Neurologie und Schlafstörungen: Neue Therapien und Hands-On Diagnostik für den klinischen Alltag

Priv.-Doz. Dr. med. Anna Heidbreder
Anna.Heidbreder@kepleruniklinikum.at

Fahrplan

- Was sollte ich als Neurolog:in aus der Schlafmedizin kennen?
- Wann brauche ich ein „Schlaflabor“?
- Warum ist Schlaf-Neurologie?

Funktionen des Schlafes:
Regeneration
Lernen
Immunsystem
Reinigungsprozesse
Wachstum
…

Schlaf ist eine Funktion des Gehirns und dient dem Gehirn

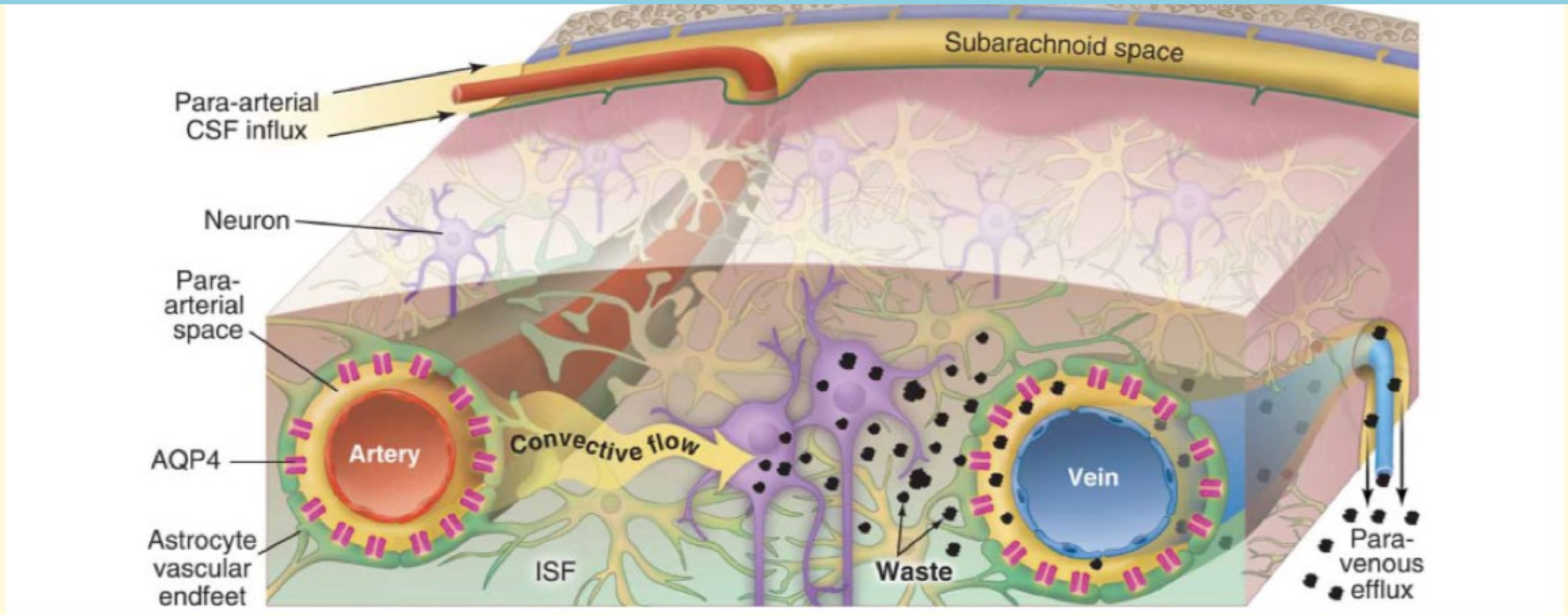
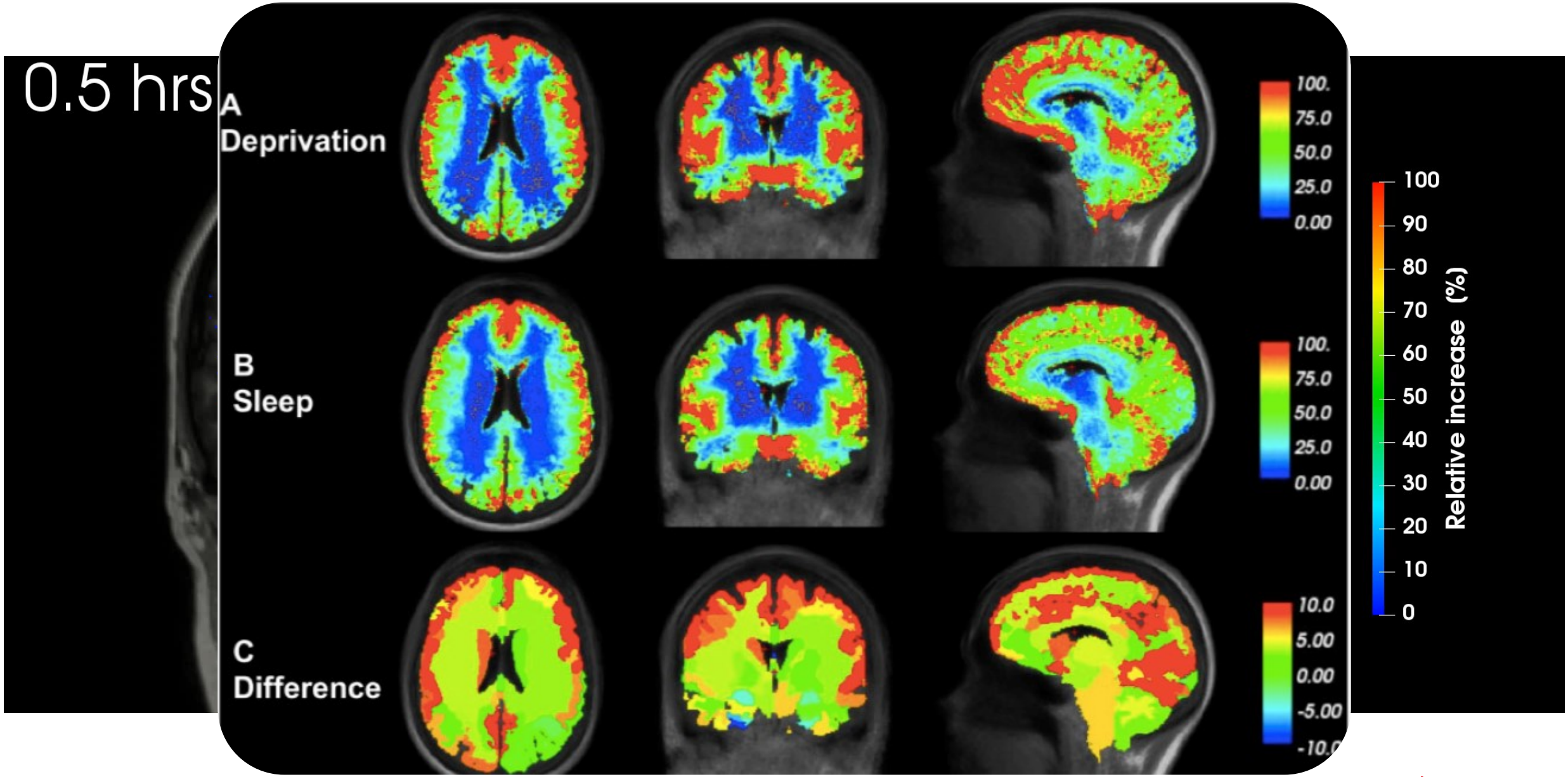


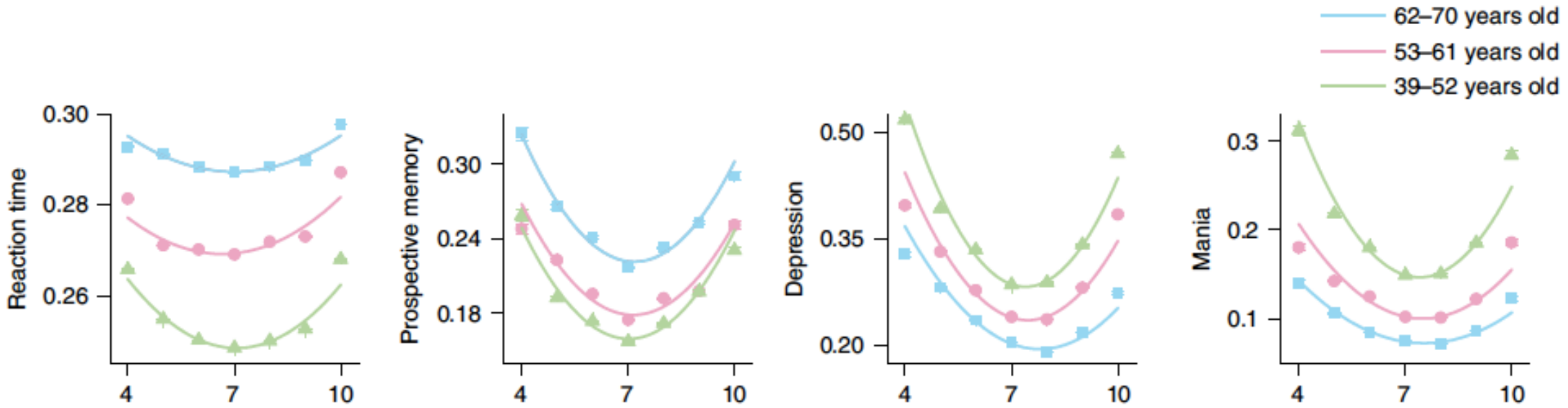
Figure 1 Schematic representation of the glymphatic system. Aquaporin 4 (AQP4) on the vascular endfeet of astrocytes facilitates convective flow from the para-arterial space into the interstitial space, where CSF exchanges with the interstitial fluid (ISF), which then enters the paravenous space. In this way, vectorial convective fluxes drive waste products away from the arteries and towards the veins. From Nedergaard, 2013. Science 340:1529–30. Reprinted with permission from AAAS.


