Snoring isn’t always benign :

understanding sleep apnoea in children

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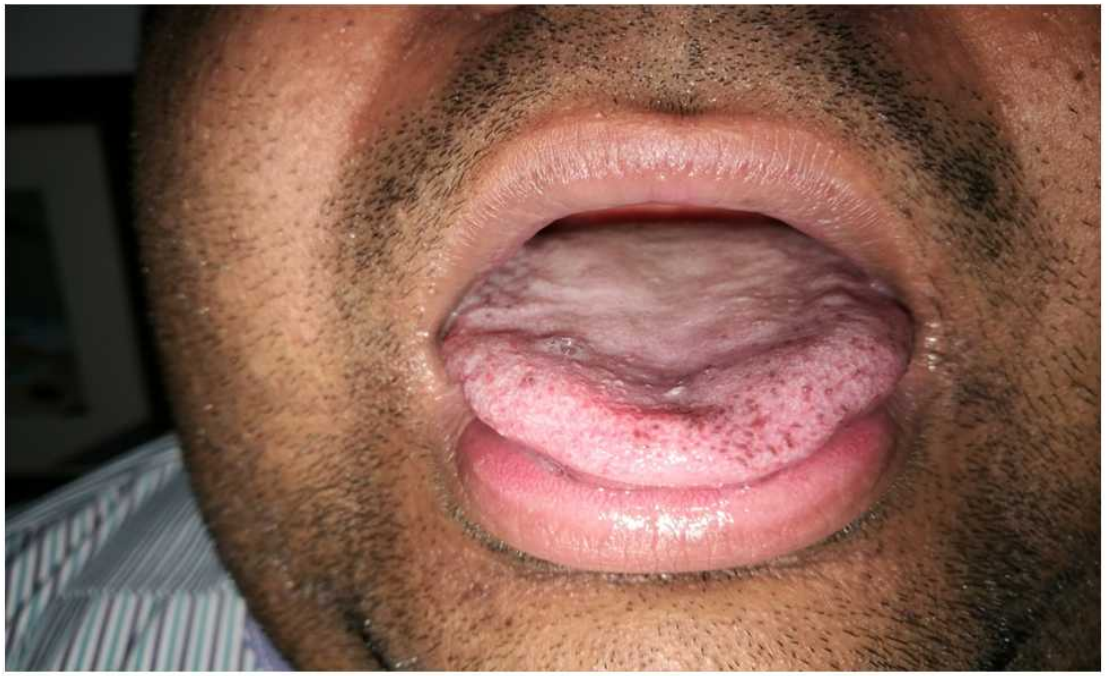
Ocean Creek Paediatric Meeting, 3 May 2026

Mallampati 4 +++



Sleep Disordered Breathing : Spectrum of disease

- **Obstructive sleep apnoea**
- Central sleep apnoea
- Nocturnal hypoventilation syndromes
- Not uncommon – often missed or neglected

Obstructive sleep Apnoea

- Simple snoring
- Upper Airway Resistance syndrome
- Full blown Obstructive sleep apnoea hypopnea syndrome (OSAHS)

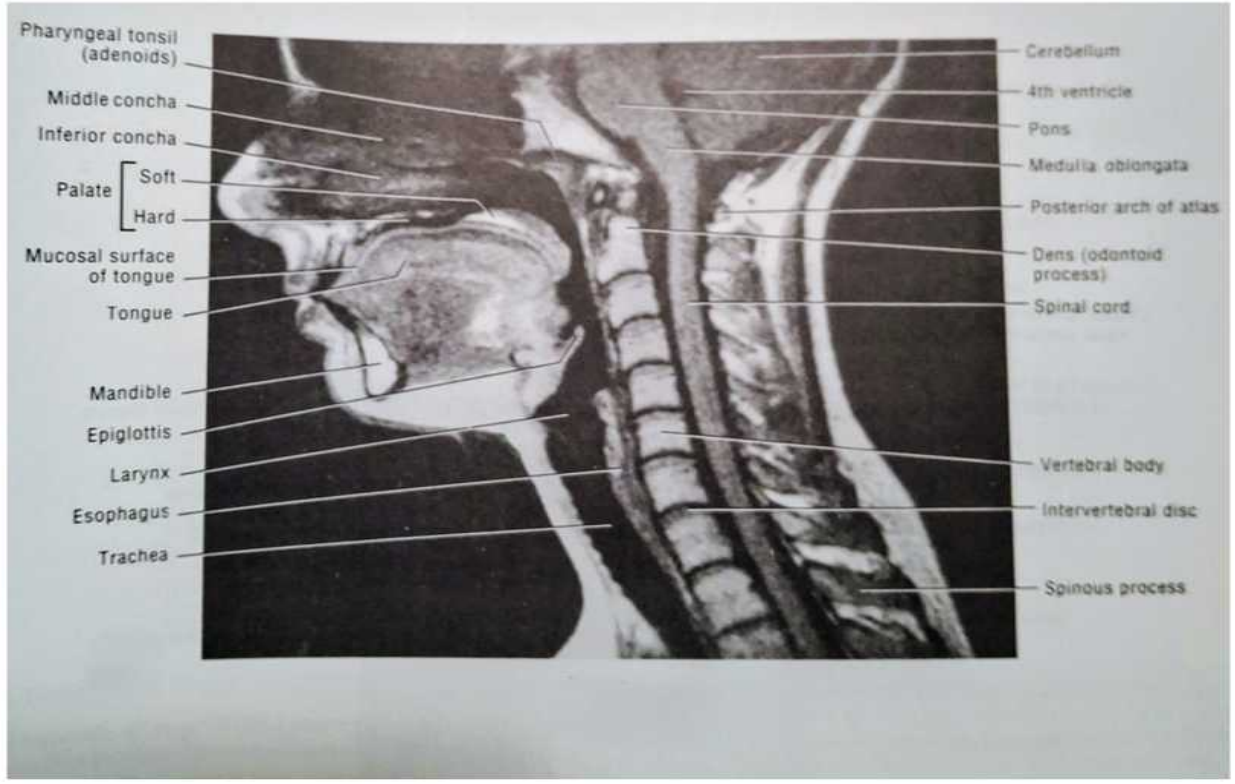
Prevalence OSAHS

- 0.7 t0 10.3 % paediatric population
- Peak from ages 5 – 10 yrs
- No data for infants and toddlers

Natural History

- Essentially unknown
- Some studies show resolution in adolescence
- Adolescent OSAHS probably persists into adulthood

Pathophysiology OSA



Abnormal Craniofacial Skeleton

- Pierre Robin Sequence
- Achondroplasia
- Crouzon’s syndrome
- Other milder midface and palatal hypoplastic syndromes

Soft tissue excess / hypertrophy

- Simple fat
- Big tonsils and adenoids
- Severe allergic rhinitis with enlarged turbinates

Neuromuscular impairment

- Impaired neuromuscular tone and reflexes of the upper airway

- From a variety of neurological insults and syndromes cerebral palsy, brain bleeds, Arnold Chiari

Pathophysiology

- Obstructed airway
- Increasing respiratory effort
- Hypoxia
- Microawakening
- Severely disturbed sleep cycle

