GLOBAL NEUROPSYCHIATRY GROUP (GNG)

Monthly Newsletter

June 2026

Welcome to the June 2026 edition. This issue brings together the month's survey results, academic case presentations, clinical discussions, shared literature, and upcoming events — compiled from the group's conversations in chronological order. If you would like to run a survey, present a case, or contribute an item for a future issue, please get in touch. Happy learning!

In this issue

1. Survey & Poll Results

Several poll series ran through June. Results are summarised below (vote counts as displayed at close). These trends help the group identify areas of consensus, uncertainty, and educational need.

Survey series: Epilepsy & ADHD

Q1. Preferred pharmacological agent for a patient with stable epilepsy recently diagnosed with ADHD.

Votes: Methylphenidate 15 (group preference), Atomoxetine 5, prefer none 5, Guanfacine 1, Modafinil 1, other 1; Dexamfetamine and Clonidine 0.

Take-home: Methylphenidate was the clear group preference, consistent with the shared literature (Wiggs 2018, Brikell 2019, Santos 2013) and ILAE 2018 guidance that stimulants — MPH in particular — are not contraindicated in well-controlled epilepsy. A notable minority would use no pharmacological agent, and thorough assessment before starting was emphasised.

Poll series: Mild traumatic brain injury (mTBI)

A ten-question teaching series on mTBI; consensus answers and vote counts summarised below.

- **Q1 Definition:** brain injury with GCS 13–15 and mechanically-caused neurological dysfunction (18).
- **Q2 LOC required to diagnose:** False (24).
- **Q3 Routine imaging may be normal:** True (24).
- **Q4 Develop persisting post-concussion symptoms:** 10–15% (9).
- **Q5 Commonest neurological symptom:** fatigue (9).
- **Q6 New psychiatric disorder in ~20–30% within 12 months:** True (12).
- **Q7 Commonest psychiatric disorders:** depression (12), GAD (6), PTSD (4).
- **Q8 Commonest headache type:** tension-type (5).
- **Q9 Predictors of delayed recovery:** pre-existing psychiatric history, older age, PTA duration, poor social support, low expectation of recovery.
- **Q10 FDA-approved biomarker:** GFAP, partnered with UCH-L1 (1).

Note: The FDA-cleared blood biomarker panel for acute mTBI comprises GFAP and UCH-L1 (used together to help decide whether CT is needed). GFAP was the group's selected answer; UCH-L1 is its established partner. S100B is used in some European guidelines but is not FDA-approved for this indication.

Poll: Non-affective fatigue in Parkinson's disease

"What about non-affective fatigue of PD — patient already on Madopar, other non-pharmacological measures not helping. What's your choice of drug?"

Votes: Modafinil 4 (top choice), Pramipexole 1, methylphenidate 1; venlafaxine, doxepin, rasagiline and others 0.

Poll: Clozapine, weight loss & levels

"A man on clozapine loses 10 kg (dedicated exercise and dietary change), takes his medication regularly, nothing else changed, and begins to notice increased paranoia. You do levels — what do you expect?"

Votes: "Levels will go down" 10 (unanimous); up 0; no change 0.

Explanation: Clozapine is highly lipophilic with a large volume of distribution. Loss of adipose reserves can reduce plasma levels and precipitate symptomatic worsening — the same principle would apply to GLP-1–induced weight loss. See the shared Journal of Psychiatry & Neuroscience report on weight loss reducing clozapine levels.

Poll: Genetic testing in ID and/or ASD

Q1. Do you routinely conduct genetic screening for individuals with ID and/or ASD?

Votes: No 16; Yes 0 — no respondent routinely screens genetically in ID/ASD.

Take-home: No respondent routinely performs genetic screening in ID/ASD — highlighting a potential gap, given that recognised microdeletion syndromes (e.g. 22q11.2) commonly present with neuropsychiatric phenotypes and are frequently missed in adults.

Survey series: DiGeorge (22q11.2 deletion) syndrome

A survey framed around DiGeorge syndrome as a prototype for neuropsychiatric disorders. Vote counts as displayed at close.