0.5 hrs
A
Deprivation
B
Sleep
C
Difference
100.
75.0
50.0
25.0
0.00
100.
75.0
50.0
25.0
0.00
10.0
5.00
0.00
-5.00
-10.0
100
90
80
70
60
50
40
30
20
10
0
Relative increase (%)

nature aging

ARTICLES

https://doi.org/10.1038/s43587-022-00210-2



Practice points

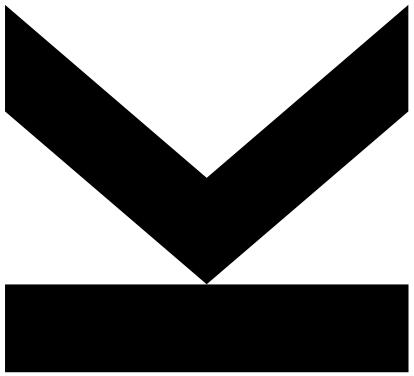
1. [redacted] largely due to significant variability in the methodology and quality of published studies.
2. [redacted] influence of individual trait characteristics. This makes large group generalizations regarding sleep quality difficult and highlights the importance of individualizing assessments of subjective sleep quality and relationships with physiologically measured sleep.
3. [redacted] polysomnography.
4. [redacted] motor performance and subjectively perceived sleep quality.
5. [redacted] contribution to executive function and subjectively perceived sleep.
6. [redacted] performance.
7. Circadian variation in performance, chronotype and sleep inertia are important confounders to account for in studies of sleep and performance.

CLINICAL REVIEW

Physiological markers of sleep quality: A scoping review

Stuart J. McCarter [a, b, *], Philip T. Hagen [c], Erik K. St. Louis [a, b, c], Thomas M. Rieck [e], Clifton R. Haider [f], David R. Holmes [f], Timothy I. Morgenthaler [a, c, d]

Was sollte ich als Neurolog:in von der Schlafmedizin kennen?

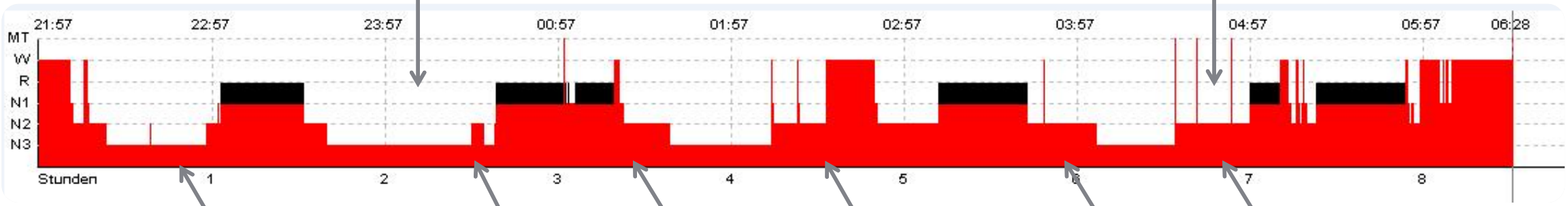


NON-REM-PARASOMNIA

CONFUSIONAL AROUSAL
SLEEP WALKING
SLEEP TERROR
SLEEP RELATETD EATING

REM-PARASOMNIA

REM-SLEEP BEHAVIOR DISORDER
ISOLATED SLEEP PARALYSIS
VIVID DREAM (DISORDER)



SLEEP REALTED MOVEMENT DISORDERS

RESTLESS LEGS SYNROME
BRUXISM
RHYTHMIC MOVEMENT DISORDERS
HYPNAGOGIC FOOT TREMOR FUSSTREMOR
SLEEP START MYOCLONUS
PROPRIOSPINALE MYOCLONUS
PERIODIC LIMB MOVEMENTS DURING SLEEP
EXZESSIVE FRAMENTARY MYOCLONUS

SLEEP DISORDERED BREATHING

OBSTRUKTIVE SLEEP APNEA
CENTRAL SLEEP APNEA
HYPOVENTILATION SYNDROME

Parasomnien

Idiopathic REM sleep behaviour disorder and neurodegeneration — an update

Birgit Högl[1], Ambra Stefani[1] and Aleksandar Videnovic[2]

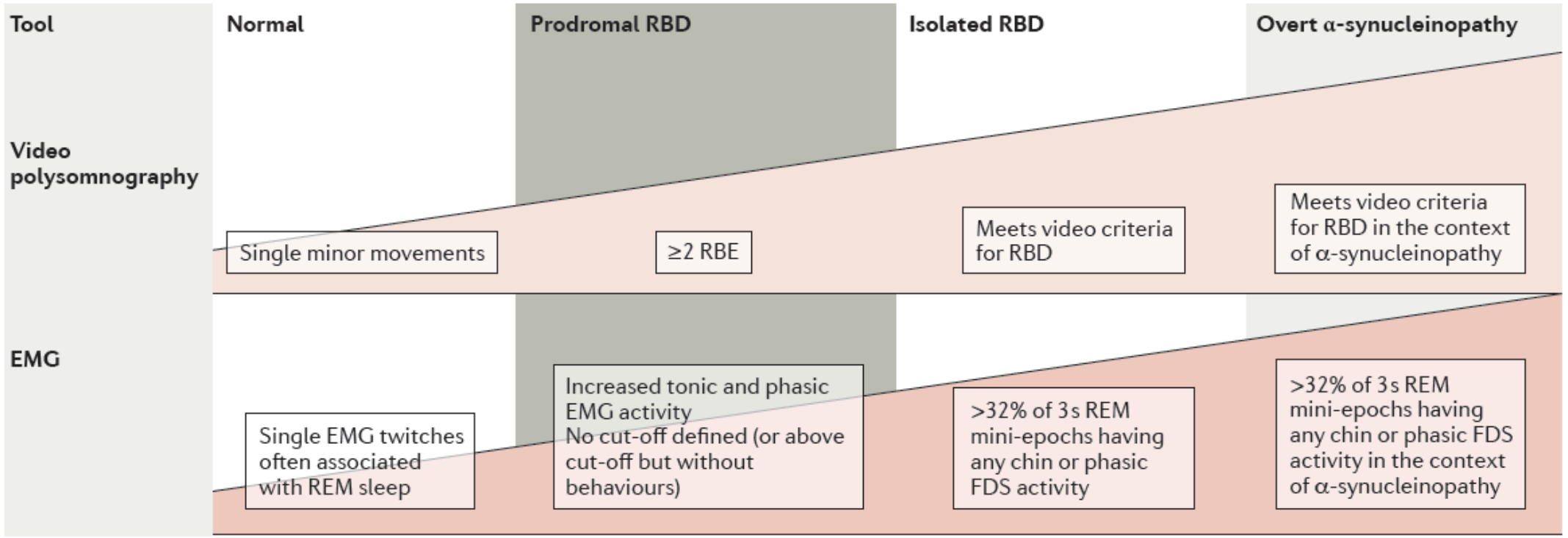
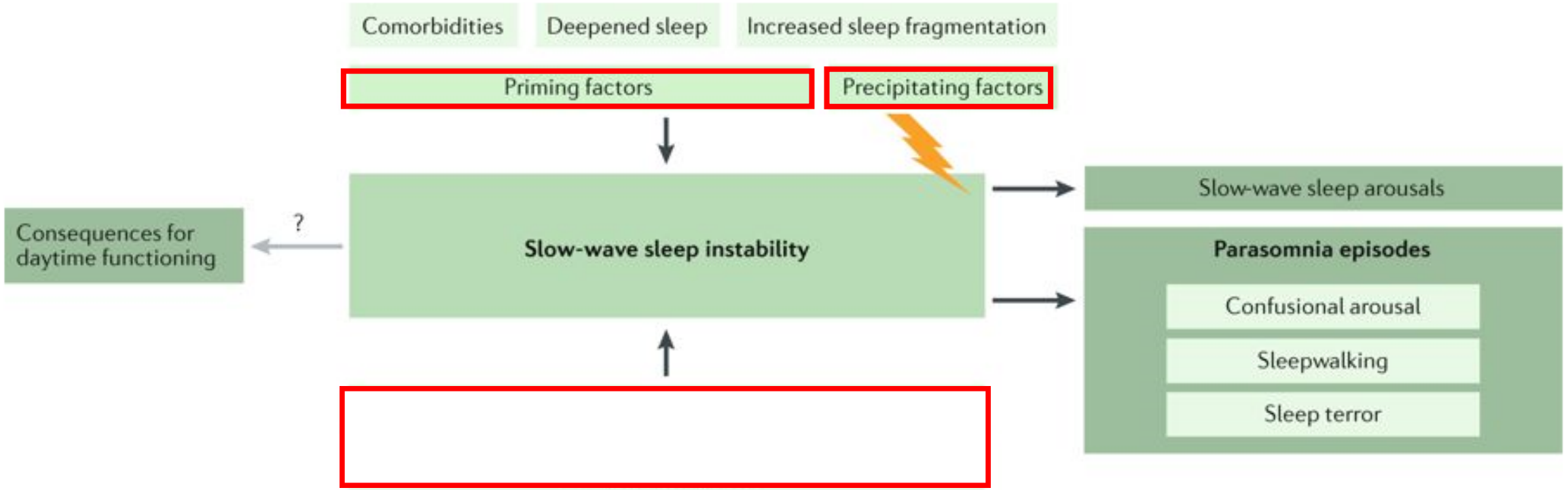


