Neuromodulation & Sleep

Conference Summary & Reference List

RANZCP SoN Neuropsychiatry Conference

15th Annual Conference · Day 3 · November 2026 · Gold Coast

GLOBAL NEUROPSYCHIATRY GROUP

Part 1 · Conference Summary

OVERVIEW

Day 3 covered sleep medicine for psychiatrists across five talks, followed by a neuromodulation workshop. The sleep talks covered sleep physiology and OSA, dreams and parasomnias, narcolepsy and the new orexin agonists, whether excessive daytime sleepiness is psychiatric, and insomnia. The workshop covered DBS, personalised TMS for Alzheimer disease, home-based tDCS after mild TBI, and the practicalities of delivering neurostimulation in Australia.

Take-home messages

- Sleepiness is not the same as fatigue, and subjective sleepiness in mood disorders often outstrips objective measures. Take a 24-hour history and check sleep opportunity with actigraphy before labelling hypersomnia.
- Psychiatric hypersomnia is long sleep with preserved efficiency, not rapid sleep onset. It tracks the mood episode and signals treatment resistance and suicide risk.
- CBT-I is first line for insomnia. DORAs are a newer licensed option that does not cause physiological dependence.
- Oral orexin-2 agonists are a step-change for narcolepsy type 1, and wider uses for alertness are likely.
- CPAP's cardiovascular benefit appears limited to high-risk OSA, meaning high hypoxic burden or a high heart-rate response.
- Neuromodulation is moving towards personalised, network-based targeting: the volume of tissue activated in DBS, and gamma-tuned, fMRI-guided TMS.

TALK 1 — SLEEP MEDICINE OVERVIEW

A/Prof Eli Dabscheck, respiratory and sleep physician

Why sleep matters

- Sleep serves glymphatic clearance, memory consolidation, immunity, cardiovascular health and hormone regulation.
- Three weeks of 4-hour nights in healthy adults increased energy intake and abdominal visceral fat.

- In the Nurses' Health Study, women sleeping 5 hours or less had a pneumonia rate ratio of 1.39. Better sleep also improves antibody responses to vaccination.
- In the UK Biobank (over 400,000 people), a better sleep score was linked to lower arrhythmia risk, independent of sleep apnoea.

Circadian rhythms

- The suprachiasmatic nucleus is the master clock and is set by light. Light suppresses melatonin from the pineal gland.
- **Delayed sleep phase:** morning bright light (a 10,000 lux lightbox) plus low-dose melatonin several hours before the desired bedtime (around 7–8 pm for a midnight bedtime).
- **Advanced sleep phase:** common in older people. Treat with evening bright light after dinner.
- **Non-24-hour rhythm:** rare outside congenital blindness. It can be anchored with a micro-dose of melatonin taken at the same time each day.

Obstructive sleep apnoea

- Recurrent airway obstruction causes desaturation and arousals, leading to sleep fragmentation (sleepiness) and sympathetic activation (cardiovascular risk).
- An AHI above 5 is abnormal. STOP-Bang is a useful screen.
- In HypnoLaus, about 1 in 10 men and 1 in 20 women had moderate to severe OSA with sleepiness.
- **Treatment:** CPAP remains the gold standard, and mandibular advancement splints are an option. In SURMOUNT-OSA, tirzepatide resolved OSA in about half of participants, with roughly 20% weight loss.
- **CPAP and the heart:** SAVE, RICCADSA and ISAACC showed no cardiovascular benefit. A pooled post-hoc analysis suggests benefit only in high-risk OSA, and possible harm in low-risk, non-sleepy patients. That finding is unexplained and needs a prospective trial.

TALK 2 — DREAMS AND PARASOMNIAS

Dr Hetal Mehta, consultant neuropsychiatrist

Normal sleep and drug effects

- About 75–80% of sleep is NREM and 20–25% is REM. NREM dominates the first third of the night (homeostatic drive) and REM the last third (circadian drive). Slow-wave sleep declines with age.
- Benzodiazepines reduce slow-wave sleep.
- Antidepressants suppress and delay REM and increase motor tone.
- Alcohol increases slow-wave sleep early in the night, then causes REM rebound.

Dreams

- Dreams occur in both REM and NREM sleep.
- REM dreaming involves reduced dorsolateral prefrontal activity and increased limbic activity (amygdala and anterior cingulate). NREM dreaming involves hippocampal and medial prefrontal activity, linked to memory consolidation.

- Lesions at the parieto-temporo-occipital junction can stop dreaming.

