GNG Monthly Discussion: Oct 24

Thank you all for being a vital part of the GNG community. This is our second monthly discussion roundup, designed to help everyone revisit the summary of conversations we've had throughout October 2024. We are also working on a newsletter with INA.

Papers shared

Thanks to everyone for sharing. Some of the links are attached. All the papers will be available under the educational section of the GNG website; you can log into the members section to see them here: https://globalneuropsychiatry.org/members-area/educational-resources/

Topics Discussed

1. Dementia
2. Parkinson Disease
3. Neuroimmunology
4. Neuromodulation
5. Catatonia
6. Primary Psychiatric Disorders Functional Neurological Disorder
7. Neurodevelopmental Disorders
8. Sleep
9. Neuroanatomy
10. General Neuropsychiatry
11. Books

Neurodegeneration

1. Aging-related Pathology in consecutive posts.pdf

Aging-related Pathology in consecutive posts.pdf

Prof. Sheldon shared that one of our alumni recently forwarded an intriguing paper from Sweden. The study involved 1,610 unselected subjects who underwent post-mortem examinations for various causes. Remarkably, only 2% of these subjects did not have hyperphosphorylated tau (hpTau). Most exhibited multiple proteinopathies, and very few had none at all. Unfortunately, it seems we all accumulate pathological proteins as we age.

2. Itching Frequency and Neuroanatomic Correlates in Frontotemporal Lobar Degeneration

Itching Frequency and Neuroanatomic Correlates in Frontotemporal Lobar Degeneration.pdf

Itching of undetermined origin may indicate the presence of a neurodegenerative process. In this study, a higher occurrence of itching was observed in patients with frontotemporal

degeneration spectrum disorder compared with Alzheimer disease.

3. **Plasma and CSF neurofilament light chains distinguish neurodegenerative from primary psychiatric conditions in a clinical setting.**
   Plasma and CSF neurofilament light chains distinguish neurodegenerative from primary psychiatric conditions in a clinical setting.pdf
   Dr Dhama Eratne shared this paper for interest and as a follow up from his talk a couple of months ago, where he presented some of this work. Our study has just been published, where we found strong diagnostic performance of NfL as a screening blood test for neurodegeneration, particularly in younger people, to distinguish dementia from primary psychiatric disorders in a clinical setting.

4. **Blood Biomarkers to Detect Alzheimer Disease in Primary Care and Secondary Care**.
   Blood Biomarkers to Detect Alzheimer Disease in Primary Care and Secondary Care.pdf**.**
   Potential for early detection and better clinical care!

   Study Aim:
   * To prospectively evaluate a clinically available AD blood test in primary care and secondary care using predefined biomarker cutoff values.

   Design:
   * N = 1213 patients.
   * Patients underwent clinical evaluation due to cognitive symptoms who were examined between February 2020 and January 2024 in Sweden.
   * The biomarker cutoff values had been established in an independent cohort and were applied to a primary care cohort (n = 307) and a secondary care cohort (n = 300);
   * 1 plasma sample per patient was analyzed as part of a single batch for each cohort.
   * The blood test was then evaluated prospectively in the primary care cohort (n = 208) and in the secondary care cohort (n = 398);
   * 1 plasma sample per patient was sent for analysis within 2 weeks of collection.

   Exposure:
   * Blood tests based on plasma analyses by mass spectrometry to determine the ratio of plasma phosphorylated tau 217 (p-tau217) to non–p-tau217 (expressed as percentage of p-tau217) alone and when combined with the amyloid-β 42 and amyloid-β 40 (Aβ42:Aβ40) plasma ratio (the amyloid probability score 2 [APS2]).

   Outcomes:
   * The primary outcome was AD pathology (determined by abnormal cerebrospinal fluid Aβ42:Aβ40 ratio and p-tau217).
   * The secondary outcome was clinical AD.
   * The positive predictive value (PPV), negative predictive value (NPV), diagnostic accuracy, and area under the curve (AUC) values were calculated.

   Results
   * The mean age was 74.2 years (SD, 8.3 years),
   * 48% were women, 23% had subjective cognitive decline, 44% had mild cognitive impairment, and 33% had dementia.
   * In both the primary care and secondary care assessments, 50% of patients had AD pathology.

   Conclusions:
   * The APS2 and percentage of p-tau217 alone had high diagnostic accuracy for identifying AD among individuals with cognitive symptoms in primary and secondary care using predefined cutoff values.
   * Future studies should evaluate how the use of blood tests for these biomarkers

influences clinical care.

Comments:
Prof. Alan Carson: While this approach is interesting and promising, it seems somewhat circular. They use a cross-sectional diagnosis as the comparator standard, but that standard is heavily influenced by the presence of blood biomarkers. It is essential to evaluate how these findings hold up in long-term follow-up, which I consider the current gold standard.

Jas: Thanks, Alan, for the prompt reply. I agree, but as you said, it is promising and exciting. If it happens to be a good biomarker that is readily available, it will potentially help improve early detection and clinical care and would save overall health costs significantly! Watch the space,

5. **MEDIA RELEASE: A critical set back in Alzheimer's treatment for Australians**
https://www.australiandementianetwork.org.au/2024/10/17/media-release-a-critical-set-back-in-alzheimers-treatment-for-australians/

Sad News for dementia patients in Australia! Please share your experiences in your respective countries.

The Australian Dementia Network (ADNeT) is disappointed with the TGA's decision to reject the use of lecanemab in Australia.

The TGA based its decision on concerns about the safety risks of lecanemab, particularly the occurrence of amyloid-related imaging abnormalities (ARIA) seen in clinical trials, despite its demonstrated efficacy in slowing cognitive decline.

Comments:
So sad. So excited to learn of its debut elsewhere. As I understand phase 3 trials were completed. Certainly a great disappointment for many hopeful early dementia sufferers in Australia

6. **Alzheimer's drug rejected for widespread NHS use in England – Article in the Guardian**
https://www.theguardian.com/society/2024/oct/23/alzheimers-drug-donanemab-rejected-widespread-nhs-use-england

NICE has rejected the widespread use of Donanemab and Lecanemab in the NHS.

The decision to approve specific Alzheimer's drugs has raised concerns about creating a two-tier system, where individuals who can afford to pay privately can access medication, while those reliant on NHS care may not have the same opportunities.

Both Donanemab and Lecanemab work by targeting amyloid, a protein that accumulates in the brains of Alzheimer's patients. These drugs aim to clear this buildup and slow cognitive decline.

Donanemab, also known as Kisunla, has shown promising results in clinical trials. It could potentially slow the rate of memory and cognitive decline by over 20% and reduce the decline in daily activities by 40%. However, the drug is administered via intravenous drip every four weeks and carries the risk of serious side effects, including brain swelling and micro-bleeds.

While both drugs have received approval for use in the US, Lecanemab was rejected earlier this year by the European Medicines Agency and by Australian health regulatory bodies recently, indicating global concerns regarding its safety and efficacy.

7. **John Joska(Esteemed GNG-INA member) and Colleagues have written an interesting article complementing the above-read on Donanemab- a nice refresher!**
https://www.dailymaverick.co.za/article/2023-06-20-despite-promising-signs-the-search-for-alzheimers-treatment-is-ongoing-and-complex/

The observed decline in patients is associated with two key hallmarks involving proteins typically found in the brain. Disruptions in producing, distributing, and removing these proteins can lead to disease.

In medical research, proponents of the two-protein pathology theories are referred to as "Baptists" and "Taoists." Baptists believe that beta-amyloid forms plaques that disrupt nerve function, while Taoists argue that tau protein loses its structure, resulting in non-functional tangles.

Amyloid and tau are central to many clinical trials aimed at developing disease-modifying treatments. One approach has involved the use of monoclonal antibodies, which stimulate the immune system to clear amyloid plaques.

Donanemab, which was modified from a mouse model, shows promise in preventing new plaque formation and removing existing plaques. Eli Lilly sponsored a trial in the U.S. and Canada involving 257 participants aged 60 to 85, who received either Donanemab or a placebo.

