

BSS International Sleep Medicine Course 2026

15th - 18th September | Newcastle



British Sleep Society



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ESRS SOMNOLOGIST EXAMINATION · BSS ISMC 2026

Sleep-Related Movement Disorders

Classification, diagnosis and management — updated with current AASM and international guideline evidence

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Conflict of Interest Disclosures for Speakers

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1. I do not have any potential conflicts to disclose.

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2. I wish to disclose the following potential conflicts of interest:

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4. This talk presents material that is related to one or more of these potential conflicts, and the following objective references are provided as support for this lecture:

- 1.
- 2.
- 3.

Learning objectives

1

Classify

Apply the ICSD-3-TR structure for sleep-related movement disorders.

2

Diagnose

Review the core diagnostic criteria for each disorder in the category.

3

Score

Apply AASM PSG scoring rules for periodic limb movements and RMMA.

4

Manage

Apply current international guidelines — AASM, IRLSSG and beyond.

5

Differentiate

Distinguish these disorders from mimics — built for exam preparation.

Introduction

Definition

Sleep-related movements or motor manifestations — physiological, or pathological when they cross into disorder

Characteristics

Simple, stereotypical, involuntary movements

Consequences

Sleep disturbance and daytime symptoms for the patient and/or bed partner

Aetiology

Primary (idiopathic) versus secondary (associated with another medical, neurological or iatrogenic cause)

Importance

Highly prevalent in the general population — most are under-recognised in routine practice

ICSD-3-TR: sleep-related movement disorders

MAIN CATEGORY (9 diagnoses)

- Restless legs syndrome (Willis-Ekbom disease)
- Periodic limb movement disorder (PLMD)
- Sleep-related leg cramps
- Sleep-related bruxism
- Sleep-related rhythmic movement disorder
- Benign sleep myoclonus of infancy
- Propriospinal myoclonus at sleep onset
- Movement disorder due to a medical disorder / medication / substance
- Movement disorder, unspecified

ISOLATED SYMPTOMS & NORMAL VARIANTS

- Excessive fragmentary myoclonus
- Hypnagogic foot tremor & alternating leg muscle activation
- Sleep starts (hypnic jerks)
- Isolated (benign nocturnal) limb cramps

Not a disorder — coded as diagnosis only if frequent/clinically significant.

Recent international guidelines and evidence

2018

IRLSSG iron treatment guideline

Evidence-based/consensus criteria for oral and IV iron therapy in RLS/WED across adults and children (Allen et al., Sleep Med 2018) — still the reference for iron-specific dosing detail.

2024

German DGN/DGSM S2k guideline

National European guideline (Trenkwalder, Stefani, Heidebreder et al.) updates RLS diagnosis and treatment, including pregnancy- and childhood-specific guidance.

2024–25

AASM RLS/PLMD guideline — a paradigm shift

Winkelman et al. (J Clin Sleep Med 2025) replace the 2012 practice parameter: alpha-2-delta ligands now carry STRONG first-line recommendations; standard/long-term dopaminergic use is conditionally recommended AGAINST due to augmentation.

2025

IV iron recommendation strengthened

The same AASM guideline gives a strong recommendation for IV ferric carboxymaltose (appropriate iron status) — the first RLS drug therapy in the guideline to reach top-tier evidence grading.

2025

RLS Foundation updated algorithm

A revised SMAB practical algorithm (successor to 2004/2013/2021 versions) complements the AASM guideline but uses different iron thresholds (TSAT <45%) — expert bodies do not fully align on numeric cut-offs.

2025 (in prep.)

First joint European RLS guideline

EAN, ESRS, European RLS Study Group, EPNS and MDS-ES are preparing a joint European guideline (announced at EAN 2025), expected to echo the shift away from routine dopaminergic first-line therapy.

RLS/WED — overview

DEFINITION

A sensorimotor disorder characterised by an urge to move the legs, usually accompanied by sensory discomfort, worse in the evening or at rest, and totally or partially relieved by movement.

Two recognised subtypes

Early-onset

Strong genetic loading, slow progression, often normal ferritin

Late-onset

Faster progression, often secondary, iron-deficient

Highest reported prevalence in European populations, intermediate in Asian cohorts, lowest in African populations — the reasons remain poorly understood.