OSA- polysomnographic study

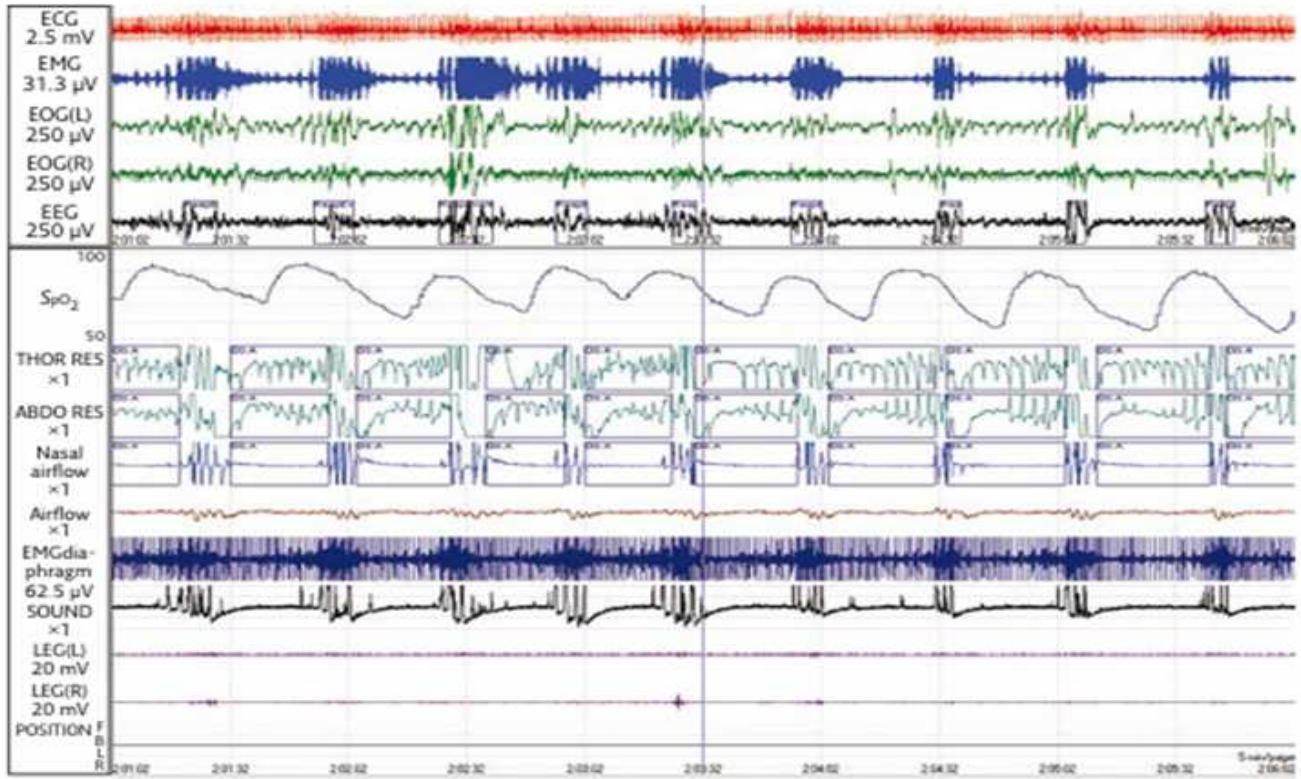


Figure 7. 5-min page of PSG showing obstructive respiratory events (ObA) accompanied by desaturations and arousals. THOR RES: thoracic respiratory band; ABDO RES: abdominal respiratory band; F: front; B: back; L: left side; R: right side. Reproduced and modified from Riha (2012) with permission.

Consequences of OSA

- Disabling symptoms
- Chronic inflammatory state (ROS, hs CRP)
- Brain development, impaired learning
- Hypertension
- LVH, stiff LV
- Pulmonary hypertension and cor pulmonale

ERS- OSA categorisation by age

- Up to 2 years

- Age 2 to 18 yrs

Childhood OSA - symptoms

Table 1. Daytime and night-time symptoms suggestive of SDB

Night-time symptoms
Snoring
Apnoeas
Nocturnal sweating
Nightmares
Talking in sleep
Bruxism
Bedwetting
Sleepwalking
Restless sleep
Frequent arousals
Oral breathing
Daytime symptoms
Sleepiness
Hyperactivity
Inattention
Headache
Learning difficulties
Oral breathing

Reproduced and modified from Simonds *et al.* (2012) with permission.

Older Children

- Symptoms similar to adults
- Snoring
- Unrefreshing sleep
- Daytime somnolence
- Poor school performance

Clinical Examination

- Obese / small for age , **Blood Pressure**
- Syndromic disorders
- Adenoid facies- long face, high arched palate. Mouth breathing
- Dental malocclusion and cross bite
- **Nose (metal spatula**
- **Throat** (Mallampati, tonsils grade)

Causes of OSA first 2 yrs of life

- Choanal atresia, nasal pyriform aperture stenosis
- Laryngomalacia, subglottic stenosis, vocal cord paralysis
- Isolated large adenoids
- Congenital craniofacial abnormalities
- Neuromuscular and syndromic disorders

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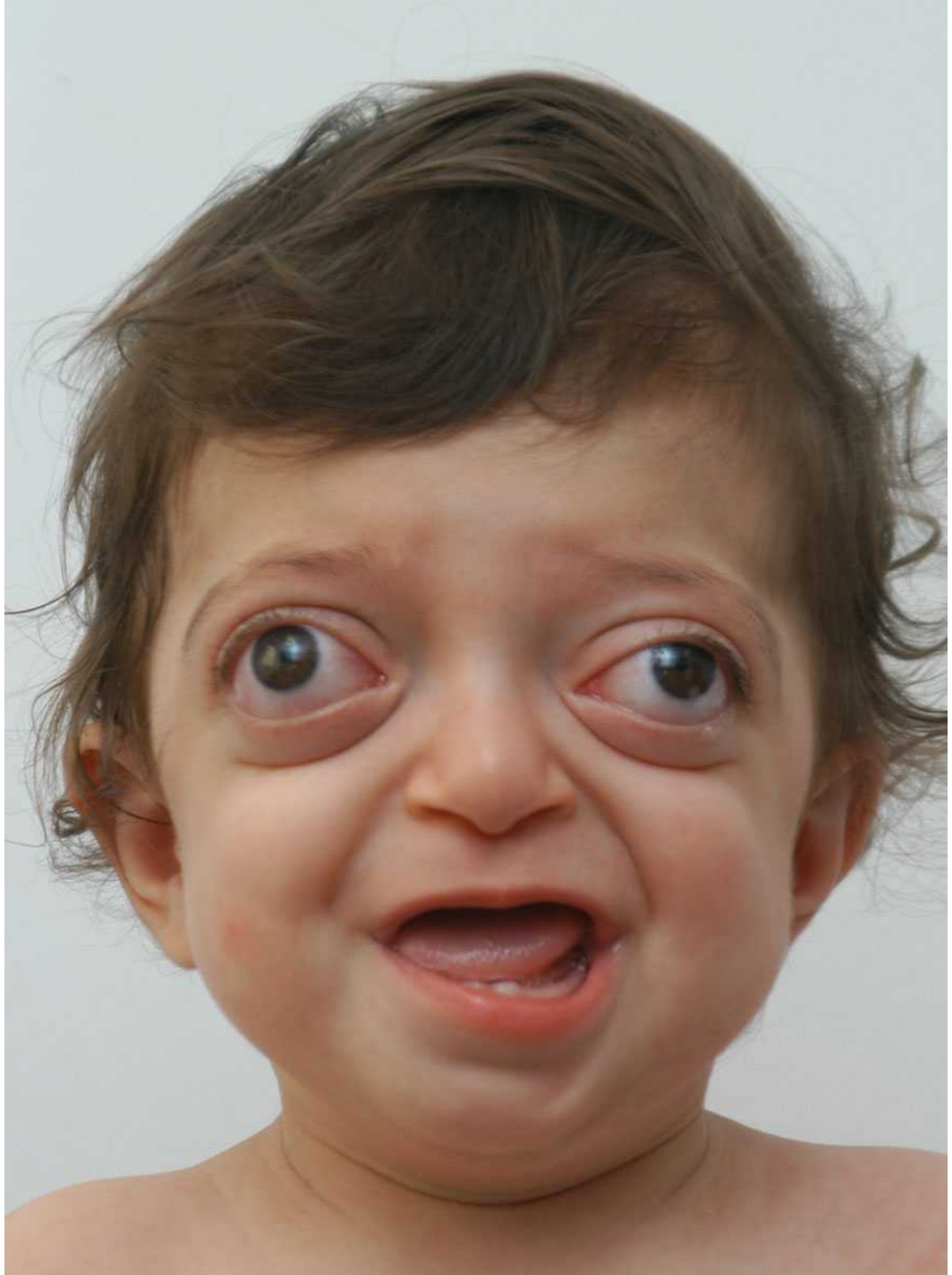
Pierre Robin sequence