Researchers assessed cognitive performance and daily functioning using the Integrated Alzheimer's Disease Rating Scale (iADRS). Initially, both groups scored 106 on the iADRS.

After 76 weeks, participants treated with Donanemab experienced a decline of 6.86 points, while the placebo group declined by 10.06 points, indicating a 30% slower rate of decline. However, this did not meet the goal of halving disease progression.

Approximately 25% of participants receiving Donanemab developed amyloid-related brain imaging abnormalities, with 22% experiencing symptoms.

Reducing amyloid plaques did not lead to improvements in clinical symptoms, and there was no significant difference in tau load between the two groups.

This study raises questions about the relationship between amyloid reduction and disease progression in Alzheimer's disease.

The authors also noted limitations, including a small and predominantly white sample, which raises concerns about the generalizability of the findings, particularly for diverse populations.

In conclusion, while the results regarding Donanemab are promising, further research is essential to assess its clinical significance and applicability across different populations in the ongoing search for effective Alzheimer's treatments.

8. **Neuropsychiatric symptoms as a sign of small vessel disease progression in cognitive impairment.pdf**
https://www.sciencedirect.com/science/article/pii/S266624502200006X

Prof Perminder Sachdev gave a fascinating talk on Small Vessel Disease and White Matter Hyperintensities. See below.

Parkinson Disease

1. **The neuropsychiatry of Parkinson's disease: advances and challenges.pdf**
   https://pmc.ncbi.nlm.nih.gov/articles/PMC8800169/

2. **The Neuropsychiatry of Parkinson Disease: A Perfect Storm.pdf**
   https://pmc.ncbi.nlm.nih.gov/articles/PMC7015280/

Neuroimmunology

1. **The immunology underlying CNS autoantibody diseases**
   https://www.sciencedirect.com/science/article/abs/pii/S0035378724005812
   Hot off the press. The full article is available in the GNG members section.

2. **Recent advances in the diagnosis and management of neuropsychiatric lupus**
   https://www.nature.com/articles/s41584-024-01163-z

   Neuropsychiatric disease is common among individuals living with systemic lupus erythematosus (SLE), and neuropsychiatric SLE (NPSLE) has extremely diverse clinical manifestations.
   Determining whether neuropsychiatric events have SLE-associated or alternative causes is a major challenge. Currently, there are no laboratory or neuroimaging biomarkers to diagnose NPSLE accurately.

   Owing to a paucity of clinical trials, high-quality evidence to guide the treatment of NPSLE is lacking.

   Management of NPSLE is guided by the severity of the clinical neuropsychiatric syndrome and the suspected relative contributions of inflammatory and ischaemic mechanisms to pathogenesis.

   Although NPSLE outcomes are generally favourable, they are associated with a substantial negative impact on health-related quality of life, economic burden, and increased mortality risk.

3. **Absence of Neuronal Autoantibodies in Neuropsychiatric Systemic Lupus Erythematosus**
   https://onlinelibrary.wiley.com/doi/full/10.1002/ana.25908

   The findings suggest that autoantibodies have very limited clinical value in the definition or diagnosis of NPSLE and should prompt a search for novel biomarkers.

   IFNα is one such plausible candidate.

   Observations suggest that the brain disease associated with SLE is likely to have pathological drivers other than neuronal surface autoantibodies.

4. AE Diagnostic Guidelines

Table 1 Autoimmune Encephalitis Diagnostic Guidelines

Criteria	Main AE syndrome	AE-typical supportive features						AE-specific Ab	Exclusion other causes
		New focal CNS finding	New seizures	CSF	MRI	EEG	Brain biopsy		
Possible	Subacute onset ≤3 mo of memory deficits/AMS/psych	Req[a]	Req[a]	Req-Pleo[a]	Req[a]				Req
Those meeting "Possible" criteria can then be assessed for further categorization, below									
Definite LE	Subacute onset ≤3 mo of memory deficits/AMS/psych suggestive of limbic system disorder			Req-Pleo[a]	Req-LE	Req-Temp[a]		Opt	Req
Probable Anti-NMDA-R	≥4 of 6 of cognitive or psych., speech dysfunction, movement disorder, decreased consciousness, dysautonomia, or hypoventilation			Req-Pleo/OCB[a]		Req-E[a]		NMDA-R	Req
Definite, other	Meet at least possible criteria. Diverse disorders (e.g., LGI1 encephalitis, CASPR2 encephalitis, and $GABA_A$-R encephalitis)							Req	Req
Definite ADEM	First multifocal CNS event, inflammatory, demyelinating cause, & encephalopathy not explained by fever & no new findings after 3 mo				Req-AD				Req
Bickerstaff's BE	Subacute onset decreased consciousness, bilateral external ophthalmoplegia & ataxia							GQ1B	Req
Hashimoto	AE with seizures, myoclonus, hallucinations or stroke-like episodes & subclinical or mild thyroid disease				Req-N			ND, Req-Thyr	Req
Probable	Subacute onset ≤3 mo of memory deficits/AMS/psych w/o other well-defined AE syndrome			Req-Pleo/OCB/IgGI[b]	Req[b]		Req[b]	ND	Req

Abbreviations: AD = typical ADEM MRI findings (diffusely poorly demarcated, large [>1–2 cm] lesions predominantly involving the cerebral white matter; T1-hypointense lesions in the white matter in rare cases; deep gray matter abnormalities [e.g., thalamus or basal ganglia] can be present, resolving); E = focal or diffuse slow or disorganized activity, epileptic activity, or extreme delta brush; GQ1B = anti-GQ1B optional for diagnosis if bilateral external ophthalmoplegia not present or ataxia not assessable; IgGI = elevated CSF IgG index; LE = bilateral abnormalities on T2-weighted fluid-attenuated inversion recovery MRI highly restricted to the medial temporal lobes); N = normal MRI; ND = neuronal Ab not detected in the serum or CSF; NMDA-R = NMDA-R Ab optional for diagnosis (definite diagnosis can be made with Ab positivity in the presence of ≥1 of the 6 major groups of symptoms); OCB = abnormal CSF oligoclonal band numbers; Opt = optional (if present, only 1 of MRI or supportive CSF or EEG findings required); Pleo = pleocytosis (elevated white cell count) in the CSF; psych = psychiatric symptoms; Req = required; Temp = EEG with epileptic or slow-wave activity involving the temporal lobes; Thyr = thyroglobulin or thyroid peroxidase antibodies.
[a] ≥1 of supportive features required.
[b] ≥2 of supportive features required.

5. Autoimmune Encephalitis Criteria in Clinical Practice.
https://www.neurology.org/doi/10.1212/CPJ.0000000000200151

6. Smouldering-Associated Worsening in Multiple Sclerosis: An International Consensus Statement on Definition, Biology, Clinical Implications, and Future Directions.
https://onlinelibrary.wiley.com/doi/10.1002/ana.27034

An interesting paper looking at SAW(Smouldering-Associated Worsening in Multiple Sclerosis) and the role of NfL as a biological marker(discussed by Dhama in neurodegenerative disorders earlier)

MS evidence shows early progression, requiring revised descriptors. Use relapsing or

progressive as non-diagnostic descriptors. ‘Clinically stable’ individuals may worsen in physical and cognitive domains. Authors have set up probing questions to uncover SAW in routine clinical practice.

The impact of smouldering disease in MS can also be potentially minimized by implementing a holistic management approach. This involves addressing MS-specific processes, preventing and treating comorbidities, and modifying factors such as smoking, lack of exercise, unhealthy diet, social isolation, and low social capital. Efforts should focus on promoting brain health by optimizing lifestyle factors, including regular exercise, stopping smoking, maintaining a healthy diet, and good sleep patterns. Yoga has gained interest as an intervention to improve symptoms and quality of life among pwMS, empowering them in disease management.

CSF and blood biomarkers in MS, such as neurofilament light (NfL), can indicate disease progression. Peak NfL levels correlate with relapses, new MRI lesions, and long-term disability accumulation. Higher NfL levels are also associated with negative prognostic indicators like paramagnetic rim lesions (PRLs) on MRI.