Disorders of arousal (ICSD-3)

- These include confusional arousals, sleepwalking and sleep terrors. They arise from N3 sleep in the first third of the night, with incomplete awakening and amnesia for the event.
- They are common in children and often self-limiting.
- Sexsomnia is a variant of confusional arousal. In forensic cases, identifying predisposing and precipitating factors is critical.

REM sleep behaviour disorder

- Loss of REM atonia leads to dream enactment, which can be violent.
- RBD is strongly linked to alpha-synucleinopathies, with about 6–8% a year converting to a motor or cognitive synucleinopathy.

Cases (anonymised)

- **Forensic sexsomnia:** a defendant with a diagnosed arousal disorder said he was hard to wake, but his past history showed he could rouse quickly and act purposefully when needed. The court found his account inconsistent with a true parasomnia.
- **Sexsomnia on PSG:** an adult with a several-year history of the behaviour. The motor activity arose from N3 with no arousal before or after.
- **RBD on video:** violent punching and kicking during REM sleep, with loss of atonia on EMG.

TALK 3 — HYPERSOMNIA AND NARCOLEPSY

A/Prof Eli Dabscheck, respiratory and sleep physician

Insufficient sleep syndrome

- The commonest mimic of a hypersomnia disorder. Actigraphy confirms short sleep.
- Case: a young working adult with a new baby was sleeping only 5–5.5 hours on weeknights.

Narcolepsy type 1

- Cataplexy is the cardinal feature: brief loss of muscle tone triggered by emotion, usually laughter, with consciousness preserved.
- It is caused by loss of orexin (hypocretin) neurons in the lateral hypothalamus.
- Other features are severe sleepiness, fragmented night sleep, hypnagogic and hypnopompic hallucinations, and sleep paralysis.
- It is strongly linked to HLA-DQB1*06:02, with a possible link to H1N1 infection and vaccination.

Narcolepsy type 2 and idiopathic hypersomnia

- **NT2:** no cataplexy and normal orexin, with sleep-onset REM periods (SOREMPs) on the MSLT.

- **Idiopathic hypersomnia:** no SOREMPs. It is often linked to long sleep (more than 11 hours), severe sleep inertia and unrefreshing naps.
- The boundary between the two is blurry.

Investigations

- **PSG:** rules out OSA and other disorders.
- **Actigraphy:** at least 2 weeks before the MSLT, to exclude insufficient sleep.
- **MSLT:** 4–5 naps at 2-hour intervals. A mean sleep latency of 8 minutes or less is abnormal, and 2 or more SOREMPs support narcolepsy.
- **CSF orexin:** very low levels are diagnostic of NT1, but the test has limited availability in Australia.

Treatment

- In Australia, dexamfetamine is first line on the PBS. Modafinil and armodafinil require failure of or intolerance to dexamfetamine.
- Sodium oxybate is very effective for cataplexy and sleepiness but is costly and accessed through the SAS.
- **Oral orexin-2 agonists:** in two phase 3 trials, Takeda's oveporexton nearly normalised wakefulness (MWT) and sleepiness (ESS) in NT1. Urinary frequency and transient insomnia were common side effects. Heavy industry investment, such as Lilly's purchase of Centessa, suggests use beyond narcolepsy.

Case (anonymised)

- A working-age adult presented with abrupt hypersomnia (19 hours a day), frequent cataplexy and diplopia, and a classic narcolepsy MSLT. The final diagnosis was with treatment-resistant autoimmune encephalitis.

TALK 4 — EXCESSIVE DAYTIME SLEEPINESS: IS IT PSYCHIATRIC?

A/Prof Eli Dabscheck and Dr Curtis Gray

A/Prof Dabscheck

- Separate sleepiness from fatigue, although the line blurs. Women with OSA, for example, often report fatigue rather than sleepiness.
- Depression and OSA overlap in fatigue, cognition, weight and sleep disturbance.
- An Alfred Hospital study using the MINI found high rates of depression and anxiety in sleep clinic referrals. Routine HADS screening followed, with cut-offs of HADS-D above 8 and HADS-A above 11.
- In 220 patients who had an MSLT, those with high HADS scores reported more sleepiness on the ESS, but HADS scores did not correlate with objective MSLT sleepiness.
- About a third of people with narcolepsy have depressive symptoms, and many are misdiagnosed with depression for years.