5–10%

prevalence in Caucasian/European adults

2:1

female-to-male ratio

2–3%

clinically significant, moderately severe RLS

RLS diagnosis



DIAGNOSTIC CRITERIA

A. Urge to move the legs due to discomfort/unpleasant sensation

- Begins/worsens during rest/inactivity
- Partially/totally relieved by movement
- Exclusively/predominantly occurs in the evening/night

B. Not solely accounted for by another condition

C. Causes concern, sleep disturbance, or impairment in daytime function

Often described during restful activities — eating, watching TV, on a flight or train. Frequently causes insomnia, particularly at sleep onset. Often bilateral/symmetrical; can spread to the abdomen or arms.

SUPPORTIVE FEATURES

- Periodic limb movements on PSG/actigraphy (present in ~80–90% of RLS)
- Positive family history
- Dramatic response to dopaminergic therapy

Validated scales (IRLSSG severity scale) are helpful for diagnosis and monitoring treatment response, but are not required for diagnosis.

RLS mimics

Nocturnal leg cramps

Sudden, painful muscle contraction; little relief with movement

Positional discomfort

No urge to move; relieved by repositioning

Peripheral neuropathy / neuropathic pain

Burning distribution; no clear rest/circadian pattern

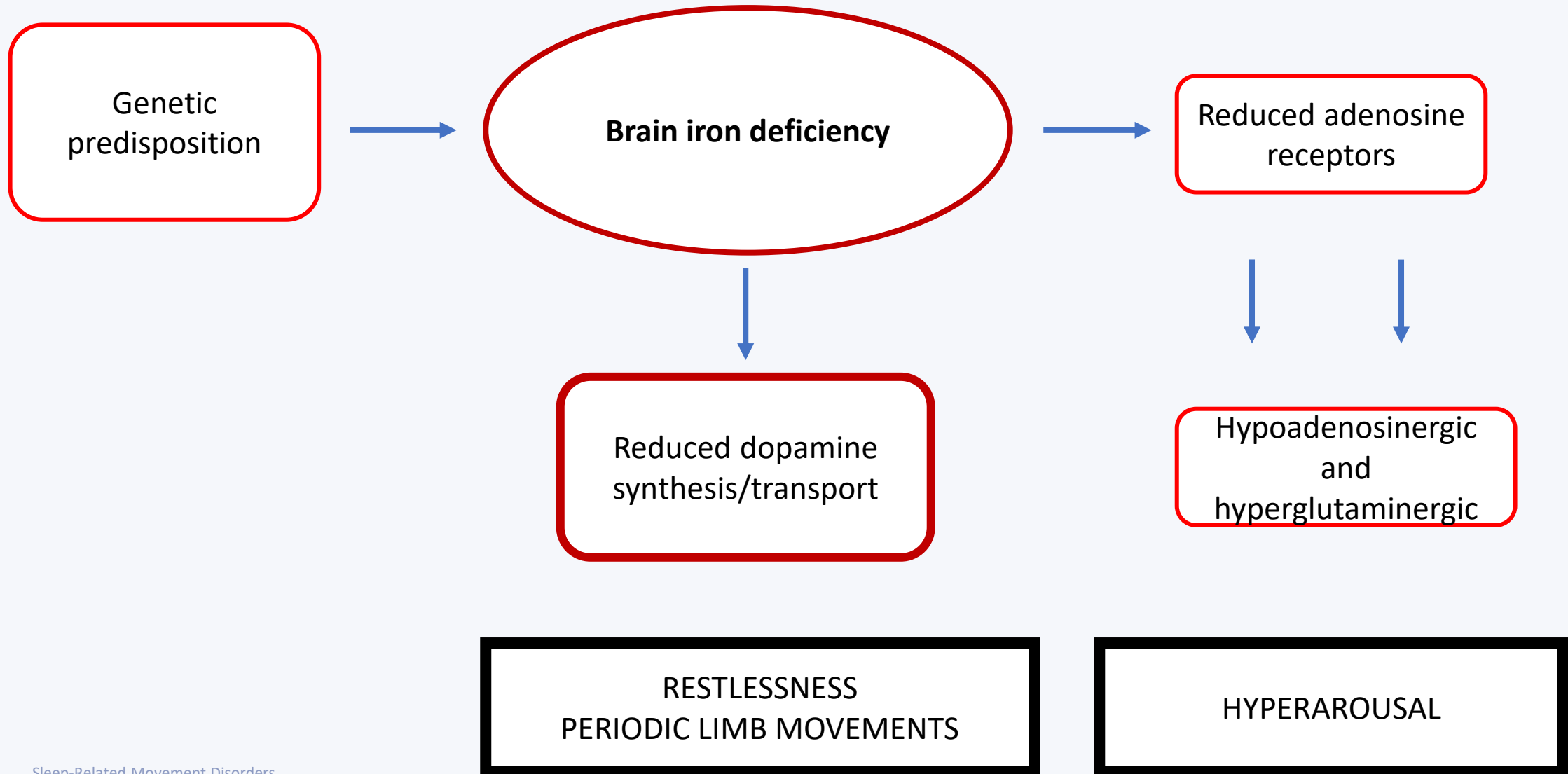
Habitual foot tapping

No distinct urge-to-move sensory component

Venous insufficiency / peripheral vascular disease

Worse with standing, relieved by leg elevation (rather than movement)

RLS pathophysiology



Conditions associated with RLS

Fe

Iron deficiency

AASM 2025: treat if ferritin ≤ 75 $\mu\text{g/L}$ or TSAT $< 20\%$; IV iron only if ferritin is 75–100 $\mu\text{g/L}$ (poor oral absorption in that range)

Preg

Pregnancy

Prevalence 20–30%, peaks in 3rd trimester, usually resolves postpartum

Kid

End-stage kidney disease

Prevalence 20–30% in dialysis patients; improves after renal transplantation

Nrv

Peripheral neuropathy

Small-fibre neuropathy; burning/painful presentation

Rx

Medications

Antidepressants (mirtazapine, SSRIs, SNRIs), antihistamines, antidopaminergics/antiemetics

Age

Age

Prevalence and PLM index both rise with advancing age

RLS management — non-pharmacological

STRATEGIES FOR DISCOMFORT / DAYTIME ALERTNESS