Q1. Have you seen a case of DiGeorge syndrome?

Votes: Yes 17; No 5.

Q2. Which common neuropsychiatric disorders do you know the condition to present with? (select one or more)

Votes: intellectual disability 13, schizophrenia 12, ASD 10, ADHD 5, anxiety 5, epilepsy 5, mood 4; Parkinson's 0.

Q3. Which physical or medical features do you most commonly associate with DiGeorge syndrome? (select one or more)

Votes: heart defects 10, palatal anomalies 6, hypoparathyroidism/hypocalcaemia 5, dysmorphic facial features 5, thymic hypoplasia 3.

Take-home: 22q11.2 deletion syndrome is the most common microdeletion syndrome and among the most clinically variable (>180 associated features). The group most readily linked it to intellectual disability and schizophrenia, and, physically, to congenital heart defects — mirroring the CATCH-22 mnemonic (Cardiac, Abnormal facies, Thymic hypoplasia, Cleft palate, Hypocalcaemia). Its wide neuropsychiatric phenotype makes it a useful prototype for thinking about genotype–phenotype links in neuropsychiatry.

2. General Academic Discussions

A summary of the month's wide-ranging academic threads. Contributors are named where they shared expertise; patient details are anonymised throughout.

mTBI as a neuropsychiatric disorder

A shared presentation framed mTBI as a significant, under-recognised neuropsychiatric condition. Key points: DTI is not endorsed by any guideline as a diagnostic tool (findings depend heavily on control-group choice); cognitive deficits beyond three months should prompt screening for functional cognitive disorder (e.g. the Cabreira tool); and ICD-11 has removed post-concussion syndrome, with DSM-5 not

carrying forward the DSM-IV research criteria. Consensus favoured treating psychiatric and neurological sequelae separately rather than under a PCS umbrella, plus ED head-injury liaison nurses, psychoeducation and signposting.

Modafinil, fatigue and apathy: mechanisms and evidence

Prompted by the PD-fatigue poll, members debated whether wake-promoting agents genuinely treat fatigue and apathy or merely unmask sleep deprivation. Apathy, anergia and fatigue can each arise from dysfunction in effort-based decision-making, with dopaminergic and noradrenergic systems exerting overlapping but distinct effects — which may explain why an agent helps fatigue but not motivation. Evidence: reasonable support for methylphenidate in apathy, weaker for modafinil; the only head-to-head fatigue trial (TRIUMPHANT-MS) found amantadine, modafinil and methylphenidate no better than placebo, with a signal only in excessive daytime sleepiness. A pragmatic MS-fatigue ladder (amantadine → modafinil → methylphenidate → dexamfetamine, after excluding reversible causes) was shared.

Sleep assessment in neuropsychiatric populations

Members stressed that fatigue/apathy assessment must include a thorough sleep evaluation. Suggested tools: SDS-CL-25 as a broad screener, with ESS, STOP-Bang, RIS, PSQI and the Sleep Wake Bed Partner Questionnaire for targeted use, and onward investigation (sleep diaries, actigraphy, oximetry, polygraphy, PSG, MSLT) as indicated. Common fatigue scales: FAS, FSS, MFIS.

Sex differences in Alzheimer's disease and ageing

A shared JAMA figure explored why women show a greater tau burden alongside greater clinical resilience, then faster decline after symptom onset — with menopause and X-chromosome biology proposed as contributors. A linked Babraham Institute seminar on hormetic pathways broadened this to the sex bias across ALS, PD, AD and hearing loss.

Autoimmune encephalitis in rapidly progressive dementia (RPD)

A Netherlands prospective multicentre cohort (2019–2024, n=147 adults with RPD) found autoimmune encephalitis the commonest treatment-responsive cause of RPD (39% of cases; 61% of treatable diagnoses), led by anti-LGI1, seronegative AE and GFAP astrocytopathy, with CJD among leading non-treatable causes. Learning points: think autoimmune first; new-onset seizures warrant autoantibody testing; early movement disorders shift suspicion from CJD; high CSF tau does not exclude AE; seronegative AE mimics neurodegeneration and delayed immunotherapy worsens outcome; paired serum and CSF testing is essential. A member contributed an anti-LGI1 case with brachiofacial dystonic seizures mistaken for hypnic jerks that responded to corticosteroids.