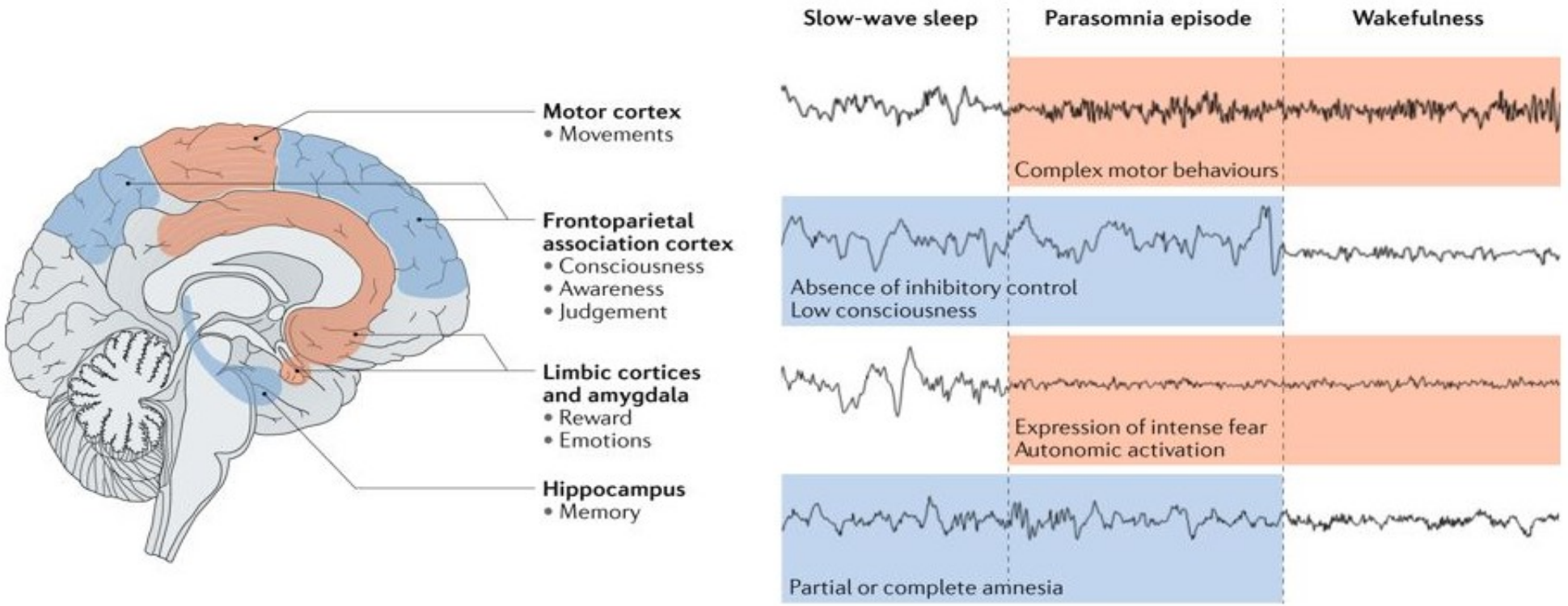
Figure 2 | **The new concept of prodromal RBD.** Neurophysiological and behavioural findings on polysomnography and EMG (electromyography) progress along a continuum over time. From initially normal findings, patients enter a prodromal stage of rapid eye movement (REM) sleep behaviour disorder (RBD) that progresses into isolated RBD, and eventually into RBD with overt α-synucleinopathy. FDS, flexor digitorum superficialis; RBEs, REM sleep behavioural events.

Pathophysiologische Konzepte

Pathophysiologisches Konzept der NREM-Parasomnie



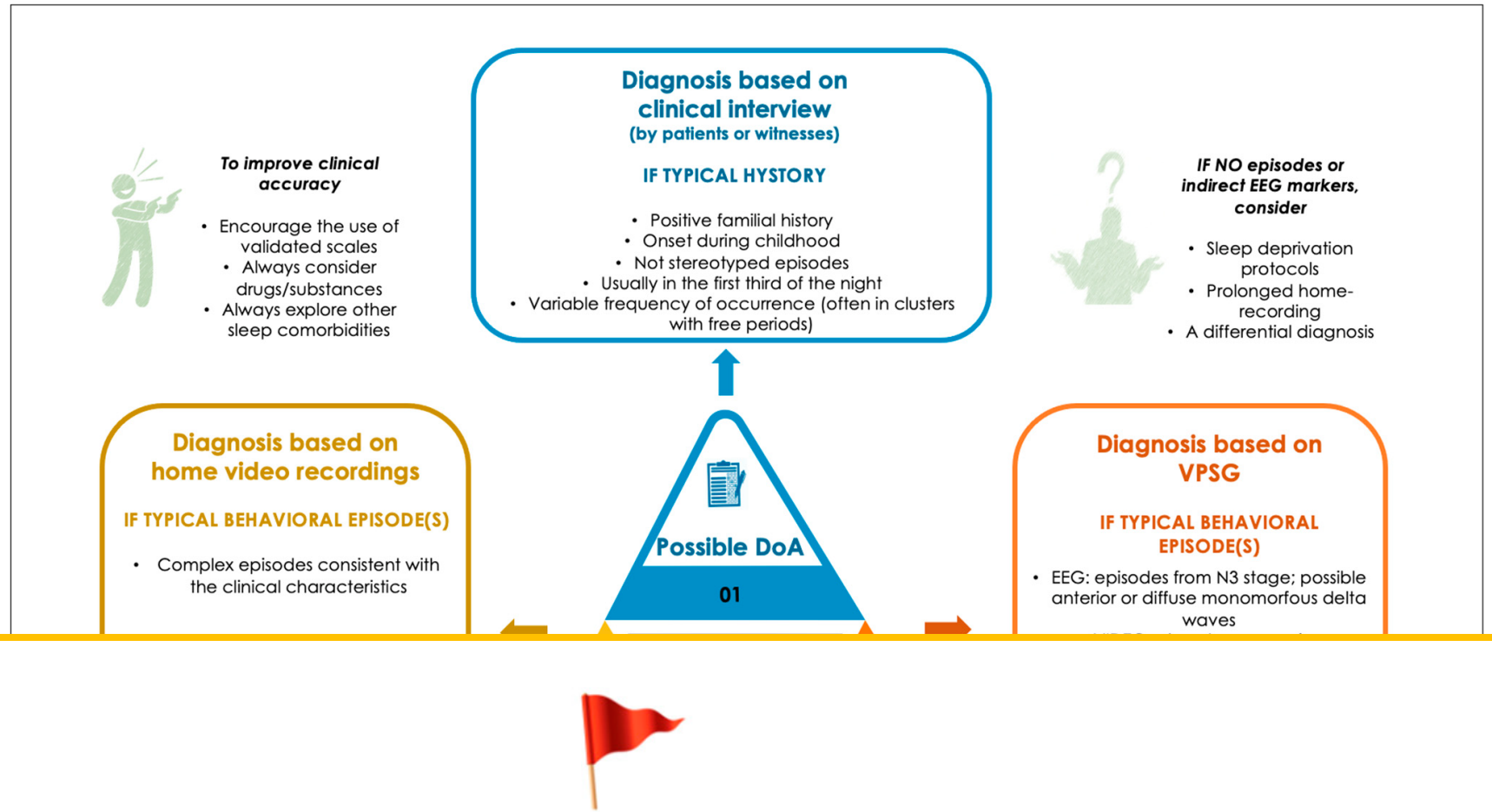
Nature Reviews Castelnovo, 2018



The coexistence of non-rapid eye movement sleep-like patterns in the frontoparietal associative cortices and hippocampus (blue shading) and wake-like patterns in the motor cortex and limbic structures (red shading) might underlie behavioural and cognitive dissociation during parasomnia episodes.

Nature Reviews Neurology Castelnovo, et al. 2018

Diagnose-Differenzierung



IF RED FLAGS AT THE CLINICAL HISTORY OR AT HOME-VIDEOS, VPSG IS ALWAYS RECOMMENDED !

Adult onset; presence of stereotypy; presence of hyperkinetic movements or dystonic postures; no ambulation or complex patterns; high episode frequency; episodes of brief duration; no familial history; episodes in any or predominantly second part of the night; choking sensation; other suspected sleep disorders; presence of dissociative symptoms during daytime/other major psychiatric comorbidities.

	Schlafwandeln	Sleep related hypermotor/ sleep related epilepsy (SHE/SRE)	REM-Schlaf-Verhaltensstörung
Manifestationsalter	Kindheit	variabel	>50 Jahre
Familienanamnese	69–90 %	<40 %	selten
Auftreten in der Nacht	1. Drittel der Nacht	jederzeit	letzte Hälfte der Nacht
Schlafstadium	Non-REM Stadium 3 (Tiefschlaf)	Non-REM Stadium 2 (Leichtschlaf)	REM-Schlafstadium
Dauer der Episoden	1–30 min	wenige Sekunden bis 3 min	wenige Minuten (<10 min)
Frequenz der Episoden	0–3/Woche	häufig (oft jede Nacht)	sehr variabel
Motorik, Handlungen	einfach bis komplexe Handlungsabfolgen mit geöffneten Augen	Stereotype und für den Betrachter sinnlose Handlungen mit geöffneten Augen	kurze ausfahrende Bewegungen, oft myokloniform (sog. jerks). Passend zu Traumerleben, Augengeschlossen
Verlassen des Bettes	häufig	nicht im Anfall aber möglich in der Phase der EEG-Normalisierung	häufig
Spontanes Erwachen aus der Episode	möglich	selten	häufig
Traumerinnern	möglich	nein	häufig
Weck-Schwelle	hoch	nicht möglich im Anfall	niedrig
Autonome Aktivierung	wenig	stark häufig bevor Motorik zu beobachten	keine
PSG	häufig Mikroarousal aus Non-REM Stadium 3 (Tiefschlaf)	nur in 10 % epilepsietypische Muster	keine Muskelatonie im REM oder rezidivierende phasische Muskelaktivität im REM-Schlafstadium
Gefahr der Fremd- und/oder Eigenverletun	möglich	möglich	möglich

Modifiziert nach Möddel et al. Akt. Neurol 2014

Standard procedures for the diagnostic pathway of sleep-related epilepsies and comorbid sleep disorders: A European Academy of Neurology, European Sleep Research Society and International League against Epilepsy-Europe consensus review

Lino Nobili[1,2] | Al de Weerd[3] | Guido Rubboli[4,5] | Sándor Beniczky[6,7,8] | Christopher Derry[9,10] | Sofia Eriksson[11] | Peter Halasz[12] | Birgit Högl[13] | Joan Santamaria[14] | Ramin Khatami[15,16] | Philippe Ryvlin[17] | Jan Rémi[18] | Paolo Tinuper[19,20] | Claudio Bassetti[21] | Raffaele Manni[22] | Michalis Koutroumanidis[23] | Luca Vignatelli[20]

J Sleep Res. 2020;29:e13184

Diagnostic step (evidence base)	Statement
2.1 Role of clinical history (clinical practice guidelines; expert opinion)	1. Clinical history for assessing comorbid sleep disorders should be obtained from patients and bed partners in all subjects with SRE (even if the patient is not complaining of any sleep-related problems). 2. Most symptoms and signs suggestive of comorbid sleep disorders by ICSD current criteria can be assessed by history. 3. Daytime sleepiness should be assessed in specific situation such as in patients with driving licence. 4. Clinical history for assessing comorbid sleep disorders should start with open questions on general aspects of
2.4 Treatment for SDB, insomnia, RLS effective in patients with SRE (Class II and III studies; clinical practice guidelines; expert opinion)	1. Any condition leading to sleep disruption and sleep deprivation should be recognised and treated according to standard procedures. Considering the high frequency of the disorders we highlight the following situations: 2. SDB (Class II, III; expert opinion). a. To treat comorbid SDB in SRE is likely to be beneficial for seizures control. b. Treating SDB should be considered independently of its severity in non-seizure-free patients. Benefit of SDB treatment on seizure reduction and sleepiness must be controlled by follow-up. c. Antiepileptic drugs (causing sedation, muscle relaxation and/or weight gain) and VNS may worsen (or induce) SDB. 3. Insomnia (clinical practice guidelines; expert opinion) a. CBT and chronobiologically based therapy are considered as first-line choice for treatment of insomnia in subjects with SRE. b. Caution should be adopted in using sleep restriction procedure as it may induce sleep deprivation that may provoke seizures in patients with epilepsy. c. Short-term treatment of chronic insomnia may include pharmacological treatment avoiding drugs lowering seizure threshold. 4. RLS (clinical practice guidelines; expert opinion). a. RLS should be treated, if they are associated with sleep deprivation or fragmentation according to current standard recommendations.

