

Sleep and Epilepsy

15th - 18th September | Newcastle

Ivana Rosenzweig • BSS ISMC 2026 • Thursday 17 September • 13:00–13:30



British Sleep Society



UK Multidisciplinary Sleep Professionals



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- 2.
- 3.

ORIENTATION

Sleep and epilepsy form a closed loop

Sleep state shapes epileptic activity; epilepsy reshapes sleep

The clinical task is to identify the direction of causality we can change.

State

Timing

Comorbidity

Safety

This lecture maps to ESRS Textbook chapter M2 and the EAN–ESRS–ILAE-Europe diagnostic consensus.

Sources: <https://esrs.eu/wp-content/uploads/2021/07/Table-of-Contents-and-Preface-Sleep-Medicine-Textbook-2nd-edition-2021-ESRS.pdf>;
https://www.ean.org/fileadmin/user_upload/ean/ean/research/EAN_Guidelines/Guideline_Reference_Center/Guidelines/Sleep_guideline.pdf

NREM synchrony facilitates; REM sleep desynchrony constrains

NREM

A permissive network state

- Greater neuronal synchrony
- Activates many interictal discharges
- Many sleep-related seizures arise from NREM
- Slow oscillations can organise pathological activity

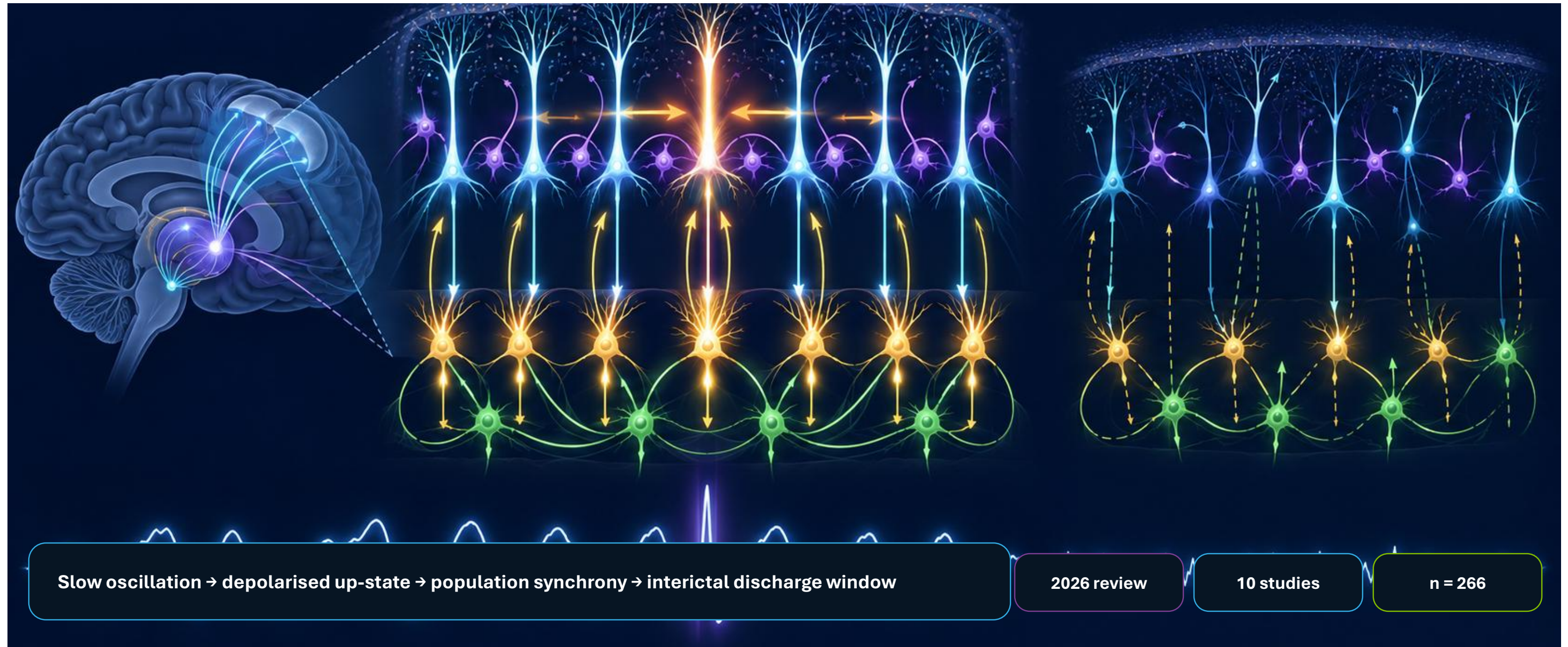
REM

A relatively suppressive state

- Cortical desynchronisation
- Lower interictal discharge burden
- Phasic REM may be especially suppressive
- Rare REM discharges can aid localisation

Sleep is not simply a pro-convulsant: effects depend on state, network and syndrome.

