



# Unmasking of Periodic Limb Movements During CPAP Titration in a Non-obese Older Woman with Positional Obstructive Sleep Apnea: A Case Report

Fan-Hsuan Kung, M.D.<sup>1</sup>, Chuan-Yen Sun, M.D.<sup>2</sup>, Tzu-Hui Hsiao, M.D.<sup>2</sup>, Kun-Ta Chou, M.D., Ph.D.<sup>2\*</sup>

<sup>1</sup> Department of Psychiatry, Tri-Service General Hospital Beitou Branch, National Defense Medical University, Taipei, Taiwan  
<sup>2</sup> Division of Clinical Respiratory Physiology, Department of Chest Medicine, Taipei Veterans General Hospital, Taipei, Taiwan  
\* Corresponding author

## Background and Introduction

Periodic limb movements during sleep (PLMS) are stereotyped, repetitive movements—typically great-toe extension with ankle dorsiflexion—scored from anterior tibialis EMG on polysomnography (PSG) according to AASM rules: each movement lasts 0.5–10 s with amplitude ≥8 μV above baseline, ≥4 consecutive movements occur 5–90 s apart, and movements within 0.5 s of a respiratory event are *not* scored [1,2]. PLMS are a PSG **finding**, not a disease: their prevalence rises steeply with age (PLM index [PLMI] >15/h in 28–33% of middle-aged adults and in 45–61% of adults over 65) and they accompany restless legs syndrome (RLS) in 80–90% of cases [3–5]. Periodic limb movement disorder (PLMD) additionally requires PLMI >15/h in adults **and** clinically significant sleep disturbance or daytime impairment that is not better explained by another disorder (RLS, OSA, narcolepsy, RBD, medication)—a diagnosis of exclusion whose validity in adults remains debated [1,6,10].

In obstructive sleep apnea (OSA), leg movements are frequently coupled to respiratory events and arousals. Once the airway is stabilized with continuous positive airway pressure (CPAP), PLMS may fall (respiratory-induced movements) or, conversely, may be **unmasked** and increase—especially in severe OSA, low baseline PLMI and non-obese patients [7–9]. We report a woman with moderate positional OSA in whom significant PLMS emerged only during CPAP titration, and discuss how such a finding should be interpreted and managed.

## Case Presentation

A 65-year-old non-obese woman (BMI 20.3 kg/m<sup>2</sup>, neck circumference 31 cm) with hypertension, coronary artery disease, cerebral aneurysm, allergic rhinitis and diet-controlled hyperlipidemia (non-smoker) presented to the chest out-patient department with habitual snoring, excessive daytime sleepiness and fragmented, non-restorative sleep. Diagnostic PSG (August 2025) showed **moderate, strongly positional OSA** (AHI 27.0/h; supine 37.7/h vs. lateral 0.0/h; REM 40.8/h) without significant hypoxemia (nadir SpO<sub>2</sub> 90%, ODI 17.4/h), poor sleep efficiency (77.5%, WASO 71 min), an arousal index of 23.3/h and only sparse limb movements (LM index 11.0/h; **PLMI 0.8/h**). Skull films showed no micrognathia but extensive dental implants with posterior tooth loss, so CPAP titration was arranged.

Whole-night CPAP titration (February 2026; clonazepam 0.5 mg at bedtime) achieved optimal control at **5.0 cmH<sub>2</sub>O** (AHI 0.5/h overall, 0.0/h at 5 cmH<sub>2</sub>O; SpO<sub>2</sub> mean 95%, minimum 90%; sleep efficiency 91.1%; arousal index 4.8/h; ESS 5; BP 114/68 → 112/69 mmHg). Unexpectedly, the study revealed **significant periodic limb movements—PLMI 47.9/h (270 episodes)—together with REM sleep without atonia**, i.e., a >50-fold rise in PLMI once respiratory events were abolished. Iron studies, medication review, screening for RLS/dream-enactment and symptom-guided follow-up were planned.