Therapie- Parasomien

Therapie- besser Management

- Nicht-pharmakologische Interventionen
  - Aufklärung
  - Bettsicherung/Schlafumgebungssicherung
  - Verhaltenstherapeutische Interventionen (Entspannung, Stimuluskontrolle, Antizipation, Hypnose)
- Management von *priming* (auslösenden) und *precipitating* Faktoren
  - Regelmäßige und ausreichende Bettliegezeiten
  - Behandlung ko-morbider Schlaferkrankungen
  - Vorsicht mit Medikamenten/Substanzen die Parasomnie triggern könnten
- Pharmakologische Interventionen
  - Keine RCT
  - Clonazepam
  - Melatonin? Melatonin-Rezeptor-Agonisten?

Cordani R et al. Sleep Med Clin 19 (2024) 43–54

Insomnie

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REVIEW ARTICLE

Journal of Sleep Research

The European Insomnia Guideline: An update on the diagnosis and treatment of insomnia 2023

Dieter Riemann[1,2] | Colin A. Espie[3] | Ellemarije Altena[4] | Erna Sif Arnardottir[5,6] | Chiara Baglioni[7] | Claudio L. A. Bassetti[8] | Celyne Bastien[9] | Natalija Berzina[10] | Bjørn Bjorvatn[11] | Dimitris Dikeos[12] | Leja Dolenc Groselj[13] | Jason G. Ellis[14] | Diego Garcia-Borreguero[15] | Pierre A. Geoffroy[16] | Michaela Gjerstad[17] | Marta Gonçalves[18] | Elisabeth Hertenstein[19] | Kerstin Hoedlmoser[20] | Tuuliki Hion[21] | Brigitte Holzinger[22] | Karolina Janku[23] | Markus Jansson-Fröjmark[24,25] | Heli Järnefelt[26] | Susanna Jernelöv[24,25] | Poul Jørgen Jennum[27] | Samson Khachatryan[28] | Lukas Krone[3,8,19] | Simon D. Kyle[3] | Jaap Lancee[29] | Damien Leger[30] | Adrian Lupusor[31] | Daniel Ruivo Marques[32,33] | Christoph Nissen[34] | Laura Palagini[35] | Tiina Paunio[36] | Lampros Perogamvros[34] | Dirk Pevernagie[37] | Manuel Schabus[20] | Tamar Shochat[38] | Andras Szentkiralyi[39] | Eus Van Someren[40,41] | Annemieke van Straten[42] | Adam Wichniak[43] | Johan Verbraecken[44] | Kai Spiegelhalder[1]

Akute Insomnie

- Definition über die Zeit < 4 Wochen Dauer
- Keine klaren Behandlungsempfehlungen für diesen Zeitraum
- Hit early and hard?
- Leave it alone?- no need to treat?

- **Pragmatisches Vorgehen:**
  - vorübergehende Behandlung sinnvoll
  - edukative Maßnahmen sinnvoll
  - schlafhygienische Maßnahmen sinnvoll
  - Hinweis auf vorübergehenden Charakter der Behandlung

Chronische Insomnie

A. The patient reports, or the patient's parent or caregiver observes, one or more of the following:

1. Difficulty initiating sleep
2. Difficulty maintaining sleep
3. Waking up earlier than desired
4. Resistance to going to bed on appropriate schedule
5. Difficulty sleeping without parent or caregiver intervention

B. The patient reports, or the patient's parent or caregiver obs one or more of the following related to the nighttime sleep difficulty:

1. Fatigue/malaise
2. Attention, concentration or memory impairment
3. Impaired social, family, occupational or academic performance
4. Mood disturbance/irritability
5. Daytime sleepiness
6. Behavioural problems (e.g. hyperactivity, impulsivity, aggressior
7. Reduced motivation/energy/initiative
8. Proneness for errors/accidents
9. Concerns about or dissatisfaction with sleep

C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e. enough time is allotted for sleep) or inadequate circumstances (i.e. the environment is safe, dark, quiet and comfortable) for sleep

D. The sleep disturbance and associated daytime symptoms occur at least three times per week

3

E. The sleep disturbance and associated daytime symptoms have been present for at least 3 months

F. The sleep/wake difficulty is not better explained by another sleep disorder

ICSD-3 (AASM, 2014)

Komorbide Insomnie

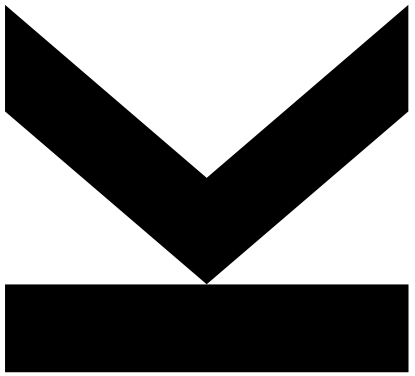
Insomnie als ko-morbide Erkrankung

Mental	Medical	Neurological	Substance use/dependence
Depressive disorders	Cardiovascular disorders	Neurodegenerative diseases	Alcohol
Bipolar disorders	Diabetes mellitus	Cerebrovascular diseases	Nicotine
Anxiety disorders	Chronic kidney diseases	Traumatic brain injury	Caffeine
Borderline personality disorder	Chronic obstructive pulmonary diseases	Multiple sclerosis	Tetrahydrocannabinol /marihuana
Posttraumatic stress disorder	Rheumatic disorders	RLS/PLMD	Opioids
Schizophrenia	Chronic pain	Fatal familial insomnia	"Designer" drugs
Substance use disorders	Any kind of malignant disorder		Cocaine
	SRBD/OSA		Amphetamines

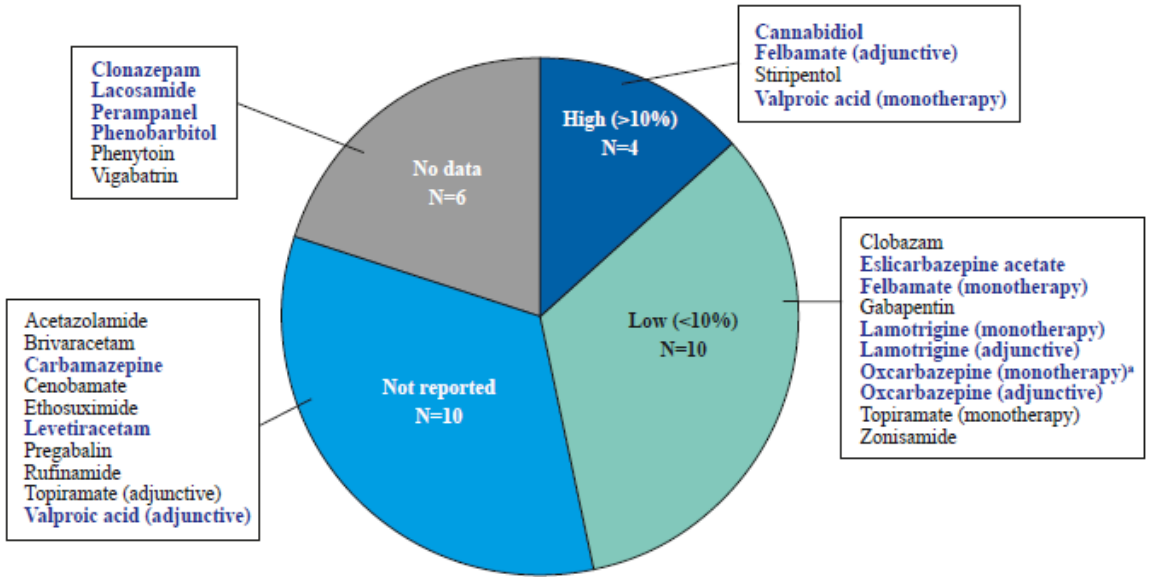
Bidirektionale Zusammenhänge v.a. für die Neurologie
Insomnie als unabhängiger Risikofaktor für Schlaganfall und Demenz

Riemann et al. 2023, Damsgard et al., 2022; Zheng et al., 2019

Ko-Medikation



Insomnie unter Anfalls- supprimierende Medikation



- **Perampanel:** geringe Inzidenz von Insomnie
- **Lacosamid:** geringe Inzidenz von Tagesschläfrigkeit
- **Clonazepam, Felbamat, Lamotrigin, Oxcarbazepin, Phenobarbital:** verschlechtern den Schlaf oder hatten keine Auswirkungen auf den Schlaf.
- **Lamotrigin:** Risiko für Insomnie
- **Phenobarbital:** Risiko für erhöhte Tagesschläfrigkeit.
- **Valproinsäure:** Datenlage uneinheitlich.
- **Cannabidiol, Carbamazepin,Levetiracetam:** keine Auswirkungen auf den Schlaf