Crouzon syndrome



Older Children > 2 years

Adenoid Facies – long face appearance

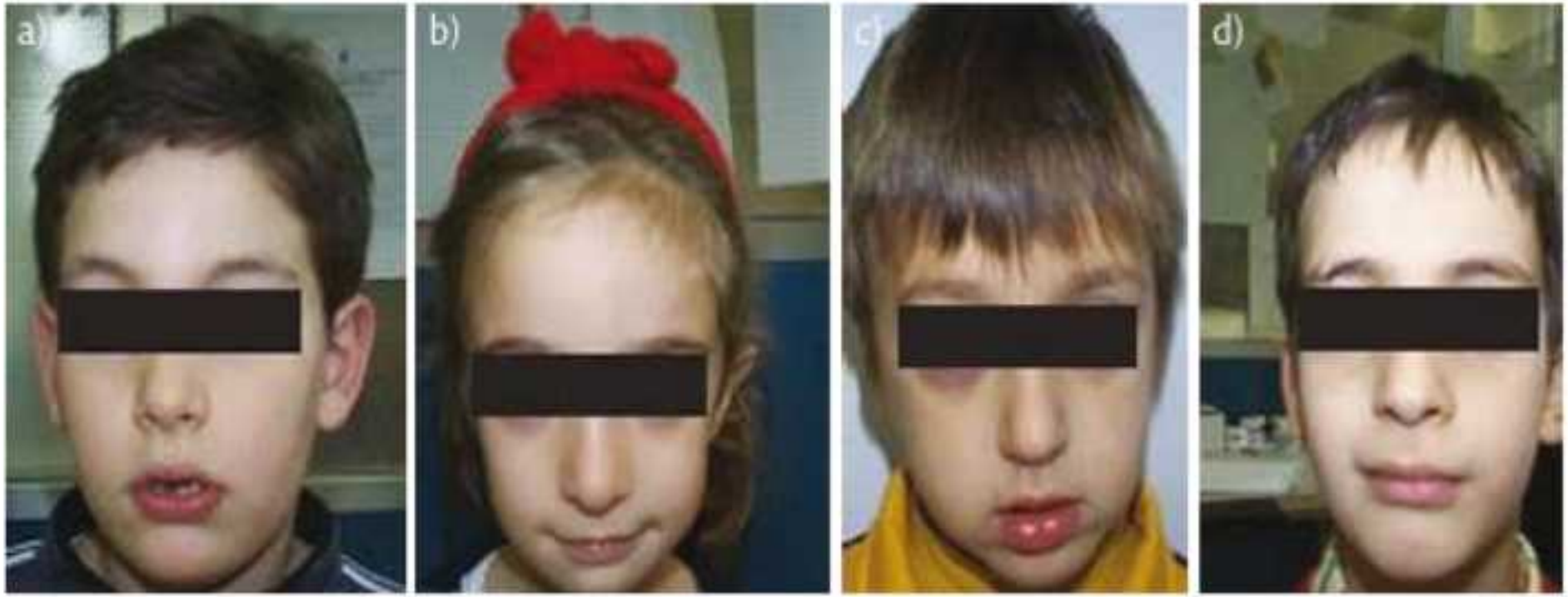
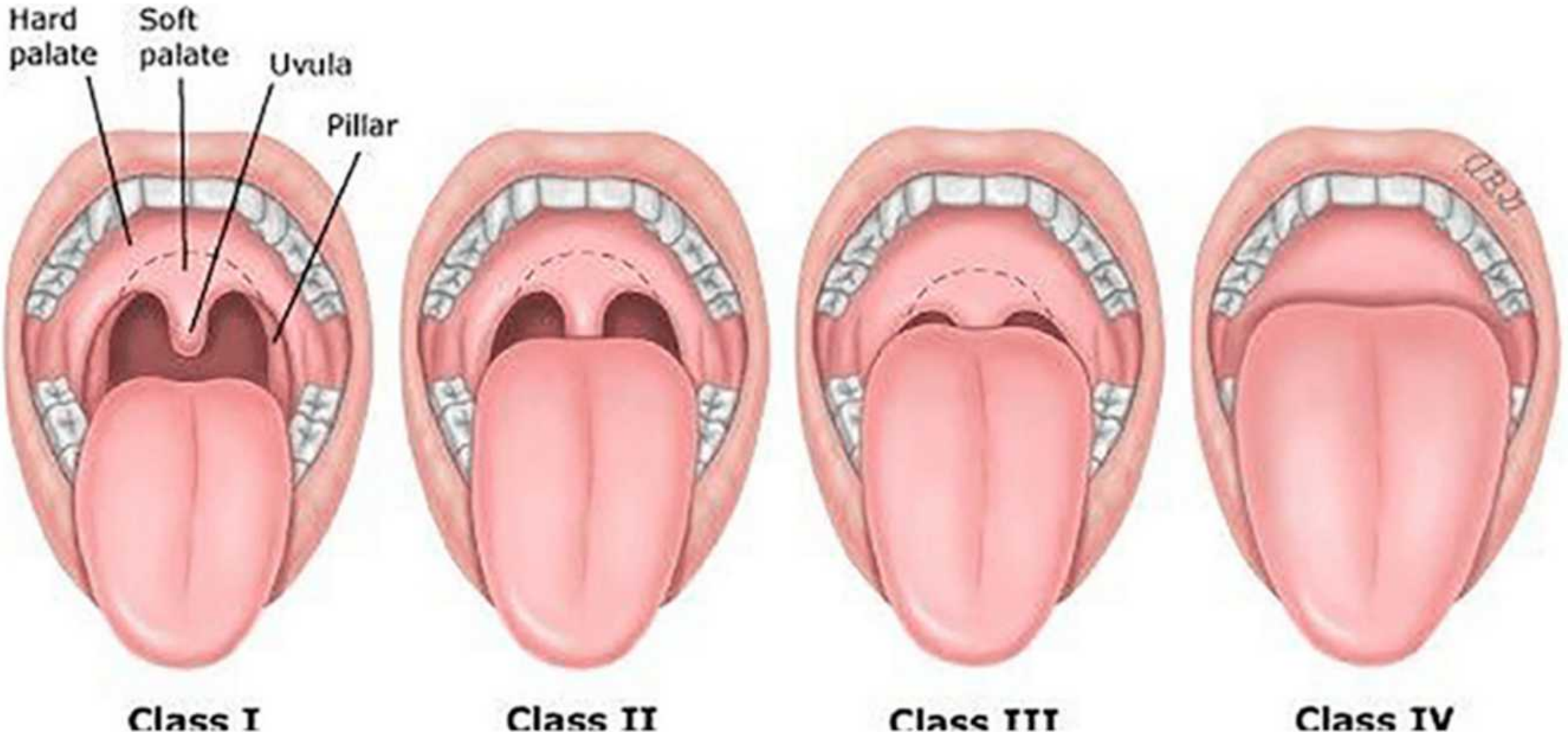


Figure 1. Examples of common craniofacial phenotypes in children with OSA. a) High-arched palate and oral breathing; b) narrow maxilla and retrognathia; c) hypotonic lips; d) increased lower facial height. Reproduced and modified from Simonds et al. *(2012) with permission.*

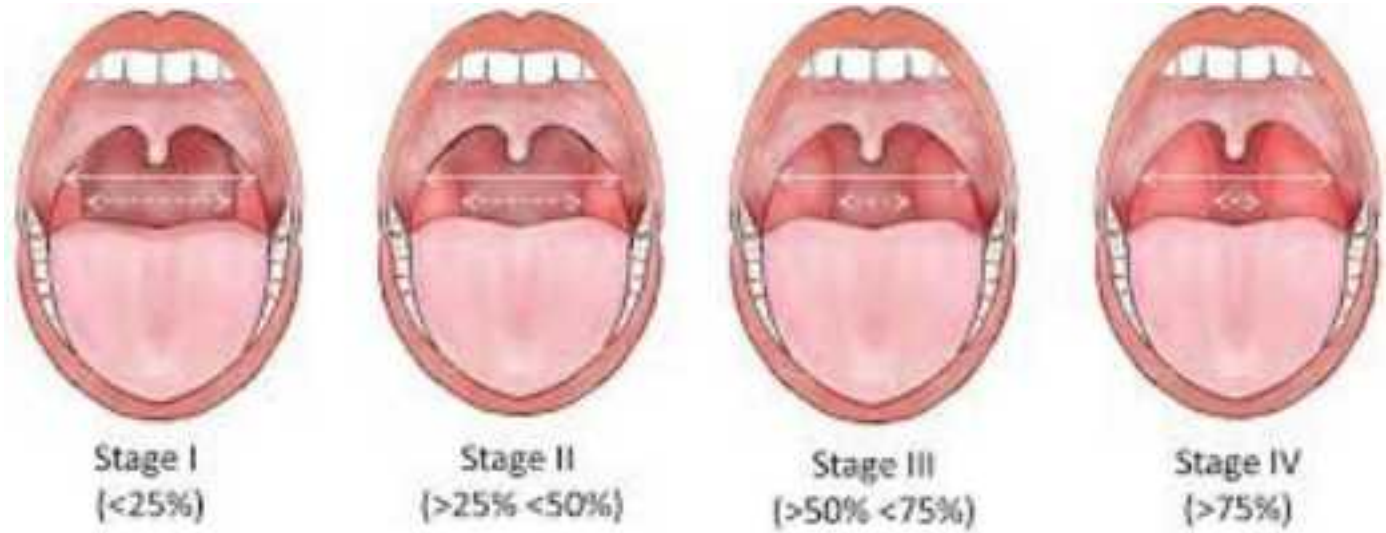
Pathognomonic OSA

Dr K Pillai

Mallampati Score



Tonsils grading scale- after Brodsky



OSA- confirming the diagnosis

- Polysomnography (gold standard)
- Polygraphy
- Questionnaires (Sleep Clinical Record)and oximetry (low resource settings)