Neuromodulation

1. **Rumination symptoms in treatment-resistant major depressive disorder, and outcomes of repetitive Transcranial Magnetic Stimulation (rTMS) treatment :**
   https://pmc.ncbi.nlm.nih.gov/articles/PMC10491586/
   Shared by ~Jesús Ramírez-Bermúdez

2. **The Psychiatric and Neuropsychiatric Symptoms After Subthalamic Stimulation for Parkinson’s Disease.**
   https://psychiatryonline.org/doi/full/10.1176/appi.neuropsych.14040069

3. **Fundamentals of deep brain stimulation for Parkinson’s disease in clinical practice: part 1-.pdf.**
   https://pmc.ncbi.nlm.nih.gov/articles/PMC11039067/

4. **'Patients with Parkinson's, schizophrenia, and addiction, will benefit from our device': Dopameter inventor Shalini Menon**
   http://dhunt.in/XfZdU By The Week via Dailyhunt

5. **Fascinating talk by Valerie Voon. Paper**: **Modelling impulsivity and risk aversion in the subthalamic nucleus with deep brain stimulation.pdf**
   https://www.nature.com/articles/s44220-024-00289-z

   Modelling impulsivity and risk aversion in the subthalamic nucleus with deep brain stimulation.
   Risk evaluation is ubiquitous in decisions.
   ~Deep brain stimulation of the subthalamic nucleus is effective for Parkinson’s disease and OCD but may lead to impulsivity and hypomania.
   *Subthalamic stimulation has complex effects on impulsivity, enhancing conflict-induced impulsivity while reducing risk-taking. *
   In a study using a card gambling task and intracranial recordings from 25 participants, acute stimulation of the right subthalamic nucleus was tested in 15 individuals. *Results showed that stimulation decreased risk-taking ($P = 0.010$, Cohen’s $d = 0.72$) and increased subthalamic theta activity ($P < 0.001$, Cohen’s $d = 0.72$).
   * Notably, stimulation altered the relationship between subthalamic physiology and evidence accumulation, similar to outcomes seen in conflict processing. *This indicates the diverse nature of impulsivity and connects the mechanisms behind stimulation-induced conflict and risk. *

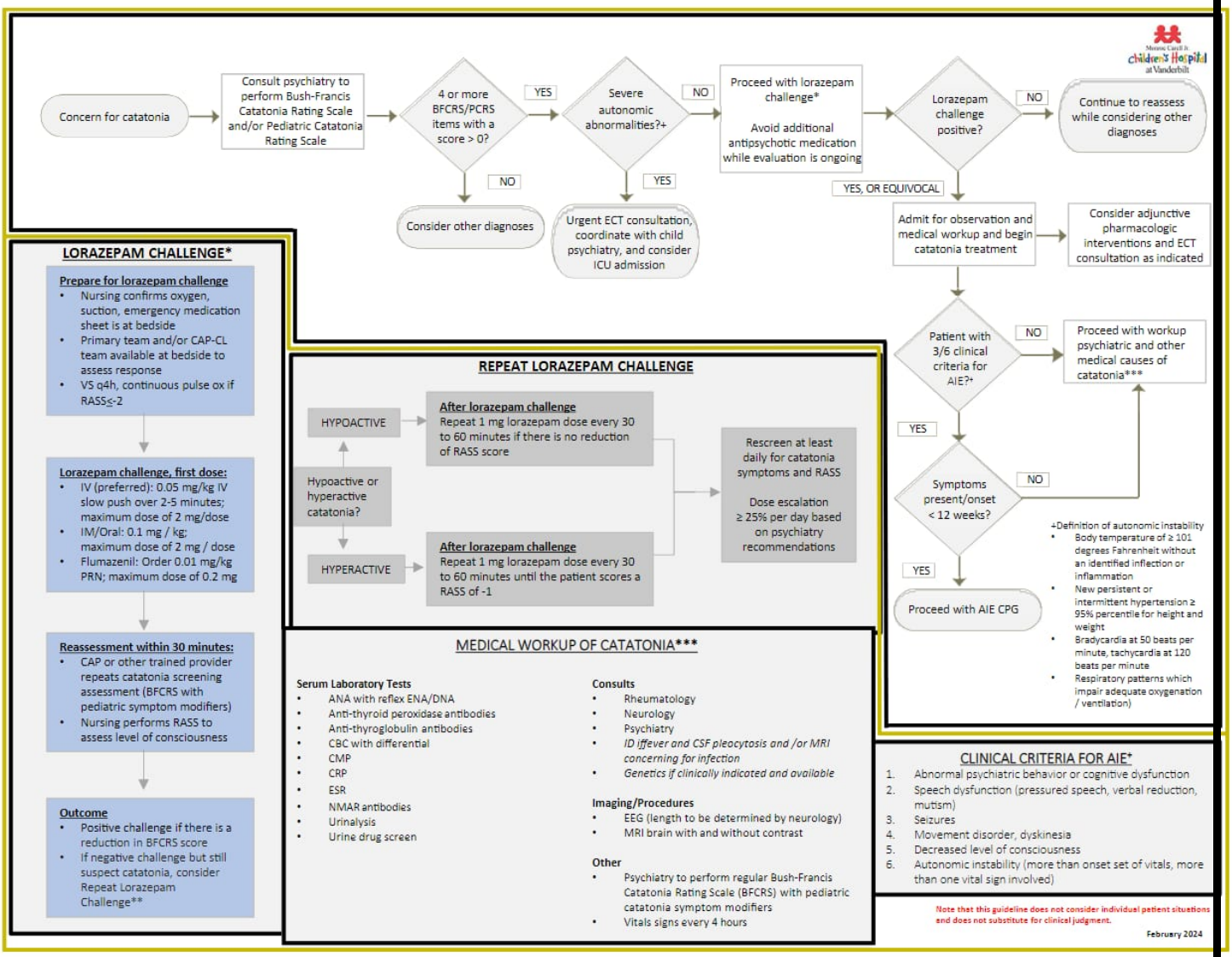
Additionally, risk-seeking behavior linked to the ventral subthalamic nucleus suggests a distinct functional connectivity with the mesial prefrontal cortex.

These findings have implications for understanding impulsivity and potential clinical applications for neuropsychiatric disorders.~

Catatonia

The Development of a Pediatric Catatonia Clinical Roadmap for Clinical Care at Vanderbilt University Medical Center

Shared by ~Jesús Ramírez-Bermúdez



Functional Neurological Disorder

1. **Functional-neurological-disorder-in-pregnancy-labour-and-the-postpartum-period-systematic-review**
   https://www.cambridge.org/core/journals/bjpsych-bulletin/article/functional-neurological-disorder-in-pregnancy-labour-and-the-postpartum-period-systematic-review/7D0C19F8CB0F0112A4EA57990054D917

Functional Neurological Disorder in Pregnancy, Labor, and Postpartum: A Summary

The review analyzed 36 studies, which included 34 case reports and 2 case series, involving a total of 43 patients.

Six subtypes of Functional Neurological Disorder (FND) were identified: functional seizures, motor weakness, movement disorders, dissociative amnesia, speech disorders, and visual symptoms.

New cases of perinatal FND were found to be more prevalent during the third trimester and afterward.

Half of the cases involved pre-existing psychiatric or neurological disorders and adverse events.

Functional motor symptoms predominantly occurred during the third trimester, labor, and the postpartum period, often following spinal and epidural anesthesia.

Some women received unnecessary anti-seizure medications and intensive care.

Psychological therapy and physiotherapy were the primary treatments employed.
Kudos to @~Verónica Cabreira and @~Ingrid Farquhar for their leadership in this important study!

2. **A Framework for Understanding the Pathophysiology of Functional Neurological Disorder**
https://pmc.ncbi.nlm.nih.gov/articles/PMC7930164/

Attending INA-RANZCP conference in Melbourne was a fascinating session on FND covering length and breadth of FND. Psychedelics in FND is an area to watch for.