NREM up-states open windows for epileptiform synchrony



The mechanistic claim is probabilistic. NREM sleep synchrony facilitates many IEDs; REM sleep is relatively suppressive. A 2026 systematic review of 10 observational studies and 266 patients found higher IED rates in NREM than REM sleep, but heterogeneity prevented claims that one specific stage is diagnostically superior.

Watch synchrony recruit a traveling wave

1 • Oscillators lock into synchrony

64 oscillators: from independence to coordination

Coupling turns many weak clocks into one visible field

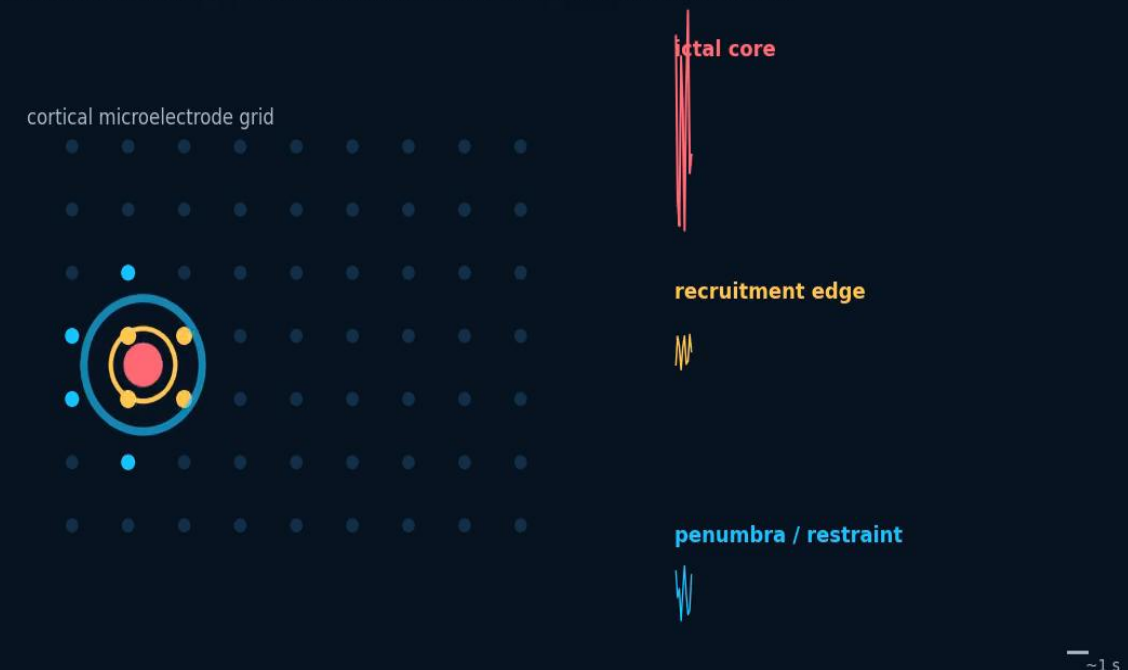


summed population signal

2 • Activity propagates across the network

A seizure is not a flash. It is a moving boundary condition.

Slow recruitment wavefront + fast discharges that travel through recruited cortex



The animation is a conceptual bridge: local excitability becomes clinically visible only when populations align and propagation succeeds

First, focus on phase alignment.

Second, see how coordinated activity can recruit neighbouring tissue and propagate.

TIMING

Seizure probability is the product of state and clock

Sleep state

NREM/REM transitions and micro-arousals change network synchrony.

Circadian phase

Independent biological timing can modulate seizure likelihood.

Syndrome / network

Different epilepsies show different 24-hour distributions.

A seizure at night is not necessarily caused by sleep; clock time and sleep stage are partially separable influences.

DIAGNOSIS

Sleep is the most powerful physiological activator of IEDs

A normal awake EEG does not end the investigation when the history is convincing

Capture sleep with an appropriate recording strategy; do not equate 'sleep-deprived EEG' with casual lifestyle sleep loss.

Routine + sleep

Sleep-deprived EEG

Ambulatory EEG

Video EEG / PSG

Joint IFCN/ILAE standards support sleep EEG as part of diagnostic recording. Sleep deprivation can increase yield but also carries seizure risk and needs a clinical protocol.

