cluster of functional symptoms, usually without an identifiable stressor (bereavement triggered one relapse — the exception). A contributor named this the "whack-a-mole" phenomenon: resolving one functional symptom is followed by another, raising the question of whether the underlying drivers are being addressed. The group favoured a comprehensive psychological formulation — asking what the symptoms are doing for the patient and what perpetuates them. Notably, the symptoms did not confer an alternate social role (she continued usual activities during symptomatic periods), making a simple "illness-role" account less compelling.

Discussion points. Sequential symptom-cluster replacement should prompt evaluation of underlying drivers rather than surface symptoms; absence of an obvious stressor does not exclude FND (the stress–FND link is probabilistic); formulation should ask what function symptoms serve, what perpetuates them, and what illness enables or constrains; a maintained functional role is relevant but not conclusive; bereavement is a recognised relapse trigger and cultural context shapes its salience.

2. Surveys & Poll Results

Results from the group's May polls (vote counts as displayed at close), alongside an active research survey.

Active survey — Neurorehabilitation MDT: supporting autistic people after ABI (KCL)

King's College London invites neurorehabilitation professionals worldwide to the first international survey on working with autistic people following acquired brain injury. All specialities welcome; ~10 minutes; entry into a prize draw for one of ten £50 vouchers.

Survey: qualtrics.kcl.ac.uk — take the survey

Autoimmune encephalitis — global trends survey

Q1. First-onset symptoms prompting consideration of AE (multi-select, n=75 votes)

Option	Votes
Seizures	**24**
Disorders of consciousness	16
Movement disorders	13
Catatonia	12
Psychosis	6
Cognitive decline	4

Q2. Routine antibody testing in first-episode psychosis (n=34)

Option	Votes
Sometimes	**22 (65%)**
Rarely	7 (21%)
Always	3 (9%)
Never	2 (6%)

Q3. Preferred biomarker when suspecting AE (multi-select)

Option	Votes
CSF antibody testing	**39**
MRI brain	10
EEG	7
Blood antibody testing	1

The Q1 pattern is clinically significant: neurological red flags reliably prompt consideration of AE, whereas the psychiatric presentations that may be its earliest or sole manifestation — psychosis and cognitive decline — attracted far fewer votes, a known diagnostic blind spot. EEG is particularly valuable in psychiatric presentations because it provides real-time management guidance rather than confirming decisions already made.

Key points — Dalmau et al., Autoimmune Encephalitides & Autoantibodies (2026): AEs are

immune-mediated, often reversible with immunotherapy, so early recognition matters; anti-NMDAR encephalitis can present with isolated psychiatric symptoms, but purely psychiatric/cognitive presentations are only ~5%; “autoimmune psychosis” is not a validated distinct disorder — CSF antibody confirmation with supporting evidence is required; CSF testing is essential and serum-only results are a source of overdiagnosis; Hashimoto encephalitis is increasingly a diagnosis of exclusion; and recovery from prolonged states is possible even after 2–4 years.

Reference: Dalmau J et al. Autoimmune Encephalitides and Autoantibodies. Epileptic Disorders. 2026 (doi:10.1002/epd2.70041)

Post-operative delirium — assessment, screening & management

Q1. Is POD routinely screened using a validated tool? (n=22)

Option	Votes
No	**16 (73%)**
Yes — but inconsistently	4 (18%)
Yes — systematically	2 (9%)

Q2. Delirium assessment tools in use (multi-select)

Option	Votes
CAM (Confusion Assessment Method)	**10**
4AT	7
DSM-5 clinical assessment	6
CAM-ICU	2
Chart review only / none	1 / 1

Q3. Familiarity with delirium biomarkers (multi-select)

Option	Votes
None — not familiar with any	**12**
CRP	9
S100B	2
Neurofilament light (NfL)	2
TNF-alpha	1

Q4. Confidence in recognising hypoactive delirium (n=13)

Option	Votes
Moderately confident	**8 (62%)**
Slightly confident	3 (23%)
Very confident	2 (15%)

Option	Votes
Not at all / extremely confident	0

Q5. Preferred psychotropic for behavioural disturbance in delirium (excluding alcohol/sedative withdrawal)

Option	Votes
Haloperidol	**10**
Risperidone	9
Olanzapine	4
Quetiapine	3
Benzodiazepines	1
Aripiprazole	0

Routine POD screening with a validated tool is absent in nearly three-quarters of settings; hypoactive delirium (the most commonly missed subtype) attracts only moderate confidence; biomarker familiarity beyond CRP is minimal. Haloperidol and risperidone dominate and are supported by systematic-review evidence and guidelines for short-term management of agitated delirium. One contributor flagged clonidine as an overlooked option in agitated presentations.