The author have reviewed FIVE constructs that are affected in FND:
1. Emotion processing (including salience),
2. Agency
3. Attention
4. Interoception
5. and Predictive processing/inference.

Examples of underlying neural circuits include
1. Salience,
2. Multimodal integration,
3. and Attention networks.

The symptoms of each patient can be described as
a combination of dysfunction in several of these networks and related processes.

Sleep

1. **Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine clinical practice guideline**
https://jcsm.aasm.org/doi/10.5664/jcsm.11390

Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine clinical practice guidelines are OUT!

All patients with clinically significant Restless Legs Syndrome (RLS) should have their serum iron studies regularly tested, including ferritin and transferrin saturation (the latter calculated from iron and total iron binding capacity, TIBC). Ideally, the test should be conducted in the morning, and patients should avoid all iron-containing supplements and foods at least 24

hours prior to the blood draw.

The first step in managing RLS is to address any exacerbating factors, such as alcohol, caffeine, antihistamines, serotonergic and anti-dopaminergic medications, as well as untreated obstructive sleep apnea (OSA).

RLS is common during pregnancy, and it is essential to monitor the risk profiles of medications.

The following recommendations are made regarding the use of medications for RLS:

- Gabapentin enacarbil is recommended over not using it.
- Gabapentin is recommended over not using it.
- Pregabalin is recommended over not using it.
- The standard use of levodopa is not recommended.
- The standard use of pramipexole, ropinirole, clonazepam, transdermal rotigotine, bupropion, and carbamazepine is not recommended.

Additionally, for periodic limb movement disorder (PLMD), the American Academy of Sleep Medicine (AASM) suggests against using triazolam and valproic acid.

For more details, please refer to the guidelines.

Neurodevelopmental Disorders

1. **Human-Specific Genes Reveal Link Between Brain Growth and Autism**
   https://neurosciencenews.com/autism-genetics-neurodevelopment-27858/

Others

1. **Treating Insulin Resistance with Metformin in Bipolar Disorder - Cindy Calkin, MD: https://youtu.be/MjbHfvHc5d0?si=Ha4wSDng8W1dQ0Vb**

   A Dalhousie University, Nova Scotia , Canada 🍁 CA researcher is making waves in the global medical community after a discovery that could help improve outcomes for people suffering from treatment-resistant bipolar depression.

   A recently published paper by Dr. Cynthia Calkin, a clinical researcher and associate professor in the departments of psychiatry and medical neuroscience, has been chosen by the American Society of Clinical Psychopharmacology (ASCP) Awards Committee to receive the 2023 Paul Wender Best Paper in The Journal of Clinical Psychiatry award.

2. **The Vagus Nerve and the Brain-Gut Axis: Implications for Neuropsychiatric Disorders https://psychiatryonline.org/doi/10.1176/appi.neuropsych.20240118?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed**

   Summary:

   The vagus nerve, the 10th cranial nerve (CN X), is the longest of all cranial nerves. It originates in the brainstem's medulla oblongata and extends into the gut region.

   CN X consists of both sensory (afferent) and motor (efferent) axons. It is associated with four brain nuclei and two peripheral ganglia and forms important neural circuits.

   This nerve plays a pivotal role in the brain-gut axis (BGA), providing the main autonomic

(parasympathetic) supply to most abdominal viscera and other organs. Its anatomic connection between the central nervous system (CNS) and enteric nervous system (ENS) influences crucial neural processes, brain functions, and behaviours such as feeding and mood regulation.

CN X is a key regulator of the neural mechanisms that lead to dysbiosis among gut microbial species. Its modulation of the brain-gut axis (BGA) is relevant to several neuropsychiatric conditions such as anxiety, depression, posttraumatic stress disorder, Parkinson's disease, and amyotrophic lateral sclerosis.

Vagal nerve stimulation (VNS) is a neuromodulatory technique approved by the U.S. Food and Drug Administration for the treatment of epilepsy and depression among patients aged 12 and above.

In summary, significant evidence supports CN X's neuromodulatory role in the BGA relevant to mental disorders. However, many important unanswered questions relate to the pathogenesis, pathophysiology, and potential treatments for neuropsychiatric and neurodegenerative diseases.

3. **Extending insights from LeDoux: using movies to study subjective, clinically meaningful experiences in neuroscience.pdf**
   https://academic.oup.com/cercor/advance-article/doi/10.1093/cercor/bhae422/7824786

   An interesting study that assessed the clinical applicability of autoimmune encephalitis criteria. The analysis included 538 people from the Mayo Clinic. The key symptoms to look out for are subacute onset of memory deficits, altered mental status, and/or psychiatric symptoms. The three most common and definite AE-IgGs detected were LGI1 (76, 34%), NMDA-R (32, 16%), and high-titer GAD65 (23, 12%).

   The authors concluded that autoimmune encephalopathies with predominant psychiatric symptoms had presentations distinct from primary psychiatric disorders. The patients exhibited a combination of psychiatric symptoms, often accompanied by signs of cognitive impairment. Additionally, these patients consistently showed supportive findings and met the diagnostic criteria for autoimmune encephalopathies. Therefore, it may be unnecessary to create a separate category for "autoimmune psychiatric" diagnoses.

4. **Inside out the neural basis of spontaneous and creative thinking: Scientific Commentary – pdf.**
   https://academic.oup.com/brain/article/147/10/3263/7757981

   The default mode network (DMN) is a crucial brain network involved in high-level human cognition. Recent research has highlighted its significance in various internal cognitive processes, including memory, associative thinking, mind-wandering, and creativity.

   In this issue of *Brain*, Bartoli and colleagues present new evidence regarding the DMN's causal role in divergent thinking, an essential aspect of creativity, as well as mind-wandering.

   This groundbreaking study monitored a group of 13 patients using intracranial electrodes to observe changes in low- and high-frequency oscillatory power within the DMN during spontaneous thought (mind-wandering) and while performing an "alternative uses" task that required divergent thinking.

   The findings revealed that DMN theta power was higher during the stimulus period compared to the response period for both mind-wandering and divergent thinking. Conversely, DMN gamma activity displayed the opposite trend, with the most significant increase observed

during the response stage.

The authors interpreted these results to suggest that the response period in the mind-wandering task primarily engages the DMN, while the alternative uses task entails a greater degree of interaction between the DMN and other brain networks.

Overall, this study offers new causal evidence regarding the DMN's involvement in divergent thinking. It also raises important questions for future research aimed at understanding the critical cognitive processes supported by the DMN that are similarly or differently engaged in mind-wandering and divergent thinking, as well as the role of theta activity in DMN connectivity.

5. **Encephalitis lethargica: clinical features and aetiology.pdf**

The main author has done a good thread on Twitter about this:
https://x.com/drjprogers/status/1849177828270440933?s=46&t=LkSMWPSw9YPKS8Ex3vVCdg

6. **Cat ownership** is associated with a 2x odds of developing schizophrenia...are Seropositive for T.Gondi
https://x.com/NTFabiano/status/1849415031844159737?t=BI-V8bPvuWH5QfXYbNcxvw&s=08

7. **Neuroscientist finds her brain shrinks while taking birth control.**
A researcher who underwent 75 brain scans over 3 months discovered that the volume of her cerebral cortex was 1 per cent lower when she took hormonal contraceptives.
https://www.newscientist.com/article/2452737-neuroscientist-finds-her-brain-shrinks-while-taking-birth-control/#Echobox=1729868432

8. **Psychoanalysis and the brain – why did Freud abandon neuroscience?**
https://www.frontiersin.org/journals/psychology/articles/10.3389/fpsyg.2012.00071/full

9. **15 years of NHS Practitioner Health:** Learning from our patients: their stories, their outcomes, their recovery
https://www.practitionerhealth.nhs.uk/news/celebrating-15-years

Books

1. **Neurological Disorders in Famous Artists -** A great book by Bogousslavsky

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New Members: Welcome on Board

We added many members to our global community in October 2024. If you have not joined as a member of our new Global Neuropsychiatry Group website yet, please consider doing so. We are building our library of resources in the members section and encourage you to join us - https://globalneuropsychiatry.org/become-member/