Prolonged and unrecognised catatonia

A thread on the longest catatonia members had seen drew striking examples — several years' duration (one misdiagnosed as FND) responding dramatically to lorazepam or, in a learning-disability setting, to ~18 ECT sessions. Under-recognition was linked to diagnostic overshadowing, staff turnover and families' relationship with services.

Klüver-Bucy syndrome: phenotype and forensic relevance

A shared systematic review of Klüver-Bucy syndrome (KBS) — 157 cases — characterised by hypersexuality, hyperorality, hyperphagia, visual agnosia, hypermetamorphosis and emotional blunting from bilateral temporal dysfunction. Hypersexuality and hyperorality were most prevalent (84%); leading aetiologies CNS infection, TBI and autoimmune. Discussion distinguished hypermetamorphosis (temporal) from utilisation behaviour (frontal), and noted KBS is too rare for population-level offending — its forensic relevance lies in individual post-encephalitis/TBI disinhibition, with implications for culpability.

Functional neurological disorder: functional visual loss

A shared systematic review and meta-analysis of functional visual loss (44 studies, n=2284) found associations with trauma, ocular comorbidity, headache and other functional disorders; prognosis was heterogeneous (better in paediatric studies) and treatment data limited. A related query explored egodystonic, sometimes suppressible tic-like or compulsive urges to act harmfully, possibly commoner in comorbid Tourette syndrome.

DiGeorge (22q11.2 deletion) syndrome as a neuropsychiatric prototype

Prompted by an adult case, the group discussed DiGeorge / 22q11.2 deletion syndrome — the commonest microdeletion syndrome and one of the strongest genetic risk factors for schizophrenia — as a neuropsychiatric prototype. Because genetic testing predates the 1990s, undiagnosed older adults may present through psychiatric services with intellectual disability, psychosis, ASD, ADHD, anxiety or mood disorders. A shared report (Kraus et al., 2018) noted psychiatric symptoms follow distinctive developmental trajectories, with incidence rising into adulthood — supporting consideration of 22q11.2 in adult evaluations. A companion poll showed no respondent routinely screens genetically in ID/ASD.

Autism and acquired brain injury: an international clinician survey

A newly joined trainee neuropsychiatrist and PhD student introduced the first international survey of clinicians' experiences working with autistic people who have sustained an acquired brain injury (ABI). Co-designed with autistic people with lived experience, it has King's College London ethical approval, takes ~10 minutes, and offers a prize draw (ten £50 vouchers): survey link. Members were encouraged to share it; one offered to circulate it via the RADiANT network (see Papers).

Other threads in brief

- **Parkinson's ICDs, neuroimaging and DBS:** the long-term aspiration of neuroimaging-based screening to personalise drug/stimulation choice, and caution about proprietary DBS tractography tools (deterministic vs probabilistic) whose development data are not released.
- **Cocaine vaccine (Calixcoca):** a forwarded item on the Brazilian synthetic anti-cocaine vaccine drew interest but appropriate scepticism — several prior anti-cocaine vaccines have not translated into clinical effectiveness, and achieving antibody titres high enough to "soak up" cocaine would be demanding.
- **CJD (Heidenhain variant) & LATE:** a member presented a Heidenhain-variant CJD case (rapidly progressive neurodegeneration), alongside discussion of limbic-predominant age-related TDP-43 encephalopathy (LATE) as a late-age TDP-43 proteinopathy.

- **Forensic brain injury — apathy:** management options canvassed for post-injury apathy in complex forensic settings included modafinil, methylphenidate and atomoxetine (low dose), bupropion, low-dose aripiprazole, lamotrigine, emerging lithium evidence, memantine, neuromodulation (TMS) and, above all, intensive neurorehabilitation.

3. Neuromodulation

A growing strand of the group's work spans functional neurosurgery, VNS, rTMS and at-home tDCS/tACS. This section gathers the month's discussions, a new member in the field, an anonymised case, and upcoming events.