Dr Gray

- **Classification:** ICSD-3 has "hypersomnia associated with a psychiatric disorder", which treats the hypersomnia as part of the psychiatric syndrome. DSM-5's "hypersomnolence disorder" implies a comorbid diagnosis.
- **Features of psychiatric hypersomnia:** it rises and falls with the mood episode. Sleep is long with preserved efficiency, and sleep onset is not rapid. Subjective sleepiness exceeds objective findings. Distinguish it from clinophilia (taking to bed without sleeping).
- **In MDD:** hypersomnia occurs in about 25% of patients and is linked to treatment resistance, relapse and suicide risk.
- **Management:** exclude insufficient sleep, OSA and medication effects. Treat the mood disorder vigorously, favouring activating agents such as fluoxetine. Add light therapy and CBT for hypersomnia (CBT-H).
- **Circadian depression (Hickie group):** about a quarter of a large Australian cohort. Features are social jetlag, evening chronotype, hypersomnia and morning tiredness. It is linked to earlier onset, hypomania, psychosis, suicidality and poorer response to SSRIs and SNRIs. Chronotherapies (light and timed melatonin) may suit it better.

TALK 5 — INSOMNIA AND CIRCADIAN RHYTHM

Dr Curtis Gray

Understanding insomnia

- Insomnia is hyperarousal that prevents sleep despite adequate opportunity. Sleep deprivation is insufficient sleep.
- **Spielman 3P model:** predisposing, precipitating and perpetuating factors. Perpetuating behaviours are what maintain chronic insomnia.
- **Arousal model:** arousal becomes conditioned to the bed and the night. The aim is relaxation, and sleep follows.
- **DSM-5 "rule of threes":** symptoms at least 3 nights a week for at least 3 months, with distress or impairment.
- Insomnia in young adulthood predicts later depression.

Assessment

- Use a 24-hour assessment (Morin and Buysse) covering the day, the evening and the night.
- Establish the patient's chronotype first.

CBT-I (gold standard)

- **Sleep restriction:** the most powerful component.
- **Stimulus control:** get out of bed if awake, and go to bed when sleepy rather than at a set time.
- **Relaxation and cognitive therapy:** for worry and unhelpful beliefs about sleep.

Medication (second line or adjunctive)

- Licensed in Australia: benzodiazepines and Z-drugs (up to 4 weeks), melatonin (age over 55, up to 13 weeks) and DORAs such as lemborexant and suvorexant.

- DORAs block the wake-promoting orexin system and do not cause physiological dependence.
- Complex comorbid patients may warrant off-label sedating agents such as quetiapine, mirtazapine or tricyclics.

NEUROMODULATION WORKSHOP

Deep brain stimulation — Dr Phil Mosley

- Subthalamic nucleus DBS for Parkinson disease "releases the handbrake" on movement.
- About 10% of patients develop impulsivity, linked to stimulation of the anterior, limbic part of the STN.
- **OCD:** Dr Mosley's group targets the bed nucleus of the stria terminalis. Modelling of the volume of tissue activated, together with tractography, shows effective stimulation engages a network that includes the ventromedial prefrontal cortex. This work has fed international efforts to refine targeting, and an NHMRC-funded OCD DBS trial is nearing completion.
- Melancholic depression is a possible future target.

TMS for Alzheimer disease — Prof Kate Hoy

- Network dysfunction is a more direct driver of symptoms than protein pathology, and TMS can modulate it.
- **Proof-of-concept trial (n = 58):** multi-site theta burst stimulation increased gamma connectivity and improved delayed recall. The change in connectivity predicted the change in memory.
- **Current trial (n = 130):** stimulation frequency is tuned to each person's EEG peak, and stimulation sites to their own fMRI network hubs.
- Software based on scalp measurements is being developed so personalised protocols can be delivered in standard TMS clinics.

tDCS after mild TBI — Prof Karen Barlow

- tDCS is a diffuse, network-level way to promote plasticity.
- A 10-day home-based protocol was safe and well tolerated in children with TBI.
- A randomised trial is now comparing tDCS, tACS and sham, combined with online cognitive training, for attention problems after TBI.

Practical neurostimulation — Dr Grace Lang, Dr Alvin Goh and Dr Samir Ibrahim

- **Brain state matters:** lifestyle and diet affect whether the TMS "signal" change lasts.
- **Transcutaneous vagus nerve stimulation:** a 2023 systematic review found auricular tVNS superior to sham and comparable to antidepressants for mild to moderate depression. It is not ARTG-listed and is accessed through the SAS.
- **Remote tDCS:** Dr Goh demonstrated a clinic-grade device (Soterix Mini-CT) with clinician-set profiles, single-use dose codes and impedance monitoring.
- **TMS eligibility:** Australian guidance requires two failed antidepressant trials, and there is a push to reduce this to one.