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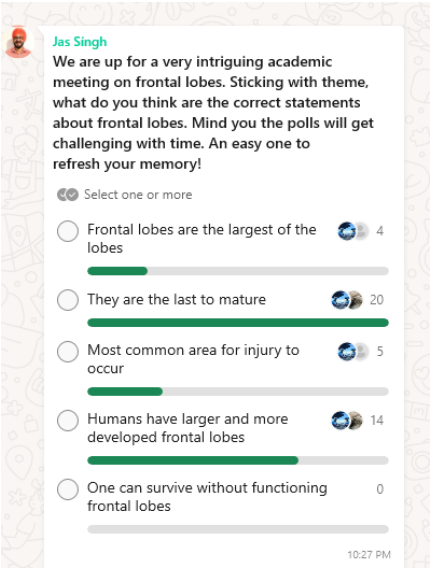
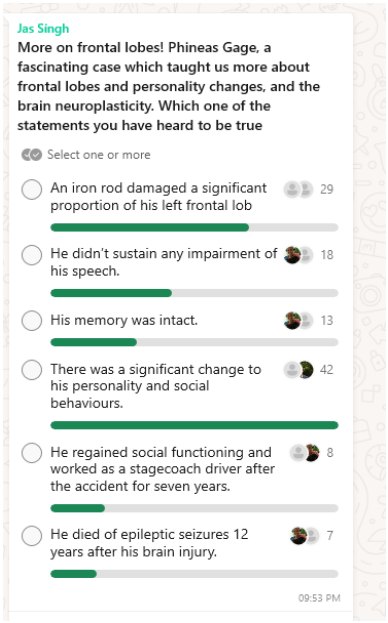
Welcome and thank you so much for being part of the journey;

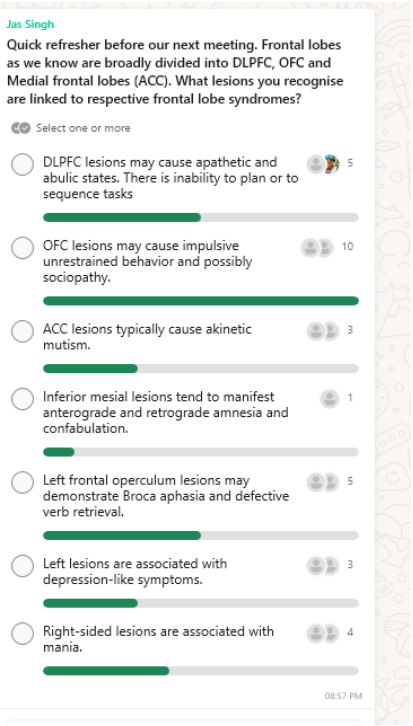
1. Dr Agnieszka Gross, Psychiatry, Golbroen, United Kingdom
2. Dr Leone Harvey-Smith, Psychiatry, Googong, Australia
3. Dr Jai Carmichael, Psychology, Melbourne Australia
4. Dr John Yeoman, Psychiatry, Lismore Australia
5. Dr Venkat Naga, Psychiatry, Brisbane Australia
6. Dr Sreejayan Kongasseri, Psychiatry, Udupi India
7. Dr Matthew Lennon, Psychiatry, Epping Australia
8. Dr Jaspreet Singh, Psychiatry, Bella Vista Australia
9. Dr Shail Bethi, Psychiatry, Gold Coast Australia
10. Dr Amita Ingole, Psychiatry, Adelaide Australia
11. Dr Samantha Broyd, Psychology, Wollongong Australia
12. Dr Danielle Feros, Psychology, Wollongong Australia
13. Dr Kyle Williams, Psychiatry, Hobart Australia
14. Dr Asmaa Ebraheem, Psychiatry, Hobart Australia
15. Dr Ian Navin, Psychiatry, Hobart Australia
16. Dr Louise Rigney, Neurology, Sydney Australia
17. Dr Hanne Gudiksen, Psychiatry, Brisbane Australia
18. Dr Anita Barbey, Neurology, Lausanne Switzerland
19. Prof Leigh van den Heuvel, Psychiatry, Cape town South Africa
20. Dr Thi Hoa Nguyen, Psychiatry, Hanoi Vietnam
21. Dr Shirlony Morgan, Psychiatry, Brisbane Australia - *UK trained, Brisbane based older adult psychiatrist.*
22. Dr Gavin McKay, Neuropsychiatry, London United Kingdom.
23. Dr Omar Ghaffar, Psychiatry, Toronto Canada - *CL and Geriatric psychiatrist, and a behavioral neurology and neuropsychiatry diplomate, working as medical lead of a CL psychiatry service at a 500 bed rehabilitation hospital in Toronto, Hennick Bridgepoint Hospital, part of Sinai Health and the University of Toronto.*
24. Dr Ganapathiram Nambi, Neuropsychiatry, Charleston SC United States
25. Dr Mihai Pop, Psychiatry, London United Kingdom
26. Trung Ngo, Brisbane, Australia
27. Ram Nambi, *currently doing a combined Neuro/Psych residency at MUSC in Charleston SC in the US.*
28. Kirsten Rowe - *a final year Psychiatry registrar at Stellenbosch University in Cape Town, South Africa. I have a special interest in Neuropsychiatry. I previously did a DPhil exploring executive function (EF) in adolescents with HIV & piloting a music intervention targeting EF &mood in this population.*
29. Luca Gosse - *An academic resident doctor from the UK (FY1) interested in Neuropsychiatry!*
30. Dr. Githua *a neurology resident at University of Cape Town with an interest in Neuropsychiatry*
31. Peter Gallagher - *Peter neuropsychologist and deputy dean of postgraduate research at Newcastle university*

Group Discussions

POLLS on Frontal Lobes







Discussion: Advice sought from Dr Ramya Vadievel

Dr Ramya Vadivel posted: I have a request with the group and hoped to get some direction in this area.

I am interested in the neuropsychiatric sequelae post TBI, more so in a niche demographic - the impact TBI /concussion has on medical practitioners with a lived experience of head injury. I have been looking for any specific data in this area but find lots of this work in the sports industry.

Would you be able to direct me to any resources , or anyone with a special interest in this area? My interest is in monitoring the impact of deficits in our profession which has a very high cognitive demand.

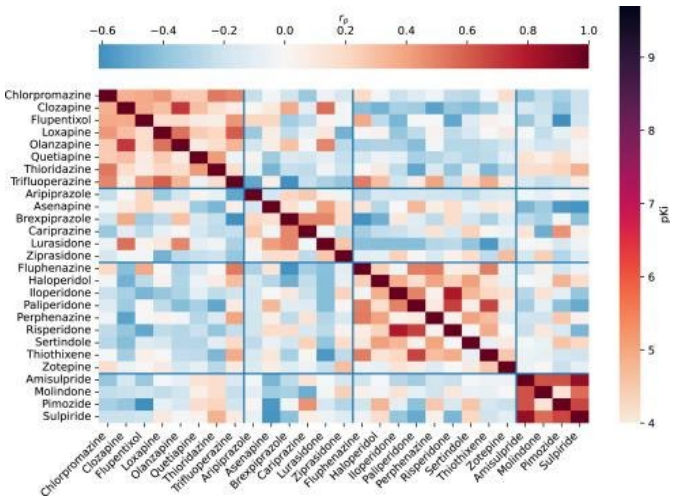
Dr Vadievel also asked*:* Looking for some advice from the group
I have a referral for medication advice - 45yr gentleman with cerebral palsy, with convulsions and is non verbal. Has been prescribed Pimozide since 1997 for behavioral issues. Also on Carbamazepine for convulsions. Pimozide is no longer available locally and the GP has requested advice on cessation and switching him to another medication.

To be honest, I have never worked with Pimozide and wanted to get advice on how to proceed with this.