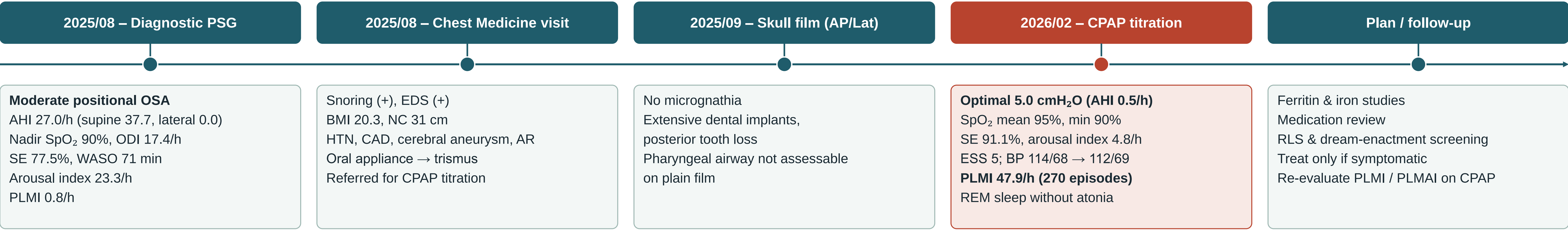
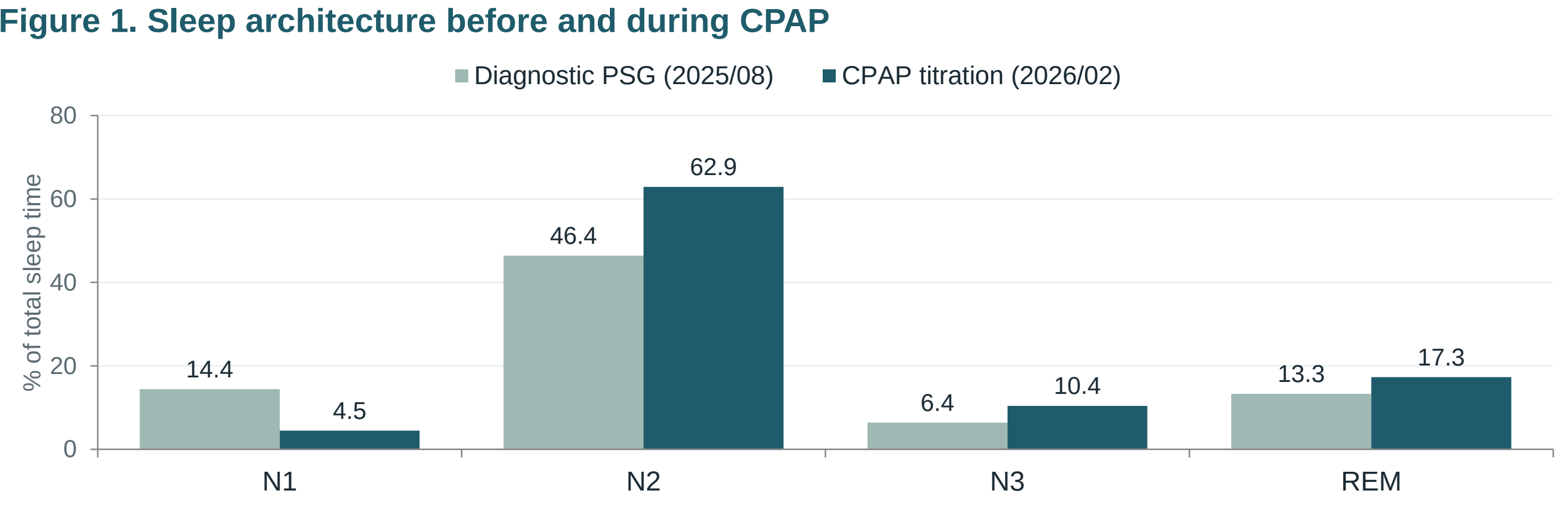
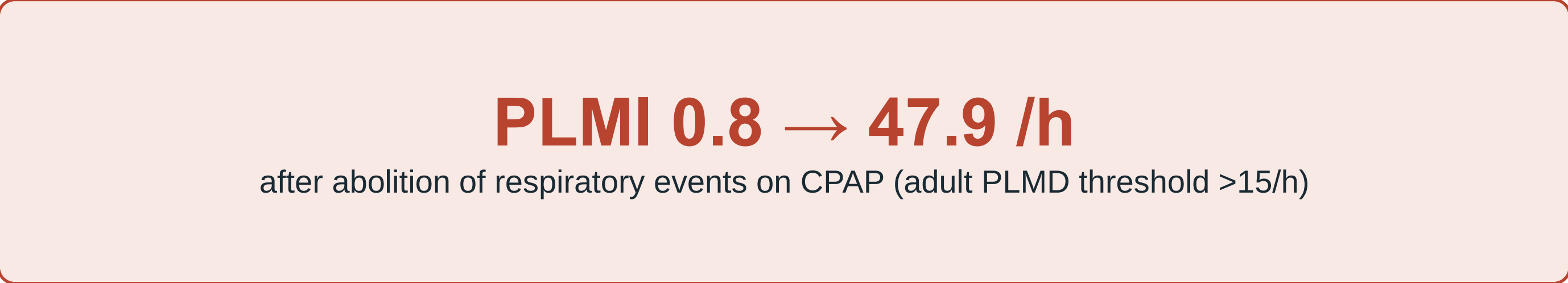