- Good sleep hygiene and a regular sleep schedule
- Massage, hot baths
- Tactile/temperature stimulation of the legs

ADDRESS CONTRIBUTORS

- Medicines review — stop/switch aggravating drugs
- Treat comorbid OSA — may reduce RLS control difficulty
- Screen for and correct exacerbating factors (caffeine, alcohol)

IRON REPLACEMENT

- Oral iron typically sufficient — first choice when ferritin ≤ 75 $\mu\text{g/L}$ and TSAT $< 20\%$
- Move to IV iron if: not responding appropriately, problems with oral formulation/tolerance, severe symptoms needing a more rapid response, or ferritin already 75–100 $\mu\text{g/L}$ (poor oral absorption)
- AASM 2025: treat if ferritin ≤ 75 $\mu\text{g/L}$ or TSAT $< 20\%$; IV iron only if ferritin is 75–100 $\mu\text{g/L}$ (poor oral absorption in that range); strong recommendation for IV ferric carboxymaltose (appropriate iron status) — the guideline's only top-tier drug recommendation alongside the alpha-2-delta ligands

RLS management — pharmacological

Alpha-2-delta ligands

- Pregabalin 150–450 mg
- Gabapentin 900–3600 mg (gabapentin enacarbil also strong)
- S/Es: weight gain, drowsiness, dizziness, depression
- AASM 2025: STRONG recommendation for each individually — moderate-certainty evidence

Dopamine agonists

- Pramipexole 0.125–0.75 mg at night
- Ropinirole 0.25–4 mg at night
- Rotigotine patch 1–3 mg/24h
- S/Es: augmentation, impulse control difficulties, nausea, headache, fatigue, dizziness
- AASM 2025: conditional recommendation AGAINST standard/long-term use (augmentation risk); cabergoline — strong recommendation against (cardiac valvulopathy)

Refractory disease

- Low-dose opioids, sedation (benzodiazepines, Z-drugs, daridorexant)
- AASM 2025 conditional options: extended-release oxycodone, dipyridamole, bilateral high-frequency peroneal nerve stimulation (new)

Augmentation

DEFINITION

A gradual worsening of RLS symptom intensity and duration with dopaminergic treatment — at least one of the following, relative to before treatment:

- Earlier symptom onset than prior to dopaminergic treatment
- Reduced latency to symptom onset when at rest
- Extension of symptoms to other parts of the body

Onset typically after several months to years of dopamine-agonist exposure, and worse with higher doses.