Liguori C et al.,Sleep Medicine Reviews 60 (2021) 101559

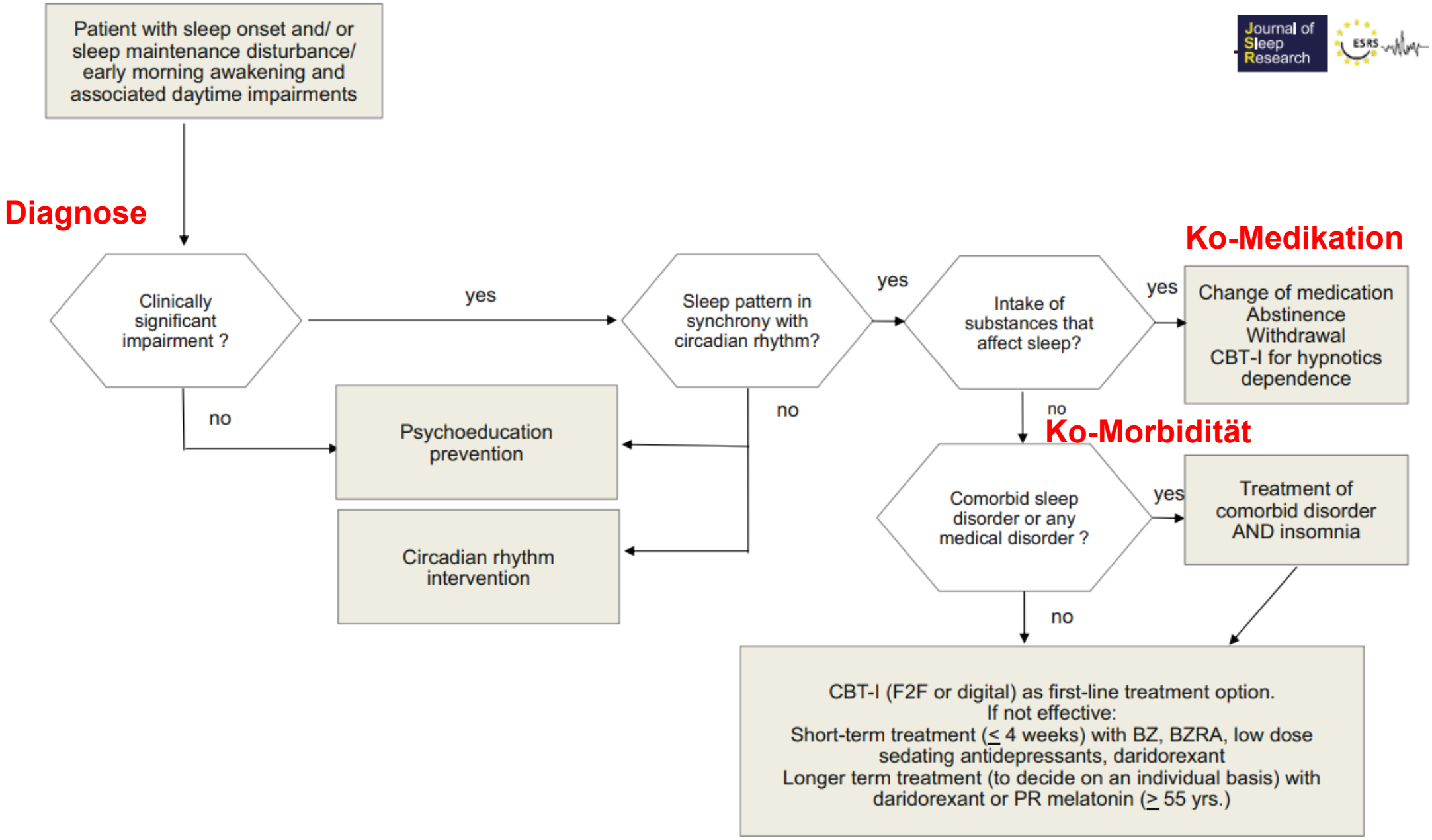
Antidepressiva

Antidepressant Class	Drugs	REM Sleep	ROL	SWS	Sleep Continuity
TCA (sedative)	amitriptyline, doxepin	↓	↑	↑	↑
	trimipramine	0/↑	0/↓	↑	↑
TCA (activating)	desipramine, imipramine, nortriptyline, protriptyline	↓	↑	↓	↓
	clomipramine	↓↓↓	↑↑↑	↓	↓
TeCA	amoxapine	↓	↑	0/↑	0
	maprotiline	0/↓	0	↑	0
	mianserin	↓	↑	↑	↑
	mirtazapine	0	0/↑	↑	↑
MAOI (reversible)	moclobemide	↓	↑	0/↑	↑
MAOI (irreversible)	phenelzine, tranylcypromine	↓↓↓	↑↑↑	0	0/↓
SSRI	citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline	↓	↑	0	↓
	duloxetine, venlafaxine	↓	↑	↑	↓
SNRI	milnacipran	0	0/↑	0	0/↓
DNRI	bupropion	0	0	0/↑	0/↓
SARI	trazodone	0/↓	0/↑	↑	↑
	nefazodone	0/↑	0	↑	↑
DARI	amineptine	↑	0/↓	↓	↓
NARI	atomoxetine, reboxetine, viloxazine	↓	↑	0/↓	0/↓
Novel drugs	agomelatine	0	0	↑	↑
	ketamine	↓	↑	↑	↑
	tianeptine	0	0	0	0
	vilazodone	↓	↑	↑	↓
	vortioxetine	↓	↑	0	0/↓

Modified according to Wichniak et al. [10]; REM—rapid eye movement, ROL—rapid eye movement onset latency, SWS—slow-wave sleep, TCA—tricyclic antidepressants, TeCA—tetracyclic antidepressants, MAOI—monoamine oxidase inhibitors, SSRI—selective serotonin reuptake inhibitors, SNRI—serotonin norepinephrine reuptake inhibitors, DNRI—dopamine noradrenaline reuptake inhibitors, SARI—serotonin antagonist and reuptake inhibitors, DARI—dopamine reuptake inhibitor, NARI—noradrenaline reuptake inhibitors, ↑—increase, ↓—decrease, 0—no or minimal effect.

Ihutka P et al. nt. J. Mol. Sci. **2021**, 22, 1333. https://doi.org/10.3390/ijms22031333

Behandlungspfad


Patient with sleep onset and/ or sleep maintenance disturbance/ early morning awakening and associated daytime impairments
Diagnose
Clinically significant impairment ?
yes
Sleep pattern in synchrony with circadian rhythm?
yes
Intake of substances that affect sleep?
yes
Ko-Medikation
Change of medication
Abstinence
Withdrawal
CBT-I for hypnotics dependence
no
Psychoeducation prevention
Circadian rhythm intervention
no
Ko-Morbidität
Comorbid sleep disorder or any medical disorder ?
yes
Treatment of comorbid disorder AND insomnia
no
CBT-I (F2F or digital) as first-line treatment option.
If not effective:
Short-term treatment (≤ 4 weeks) with BZ, BZRA, low dose sedating antidepressants, daridorexant
Longer term treatment (to decide on an individual basis) with daridorexant or PR melatonin (≥ 55 yrs.)

Nicht medikamentöse Therapie

Components of Cognitive Behavioral Therapy for Insomnia.

Table 2. Components of Cognitive Behavioral Therapy for Insomnia.

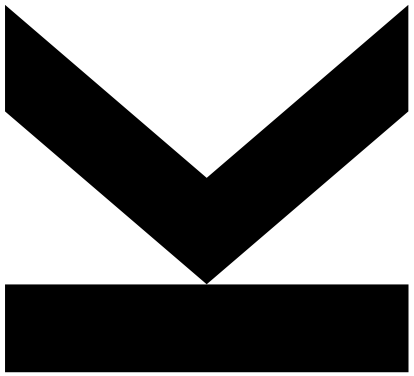
Component	Intended Effect	Specific Directions for Patients
Sleep restriction	Increase sleep drive and stabilize circadian rhythm	Reduce time in bed to perceived total sleep time (not less than 5–6 hours), choose specific hours on the basis of personal preference and circadian timing, increase time in bed gradually as sleep efficiency improves
Stimulus control	Reduce arousal in sleep environment and promote the association of bed and sleep	Attempt to sleep when sleepy, get out of bed when awake and anxious at night, use the bed only for sleep or sexual activity (e.g., no watching TV in bed)
Cognitive therapy	Restructure maladaptive beliefs regarding daytime and health consequences of insomnia	Maintain reasonable expectations about sleep; review previous insomnia experiences, challenging perceived catastrophic consequences
Relaxation therapy	Reduce physical and psychological arousal in sleep environment	Practice progressive muscle relaxation, breathing exercises, or meditation
Sleep hygiene	Reduce behaviors that interfere with sleep drive or increase arousal	Limit caffeine and alcohol, keep bedroom dark and quiet, avoid daytime or evening napping, increase exercise (not close to bedtime), remove bedroom clock from sight

Winkelman JW. N Engl J Med 2015;373:1437-1444

F2F- digital

Language (country of origin)	Name of product	Web address	Computer/app based	Reimbursement by national healthcare	Reference (s)
Dutch The Netherlands	i-Cycle	https://slaapregister.nl	Yes/Yes (partly) + assistance[a]	No	Leerssen et al. (2022)
Dutch The Netherlands	Somnio	https://somnio.nl	Yes/Yes + assistance[a]	Yes	Dekker et al. (2020)
Dutch The Netherlands	i-Sleep	https://www.minddistrict.com https://therapieland.nl	+ assistance[a]	Yes	Baka et al. (2022); Van der Zweerde, van Straten, et al. (2019); Van der
German Austria	NUKKUAA	www.nukkuaa.com	No/Yes	No	Schabus et al. (2022); Eigl et al. (2022); Hinterberger et al. (2022, 2023)
German Germany	SOMNIO	https://somn.io/	Yes/Yes	Yes	Lorenz et al. (2019)
German Germany	HelloBetter Schlafen/ HelloBetter sleep	https://hellobetter.de/online-kurse/schlafen/	+ assistance[a]	Yes	Behrendt et al. (2020); Ebert et al. (2015); Thiart et al. (2015)
German Switzerland	Meinstresscoach/ SweetDreams	https://www.meinstresscoach.ch/kurse	No/Yes + assistance[a]	No	Hürlimann et al. (2023)
Swedish Sweden	Internetpsykiatri Sömnproblem—insomni	https://www.internetpsykiatri.se/behandling/somnproblem-insomni/	Yes/No + assistance	Yes	Kaldo et al. (2015); Blom et al. (2016)
Swedish Sweden	Livanda Sömnproblem (insomnia)	https://www.livanda.se/kbt-internetterapi-somnproblem.aspx	Yes/No + assistance[a]	No	Ström et al. (2004)

Medikamentöse Therapie



Medikamentöse Therapie

Studien 6-12 Monate aber CAVE

TABLE 11 Major drug classes used to treat insomnia in Europe

BZ	Diazepam, flunitrazepam, flurazepam, lormetazepam, nitrazepam, oxazepam, temazepam, triazolam	Für maximal 4 Wochen, CAVE Nebenwirkungen
BZRA	Zaleplone, zolpidem, zopiclone, eszopiclone	Für maximal 4 Wochen, CAVE Nebenwirkungen
Sedating antidepressants	Agomelatine, amitriptyline, doxepin, mianserin, mirtazapine, trazodone, trimipramine	Off- label, keine Zulassung für die Behandlung der Insomnie, nur für die Behandlung der Komorbidität
Antipsychotics	Chlorprothixene, levomepromazine, melperone, olanzapine, pipamperone, prothipendyl, quetiapine	
Antihistamines	Diphenhydramine, doxylamine, hydroxyzine, promethazine	Fehlende Evidenz, Effekt auf Mortalität
Phytotherapeutics	Hops, kava-kava, melissa, passiflora, valerian, lavender	Evidenz?, unzureichende Studienlage
Melatonin receptor agonists	Fast-release melatonin, ramelteon, PR melatonin	Retardiertes Melatonin bei Menschen > 55 J.
Orexin receptor antagonists	Daridorexant	Einziges zur Zeit für längere Behandlungsdauer empfohlenes Medikament (3 Monate)

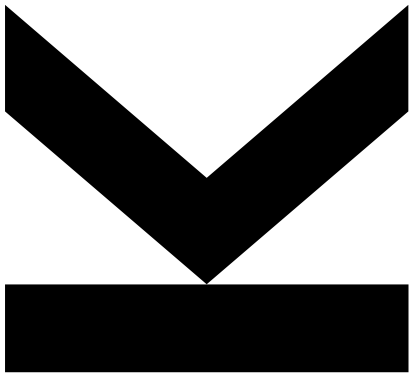
Abbreviations: BZ, benzodiazepines; BZRA, benzodiazepine receptor agonists; PR, prolonged-release.