Psychiatry and neurosurgery: cutting through stigma

A shared guest editorial ("Cutting through stigma: psychiatry and neurosurgery working together") argued that functional neurosurgery can modulate the brain circuits underlying psychiatric symptoms — safe and evidence-based for select disorders (e.g. refractory OCD, depression) and promising for others — yet referrals remain strikingly low relative to the burden of refractory illness. The authors call for closer psychiatry–neurosurgery collaboration and de-stigmatisation of neurosurgical options.

New member: a vagus nerve stimulation (VNS) clinic for depression

The group welcomed Tiago Costa, who runs a vagus nerve stimulation (VNS) clinic for treatment-resistant depression. VNS sits alongside rTMS and functional neurosurgery in the escalating ladder for refractory mood disorder; Tiago will speak on VNS at the GNG Neuromodulation Therapy Day (below).

Anonymised case: exploring neuromodulation in later-life cognitive impairment

A member sought opinion on a patient in their mid-70s with mild cognitive impairment and a question of early neurodegeneration, in whom neuromodulation was being considered. Imaging prompted debate: FDG-PET showed the cerebellum relatively spared (a reference region preserved in Alzheimer's), while structural imaging showed some bifrontal and temporal atrophy on the GCA scale — though neuroradiology felt "nothing reeks of a neurodegenerative process," cautioning against over-reading age-expected change.

Management options canvassed: an acetylcholinesterase inhibitor (e.g. donepezil) if a neurodegenerative process were supported; guanfacine as a pro-cognitive agent (with a rationale for combining it with a stimulant); and rTMS, including a dual-site precuneus + left DLPFC protocol (referencing Bionics Institute / Prof Kate Hoy work). Members stressed an accurate formulation before committing to a neuromodulation pathway. Contributors: Zubair Khan, Raman Sakhuja and Shobit Garg.

At-home tDCS/tACS devices: promise and pitfalls

A practical thread examined the expanding market of at-home tDCS/tACS devices (Flow Neuroscience, Mave Health, Plato). Enthusiasm for accessible neuromodulation was tempered by safety concerns over cheap, unregulated stimulators (e.g. via general marketplaces) and by potential conflicts of interest where a device is gated behind a prescriber with a commercial stake. Consensus: promising, but needing regulation, dosing standards, oversight and transparency.

Neuromodulation events & education

- **Maudsley Neurotechnology (Neuromodulation) Meeting — 18 June 2026:** included Prof Marcus Kaiser on transcranial ultrasound stimulation and network-informed targeting.
- **"Neuromodulation for Psychiatry" webinar — 9 July 2026, 8:00 pm:** an introductory online session on neuromodulation techniques and their psychiatric applications.
- **GNG Neuromodulation Therapy Day — 17 December 2026, 8:00 am–4:00 pm, London (6 hours CPD):** a full-day meeting spanning rTMS, tDCS/tACS, VNS and functional neurosurgery, with Tiago Costa speaking on VNS for treatment-resistant depression. See the Events section for the full listing.

4. Child Neuropsychiatry

A new section reflecting the group's growing paediatric neuropsychiatry discussion. Cases are anonymised; contributors are named where they shared expertise.

Anonymised case: recurrent post-febrile neuropsychiatric episodes in a 13-year-old (PANDAS/PANS spectrum)

A member presented a 13-year-old boy with recurrent, episodic neuropsychiatric symptoms, each following a febrile illness. Episodes (from age 8) featured post-pharyngitis irritability, hypersomnolence, school refusal and, in one, marked skin excoriation to bleeding; ASO titres were raised in earlier episodes (up to ~800 IU/mL) and symptoms responded to azithromycin, oral steroids and penicillin prophylaxis. In the current episode ASO was negative with throat swab showing Gram-positive cocci in pairs. MRI, EEG, an autoimmune panel and CSF were all normal.

The post-pharyngitis pattern, raised ASO and antibiotic/steroid responsiveness raised a PANDAS/PANS-spectrum possibility, though atypical — dominated by hypersomnolence, withdrawal, irritability and school refusal rather than OCD or tics — and the presenter sought views on workup, immunomodulation and differentials.