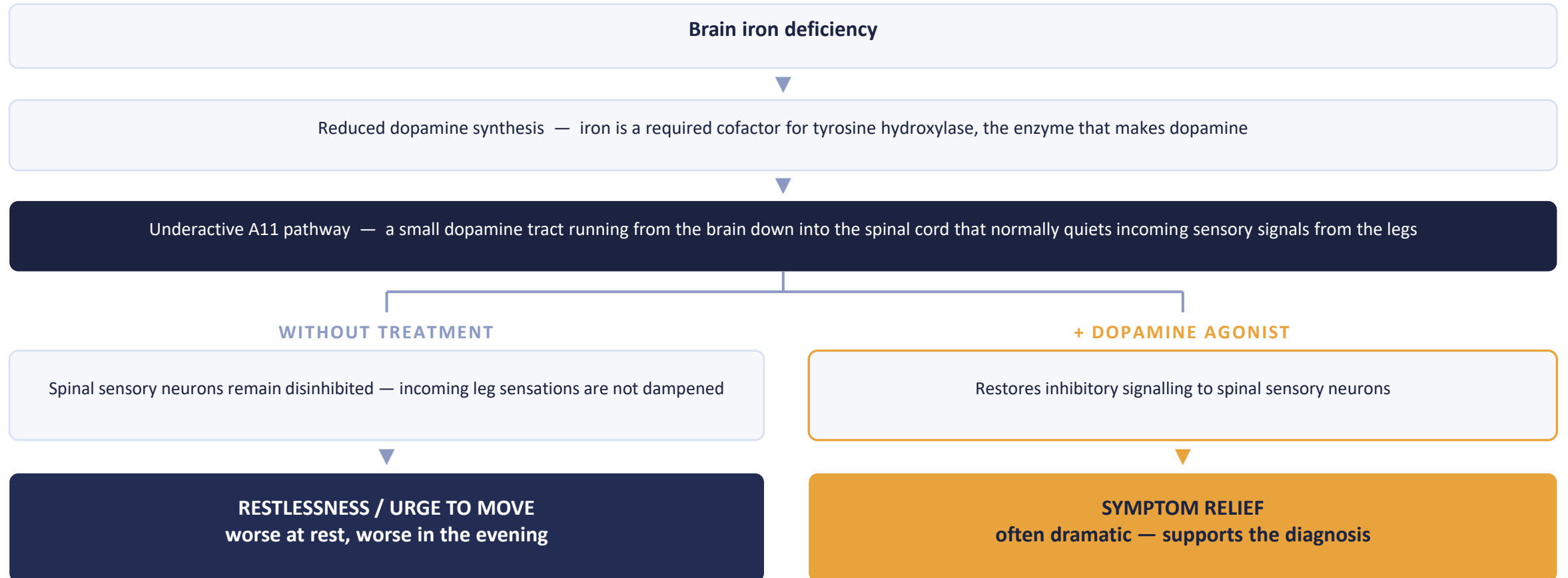
RISK FACTORS

- Duration/dose of dopamine-agonist exposure
- Short-acting agents > long-acting
- More severe RLS symptoms at baseline
- Iron deficiency

MANAGEMENT

- Correct iron deficiency
- Wean/withdraw dopamine agonist with cross-titration to an alpha-2-delta ligand
- AASM 2025: best prevention is avoiding standard/long-term dopaminergic therapy from the outset

Why dopamine agonists work initially



This is why a dramatic response to a dopamine agonist is itself a supportive diagnostic feature for RLS — but the same mechanism that produces this early relief sets up the problem explained on the next slide.

Why the relief reverses over time

Chronic dopamine agonist exposure — risk rises with higher doses, short-acting agents, and longer duration of use

Receptor downregulation

Dopamine receptors turn down their own sensitivity in response to constant, non-physiological stimulation

Spinal sensory sensitisation

A11 inhibition fails sooner and less completely; spinal sensory neurons become more excitable — a lower threshold triggers symptoms

Worsening brain iron

Dopamine agonists may further reduce brain iron availability, deepening the original deficit that caused RLS

NET EFFECT: disinhibited, over-sensitive spinal sensory neurons

Earlier onset in the day

Faster onset at rest

Spreads beyond the legs

The trap: raising the dose relieves symptoms briefly but drives further receptor downregulation — worsening augmentation. Correct approach: cross-titrate to an alpha-2-delta ligand, and correct iron deficiency; never simply increase the dopaminergic dose.