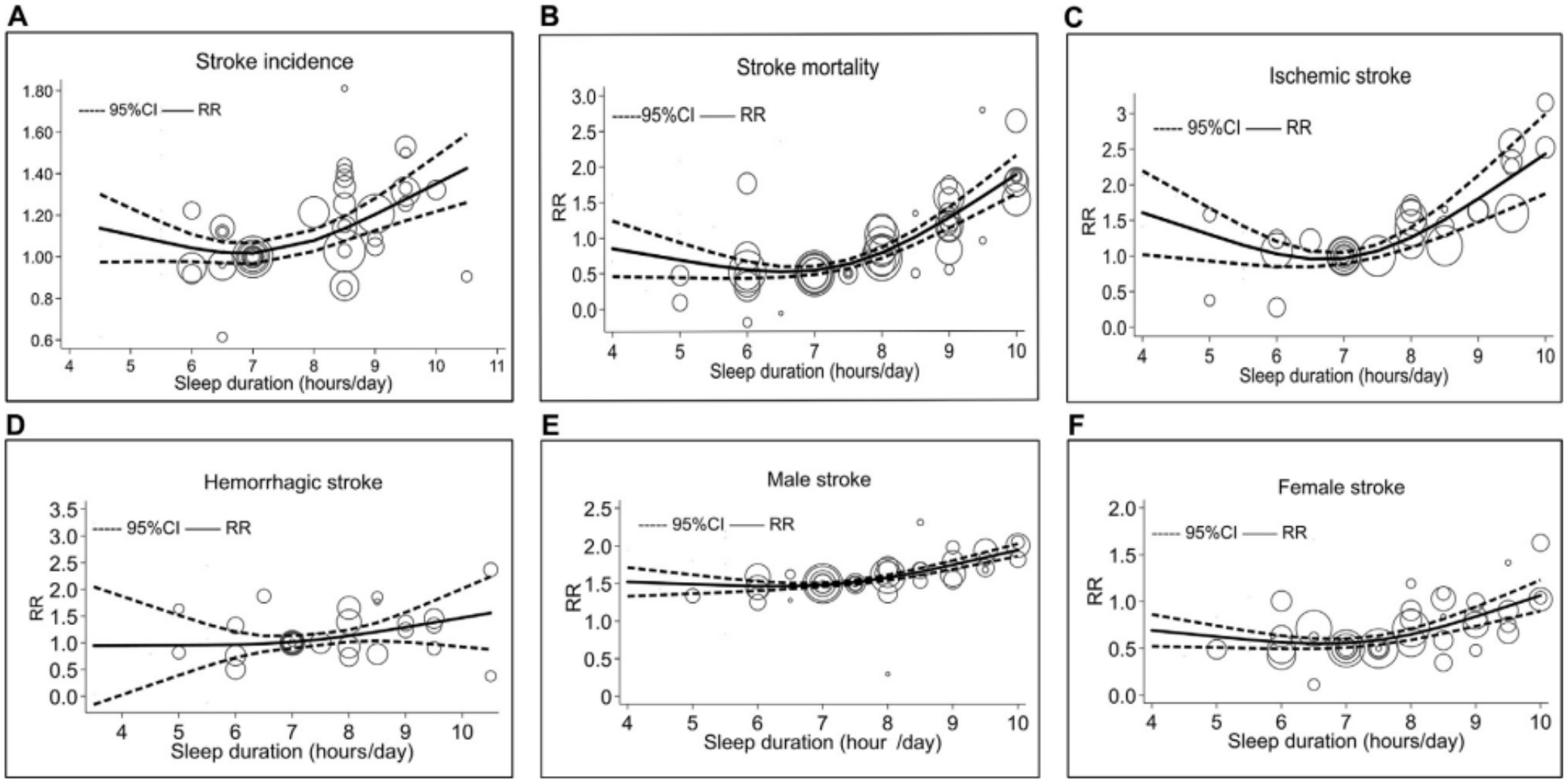
Riemann et al. 2023

Schlaf-Stroke

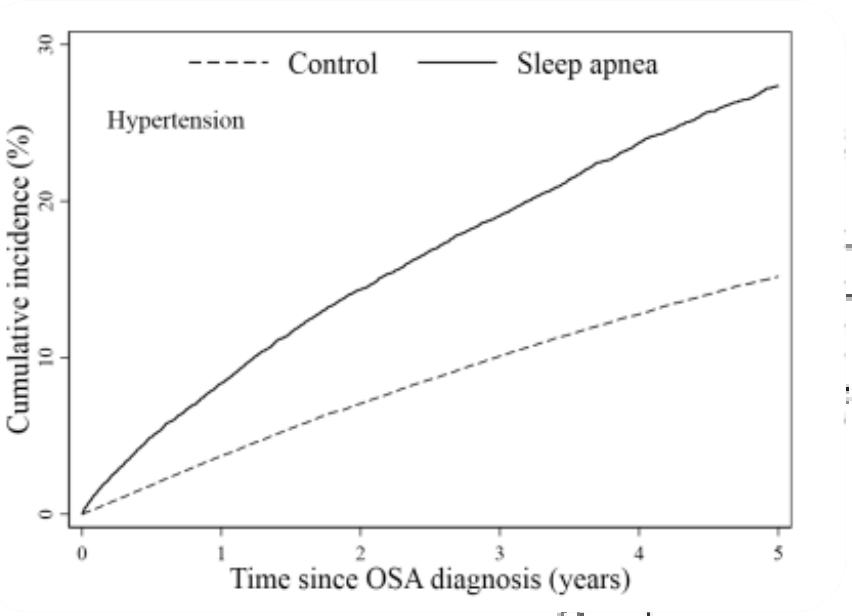
Joynt Maddox KE, Circulation. 2024;149:e00–e00. DOI: 10.1161/CIR.0000000000001256

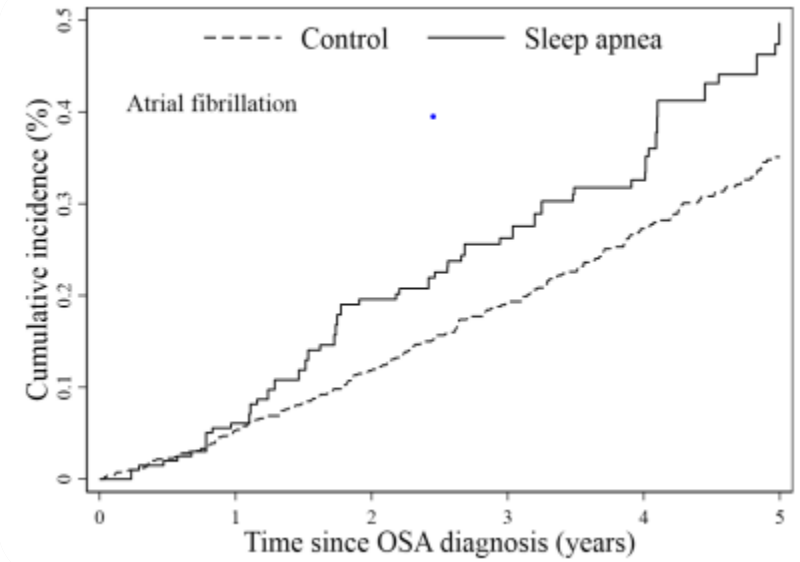
Schlafdauer

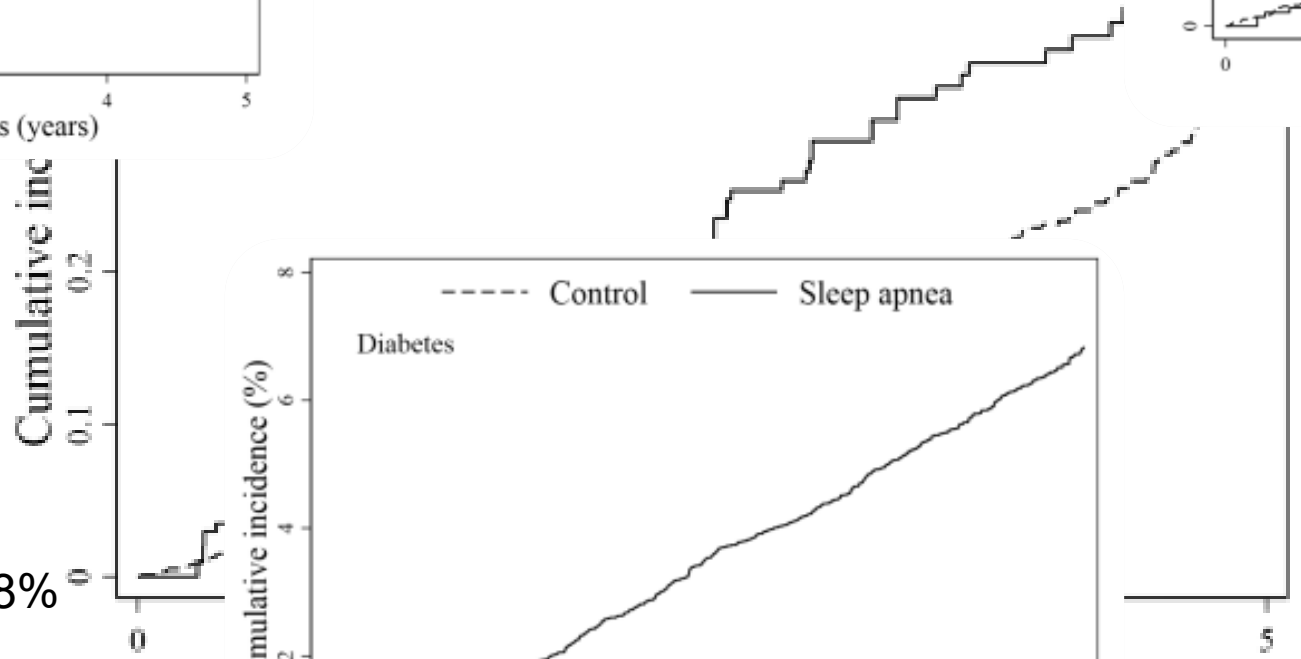



A
Stroke incidence
----- 95%CI —— RR
Sleep duration (hours/day)
B
Stroke mortality
-----95%CI —— RR
RR
Sleep duration (hours/day)
C
Ischemic stroke
----- 95%CI —— RR
RR
Sleep duration (hours/day)
D
Hemorrhagic stroke
----- 95%CI —— RR
RR
Sleep duration (hours/day)
E
Male stroke
----- 95%CI —— RR
RR
Sleep duration (hour /day)
F
Female stroke
----- 95%CI —— RR
RR
Sleep duration (hours/day)

Schlafatmungsstörung







After 5-year follow-up, 31.8% of the patients with OSA developed any cardiovascular event compared with 16.5%

20240 patients aged ≤50 years with OSA (19.6% female; mean±SD age 39.9±7.7years) and 80314 controls

Albertsen IE. J Am Heart Assoc. 2024;13:e033506.