References: Nikooie et al. Antipsychotics for delirium (Ann Intern Med 2019) · Burry et al. Cochrane Review 2018

Antidepressant choice in Parkinson's disease — when SSRIs fail

Preferred antidepressant in PD following SSRI non-response (multi-select, n=48 votes)

Option	Votes
Venlafaxine	**19**
Mirtazapine	18
Agomelatine	6
Pramipexole	3
Bupropion	2
Nortriptyline	0
Rasagiline	0

Venlafaxine (SNRI) and mirtazapine (NaSSA) together account for 77% of responses — a shared logic of dual-mechanism action beyond serotonin reuptake, consistent with network meta-analytic data favouring SNRIs and NaSSAs in PD depression, where anticholinergic load, falls and motor effects constrain choice. Agomelatine is a reasonable option (addressing PD sleep disruption without MAOI interactions); pramipexole targets the dopaminergic deficit directly. Nortriptyline received no votes

despite the strongest PD evidence (superior to paroxetine in the SOBHI trial), likely reflecting anticholinergic concern.

References: Liu et al. Antidepressants in PD: network meta-analysis (PLoS ONE 2013) · Richard et al. RCT of antidepressants in PD (Neurology 2012)

3. Papers

Selected papers shared and discussed this month, with brief take-home points. Full links are in the References section.

The paradox of the frontal lobe paradox — scoping review

The "frontal lobe paradox" describes patients with frontal damage who perform normally on standard executive tests yet show marked dysfunction in daily life. This scoping review of 28 documents found its empirical basis rests on a handful of case studies and outdated literature; Shallice and Burgess's early work was actually about building ecologically valid tests, not describing a paradox. Everyday difficulties reflect emotional instability, apathy and social-cognition impairment — not captured by lab tests — and analogous test–life discrepancies occur in personality disorder, autism and ADHD without invoking a paradox. A key medico-legal counterpoint: normal laboratory results cannot legitimately dismiss clinically observable executive dysfunction; the loss of ICD-10 categories F07.1/F07.2 in ICD-11 has practical consequences for people with acquired brain injury.

Reference: Newstead et al. The paradox of the frontal lobe paradox. Front Psychiatry. 2023

Post-stroke psychosis — systematic review

Post-stroke psychosis occurs in ~4.86% of patients — higher than previously assumed — with no established treatment guidelines. Onset is often delayed (~6 months), mirroring post-TBI psychosis; presentations include delusional disorder, schizophrenia-like disorder, and mood disorder with psychotic features, with persecutory delusions, Othello syndrome, reduplicative paramnesia and somatic delusions most frequent. Mean onset age ~66.6 years (more frequent in men); ~79.8% ischaemic; common right-hemisphere lesions (frontal, temporal, parietal, caudate; left retrosplenial on network mapping); average resolution ~3.5 months but long-term outcomes often poor. Antipsychotics are used but none formally evaluated; robust prospective trials are urgently needed.

Reference: Stangeland, Orgeta, Bell. Poststroke psychosis: a systematic review. JNNP. 2017

Research digest — selected papers

Performance of traumatic encephalopathy syndrome (TES) criteria for CTE (Nature Medicine, 2026)
Of 25 individuals meeting TES clinical criteria, only six (24%) showed CTE neuropathology at autopsy (PPV 24.0%). Clinical syndrome does not reliably map to neuropathology — a significant caution when counselling athletes and veterans after repetitive head impacts.

Mapping histamine pathway networks in the human brain (Nature Mental Health, 2026)
A connectome-level analysis of histaminergic network architecture across cognition and psychiatric disorders — relevant to sleep, attention and neuroinflammation, and an underappreciated neurotransmitter system in practice.

Cannabis and mental health: a review (JAMA Internal Medicine, 2026)

Highlights the mismatch between widespread self-management use and a thin evidence base; low-certainty evidence suggests THC-predominant cannabis may not improve PTSD, with poorly characterised long-term effects and risk of conflating patient-reported use with efficacy.

Psychotic disorders in patients with epilepsy (Epilepsia, 2013)

A practice-focused review proposing treatment procedures for interictal and postictal psychoses, aimed at the non-psychiatrist epileptologist who often meets these patients first.

Dopaminergic modulation and functional connectivity in FND (J Psychiatr Res, 2024)

The rationale for modafinil in FND may be enhancement of functional connectivity (task-positive/negative networks, cortical–cerebellar pathways) rather than purely dopaminergic — biologically plausible, but plausibility is not proven efficacy.

Peduncular hallucinosis — not always the brainstem (group discussion note)

Complex, vivid, formed visual hallucinations with preserved insight (distinguishing from primary psychosis), often at the wake–sleep transition. The pons–midbrain–thalamus "trilogy" is a useful framing: interruption of the thalamocortical visual loop by posterior-circulation stroke, cavernoma, tumour, migraine, Wernicke's or demyelination; management targets the cause, with atypical antipsychotics used where needed.

Plasma NfL and p-tau217 in neuropsychiatry (Eratne et al., Alzheimer's & Dementia, 2024–25)

Plasma NfL is ~2.5-fold elevated in neurodegenerative vs primary psychiatric disorders (AUC 0.86), useful to flag an organic aetiology; a 2025 memory-clinic study (n=341) found plasma p-tau217 most powerful (AD vs psychiatric 93%; AD vs FTD 96%), with NfL adding value for non-Alzheimer's neurodegeneration. Biomarker discordance requires multimodal interpretation.