Periodic limb movements in sleep (PLMS)

PLMS are periodic episodes of repetitive, stereotyped limb movements (typically extension of the great toe with dorsiflexion of the ankle, $\pm$ knee/hip flexion) occurring predominantly in NREM sleep.

Duration 0.5–10 seconds per leg movement

Amplitude $\geq 8 \mu\text{V}$ increase above resting EMG baseline

Inter-movement interval 5–90 seconds between onsets (start-to-start)

Series definition ≥ 4 consecutive movements meeting the above criteria = a PLM series

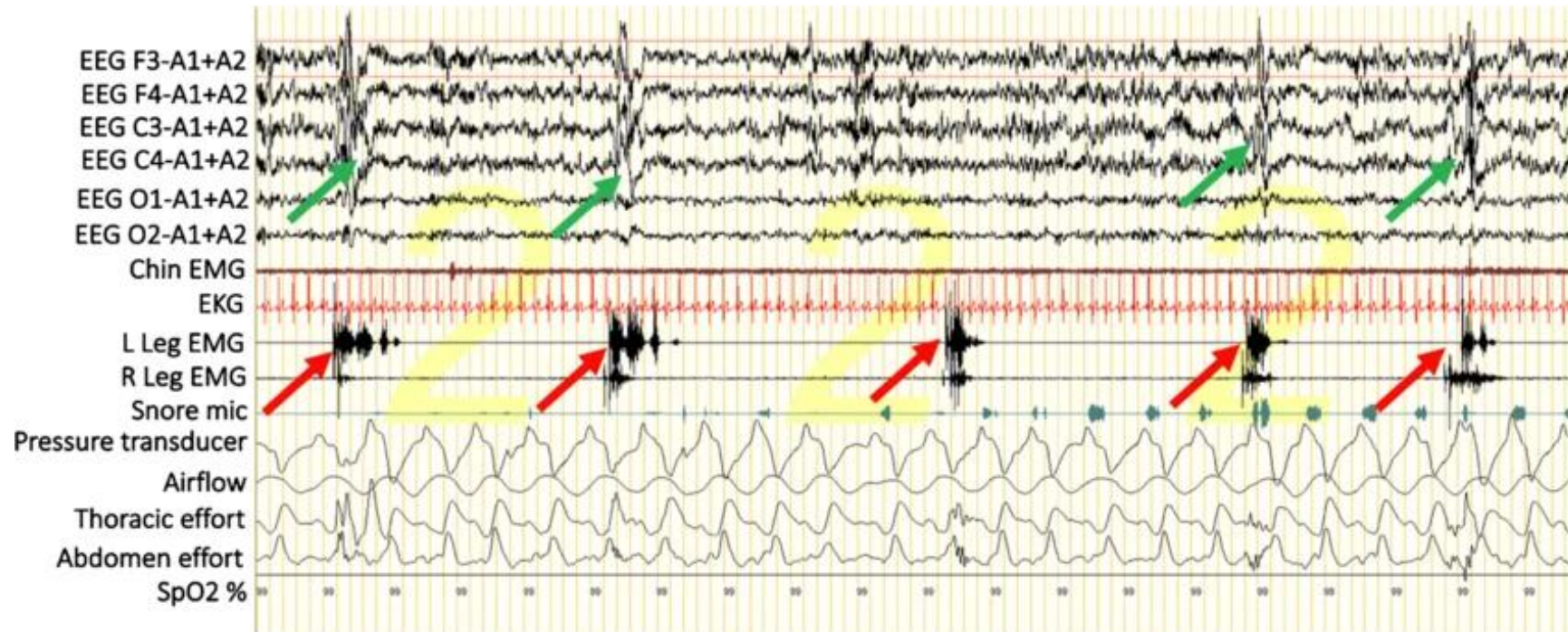
Bilateral timing rule Movements in both legs < 0.5 s apart are counted as a single movement

Arousal association Scored whether or not associated with an EEG arousal or respiratory event

Exclusion Movements occurring within 0.5 s of a respiratory event are NOT scored as PLM

Periodic limb movements in sleep (PLMS)

PLMS are periodic episodes of repetitive, stereotyped limb movements (typically extension of the great toe with dorsiflexion of the ankle, $\pm$ knee/hip flexion) occurring predominantly in NREM sleep.