Group discussion: Members agreed the child did not fully meet classic PANDAS criteria but that atypical cases occur. The main differential offered was recurrent depression (a low-dose SSRI and a family-history enquiry suggested), though the strictly post-febrile, steroid-responsive episodes pointed away from a primary mood disorder. Support for PANDAS/PANS included high ASO and steroid responsiveness; anti–basal ganglia antibodies were suggested, with options of infection vigilance, NSAIDs or short steroid courses, antibiotic prophylaxis (potentially to age 21) and tonsillectomy — IVIG/plasmapheresis felt unnecessary given steroid response. A neurometabolic differential was debated but argued against by normal early development. Contributors: Praveen Kumar, Jacob Mays and Srikanth.

Take-home: post-infectious, relapsing–remitting neuropsychiatric change in a child warrants a PANDAS/PANS-spectrum differential even when OCD/tics are absent — with mood disorder and inborn errors of metabolism as key alternatives. Steroid responsiveness and paired ASO/throat findings guide immunomodulatory strategy; anti–basal ganglia antibodies and a metabolic screen

can help where the picture is atypical.

Excoriation behaviour as a marker of a PANS flare

Complementing the case, a member shared a published report — "Scratching the Surface: Excoriation Behavior Revealing an Underlying PANS Flare" (Ragunathan, Veeraraghavan, Kaushik, Sharma; AIIMS Guwahati) — in which prominent skin-picking heralded a PANS flare with raised ASO, normal imaging/EEG and steroid/antibiotic response, highlighting excoriation as an under-recognised presenting feature (see Papers). An inborn-errors-of-metabolism overview was also circulated for the neurometabolic differential.

5. Featured Article

A member-contributed article, condensed for the newsletter. The full manuscript (with complete reference list) is available from the author on request.

The missing variable in behavioural escalation in autism: why caregiver emotional regulation deserves clinical attention

By Vasavi Yellamraju

Abstract. *Behavioural escalation in autism — aggression, dysregulation, self-injury — is among the hardest aspects of care and is well studied from the child's side (triggers, behavioural strategies). Caregivers' own emotional regulation as they respond has received far less attention. This article argues caregiver emotion regulation is an under-researched but clinically important variable that belongs in parent-mediated interventions.*

Behavioural escalation and caregiver burden

In autistic children, aggression, self-injury, dysregulation and destructive behaviour vary from episodic to chronic and reflect interacting biological, psychological and environmental factors; episodes carry real risk (restraint, ED attendance, trauma). Persistent challenging behaviour erodes caregiver wellbeing — stress, financial strain, fatigue and isolation — compounded by limited behavioural support and heavy caregiving demands.

Emotion regulation: a missing variable

Caregiver stress is well documented, and targeting it improves family wellbeing — but stress and emotion regulation differ. Emotion regulation (Gross) is how people influence which emotions they feel, when, and how they express them. During escalation caregivers feel fear, guilt, frustration or distress; regulating well means reading the child's behaviour as distress-driven, keeping self-control and enabling de-escalation. Caregivers who regulate their own emotions co-regulate the child better through calm communication and reduced demands.

Clinical implications

Current approaches centre on child vulnerabilities (triggers, environment, routines, behavioural strategies) and are not designed to help caregivers manage their own responses in crisis. Assessing caregivers' emotional responses and training them in emotion regulation could tailor interventions,

build resilience and ease burden — strengthening family co-regulation and, emerging evidence suggests, the child's outcomes. This shifts focus from child behaviour modification toward the caregiver–child relationship at moments of distress.

Future directions and conclusion

Key questions remain: how caregivers regulate emotion during escalation, which strategies work, and how these change with experience. Research has favoured caregiver stress and behavioural outcomes over real-time emotional processes; longitudinal and observational methods could advance the field. A fuller understanding could improve parent-mediated interventions, caregiver training and family-centred autism services alike.