Alzheimer's disease — drug advances and the genetic-destiny question (BMJ / The Independent, 2025)

A BMJ review maps the shift from symptomatic agents to disease-modifying anti-amyloid antibodies (lecanemab, donanemab), noting ARIA risk and that amyloid is not the whole story (tau, neuroinflammation, synaptic dysfunction). A companion piece reframes genetic predisposition as modifiable through vascular and lifestyle factors.

Brain imaging research is thriving in FND — special issue editorial (NeuroImage: Clinical, 2026)

Perez, Kozlowska and Aybek synthesise 12 articles, framing FND as both a "software" (functional connectivity/network) and emerging "hardware" (structural/microstructural) problem — a useful consolidated reading resource.

4. Books

How Doctors Think

Jerome Groopman · Houghton Mifflin, 2007.

A compelling look at the cognitive biases and affective influences that shape clinical decision-making; misdiagnosis often stems from how doctors think, not lack of knowledge. Essential reading on diagnostic reasoning.

Textbook of Neuropsychiatry and Clinical Neurosciences — 6th Edition

American Psychiatric Association Publishing, 2026.

A comprehensive reorganisation of the field's definitive reference, integrating neuropsychiatric symptoms, syndromes and treatments and updated for advances in neuroimaging, biomarkers and therapeutics.

Concise Guide to Neuropsychiatry and Behavioral Neurology — 3rd Edition

David B. Arciniegas (Ed.) · APA Publishing, 2026.

Updated for advances in neuroimaging, electrophysiology and blood/CSF biomarkers; a practical, patient-centred clinical companion.

5. Future Events

GNG–INA Monthly Academic Meeting — 25 May 2026

Chair: Wendy Phillips, Neurologist (Australia). Two clinical presentations (times AEST):

- **8:30–9:40 pm —** "An Interesting Case" — Prof Dennis Velakoulis, Neuropsychiatrist (Australia).
- **9:40–10:30 pm —** "A Complex Neuropsychiatric Case" — Chaminda Gunawardane, Psychogeriatrician (Australia).

Recordings from the May academic meeting and journal club: globalneuropsychiatry.org/webinars

Upcoming conferences 2026

Euro-Global Conference on Psychiatry, Depression & Emotional Disorders
18–19 May 2026 · Barcelona, Spain

Dedicated neuropsychiatry and brain-disorders sessions; hybrid format available.

International Neuropsychiatry Conference 2026
19–21 October 2026 · Boston, USA

Clinical advances and current research in neuropsychiatry; hybrid participation available.

2nd International Conference on Neuropsychiatry
6–7 November 2026 · Dubai, UAE

Leading experts and clinicians exploring the brain–mind interface.

6th International Conference on Neuroscience and Psychiatry
11–12 November 2026 · Bangkok, Thailand

International scientific forum on neuroscience and psychiatric research.

RANZCP Section of Neuropsychiatry 2026 Conference (GNG co-organiser)
12–15 November 2026 · QT Gold Coast, Surfers Paradise, Australia

In collaboration with the Global Neuropsychiatry Group (GNG) and SPIDD. Theme: "From neurons to narratives: the integrated science of brain and mind." Pre-conference FND workshop on 12 November.

South African Biological Psychiatry Congress 2026 (BioPsychSA / INA)
November 2026 · Century City, Cape Town, South Africa

In collaboration with the International Neuropsychiatric Association (INA). Dedicated neuropsychiatry sessions, INA workshop and AfCNP symposium.

6. References

Papers and resources cited above, with links.

1. Dalmau J et al. Autoimmune encephalitides and autoantibodies. Epileptic Disord. 2026. [link]
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3. Burry L et al. Antipsychotics for delirium in hospitalised non-ICU patients. Cochrane Database Syst Rev. 2018. [link]
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6. Newstead S et al. The paradox of the frontal lobe paradox: a scoping review. Front Psychiatry. 2023. [link]
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8. Arena JD et al. Performance of TES criteria in identifying CTE. Nat Med. 2026. [link]
9. Martens D et al. Mapping histamine pathway networks in the human brain. Nat Ment Health. 2026. [link]
10. Dopaminergic modulation and functional connectivity in FND. J Psychiatr Res. 2024. [link]
11. Garde Gonzalez J et al. Peduncular hallucinosis: clinical characteristics, etiology, and a case report. Eur Psychiatry. 2024. [link]
12. Eratne D et al. Plasma and CSF neurofilament light distinguish neurodegenerative from primary psychiatric conditions. Alzheimers Dement. 2024. [link]
13. Eratne D et al. Plasma p-tau217, NfL, GFAP diagnostic performance across AD, FTD and psychiatric disorders. Alzheimers Dement. 2025. [link]
14. Perez DL, Kozlowska K, Aybek S. Brain imaging research is thriving in FND (special issue editorial). NeuroImage Clin. 2026. [link]
15. Hunter ECM et al. Understanding and treating depersonalisation disorder. Adv Psychiatr Treat. [link]

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