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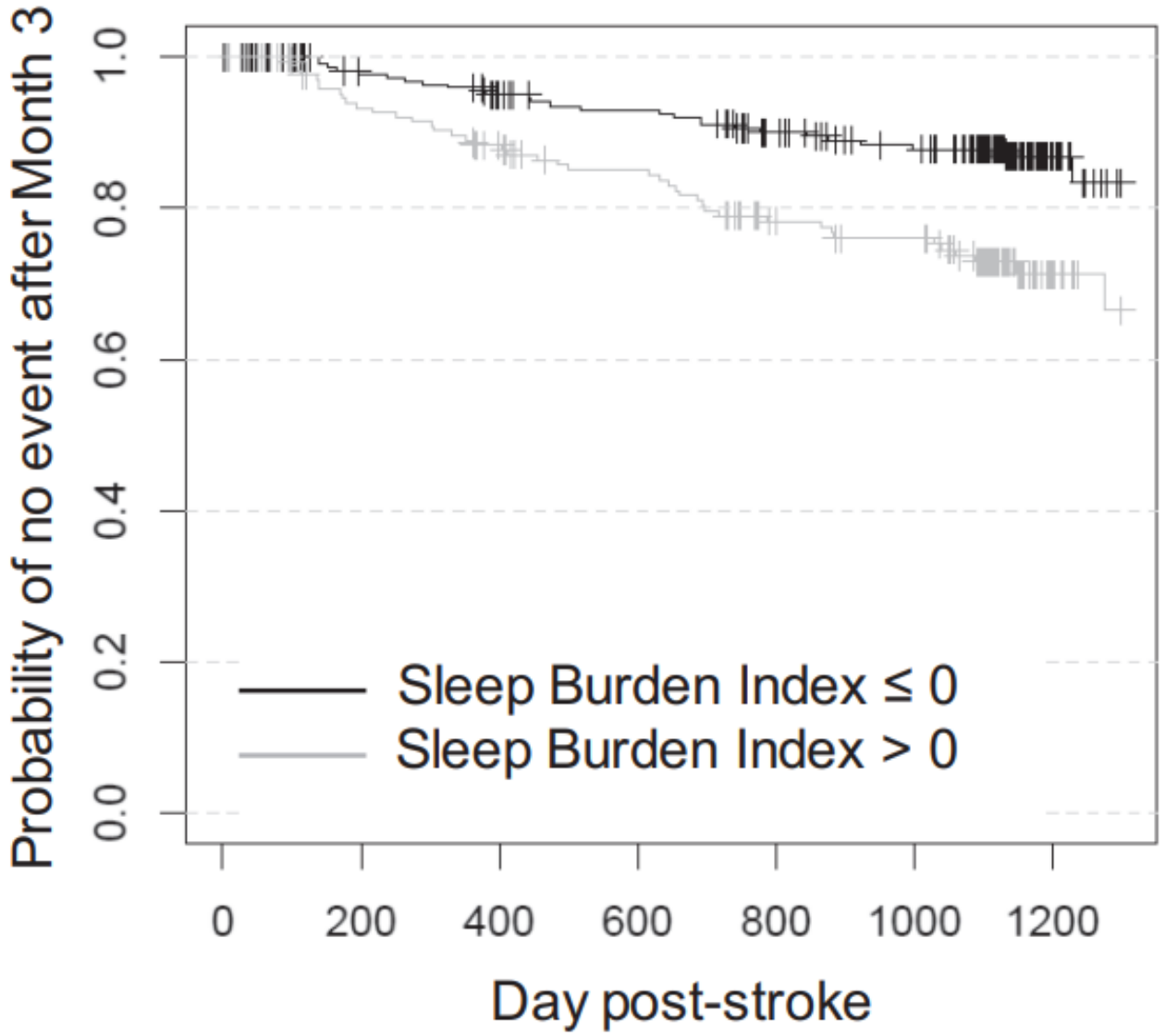


REI, ISI, sleep duration, IRLS

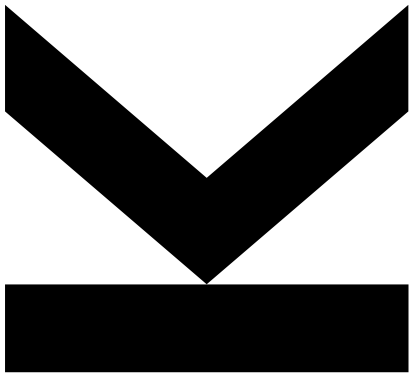
Duss SB. Eur J Neurol. 2023;30:1696–1705.

Subjective and objective features of sleep disorders in patients with acute ischemic or haemorrhagic stroke: It is not only sleep apnoea which is important

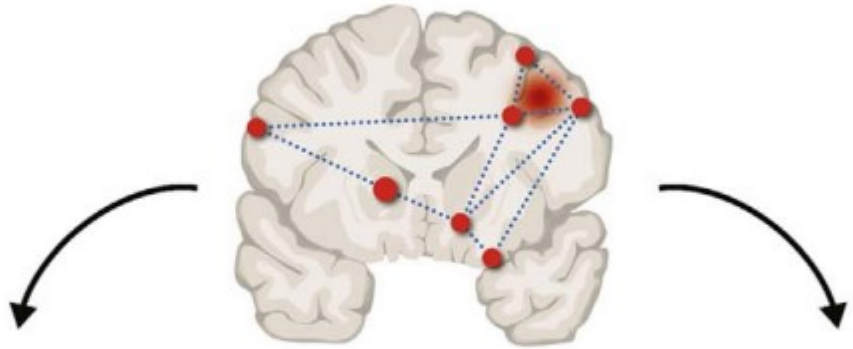
Evelina Pajediene[a,*], Adomas Pajeda[b], Gintare Urnieziute[a], Erlandas Paulekas[a], Vanda Liesiene[c], Indre Bileviciute-Ljungar[d,e,f], Giedre Jurkeviciene[a], Daiva Rastenyte[a], Kestutis Petrikonis[a]

Conclusions: Half of our acute stroke patients had at least one or more new or exacerbated sleep complaints and/or symptoms, mainly related to OSA or insomnia. In the selected PSG group almost all patients were diagnosed with a sleep disorder, half of them having non-breathing sleep disorder, such as PLMD, RBD and insomnia.

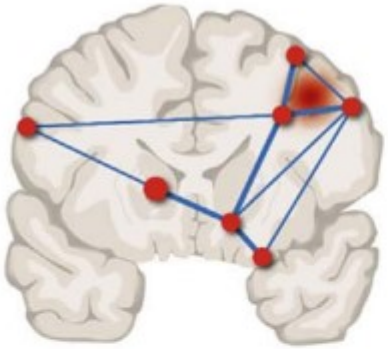
Schlaganfall→ Schlaf?



Sleep and sleep disturbances in stroke patients

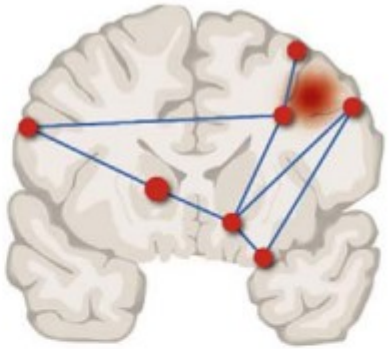


Prognosis for good recovery



- No sleep disorders or efficient treatment
- Consolidated sleep
- Few and transient post-stroke changes in sleep's macro- & microstructure
- Intact homeostatic regulation of SWA