Periodic limb movement disorder (PLMD)

1

PSG finding

Elevated PLMI on PSG — commonly cited thresholds: >15/h in adults, >5/h in children

2

Clinical impact

Sleep fragmentation / sleep-maintenance insomnia, and impairment in daytime function

3

Not better explained

AASM 2025 good practice statement: exclude other conditions with high rates of PLMS first — untreated OSA, RBD, and narcolepsy

4

RLS caveat

If criteria for RLS are also met, PLMS are usually a supportive feature — not an additional/independent diagnosis

5

Treatment evidence is thin

Largely shares management algorithm with RLS

PLM associations — a non-specific finding

RLS

Present in 80–90% of RLS

Narcolepsy

Elevated PLMI commonly seen

REM behaviour disorder

Frequent co-occurrence

OSA

Movements may follow respiratory events — or trigger respiratory instability

Medications

SSRIs, SNRIs, TCAs

Medical disease

End-stage kidney disease

Neurodegenerative disease

Parkinson's disease, MSA, dementia with Lewy bodies

Ageing

PLMI typically rises after age 50–60

Sleep-related leg cramps

DEFINITION

Sudden, involuntary, sustained and painful muscle contractions of the leg muscles (gastrocnemius, soleus, small muscles of the foot).

CLINICAL FEATURES

- Occurs during the sleep period
- Hard, contracted muscle on palpation
- Relieved by stretching
- Not explained by another disorder (e.g. RLS)

EPIDEMIOLOGY

More common in the elderly population:

Up to 1/3

in adults over 60

Up to 1/2

in adults over 80

Leg cramps — secondary causes and management

SECONDARY CAUSES TO SCREEN FOR

- Metabolic alteration — hypokalaemia, hypocalcaemia, hypomagnesaemia; dehydration
- Neurological problems — spinal stenosis, peripheral neuropathy
- Peripheral vascular disease
- Cirrhosis
- Drugs — OCP, diuretics, IV iron sucrose, long-acting beta agonists, statins

MANAGEMENT

- Address any underlying primary cause identified above
- Passive muscle stretching, three times daily
- Low-dose clonazepam for frequent, disruptive cramps
- Some historic evidence for quinine — no longer routinely recommended given its safety profile

Sleep-related bruxism

DEFINITION

Repetitive jaw-muscle activity by clenching/grinding/tapping of teeth and/or bracing/thrusting of the mandible, in light NREM and REM sleep — rhythmic masticatory muscle activity (RMMA) on PSG EMG.

Consequences

- Tooth wear / fracture
- Masticatory hypertrophy, jaw pain, TMJ disorder
- Morning headache
- Bed-partner disturbance

EPIDEMIOLOGY

Prevalence decreases over the lifetime:

Up to 20% of children

~8% of adults

~3% of the elderly

Can be associated with sleep-disordered breathing.

Management of sleep-related bruxism

1

Sleep hygiene

General measures to support consolidated, good-quality sleep.

2

Behavioural — stress management

Address a common trigger/aggravating factor directly.

3

Oral appliances / mandibular advancement devices

Protect dentition from wear and fracture — do not abolish RMMA event frequency.

4

Treat OSA if present

Bruxism episodes cluster around respiratory events and arousals — treating SDB may reduce bruxism.

5

Pharmacological options

Not usually given — limited evidence base for drug therapy.

6

Botulinum toxin

Can be considered in adults with refractory, severe bruxism.

Sleep-related rhythmic movement disorder (SRRMD)

DEFINITION

- Rhythmic, repetitive, stereotyped, large-amplitude movements of large muscle groups
- Body rocking, head banging (jactatio capitis nocturna), head rolling, body rolling
- Sleep onset through light NREM sleep

Epidemiology

- Up to 60% of infants — considered a normal developmental phenomenon
- Largely resolves by age 4–5
- Much lower persistence into adulthood; when it persists, consider ASD, learning disability, or psychiatric comorbidity

MANAGEMENT

- Usually self-limiting — reassurance is often all that is needed
- Safe sleep environment — padding, moving the bed from walls
- Sleep hygiene
- Benzodiazepines may be an option in adults with persistent, injurious disease