Alan Carson commented: It used to be popular, particularly for monosymptomatic delusional psychosis of infestation. It was very high potency. In terms of its behavior, if you think of it as a GTI version of haloperidol and act accordingly, you won't go far wrong

Harry Costello commented: This may be a helpful paper to guide alternatives - https://www.biologicalpsychiatryjournal.com/article/S0006-3223(23)01200-3/fulltext

Figure 2 suggests the pharmacological profile of pimozide is very similar to amisulpride.



Buddhi clinic Commented: I have switched people (especially Tourette's for which it was recommended) from pimozide to risperidone, also very successfully. As Alan says haloperidol is the other useful alternative as pimozide is pretty potent.

Dr Jain added: I would have checked the current serum level of CBZ (because of its auto induction) and find out any underlying ASD and details of challenging behaviour first before shifting to another molecule. Also try to differentiate between episode of challenging behaviour from simple and complex partial seizures

Discussion: World Mental Health Day

Ray Wuck posted: Today is World Mental Health Day. As you go about in your busy lives, remember to pause and take a moment to appreciate and take care of yourself. We are thinking of
you today. https://en.m.wikipedia.org/wiki/World_Mental_Health_Day

Discussion: Neuropsychiatry Summer School

Could you let me know if there is a neuropsychiatry summer school or other kind of course who can be attended next July and August?
Or is there any virtual fundamental course?

Suggestions included:

Prof Alan Carson commented: The BNPA runs one of the best fundamentals courses out there. (although its winter not summer).It was hybrid although I am unsure if it still is.

Also from Prof Alan Carson : Purely on FND the fnd society has over a hundred hours of dedicated lectures and two online courses for members.
https://www.fndsociety.org

Kanny: did the PGCert at Birmingham. It was excellent. It is geared towards people that have jobs, so they clump contact hours together. Was doable whilst also working.

Mohan shared: https://www.cardiff.ac.uk/centre-neuropsychiatric-genetics-genomics/study/summer-school?dm_i=43QS%2CJN27%2C1OO20H%2C2A4JB%2C1

Jas Singh shared: Another highly recommended course. https://www.practicalcognition.com It's hard to find short-term Courses to learn the vast field of Neuropsychiatry. There are Masters programmes as recommended by Alan and other colleagues. Often, people know it in a piecemeal way! The best way to learn is at the bedside if you can find someone or gain some experience in neurology by attending clinics like Epilepsy, movement disorders, Sleep clinics, FND, cognitive disorders, neurodevelopmental conditions and other conditions at the interface between neurology and psychiatry. We are all working on a curriculum that will hopefully give some direction. We are also collating all the available courses to create a directory for all the members.

https://cmecatalog.hms.harvard.edu/neuropsychiatry-comprehensive-update/agenda

Harvard Neuropsychiatry: A Comprehensive Update (virtually, unfortunately). I haven't attended before, and it's also a short one, but I have heard good things and I think many of the speakers are esteemed members of this group. Brain Medicine: Integrating the Clinical Neurosciences

Cambridge Dementia Course
https://cambridgedementiacourse.org/team/

Edinburgh Sleep Medicine 2024 Course
https://acnr.co.uk/event/edinburgh-sleep-medicine-2024-course/

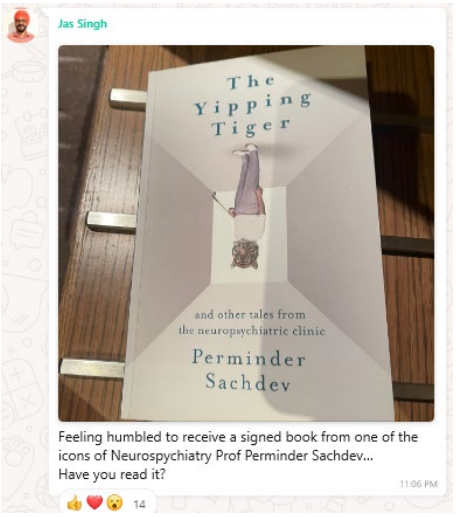
Discussion: Liver Toxicity with Amisulpride

Ahmad Almutairi asked: I'm wondering if anyone had ever seen a case of liver toxicity with Amisulpride? - I have a case of a young lady aged 26 who developed elevated LFTs while on amisulpride dose was 400 mg divided and the medical team did not find any other causes to explain her LFTs. After stopping amisulpride she improved and a rechallenge caused the lfts to rise again

I have never seen anything like it and wanted your opinion

Suggestion from Hetal: Usually, amisulpride is considered liver safe. Could it be the formulation used?

Discussion: The Yipping Tiger



GNG-INA Monthly Academic Meeting: 31st October

Topic: Long-term Outcome of Early Life Frontal Injury

- Growing Up Without Frontal Lobes: The Story of JP
- Long-term follow-up of adolescent frontal injury.

Thanks to guest Speakers Sheldon and Katarina and Session Chair Michael, for their fantastic contribution to the Group at the October Academic meeting held on 31 October 2024.

CLICK HERE TO WATCH NOW

The video was available to watch free for two weeks. After that it is archived on the INA website. Many thanks for watching. Do share any feedback and please free to share any experiences you have had with people with frontal lobe injuries. Share widely! Happy viewing.

SESSION CHAIR: Professor Michael Kopelman
Professor, Kings College London, NIHR Maudsley Biomedical Research Centre (BRC). Read more.

PRESENTERS

Professor Sheldon Benjamin
Professor of Neurology and Psychiatry, Director of Neuropsychiatry, University of Massachusetts T H Chan School of Medicine. Read more.

Growing Up Without Frontal Lobes: The Story of JP
Summary: **Download case summary here**
The case of JP, reported by Ackerly and Benton in 1948, stands as the index case of developmental prefrontal damage and its impact on social adaptation. The cause of JP's neonatal prefrontal damage was never established, nor were the etiologies of his left-sided seizures at age 4 or his progressive anterograde amnesia in middle age. Ackerly followed JP until the patient's death at age 64, but no report of either JP's later years or a post-mortem examination was ever published. With a combination of methods we were able to discover how JP's personality evolved over the rest of his life, locate film of the brain cutting and determine the most likely cause of his frontal damage.

Download the full paper here:
https://mcusercontent.com/a2bef318d5c6c6e38760b40ac/files/1dbd6f8c-d1e0-e301-6d1b-ecab19a32a5e/JP_Lifelong_Deficits_2023.pdf

Dr Katarina Hughes, MD
Psychiatry-Neurology Resident, University of Massachusetts T H Chan School of Medicine.

Long-term Follow-up of Adolescent TBI
Summary: Lifelong challenges in social adaptation faced by a woman who suffered TBI with diffuse axonal injury in a motor vehicle accident at age 14 and has been followed for 35 years in the neuropsychiatry clinic.

Don't forget to see all of our previous academic meetings, presentations, case studies and recordings visit the INA website. Please consider joining INA.

Next INA-GNG Monthly Academic Meeting (check your local time zone)

Thursday 28 November 2024

Register for zoom link:
https://us02web.zoom.us/meeting/register/tZwocOqtrjwsGNB59MNfuDEa-Ou7lG6IrnUs

Time: 10:30-12:15 pm London, UK Time
8:30-10:15 pm Brisbane Time

Session Chair: Dr Alex Lehn, University of Queensland, Australia
Bio: https://about.uq.edu.au/experts/4316 and https://globalneuropsychiatry.org/gng-advisors/

10:30 am - 11:30 am: Dr Philip Mosely
Bio: https://about.uq.edu.au/experts/3938

Topic: Deep brain stimulation for treatment-refractory psychiatric illnesses
In this talk I will discuss our work applying deep brain stimulation to treatment-refractory psychiatric conditions, beginning with obsessive-compulsive disorder and hopefully (funding permitting) moving to major depressive disorder. This work is grounded in a long history of managing neuropsychiatric symptoms in the setting of subthalamic deep brain stimulation for Parkinson's disease.
Papers: https://rdcu.be/dZO5Q https://rdcu.be/dZO57

11:30 am - 12:15 pm: Dr Nick Medford
South London and Maudsley NHS, United Kingdom
Topic: Chronic FND in hospital setting - the prognostic factors.
Presentation of a recent paper

Survey for neurologists or neurology residents/trainees

Cesar Koga is currently conducting research with the Epilepsy and the Psychiatry services at the Clinical Hospital Complex of the Federal University of Paraná (CHC-UFPR) in Brazil. It's a brief online survey aimed at better understanding neurologists' perceptions on the management of psychiatric disorders in people with epilepsy.