Oximetry

- In children with enlarged T & A , normal oximetry does **not** exclude OSA

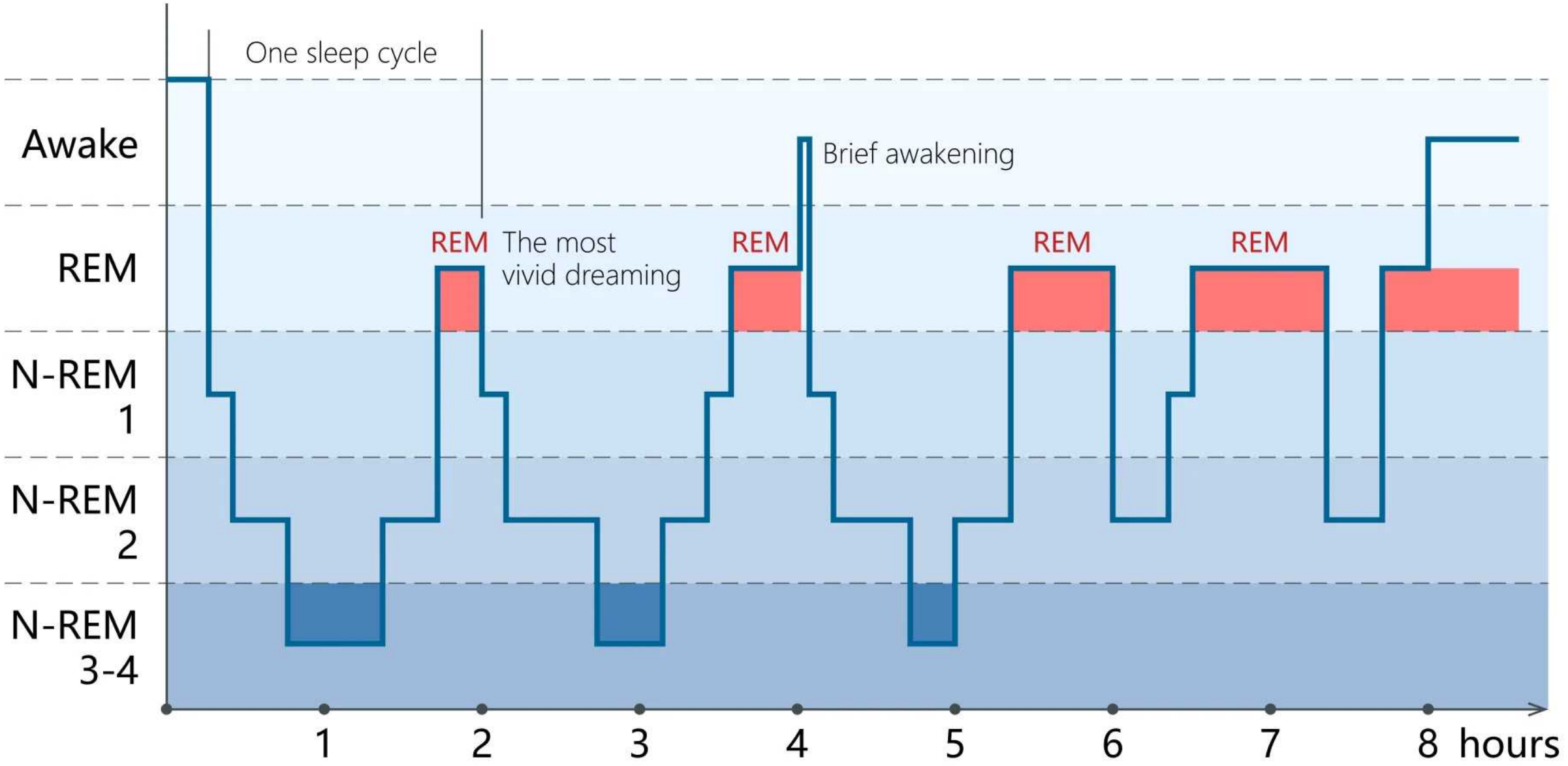
- In children with Trisomy 21 hypoxaemia could also be due to central apnoeas (Rx is different)

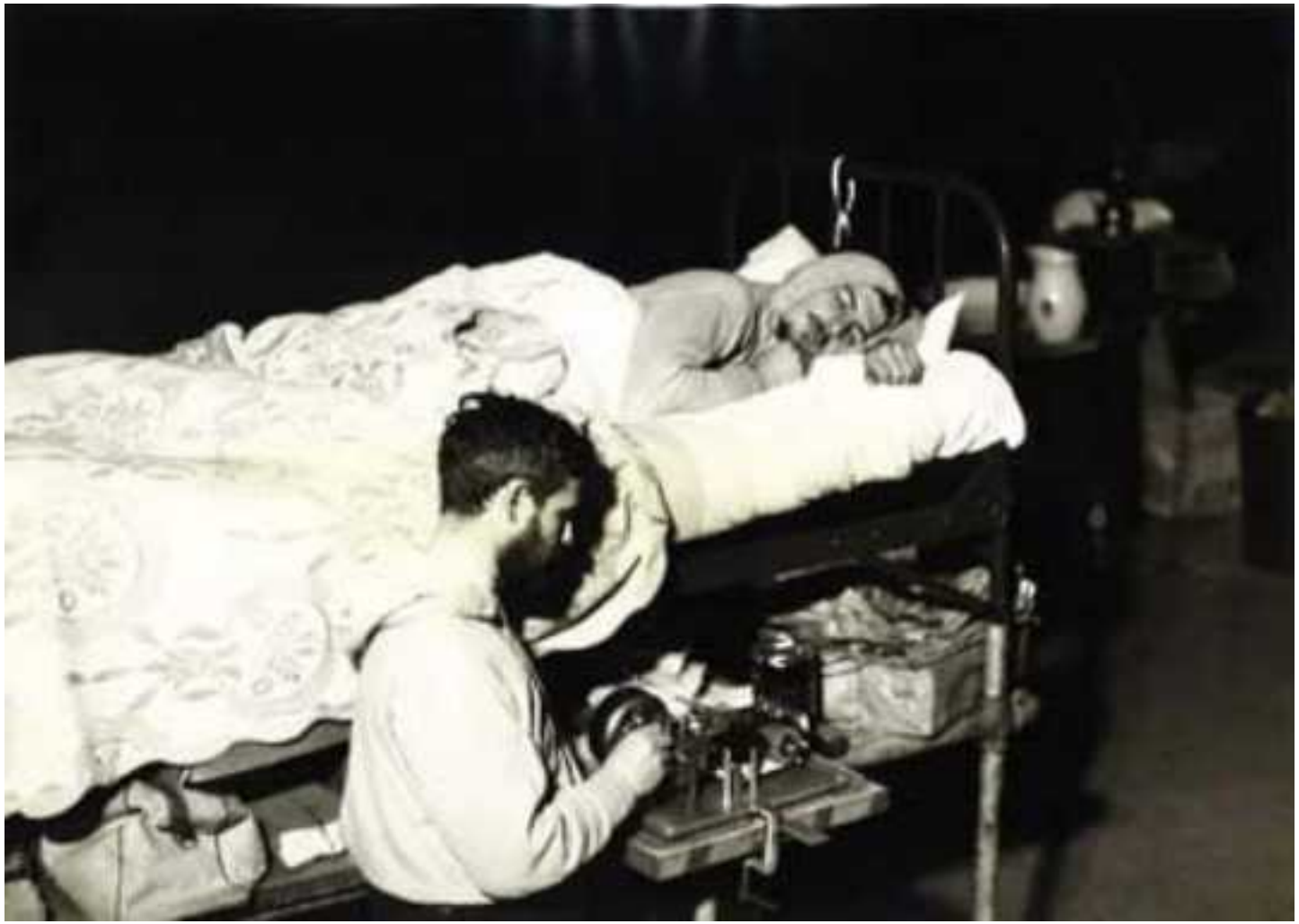
Polysomnography v Polygraphy

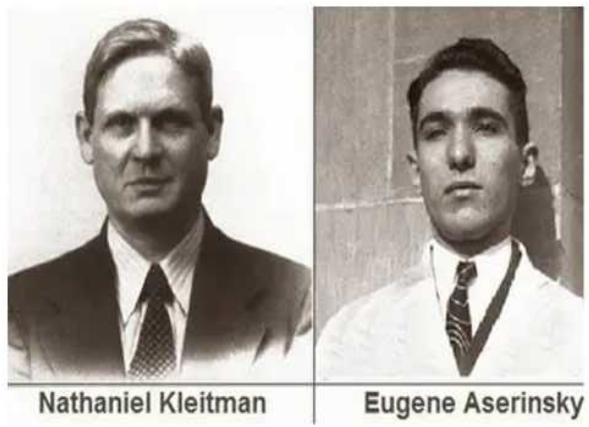
- 2016 ERS Consensus
- PG a suitable alternative to PSG for Dx of Paediatric OSA
- Good quality recordings in 95 % cases
- 90 % sensitivity