Sleep-onset & infantile myoclonus syndromes

PROPRIOSPINAL MYOCLONUS AT SLEEP ONSET

- Sudden axial myoclonic jerks (trunk/abdomen flexion, sometimes limbs) while falling asleep — 'sit-up' or 'jack-knife' movements
- Originates from a spinal cord generator, propagating slowly up/down the cord
- Rare, more often reported in men
- Can prevent sleep onset, causing significant insomnia complaints

BENIGN SLEEP MYOCLONUS OF INFANCY

- Repetitive myoclonic jerks of limbs/trunk, usually during the first few weeks of life
- Occurs exclusively during sleep; stops immediately on awakening/arousal
- Rare; more common in males (M>F); typical age range up to 3–6 months
- Normal neurological exam and normal EEG — key distinguishing point from epileptic myoclonus/infantile spasms

Isolated symptoms and normal variants

- **Excessive fragmentary myoclonus (EFM)**

Small, asynchronous twitches of fingers, toes or mouth corners on EMG, without visible movement; incidental in up to 10% of the population unless frequent/significant enough to be symptomatic

- **Hypnic jerks (sleep starts)**

Sudden, brief, single jerk of body/limbs at sleep onset, often with a sensation of falling; extremely common, no clinical significance

- **Hypnagogic foot tremor / alternating leg muscle activation**

Rhythmic EMG activity in tibialis anterior at sleep onset or in light NREM; benign PSG finding, no treatment required

- **Isolated (benign nocturnal) limb cramps**

Occasional cramps not meeting frequency/impact threshold for the disorder — common

NREM arousal vs. RBD vs. nightmare vs. movement disorder vs. NFLE

Feature	NREM disorder of arousal	RBD	Nightmare disorder	Sleep-related movement disorder	Nocturnal frontal lobe epilepsy
Timing	First third of night (slow-wave sleep)	Second half of night (REM-dense)	Second half of night (REM-dense)	Any sleep stage (disorder-specific)	Any stage; often clusters, may recur same night
Recall / responsiveness	Amnesia for event; poorly responsive during episode	Vivid dream recall; may respond once fully roused	Detailed, vivid recall; rapid full orientation on waking	Usually aware; may recall (e.g. RLS)	Variable; often retains awareness
Behaviour / motor activity	Complex, semi-purposeful (walking, confusion, terror)	Violent dream enactment, vocalisation	None — awakening only, no motor signature	Simple, stereotyped, repetitive	Brief (<2 min), stereotyped, dystonic/hyperkinetic
PSG correlate	Arousal from slow-wave sleep	REM sleep without atonia (RSWA)	Awakening from REM, no motor signature	None — motor/PSG finding (e.g. PLM criteria)	Epileptiform discharge may be absent on scalp EEG
Key work-up	History (bed-partner/parent account); video-PSG if atypical, injurious, or uncertain	Video-PSG with RSWA quantification (SINBAR limb EMG)	Clinical history; screen for REM-fragmenting pathology/PTSD/comorbid psychiatric disease	PSG with limb EMG ± ferritin/TSAT	Video-EEG-PSG, consider MRI

High-yield take-home points

1

RLS needs all of A-C met: urge to move + rest/circadian pattern + relief with movement, and exclusion of mimics

2

AASM 2025: alpha-2-delta ligands (pregabalin, gabapentin, gabapentin enacarbil) are now the only **STRONG** first-line drug class

3

Standard/long-term dopamine agonists are now a conditional recommendation **AGAINST** — augmentation risk; cabergoline is strongly against (valvulopathy)

4

Iron: treat if ferritin ≤ 75 $\mu\text{g/L}$ or TSAT $< 20\%$; use IV iron specifically when ferritin is 75–100 $\mu\text{g/L}$

5

IV ferric carboxymaltose carries a strong recommendation — the guideline's top drug-therapy grade alongside the gabapentinoids

6

PLMD is diagnosed only when RLS, OSA, RBD and narcolepsy are excluded and there is clinical impact — not from an index alone

7

PLM scoring: 0.5–10 s duration, ≥ 8 μV , 5–90 s inter-movement interval, ≥ 4 movements per series

8

Augmentation = earlier onset, shorter latency, spread to other body parts — never treat by raising the dopaminergic dose

9

A joint European (EAN/ESRS) RLS guideline is in preparation and expected to echo the AASM's shift away from dopaminergic first-line therapy

Key references

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