SLEEP DEPRIVATION

One phrase — two very different contexts

DIAGNOSTIC TOOL**Planned and supervised**

Used selectively to facilitate sleep / activate IEDs

Risk assessed and patient counselled

Recording and recovery are structured

DAILY-LIFE TRIGGER**Uncontrolled and cumulative**

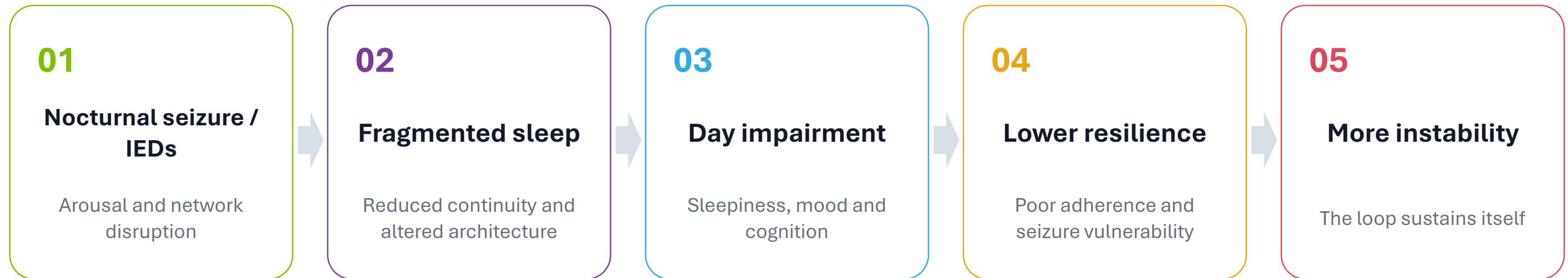
May lower seizure threshold

Often clusters with stress, alcohol or non-adherence

Prevent through regular sleep and treatment of insomnia

The exam trap is to recommend sleep deprivation as benign. In practice, distinguish a supervised diagnostic protocol from habitual restriction.

Seizures and IEDs fragment the very sleep that protects the brain



The therapeutic target is often the loop, not one isolated symptom.

Interictal activity may interfere with sleep-dependent memory processes even without obvious nocturnal seizures.

COMORBIDITY

Screen every patient with epilepsy for treatable sleep disorders

Sleep-disordered breathing

Snoring, witnessed apnoea, obesity, morning headache and resistant sleepiness.

Insomnia

Fear of seizures, conditioned arousal, mood disorder and medication effects.

RLS / PLMS

Evening urge, sensory symptoms and sleep fragmentation.

Circadian / insufficient sleep

Irregular schedules and social jet lag can destabilise control.

Parasomnias

May mimic seizures or coexist; history and video are decisive.

Medication effects

Sedation, insomnia, weight change and respiratory burden.

The 2020 consensus emphasises early diagnosis and treatment of sleep disorders, particularly respiratory disorders, because they are likely to benefit seizure control and quality of life.

https://www.ean.org/fileadmin/user_upload/ean/ean/research/EAN_Guidelines/Guideline_Reference_Center/Guidelines/Sleep_guideline.pdf

Treating OSA removes a modifiable source of fragmentation

Why suspect it

OSA symptoms may be mislabelled as antiseizure-medication sedation or postictal fatigue.

Why it matters

Fragmentation, hypoxaemia, cardiovascular risk, mood and cognition compound epilepsy burden.

What to do

Test appropriately; optimise PAP/adherence and weight-related care; reassess sleepiness and seizures.