Keywords: *autism; behavioural escalation; caregiver burden; emotion regulation; family-centred care*

Selected references

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6. Case Discussions (Anonymised)

Cases are presented with identifying details removed. They reflect collective clinical opinion shared for peer learning and do not constitute individual patient advice.

Case 1 — Parkinson's disease with nocturnal compulsive eating

A patient in their late 60s with ~10 years of Parkinson's disease (Madopar three-times daily plus an evening dose, rasagiline, an AChEI) developed three months of compulsive, binge-type night-time eating ~4–5 hours after the last levodopa dose, with mild mood/anxiety symptoms and mild cognitive impairment — distressing and compulsive rather than hunger-driven.

Group discussion: Most favoured an impulse-control disorder (ICDs occur even at low doses; rasagiline and levodopa both possible culprits), with the nocturnal pattern raising an "off"-related phenomenon. Suggested: stop rasagiline, reduce/remove the night levodopa dose, use the lowest agonist dose, verify adherence, and treat the eating behaviourally as for binge eating (small frequent meals, partner support). The self-help book *Overcoming Binge Eating* (Fairburn) was recommended.

Case 2 — Hypoxic brain injury with depression and apathy (forensic)

A patient with hypoxic brain injury presented with depression and prominent apathy on mirtazapine 45 mg nocte (sertraline not tolerated), with a background of substance use and a significant forensic history. The clinician asked about modafinil for apathy given a history of aggression.

Group discussion: Antidepressants were felt to add little. Options included low-dose methylphenidate or aripiprazole, modafinil, bupropion (if no seizure risk), atomoxetine and low-dose lamotrigine, with low-dose stimulants reasonable but cautious. Members stressed the greatest gains come from intensive neurorehabilitation and neuromodulation, and flagged post-discharge prescribing and ADHD-service access as hurdles.

Two further teaching cases discussed this month — clozapine levels falling after significant weight loss, and prolonged unrecognised catatonia — are covered under the Survey & Poll Results and General Academic Discussions sections respectively, and are not repeated here.

7. Papers & Resources Shared

Summaries of the literature and resources circulated this month, with links where available. Full citations are collated in the References section.

Reward sensitivity in Parkinson's patients with binge eating (Parkinsonism Relat Disord, 2018)
In PD, binge eating on dopaminergic therapy tracked with reduced working memory and higher depression scores rather than heightened food-reward salience — implicating cognitive mechanisms.
Shared by: Gareth McGuigan

Structural connectivity of discrete networks underlies impulsivity and gambling in Parkinson's disease (Brain, 2019;142(12):3917)
High-resolution imaging links weaker structural connectivity of the response-inhibition network to impulse-control disorders and pathological gambling in PD.
Shared by: Philip Mosley

Mental disorders as homeostatic property clusters: a narrative review (JAMA Psychiatry, 2026)
Fried reframes mental disorders as homeostatic property clusters — fuzzy, probabilistic clusters of biopsychosocial properties rather than natural kinds — recasting heterogeneity, comorbidity and transdiagnostic effects as expected.
Shared by: Vivek Misra

Modafinil alters decision-making based on feedback history (randomised, placebo-controlled study; PubMed 27649777)
A single 200 mg modafinil dose biased choice toward previously rewarded behaviour, suggesting a dopamine-mediated effect on feedback-based decision-making.
Shared by: Philip Mosley

A common alteration in effort-based decision-making in apathy, anhedonia and late circadian rhythm (eLife, reviewed preprint)
Links late chronotype to altered effort-based decision-making shared across apathy and anhedonia, implicating circadian rhythm in dimensional apathy.

Shared by: Philip Mosley

TRIUMPHANT-MS: amantadine, modafinil and methylphenidate for MS fatigue (Nourbakhsh et al., Lancet Neurology, 2021)

Randomised crossover trial (n=141): no benefit of amantadine, modafinil or methylphenidate over placebo on MS fatigue; a post-hoc signal only with excessive daytime sleepiness.

Shared by: Harry Costello

Female neuropathologic risk and clinical resilience in Alzheimer disease (JAMA, 2026)

Women show greater tau burden at equivalent amyloid, greater clinical resilience, then faster post-onset decline — with menopause and X-chromosome biology proposed as drivers.