Pioneers of Sleep Medicine

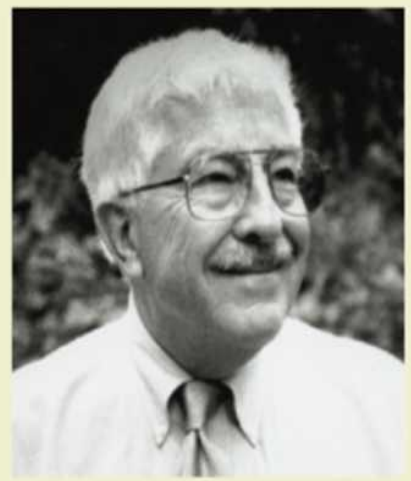
Sleep Cycles



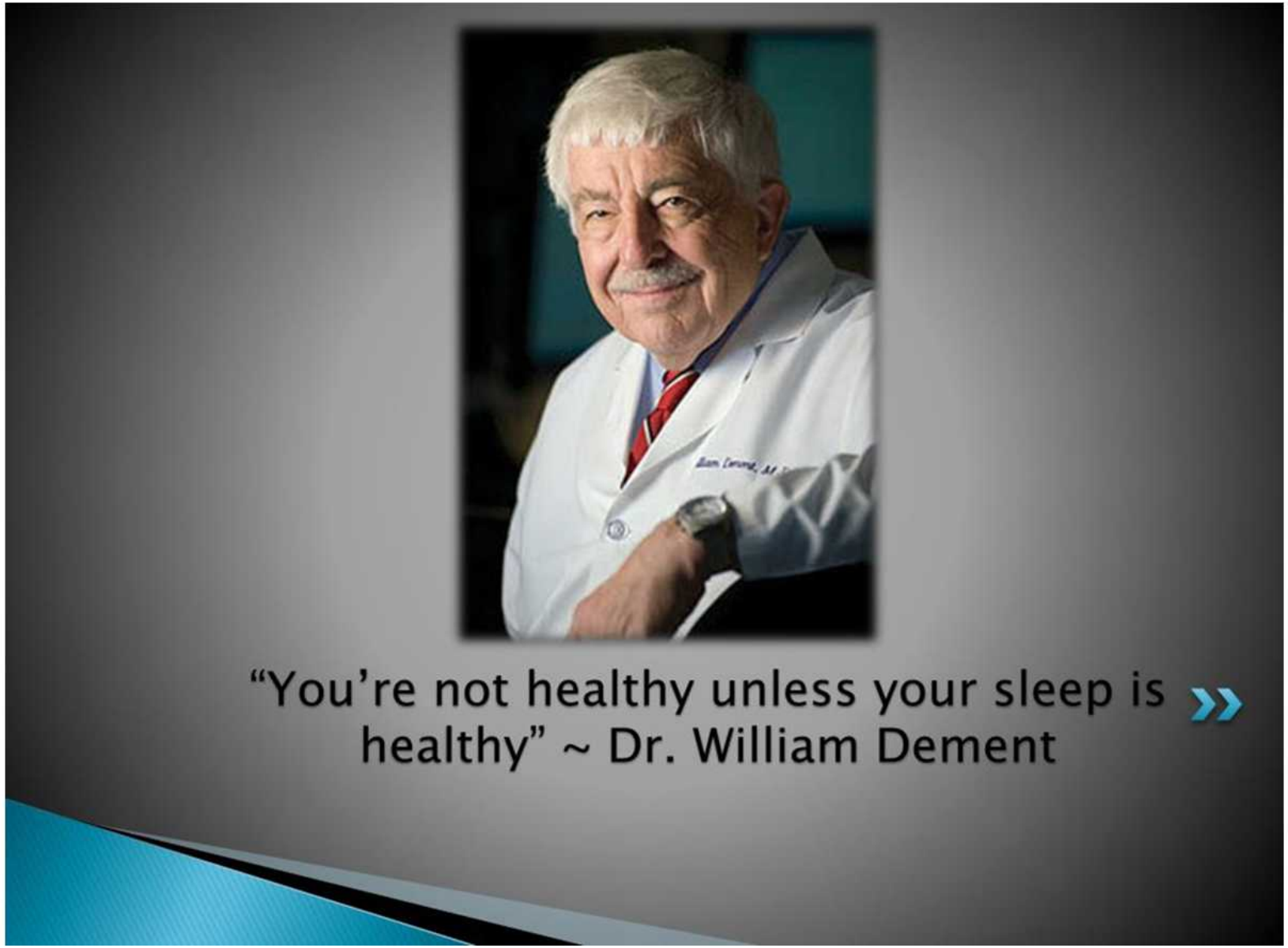




Nathaniel Kleitman | Eugene Aserinsky

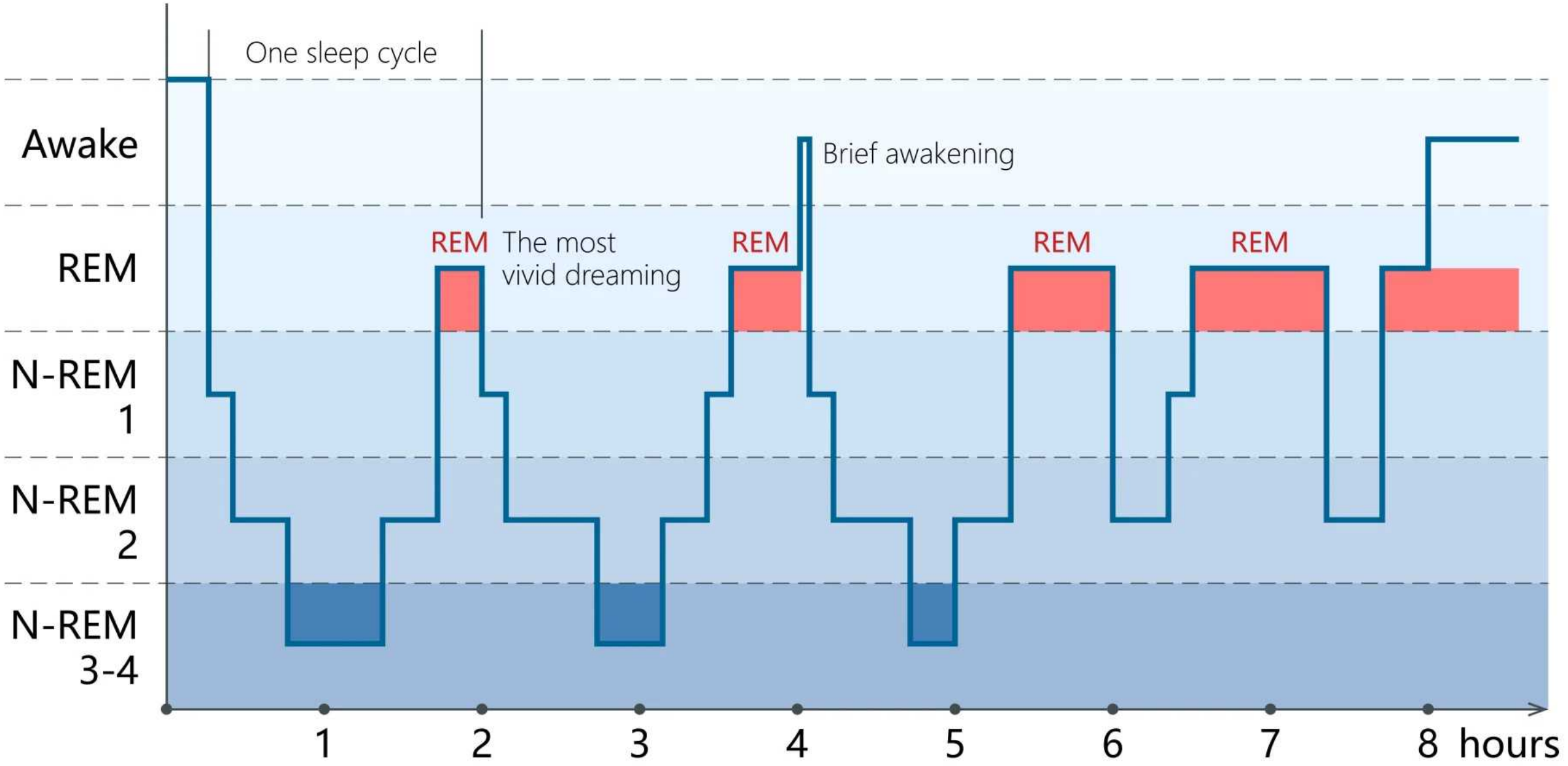


Dr. William Charles Dement


"You're not healthy unless your sleep is healthy" ~ Dr. William Dement

Christian Guilleminault

Sleep Cycles



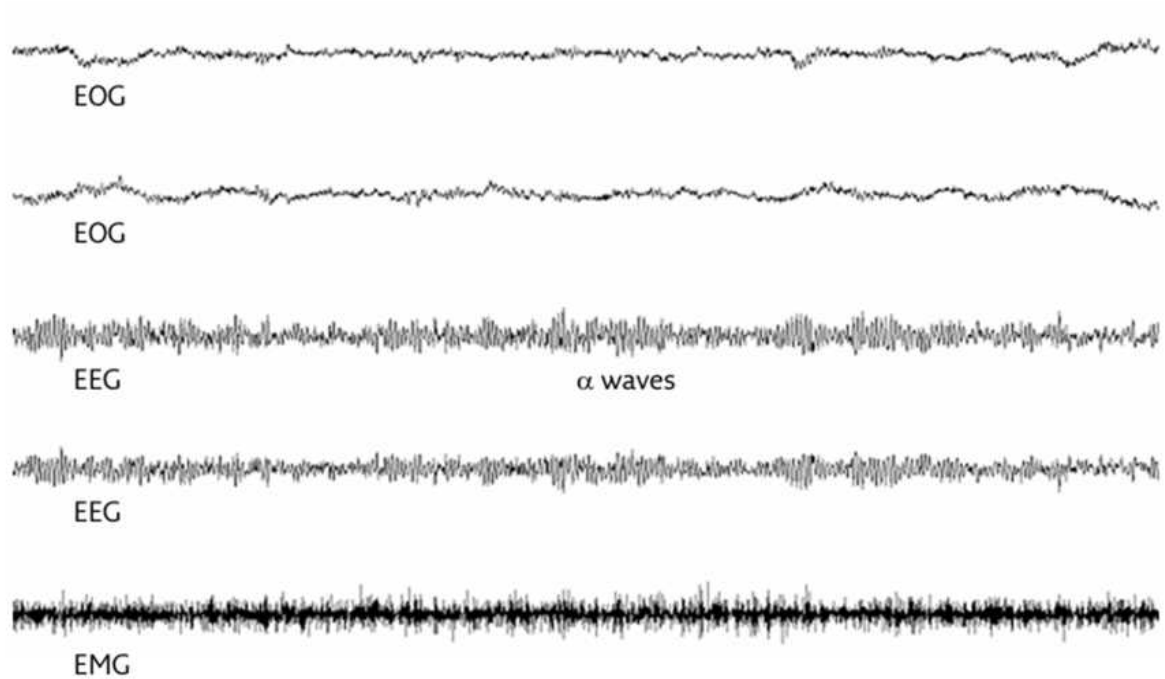


Figure 3. Stage W (Wakefulness). Note α-waves on EEG. Reproduced and modified from Riha (2012) with permission.