Table 1. Polysomnographic parameters: diagnostic PSG vs. CPAP titration night

| Parameter                           | Diagnostic PSG (2025/08)    | CPAP titration (2026/02)          |
|-------------------------------------|-----------------------------|-----------------------------------|
| AHI (events/h)                      | 27.0                        | 0.5 (0.0 at 5 cmH <sub>2</sub> O) |
| Supine / lateral AHI                | 37.7 / 0.0                  | supine only, 0.5                  |
| Mean / minimum SpO <sub>2</sub> (%) | 95.9 / 90                   | 95 / 90                           |
| Desaturation index (/h)             | 17.4                        | 0.2                               |
| Sleep efficiency (%)                | 77.5                        | 91.1                              |
| Sleep latency / REM latency (min)   | 14.0 / 193.0                | 16.0 / 99.0                       |
| N1 / N2 / N3 / REM (% sleep)        | 14.4 / 46.4 / 6.4 / 13.3    | 4.5 / 62.9 / 10.4 / 17.3          |
| Arousal index (/h)                  | 23.3                        | 4.8                               |
| Snoring episodes                    | 1093                        | 109 (index 19.4/h)                |
| Limb movement index (/h)            | 11.0                        | —                                 |
| PLM index (/h)                      | 0.8                         | 47.9 (270 episodes)               |
| Epworth Sleepiness Scale            | —                           | 5                                 |
| Other findings                      | Prominent positional effect | REM sleep without atonia          |



## Discussion and Conclusion

The >50-fold rise in PLMI (0.8 → 47.9/h) once the airway was stabilized illustrates **CPAP-emergent (unmasked) PLMS**. Three mechanisms converge. First, AASM rules exclude any leg movement within 0.5 s of a respiratory event, so movements time-locked to apneas and hypopneas at baseline are not scored, whereas the same generator produces “free” PLMS once breathing is normalized [2,7]. Second, CPAP consolidates sleep (sleep efficiency 77.5 → 91.1%, arousal index 23.3 → 4.8/h), providing more stable NREM sleep—the state in which periodic movements are expressed. Third, the phenomenon has been reproduced in cohorts: Baran et al. found PLMI increased on CPAP in severe OSA but decreased in mild OSA [7]; in the APPLES cohort PLMS were common in OSA and their trajectory on CPAP was heterogeneous [8]; and a meta-analysis of 14 studies (n = 2,938) showed a net increase in PLMI after CPAP, greatest in patients with **baseline PLMI <5/h, BMI <30 kg/m<sup>2</sup> and higher baseline AHI/arousal indices** [9]—precisely this patient’s profile (age >65, female, BMI 20, PLMI 0.8/h, arousal index 23/h). Night-to-night variability of PLMI is large, so a single titration night should be interpreted cautiously [6]; concurrent clonazepam reduces movement-related arousals but does not lower PLMI itself, and benzodiazepines are conditionally recommended *against* for RLS/PLMD in the 2025 AASM guideline [10]. REM sleep without atonia in an older adult on clonazepam warrants a targeted history of dream enactment and longitudinal follow-up.

Does this patient have PLMD? Not on present evidence: she is not sleepy on CPAP (ESS 5), and PLMS in the setting of OSA and CPAP are common incidental findings—“**treat the disorder, not the number**” [1,6,10]. PLMS are, however, relevant to her cardiovascular history: each movement is accompanied by transient heart-rate (~7–10 bpm) and blood-pressure (~22/11 mmHg) surges, and PLMS are associated with prevalent hypertension (RR 1.26, 95% CI 1.12–1.41), although causality and any benefit of suppressing PLMS remain unproven [11,12]. A guideline-concordant plan therefore includes: (1) serum ferritin and transferrin saturation—oral iron if ferritin <75 μg/L, intravenous ferric carboxymaltose (strong recommendation) if ≤100 μg/L; (2) review of PLMS-provoking drugs (SSRIs/SNRIs, TCAs, mirtazapine, antihistamines, dopamine antagonists); (3) structured screening for RLS (URGE criteria) and secondary causes; (4) pharmacotherapy—α2δ ligands (gabapentin, gabapentin enacarbil, pregabalin; strong recommendation) rather than dopamine agonists (conditional against, augmentation risk)—**only if** sleep disturbance or daytime impairment attributable to PLMS persists; and (5) reassessment of PLMI and PLM-arousal index on established CPAP therapy [10].

**In conclusion**, untreated OSA can mask periodic limb movements, and a marked rise in PLMI during CPAP titration should be anticipated rather than alarming. PLMI must be interpreted in the context of age, medications, sleep continuity and symptoms; ICSD-3 TR exclusion criteria should be applied before diagnosing PLMD; and treatment—iron repletion and α2δ ligands as first line—is reserved for symptomatic patients, with attention to the cardiovascular comorbidity that PLMS may accompany.

### References

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