Shared by: Jasvinder Singh

Turning stress into survival: hormetic pathways and sex-specific ageing (Babraham Institute seminar)

Seminar on whether sex differences in stress-response and senescence underlie women's longevity yet earlier onset of some inflammatory conditions.

Shared by: Vicky Wing

The neuropsychiatric phenotype of Klüver-Bucy syndrome: a systematic review of reported cases (J Neuropsychiatry Clin Neurosci, 2026)

Systematic review (141 articles, 157 cases): hypersexuality and hyperorality most prevalent (84%), bilateral temporal lesions in 73%; leading causes CNS infection, TBI and autoimmune.

Shared by: Jesus Ramirez & colleagues

Functional visual loss: a systematic review and meta-analysis of epidemiology, prognosis and treatment (Eye, 2026)

Systematic review/meta-analysis (44 studies, n=2284): associations with trauma, ocular comorbidity, headache and other functional disorders; heterogeneous prognosis, limited treatment data.

Shared by: Neil Ramsay (co-author) / Jasvinder Singh

Weight loss leading to reduction in clozapine levels and symptomatic worsening (Journal of Psychiatry & Neuroscience)

Significant adipose loss can lower plasma levels of highly lipophilic clozapine, risking relapse — the basis for the clozapine teaching poll.

Shared by: Raman Sakhuja

Limbic-predominant age-related TDP-43 encephalopathy (LATE) (Alzheimer's & Dementia)

TDP-43 proteinopathy of late age (>80), formerly hippocampal sclerosis of ageing — an important differential in older-age cognitive decline.

Shared by: Chaminda Gunawardana

GNG academic case handout: Creutzfeldt–Jakob disease (Heidenhain variant)

GNG academic case presentation on Heidenhain-variant CJD (rapidly progressive neurodegeneration); recordings to follow pending approval.

Shared by: Dr Chaminda Gunawardana

DiGeorge syndrome: relevance of psychiatric symptoms in undiagnosed adult patients (Wien Klin Wochenschr, 2018;130(7–8):283–287)

Short report: DiGeorge (22q11.2 deletion) syndrome carries high rates of intellectual disability, schizophrenia and ADHD; undiagnosed adults may first present via psychiatric services, symptoms rising into adulthood.

Shared by: Shared to accompany the DiGeorge survey

Methylphenidate treatment and risk of psychotic disorder (JAMA Psychiatry, 2026)
Finnish birth-cohort study (n≈697,000): no overall increase in psychotic-disorder risk with methylphenidate in ADHD, and a possible protective effect of sustained childhood treatment.

Traumatic brain injury in a forensic intellectual disability population (Psychology, Crime & Law, 2017;23(5):400–413)
Self-reported TBI in a forensic intellectual-disability population — a companion to the autism-and-ABI survey on ABI risk in neurodevelopmental groups.

Shared by: Verity Chester

Scratching the Surface: Excoriation Behavior Revealing an Underlying Pediatric Acute-onset Neuropsychiatric Syndrome Flare (case report)
AIIMS Guwahati case report in which prominent skin excoriation heralded a PANS flare (raised ASO, normal imaging/EEG, steroid- and antibiotic-responsive) — excoriation as an atypical presenting feature.

Shared by: Vishnupriya Veeraraghavan

Neurologic inborn errors of metabolism: pathophysiology, manifestations, evaluation and management (overview)
A 30-page overview of inborn errors of metabolism, circulated as a companion resource for the neurometabolic differential in the paediatric post-febrile case.

Shared by: Srikanth

8. Upcoming Conferences & Events

Selected upcoming meetings relevant to this month's topics (dates verified at time of publication; please confirm on the organiser's website before making arrangements).