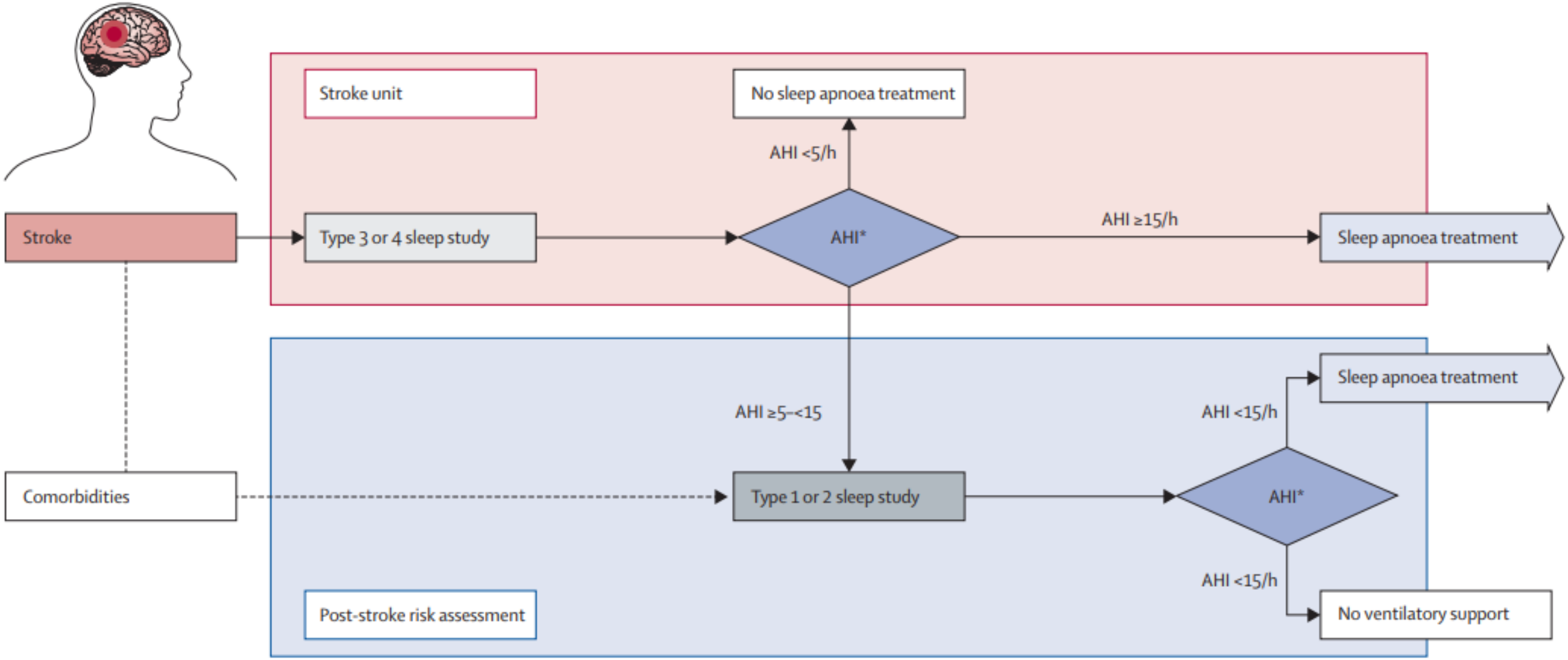
Prognosis for bad recovery



- Presence of sleep disorders or inefficient treatment
- Sleep disruption
- Severe and persistent post-stroke changes in sleep's macro- & microstructure
- Disturbed homeostatic regulation of SWA

Duss SB. Neurobiology of Sleep and Circadian Rhythms 2 (2017) 94–105

Was bedeutet das für unser praktisches Handeln?



Bassetti CL. Lancet Neurol 2022; 21: 78–88

Problem

Post-stroke OSA:

- Common and underdiagnosed
- Impedes neurologic recovery

Diagnosing OSA:

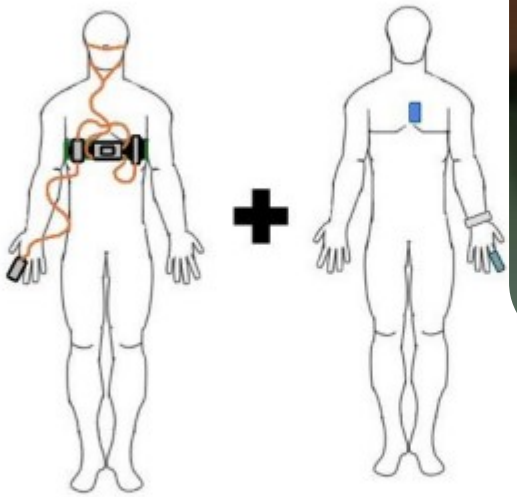
- Slow
- Cumbersome
- Long wait times

Research Question

Can we rapidly screen OSA using wearable sensors and machine learning?

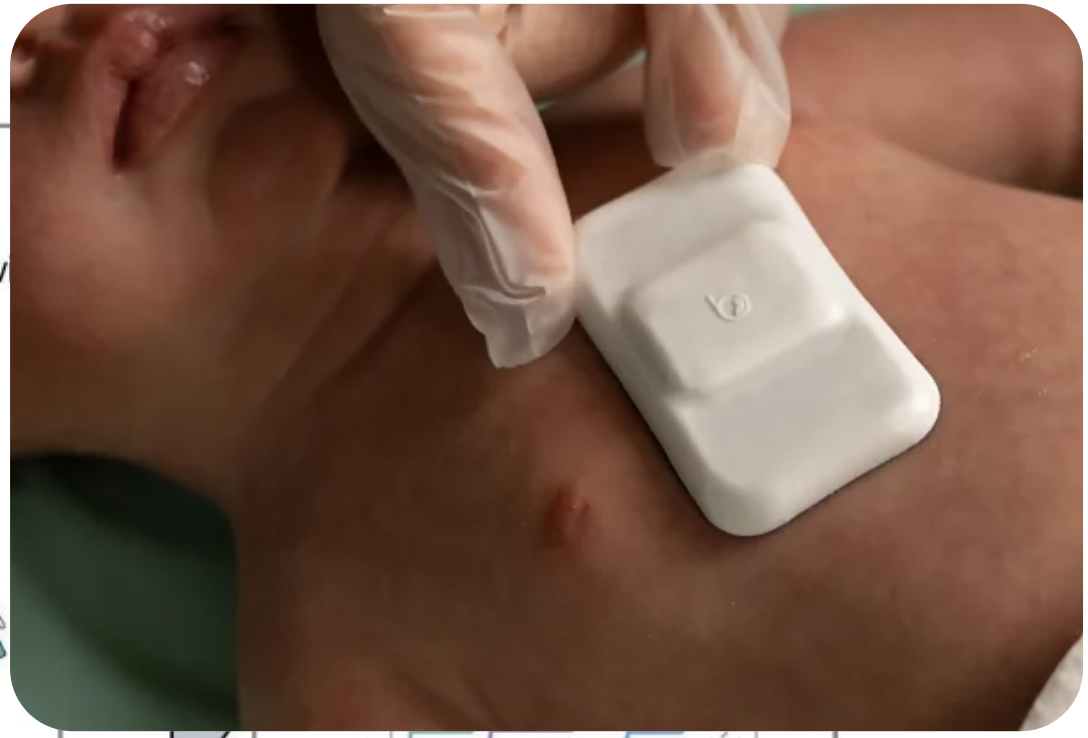
Methods

n = 76 inpatients w
post-acute stroke

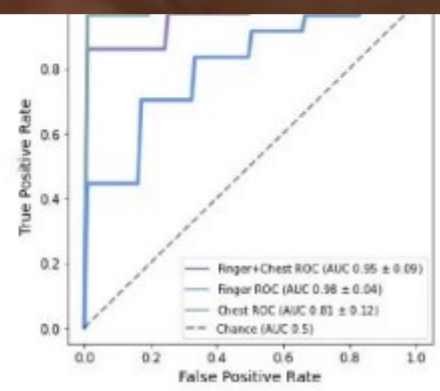


Sleep apnea diagnosis
from home sleep apnea test

Overnight biometric data
from two wireless wearable sensors (chest & finger)

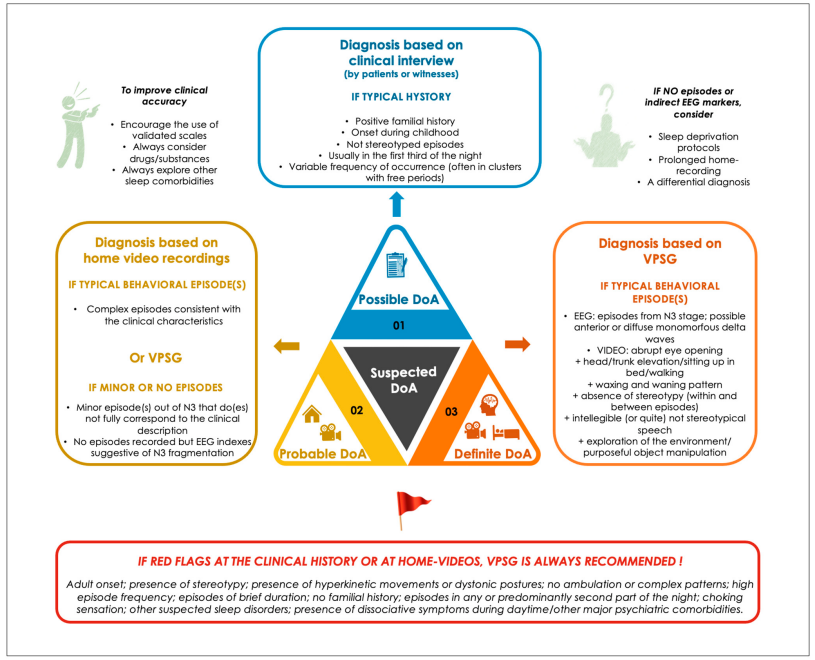


Machine Learning



*Normal vs. Moderate-Severe OSA

Sindorf J. Sleep, zsae123, https://doi.org/10.1093/sleep/zsae123 **Published:** 30 May 2024



EUROPEAN GUIDELINE:

Management of Narcolepsy in Adults & Children

Written by EAN, ESRS and EU-NN

NARCOLEPSY is an uncommon disorder of presumed autoimmune origin that usually requires lifelong treatment. Narcolepsy typically has a pleomorphic clinical presentation and produces a considerable variety of different symptoms with a variable clinical course.

TREATMENT RECOMMENDATION

The management of narcolepsy involves non-pharmacological and pharmacological approaches, with an increasing number of symptomatic treatment options for adults and children.

ADULTS

Excessive daytime sleepiness / Disturbed nocturnal sleep

- Regular sleep-wake schedule & scheduled daytime naps (strong recommendation)
- Wake promoting agents during the day: Modafinil, pitolisant & solriamfetol (strong recommendation)
- Sodium oxybate during the night (strong recommendation)
- Wake promoting during the day: Methylphenidate & amphetamine-derivates (weak recommendation)

Cataplexy

- Sodium oxybate during the night (strong recommendation)
- Venlafaxine & clomipramine during the day (strong recommendation)
- Pitolisant during the day (weak recommendation)

CHILDREN

Excessive daytime sleepiness / Disturbed nocturnal sleep

- Regular sleep-wake schedule & scheduled daytime naps (strong recommendation)
- Sodium oxybate during the night (strong recommendation)
- Wake promoting during the day: Modafinil, methylphenidate, pitolisant & amphetamine-derivates during the day (weak recommendation)

Cataplexy

- Sodium oxybate during the night (strong recommendation)
- Antidepressants during the day (weak recommendation)

Read the full guideline in the European Journal of Neurology: https://doi.org/10.1111/ene.14888

When using GRADE, panels make strong recommendation when most clinicians and patients would choose the recommended course of action. Weak recommendation indicates that clinicians and patients should consider the recommended course of action, but the final decision should be based on discussion, case-by-case risks and preferences.

Treatment choices should be tailored to each patient's symptoms, needs, comorbidities and risk of potential drug interactions.

ean european academy of neurology

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JKU JOHANNES KEPLER UNIVERSITÄT LINZ | Kepler Universitäts Klinikum

Universitätsklinik für Neurologie

Neurologie@kepleruniklikum.at
Anna.Heidbreder@kepleruniklinkum.at

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