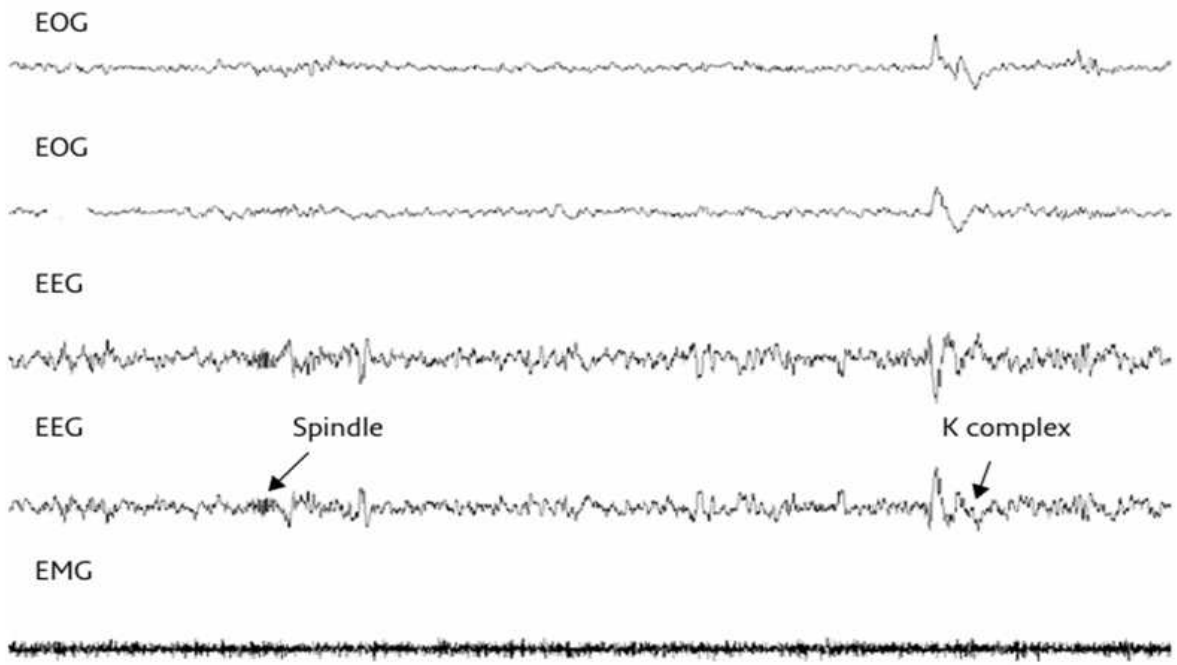


Figure 4. Stage N2 sleep (NREM 2). Reproduced and modified from Riha (2012) with permission.

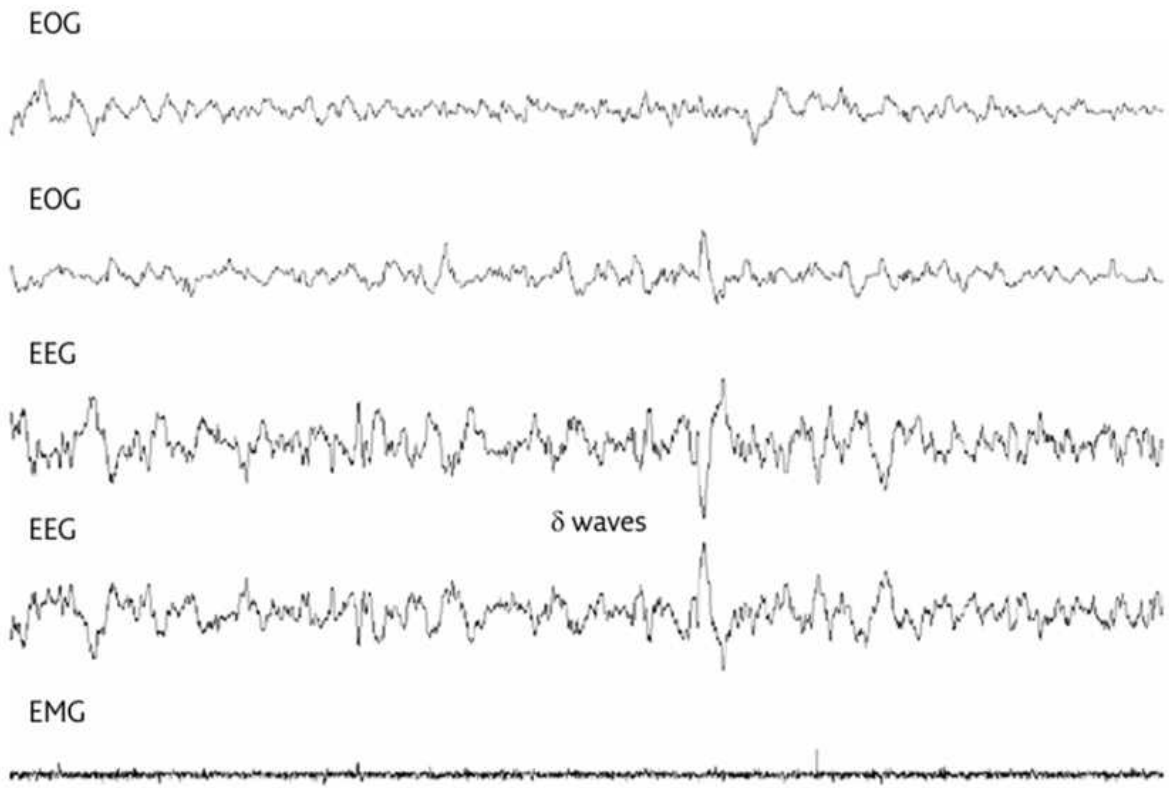


Figure 5. Stage N3 sleep (NREM 3). Note slow, high-amplitude δ-waves. Reproduced and modified from Riha (2012) with permission.