GNG Academic Meeting

GNG Monthly Academic Meeting — register here
Thursday 30 July 2026 · 11:30 am–1:30 pm UK / 8:30 pm–10:30 pm AEST · Online (please verify your local time zone)
The next GNG monthly academic meeting features Dr Rowena Mobbs (neurologist, Mater Hospital, Sydney) on "Bedside to bench: clinical learnings in probable CTE," and Prof. Juan Carlos Urizar (geriatric neuropsychiatrist, Brigham and Women's Hospital / Harvard Medical School, Boston) on "Neuropsychiatry of Normal Aging." Session chair to be confirmed. Register via the link above; contact contact@globalneuropsychiatry.org or see www.globalneuropsychiatry.org.

Conferences

Biological Psychiatry Congress 2026 (BIOPSYCHSA / SASOP) — registration
26–29 November 2026 · South Africa (in-person)

The South African Society for Biological Psychiatry congress: pre-congress workshops on 26 November — including an International Neuropsychiatry Association (INA) Neuropsychiatry Update and the AfCNP Symposium — followed by the main congress 27–29 November. Early-bird registration closes 31 July 2026.

RANZCP Section of Neuropsychiatry 2026 Conference
12–15 November 2026 · QT Gold Coast, Surfers Paradise, Australia

Presented by the RANZCP Section of Neuropsychiatry in collaboration with SPIDD and the Global Neuropsychiatry Group (GNG). Theme: "From neurons to narratives: the integrated science of brain and mind," covering neuropsychiatric and neurocognitive disorders, brain injury, sleep, neurostimulation/neuromodulation and cognitive rehabilitation, with a pre-conference FND workshop on 12 November.

MDS International Congress of Parkinson's Disease and Movement Disorders
4–8 October 2026 · COEX, Seoul, Korea

The flagship movement-disorders meeting — highly relevant to this month's Parkinson's impulse-control-disorder and DBS threads.

39th ECNP Congress
10–13 October 2026 · Munich, Germany

Europe's leading neuropsychopharmacology congress — relevant to the apathy/fatigue, ADHD and psychopharmacology discussions.

American Epilepsy Society (AES) Annual Meeting
4–8 December 2026 · Denver, Colorado, USA

Comprehensive epilepsy science and practice — relevant to the Epilepsy & ADHD survey and stimulant-safety literature.

9. New Books in Neurology & Neuropsychiatry

Recently published or newly updated titles relevant to the group's interests.

Textbook of Neuropsychiatry and Clinical Neurosciences, 6th Edition
Ed. Adrian Preda. American Psychiatric Association Publishing, 2026.

The flagship neuropsychiatry reference, fully updated — with chapters spanning the neuropsychiatry of traumatic brain injury and of Parkinson's disease / dementia with Lewy bodies, directly relevant to this month's mTBI and movement-disorder threads.

Textbook of Traumatic Brain Injury, 3rd Edition
Eds. Jonathan M. Silver, Thomas W. McAllister, David B. Arciniegas. American Psychiatric Association Publishing.

Comprehensively revised, with new chapters on biomechanical forces, biomarkers, neurodegenerative dementias, suicide and social cognition — a strong companion to the mTBI survey and discussion.

Textbook of Geriatric Neuropsychiatry, 3rd Edition
Eds. Sepideh Bajestan, Gaston Baslet, Alan Carson. American Psychiatric Association Publishing.

Updated edition covering the neuropsychiatry of later life, including traumatic brain injury — co-edited by GNG contributor Alan Carson.

Also mentioned in discussion: *Overcoming Binge Eating* (2nd Edition) by Christopher G. Fairburn — a structured self-help programme recommended in the Parkinson's nocturnal-eating case discussion.

10. References

Literature and resources cited above, with the studies underpinning the Epilepsy & ADHD stimulant-safety survey.

1. Loane C, et al. Reward sensitivity in Parkinson's patients with binge eating. Parkinsonism Relat Disord. 2018. [link]
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Epilepsy & ADHD survey — supporting evidence

Supporting evidence (stimulants in epilepsy/ADHD): Wiggs 2018 and Brikell 2019 (registry analyses — no increased seizure risk); Man/Hollis 2020 (transient first-month risk only); Liu 2018 and Santos 2013 (safe/effective); ILAE consensus (Auvin 2018); bidirectional ADHD–epilepsy risk (Chou 2013, Hesdorffer 2004).

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