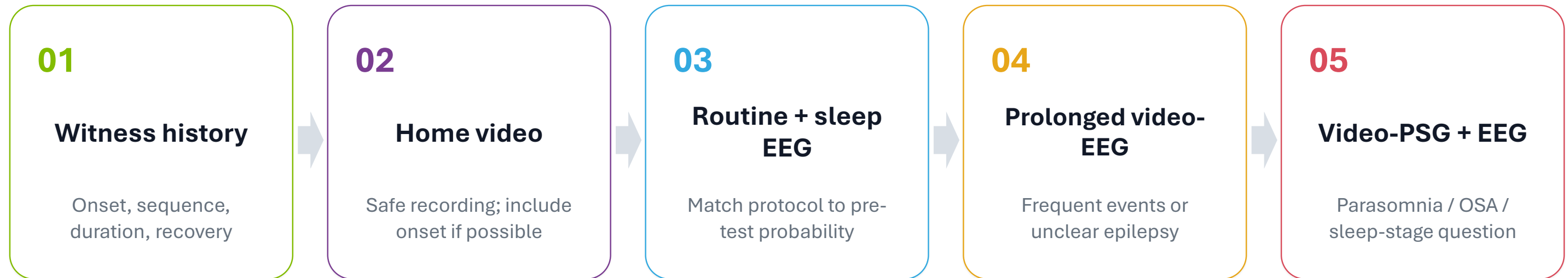
NOCTURNAL EVENTS

Stereotypy, duration and timing separate many mimics

Feature	Sleep-related seizure	NREM parasomnia	REM behaviour disorder
Pattern	Highly stereotyped	Variable / interactive	Dream enactment
Duration	Often brief; may cluster	Often longer	Seconds to minutes
Sleep timing	Any stage; often NREM	Usually first third / N3	Usually later night / REM
Motor clue	Tonic/dystonic or hypermotor	Complex wandering / autonomic fear	Punching, kicking, vocalisation
Best next evidence	Video-EEG	Home video ± video-PSG	Video-PSG with REM atonia assessment

No single feature is absolute. Multiple stereotyped brief events per night strongly raise concern for sleep-related epilepsy.

Capture the event before escalating technology



Primary question is epileptic classification; choose video-PSG with expanded EEG when sleep disorder and stage physiology are central.

Three exam patterns worth recognising

Sleep-related hypermotor epilepsy

Brief, stereotyped hypermotor events, often clustered from NREM; scalp EEG may be unrevealing.

Generalised epilepsy on awakening

Seizures cluster after waking; sleep loss is a common precipitant.

DEE-SWAS / sleep-potentiated activity

Marked activation in NREM with cognitive or behavioural regression; sleep EEG is essential.

Use patterns to guide the recording: overnight sleep may be diagnostic even when routine wake EEG is not.

ANTISEIZURE MEDICINES

Ask two questions: what does the drug do to sleep, and what does sleep do to adherence?

DIRECT EFFECTS

Sedation, insomnia and architecture

- Effects differ across medicines and patients
- Dose timing can matter
- Weight gain may increase OSA risk
- Polytherapy amplifies burden

INDIRECT EFFECTS

Seizure control can improve sleep

- Fewer nocturnal seizures and arousals
- Better confidence at bedtime
- But missed night doses may be catastrophic
- Review rather than reflexively add hypnotics

Avoid a rigid drug-by-drug memorisation table. The exam principle is bidirectional assessment and rational timing.

Night-time seizures require an explicit SUDEP conversation

Highest-yield modifiable target

Prevent generalised tonic-clonic and focal-to-bilateral tonic-clonic seizures through effective treatment and adherence.

Discuss individual risk

NICE recommends discussing SUDEP from diagnosis and specifically with people who have seizures during sleep.

Night-time supervision

Consider supervision or a monitor for selected higher-risk patients; explain limits and avoid false reassurance.