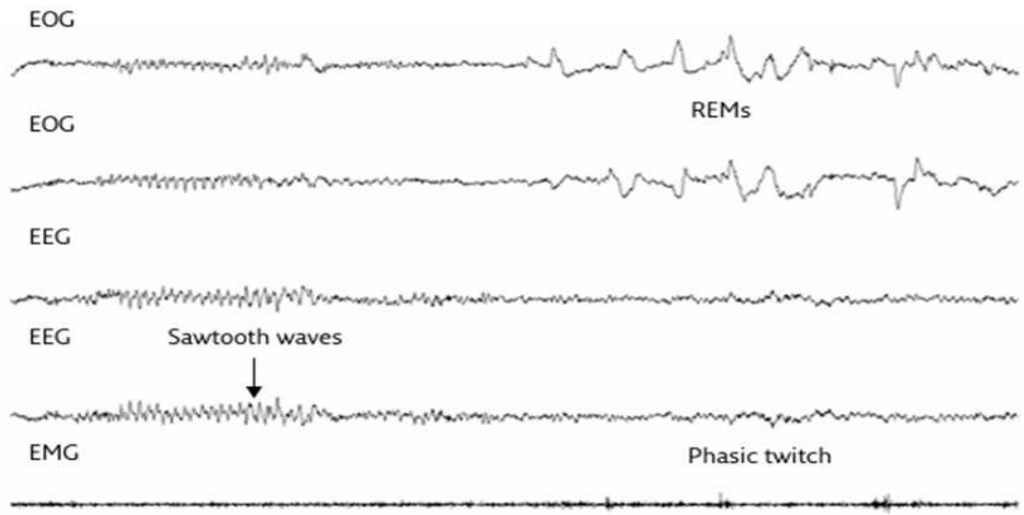
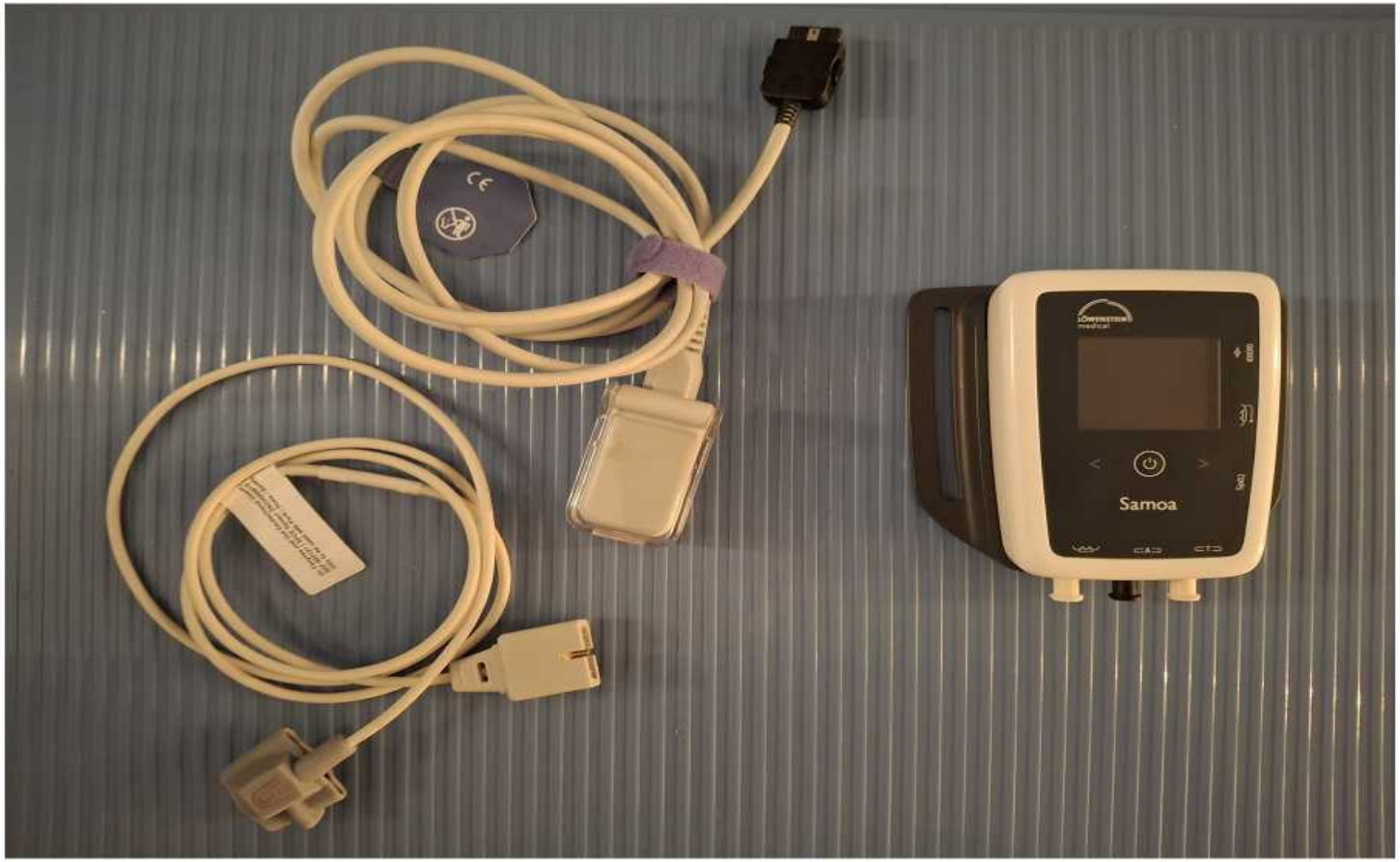


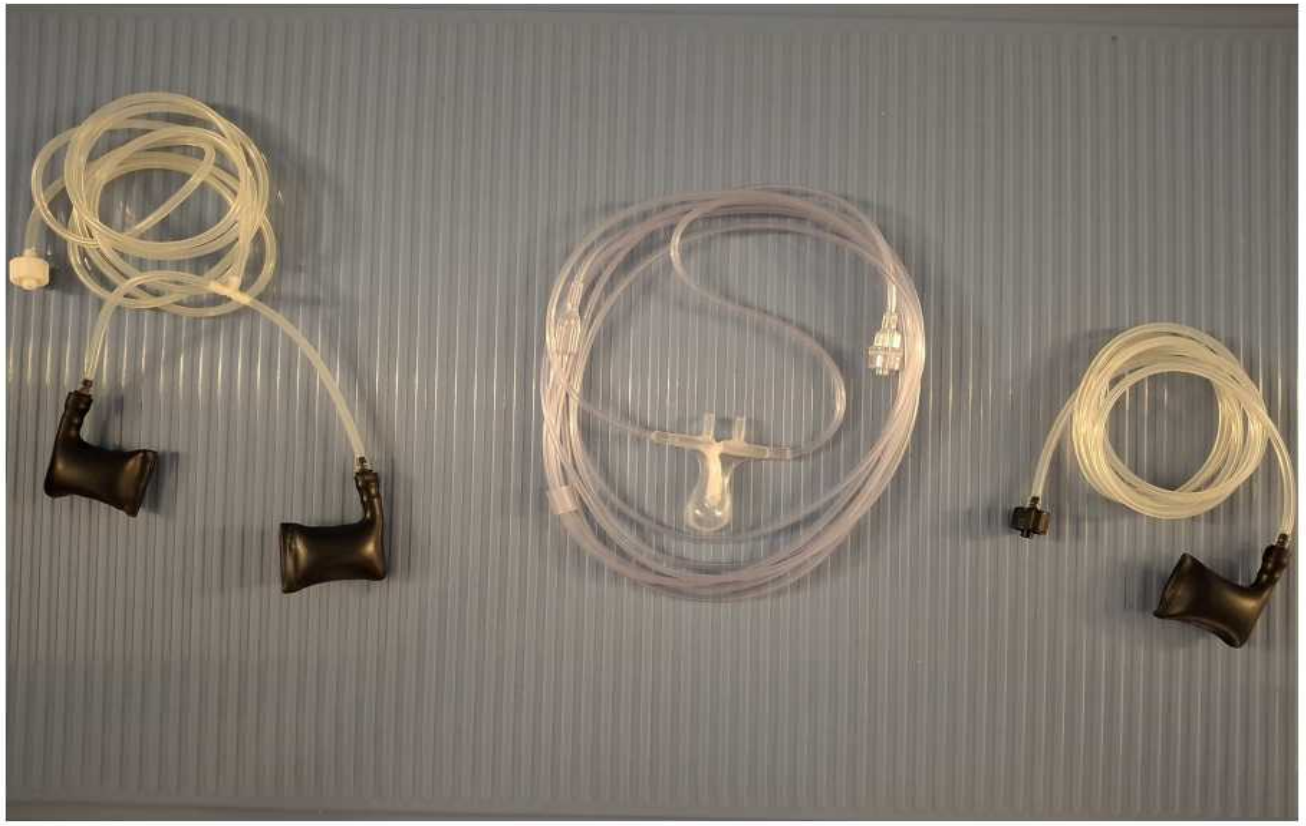
Figure 6. Stage R (REM). Reproduced and modified from Riha (2012) with permission.