- **Neuronavigation debate:** Dr Lang and Dr Ibrahim argued it is essential for accuracy. Dr Barton countered that there is no prospective evidence it beats Beam F3 scalp targeting, and that insisting on it limits access.
- **Deep TMS:** H-coil devices (BrainsWay) are used for depression, OCD and addiction.

Part 2 · Reference List by Topic

1 BRAIN STIMULATION FOR COGNITION IN ALZHEIMER DISEASE

Prof Kate Hoy, clinical neuropsychologist (Neuromodulation workshop)

> Network dysfunction is a treatable target in AD. iTBS to multiple frontoparietal sites raised gamma connectivity and improved delayed recall, and baseline gamma connectivity predicted who responded. Individual fMRI-guided targeting and accelerated protocols are the next step.

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2 DEEP BRAIN STIMULATION FOR TREATMENT-REFRACTORY OCD

Dr Phil Mosley, neuropsychiatrist (Neuromodulation workshop)

> DBS works on a network, not a single nucleus: different targets converge on a shared fronto-subthalamic tract. Responsive (closed-loop) ventral striatal DBS shows that stimulation can be triggered by the electrophysiology of obsessions. Australian authors argue DBS should be an accepted therapy for carefully selected patients in specialist centres.

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3 VESTIBULAR STIMULATION AND INSIGHT IN PSYCHOSIS

Dr Trung Ngo (Neuromodulation workshop)

> Impaired insight in schizophrenia may share right-hemisphere circuitry with anosognosia after stroke. A single session of left cold caloric vestibular stimulation transiently improved illness awareness; multi-session devices are now being tested.

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4 SLEEP AND MOOD: HYPERSOMNOLENCE, OSA AND CIRCADIAN DEPRESSION

A/Prof Eli Dabscheck and Dr Curtis Gray (Talk 4)

> Hypersomnolence affects about a quarter of people with depression and predicts treatment resistance, yet the MSLT is a poor measure of it. Treating OSA with CPAP lowers depression scores. A 'circadian' subtype (about 23% of the Australian Genetics of Depression Study) is younger, more often female and responds less well to SSRIs and SNRIs.

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5 INSOMNIA IN PSYCHIATRIC PRACTICE

Dr Curtis Gray, psychiatrist (Talk 5)

> About half of insomnia presentations are linked to a psychiatric disorder, and insomnia predicts later depression. Take a 24-hour history using the 3P (Spielman) model; questionnaires help, but investigations are rarely needed. CBT-I principles come first. In Australia, DORAs are indicated for insomnia with review at 3 months, and benzodiazepines and Z-drugs for up to 4 weeks.

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6 NARCOLEPSY, IDIOPATHIC HYPERSOMNIA AND THE OREXIN ERA

A/Prof Eli Dabscheck (Talk 3)

> NT1 is an orexin deficiency (twin discoveries in 1998, linked to narcolepsy in 2000), strongly tied to HLA DQB1*0602, with a mean diagnostic delay of about 14 years. CSF orexin testing changed the diagnosis in 4 of 15 Australian patients. Oral orexin-2 agonists are transforming treatment: TAK-994 stopped for hepatotoxicity, and oveporexton succeeded in two phase 3 trials.

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7 DREAMS AND PARASOMNIAS

Dr Hetal Mehta, consultant neuropsychiatrist (Talk 2)

> Disorders of arousal arise from N3 sleep early in the night; identifying triggers matters, especially in forensic cases such as sexsomnia. REM sleep behaviour disorder is a strong

marker of alpha-synucleinopathy, with about 6–8% a year converting to a neurodegenerative disease.

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8 OSA, CPAP AND CARDIOVASCULAR RISK

A/Prof Eli Dabscheck (Talk 1)

The big CPAP RCTs (SAVE, RICCADSA, ISAACC) were neutral for cardiovascular events, but they enrolled few typical sleep-clinic patients and adherence was poor. A meta-analysis including observational data suggests lower mortality, though healthy-user bias is a real concern. Pooled trial data point to benefit only in high-risk OSA, defined by hypoxic burden or heart-rate response. Tirzepatide is an option for OSA with obesity.

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9 WHY SLEEP MATTERS: REGULATION AND HEALTH

A/Prof Eli Dabscheck (Talk 1)

> Sleep timing comes from homeostatic pressure (Process S) and the circadian clock (Process C), switched by a hypothalamic 'flip-flop'. Short sleep weakens vaccine responses, raises pneumonia risk, increases energy intake and visceral fat, and healthier sleep patterns go with lower arrhythmia risk.

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