The main goal of this survey is to pave the way for a future larger research project that aims to highlight the importance of Neuropsychiatry among neurologists in Brazil.

This survey is exclusively for neurologists or neurology residents/trainees and was designed to be very straightforward (it takes less than 5 minutes to complete).

Cesar kindly requests the neurologists in this group to take a moment to respond if possible! It would also be greatly appreciated if everyone could share this survey with other neurologists or neurology residents/trainees. We are trying to gather as many responses as possible, globally!

Survey Link: https://forms.gle/sK9fsRTD78rBbUn7A

Feel free to contact Cesar in case of any comments or questions:
Email: cesarkogack@gmail.com Cellphone: +55 41 996955209

Meetings and Events held in October

Tim Nicolson shared: All welcome to great talk 8 October at 1pm UK time on big structural imaging biobank that's just been set up by Ash Venkataraman at the Maudsley and 2 example projects showcasing its applications at both ends of the neuropsychiatry spectrum- one comparing FND and PTSD with health controls (so looking at structural differences in functional disorders!) and one looking at the effects/interactions of social deprivation on structural changes in dementia!

We'll (finally!) be launching our KCL / IoPPN Neuropsychiatry Research and Education Group (NREG) website in the next few days so will put on there along with our back catalogue of talks from the last 4 years! Our upcoming talks are as follows which are always Tuesdays 1-2pm UK time. I will try and post links/reminders here but checkout our new website when launched and follow our Twitter/X account @IoPPN_NREG or IoPPN Events page for details and Teams links!

22/10	Luke Jelen	Ketamine Chronicles: Probing Glutamate and Opioid Mechanisms of Antidepressant Response to Ketamine	SR12
12/1	Arteen Ahmed / Paul Shotbolt	CRISP study - Effects of DBS-STN on Impulsivity and Other Psychiatric Symptoms in Parkinson's Disease:	RMA
26/11	Mary Summers	Symptoms validity tests in functional disorders	SR4
10/12	Agnes Arnold-Forster	Nostalgia: A History of a Dangerous Emotion	SR1
14/1	Tammy Hedderley	Intense Imagery Movements & Maladaptive Daydreaming	RMA
28/1	Daniel Kondziella	The evolutionary origin of near-death experiences	SR3

David Perez shared this link to the Building Clinical Programs in #FND: Opportunities & Pitfalls webinar held October 17, 2024
https://x.com/davidlewisperez/status/1840778579245707720?s=48

The RANZCP Section of Neuropsychiatry / INA Congress was held 27th-29th October in Melbourne.

https://www.ina2024.com.au

The main credit goes to Prof Richard Kanaan, Prof Valsa Eapen, Prof Tony David and all the presenters who had put in great time and effort to enlighten us with knowledge and new learning. A special thanks to Aleisha and her admin team for organising this beautiful event with the help of scientific committee. We at GNG-INA will keep working hard to provide high quality Neurospychiatry education all year through. Please do join us this coming Thursday online for another scintillating session! This is your space, let's keep sharing knowledge for the betterment of everyone. See some of the details on talks from the congress below:

Prof Angela Vincent delivered the Cajal lecture at the @INA meeting in Melbourne 27-29 October - www.ina2024.com.au/keynote-speakers.
Other GNG members presented throughout the Congress.

Fascinating talk by the legend - Prof Angela Vincent. Some highlights

- LGI1 and CASPR2 abs, IgG 4 abs
  - memory loss
  - Confusion
  - Neuromyotonia
  - Pain
  - Faciobrachial seizure non responsive to AEs but good response to steroid
  - Neuronal loss is more eminent
- NMDAR abs, IgG1
  - Subacute
  - Psychiatric presentation
  - Catatonia
  - Seizures
  - Agitation
  - Mood Disorder - interesting
  - Insomnia
  - Normal MRI findings initially
  - No significant movement disorder is explained
- GABAR Abs
  - catatonia
- GlyR abs
  - spinal cord, brain stem and autonomic nervous system
  - rigidity and myoclonus
  - Good response to immunotherapies
- IgLON5, IgG 4
  - Bulbar
  - Sleep
  - Cognitive
  - Neuromuscular

Summary from Angela Vincent's presentation

Antibodies can be predominantly IgG1(2,3) or IgG4; the subclass seems to be related to the antigen structure:
Divalent antibodies = multi-subunit protein
The Monovalent antibodies = single subunit protein

Our session is Animal models have successfully reproduced some clinical features, eg. seizures, but seldom recapitulate the key features of the human condition
- eg movement disorders in NMDAR-Ab encephalitis.

In most cases we don't know which precise cells or networks are targeted by the antibodies to cause the specific clinical features -exception GlyRs related to PERM.

Or are these disorders so variable and unpredictable in the phenotypes that it is hopeless to

even try!

Are there compensatory changes in the brain that might confuse those investigations in mice and men?

Joe Starke Presented fascinating talk on "An overview of Methylenetetrahydrofolate reductase (MTHFR) gene polymorphisms in psychiatric illness".

Here is the summary of that. Worth keeping this in mind in your clinical practice! 🙏

Folate is essential for multiple cellular and neurobiological processes.
The MTHFR enzyme is critical for converting folate into active, methylated form
MTHFR gene polymorphisms can result in impaired enzyme functioning leading to a functional folate deficiency, even if dietary intake is adequate.
Polymorphisms occur with increased frequency in psychiatric illness, specifically schizophrenia, bipolar disorder, depression, autism, and cognitive decline and are associated with earlier onset, more severe symptoms and treatment resistance.
Folic acid supplementation has value, but in the setting of MTHFR polymorphisms it may fail to adequately address folate deficiency, even though folate levels may appear normal.

In addition, unmetabolized FA is associated with negative health impacts Genetic testing for MTHFR polymorphisms and supplementation with 5 MTHF warrant increased attention as simple and relatively low cost adjunctive treatments in a range of psychiatric illnesses.

Joe commented: that he is a CL psychiatrist at eastern health in Melbourne. I have a particular interest in nutritional and functional psychiatry and the role of micronutrients. It was great to have a receptive audience today and I would be keen to connect with others in the group who are exploring in this area.

Raj commented: That's very interesting. We do supplement our neuropsychiatry patients with multivitamins (particularly Vit B1, B6, B12 and Vit.D) and multi minerals (magnesium), anti inflammatory/ antioxidants (CoQ10, astaxanthin, omega 3, Vit C, NAC, Lycopene etc) and pre- & probiotics, protein /collagen supplements, etc.

We still don't know much about utility/value for money of these supplements in particular cases and the quality of supplements available in the market (as they are not subject to stringent regulations).

It would be interesting to learn from Dr.Joe Starke on such issues!

Joe replied: I think where I am hoping this will go is that we build up individualised supplementation approaches based on testing. And yes you are right there is such a range of supplements out there. Different versions of the various nutrients. And variable quality is a concern. As more interest is focused on this we can refine our protocols.

Janardhanan: we are presenting a symposium - "**The role of non-invasive and invasive neuromodulation in Obsessive-Compulsive Disorder and Tic Disorder**" at the INA conference on 28th Oct, Monday 9am to 10am. It would be great to meet those of you who are attending the conference in Melbourne.

Trung Ngo: There is preliminary evidence that vestibulocortical stimulation (VCS) provides therapeutic relief in a case of RLS.

Happy to discuss further with anyone attending who would be interested in collaborating on a pilot VCS therapy trial in RLS (e.g., n= 6 to 12) [and/or OSA

Fascinating talk by Prof Perminder Sachdev on Small Vessel Disease and White Matter Hyperintensities.

Prof Sachdev was also awarded INA Lishman Award. A well-deserved award for all the hard work he has done and is still doing! It's a privilege for us to have him as a member of the global Neuropsychiatry community!