Protect sleep

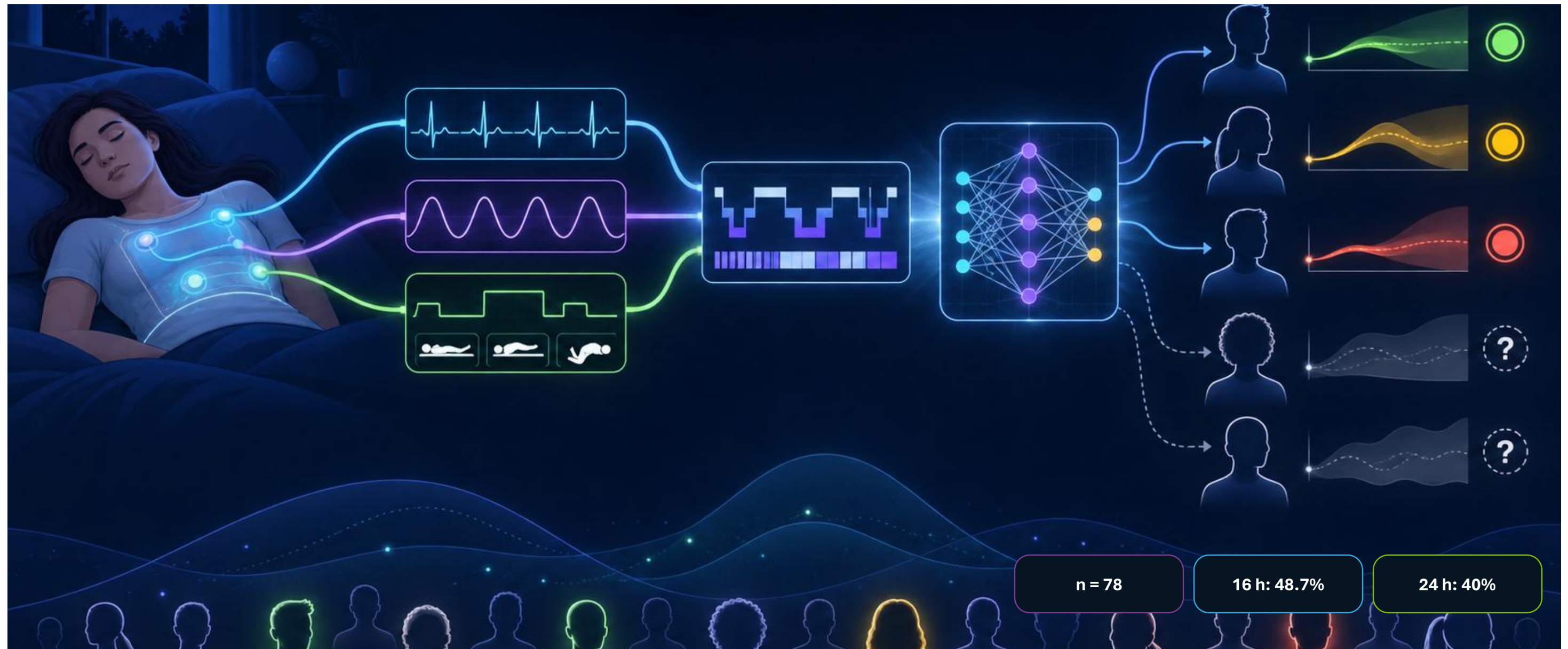
Avoid deprivation and irregular sleep, reduce alcohol/drug misuse, and treat comorbid sleep disorders.

Use calm, individualised language. NICE lists sleeping alone without supervision among potentially modifiable risk factors and recommends discussing night-time supervision for selected higher-risk patients.

Sources: <https://www.nice.org.uk/guidance/ng217/chapter/reducing-the-risk-of-epilepsy-related-death-including-sudden-unexpected-death-in-epilepsy>;
<https://www.ilae.org/patient-care/sudep>

NEW FINDING

Tonight's sleep may carry tomorrow's risk signal



In 78 inpatients wearing a biometric smart shirt, ten nocturnal sleep and physiology features fed a pseudo-prospective support-vector-machine forecast. Better-than-chance performance occurred in 48.7% for a 16-hour horizon and 40% for 24 hours. This is promising for a subset, but the recordings were short and in an epilepsy-monitoring unit. It is not ready for routine clinical risk prediction.

CASE

Case: epilepsy, parasomnia or both?

CASE

A 29-year-old has 20–40 second nocturnal episodes: abrupt arousal, asymmetric tonic posture and bicycling movements, 3–6 times per night, with rapid recovery.

Routine awake EEG normal • partner has two smartphone videos • snores loudly

What is the most useful next strategy?

Review the videos

Prolonged video-EEG

Assess OSA in parallel

Answer: all three in a coordinated pathway. The semiology suggests sleep-related hypermotor epilepsy; normal routine EEG does not exclude it; loud snoring requires parallel OSA evaluation.

EXAM CHECKPOINT

Single best answer

Which sleep state usually activates interictal epileptiform discharges most strongly?

A REM

B NREM

C Wake after sleep onset

D No state effect

Correct answer: B — NREM sleep. Synchrony and slow oscillations facilitate many IEDs, while REM is relatively suppressive.

TAKE-HOME

Five moves improve both diagnosis and outcomes

Treat sleep as a biological variable and a clinical comorbidity.

- 1: Use NREM/REM physiology to interpret seizure timing and EEG yield.
- 2: Screen actively for OSA, insomnia, RLS and insufficient sleep.
- 3: Use witness history and home video before choosing video-EEG versus video-PSG.
- 4: Review antiseizure medicines through the lens of sleep and adherence.
- 5: Discuss nocturnal risk and SUDEP explicitly, individually and constructively.

Improving sleep may improve epilepsy care even when it is not an antiseizure drug.

SOURCES

Core reading

ESRS Sleep Medicine Textbook, 2nd ed. (2021): chapter M2.

Nobili L et al. EAN–ESRS–ILAE-Europe diagnostic consensus. Eur J Neurol 2020.

IFCN–ILAE. Routine and sleep EEG minimum recording standards (2023).

NICE NG217. Epilepsy-related death and SUDEP (updated 2025).

ILAE. SUDEP clinical resource.