I have always found SVD as a mysterious condition with a heterogeneous phenotype! Lot of new learning for me today. Sharing some pointers.

Few Pointers from the talk:

Neuropsychiatric Conditions with some association

- Depression(VaD)
- Stroke
- Mortality
- Apathy
- Migraine
- Depression
- Gait disturbance
- Incontinence
- Late onset Schizophrenia/Psychosis
- Cognitive impairment
- Neurodegeneration

Management

- BP medication not recommended if BP is normal
- Aspirin no evidence
- Lipid lowering drugs - could be considered
- Lifestyle interventions are the key.
- Identifying the risk factors earlier on - prevention is the key along with maintaining healthy lifestyle.
- Appropriate glycemic control - insufficient

Other meetings held in October 2024

Prof Sheldon Benjamin shared: If anyone happens to be free, awake and interested Bruce Miller is giving a talk at noon (9 October 2024) US Mountain time (1pm Eastern US, 1800 UK time). He's a brilliant frontal dementia researcher and it will be fascinating to hear his thoughts on the genesis of psychosis at the inaugural Christopher M Filley Lectureship: Neurological Basis of Psychosis.

Link for an **ACNA meeting** link (via Monash University, Australia) for 9 October 2024 was shared with the GNG. They have very kindly agreed to share this with our group. The Topic was **a case study presentation by the ECDC Fellow, Dr Sarah Thomas**

Link for **Virtual University of Toronto Neurology Grand Rounds** held on Friday 25 October: **Functional Cognitive Disorders** was shared with GNG members by Prof Alan Carson who commented: Perhaps somewhat foolishly the University of Toronto offered me a visiting professorship this year (I was actually very honoured) anyway I give my lecture on Friday 25th of October. its online and any medic is welcome to attend. Please feel free to ignore this flagrant self promotion but if anyone wants to join you are very welcome.

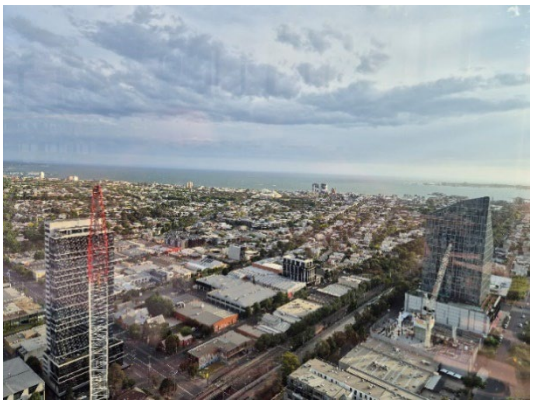
Ray Wuck Shared this image:

Social Event

The Global Neuropsychiatry Group held its first ever social dinner with GNG members who were in Melbourne, Australia for the INA Congress. The Dinner was held on Sunday 27 October with 17 GNG members joining at Strato Melbourne on the 40th floor of a building close to the Conference Venue. It was a great night with nice food and interesting conversation. It was fantastic to meet each other in person. See a few pictures below:

From bottom left and clockwise around the table: Aleisha O'Connor - Melbourne Australia, Jasvinder Singh – Brisbane Australia, Margo Lauterbach - USA, Valsa Eapen – Sydney NSW, Nicole Warren- Brisbane Australia, Niraj Agarwal, London UK, Omar, Anthony David – UK, Harry McConnell - Brisbane

From bottom left and clockwise around the table; Trung Ngo – Brisbane Australia, Wendy Phillips – Melbourne, Jay Salpekar – Baltimore USA, Angela Vincent – UK, Perminder Sachdev – Sydney Australia, Prasoon Gupte – Toowoomba Qld Australia, Aaron Hauptman - Baltimore USA, Jen Darman - Melbourne Australia



Views of the City of Melbourne and Port Phillip Bay from the restaurant.

Upcoming Events

2024

If anyone is attending the **WPC 2024 in Mexico City**, please let me know if you would like to have a small meeting of the Neuropsychiatry Interest Group during the Congress. We will discuss the work plan for the group. Pls DM on my email address P.sachdev@unsw.edu.au

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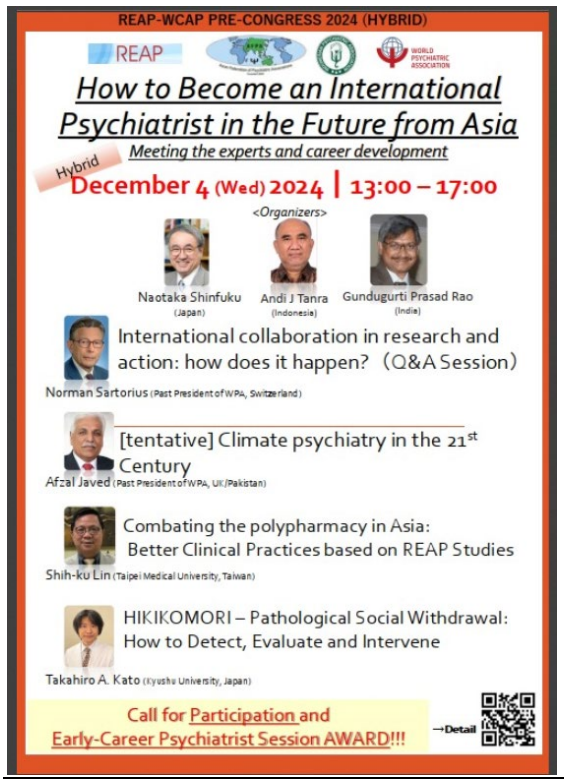
2024 Encephalitis International Conference
2nd and 3rd December 2024 at the Royal College of Physicians, London - and VIRTUALLY!
Read more: https://www.encephalitis.info/encephalitis-conference/

Registration is open now! Encephalitis 2024 is dedicated exclusively to encephalitis and covers epidemiology, pathogenesis, diagnosis, treatment and rehabilitation in both children and adults, covering both infectious and autoimmune aetiologies.

Highlights from previous years: https://www.encephalitis.info/encephalitis-conference-through-the-years/

10th World Congress Asian Psychiatry (WCAP) 2024 to be held on December 4, 2024 in the Prime Plaza Sanur Hotel, Bali – Indonesia: www.10thwcap-afpa.com
Theme: How to Become an International Psychiatrist in the Future from Asia

Registration: https://bit.ly/10thWCAP-Registration
For Poster and Freepaper Competition: https://bit.ly/PosterFreepaper-Registration
For Hotel Reservation: https://bit.ly/10thWCAP-HotelReservation



For those in Europe this **'Essentials of Neuropsychiatry' BNPA Teaching Weekend** in person in Oxford on 13-15 December 2024 is superb - designed for trainees in psychiatry, neurology and psychology but still great for a refresher for more senior people wanting to get latest info on topics from some great speakers

Read more: https://bnpa.org.uk/teaching-weekends/

It's a great 'Oxford experience' as stay and dine in a College in central Oxford. This is last year's program https://bnpa.org.uk/wp-content/uploads/2024/04/BNPA-TWE-PROGRAMME-8910-DEC-23.pdf

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2025

Harvard Medical School **Brain Medicine: Integrating the Clinical Neurosciences** **February 7, 2025** Live Virtual. **Read more:** https://cmecatalog.hms.harvard.edu/brain-medicine-integrating-clinical-neurosciences

The American Neuropsychiatric Association (ANPA) 2025 Annual meeting to be held in Montreal **12 – 15 March 2025. Read More:** https://x.com/anpadirect/status/1792708190393803106?s=48

Other business

Thank you everyone for being part of the journey! This was our second attempt to sum up monthly discussions. Any feedback is highly appreciated. If you wish to contribute, please feel free to share any interesting article, story, idea, poetry or anything related to neurosciences.

You are amazing! Keep learning and keep growing!

https://globalneuropsychiatr...

Thank you from the GNG Admin Team.

Jennifer & Jasvinder

email: globalneuropsychiatryg@gmail.com