Recording Sleep

Polygraphic sleep recorder



Paediatric Polygraph – nasal cannula and belt sensors



OSA- polysomnographic study

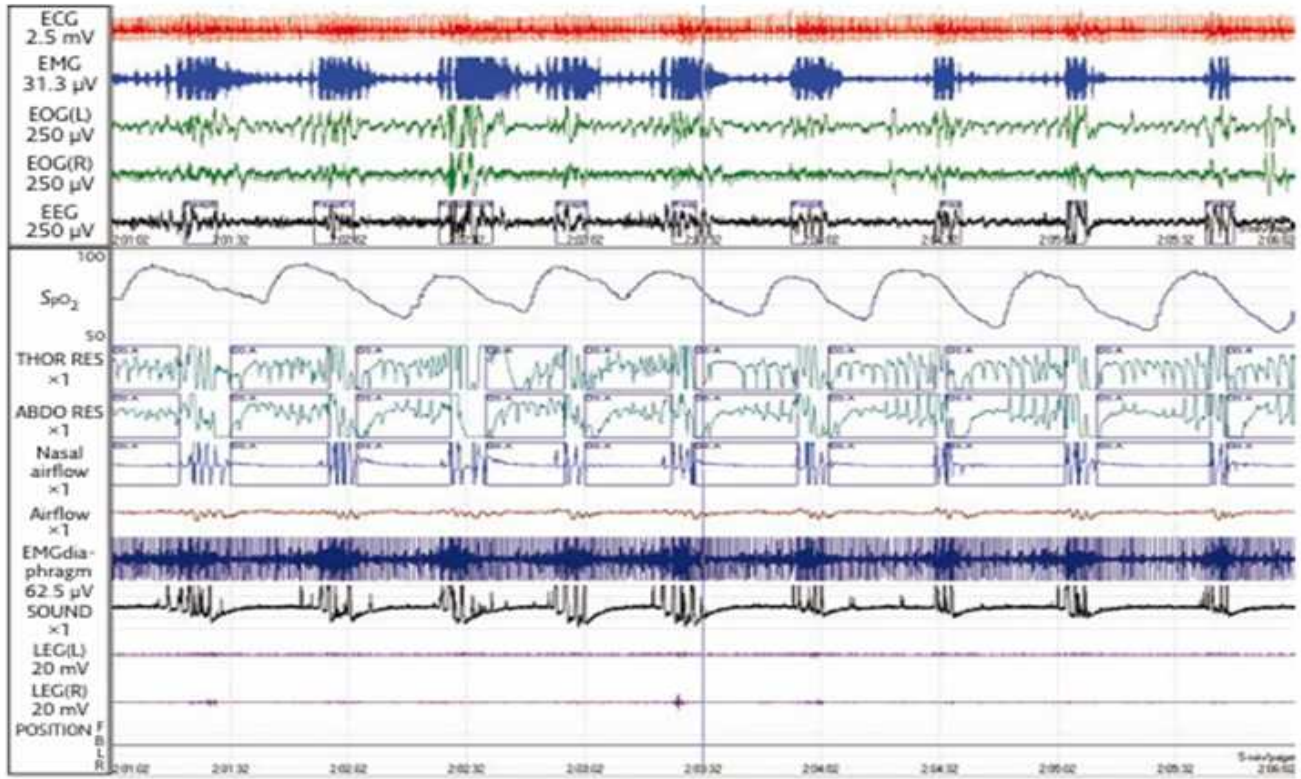
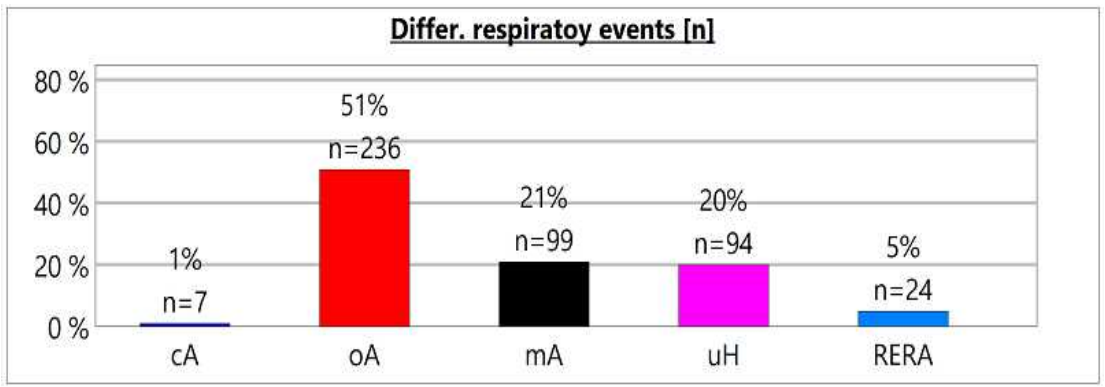


Figure 7. 5-min page of PSG showing obstructive respiratory events (ObA) accompanied by desaturations and arousals. THOR RES: thoracic respiratory band; ABDO RES: abdominal respiratory band; F: front; B: back; L: left side; R: right side. Reproduced and modified from Riha (2012) with permission.

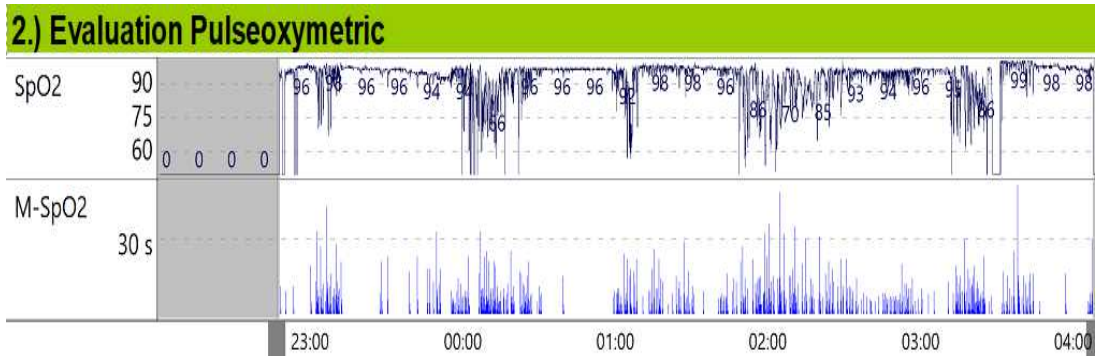
Baby X

Respiratory evaluation	Findings
AHI Per hour	**81,3**
AHI (Desat.-Cor.)[Per hour]	(49,8)
RDI Per hour	**85,8**
RDI (Desat.-Cor.) [Per hour]	(51,1)
Apnea Index AI [Per hour]	63,8
Hypopnea Index HI [Per hour]	17,5
No. of Apnea [n]	342
Of them Central: [n]	7
Mean duration of apneas [Sec]	14
No. of Hypopnea [n]	94
Total Apnea / Hypopnea time (RDT) [Hrs]	1:39:54
Apnea / Hypopnea time per hour [Min Per hour]	18:38
Longest Apnea [Min] (t=23:46:01)	1:51
Longest Hypopnea [Sec] (t=03:00:35)	54
Periodic breathing time per hour [Sec Per hour]	0
Total Periodic breathing time [Sec]	0
Snoring Index SI [Per hour]	0,0
Snoring Index SI irregular [Per hour]	0,0
Total Snoring Time ST [Sec]	0



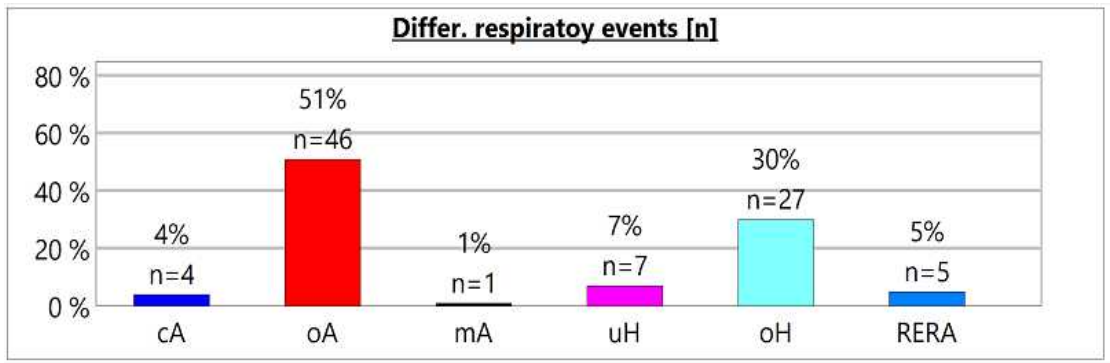
Baby X

Evaluation of SpO2 / Pulse	Findings
Desaturation-Index ODI [Per hour]	**67,3**
No. of desaturations [n]	349
No. of desaturations < 90%: [n]	182
Total time [Hrs]	1:05:05
Time per hour [Min Per hour]	12:34
Lowest Desaturation [%] (01:51:15)	53
Longest Desaturation [Sec] (03:38:29)	52
Mean Duration [Sec]	11
Mean Desaturation [%]	85,2
Mean Saturation [%]	93,5
Max. Saturation [%] (02:29:35)	100
Min. Saturation [%] (02:03:11)	51
t90 [%]	14,3
Min. Puls (23:53:28) [1/min]	57
Max. Puls (02:01:47) [1/min]	151
Mean Pulse [1/min]	120
Pulse variances [n]	246
Pulse variance index [Per hour]	47,1



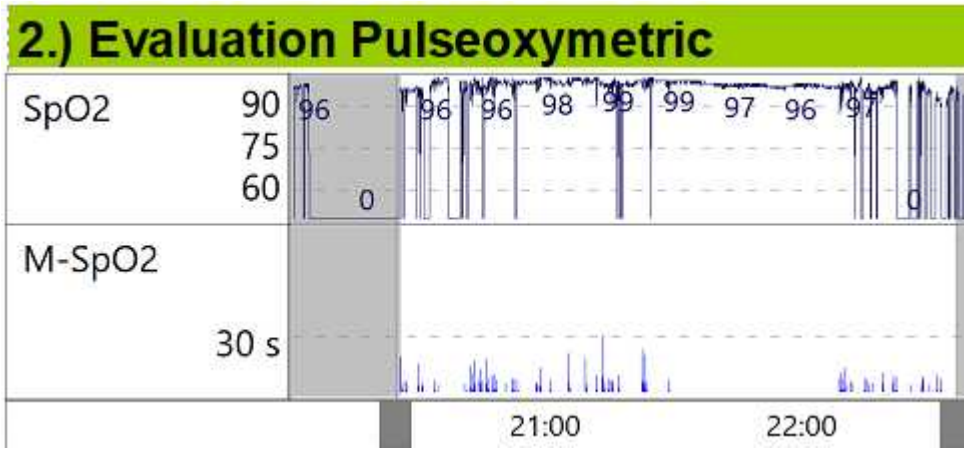
Baby Y

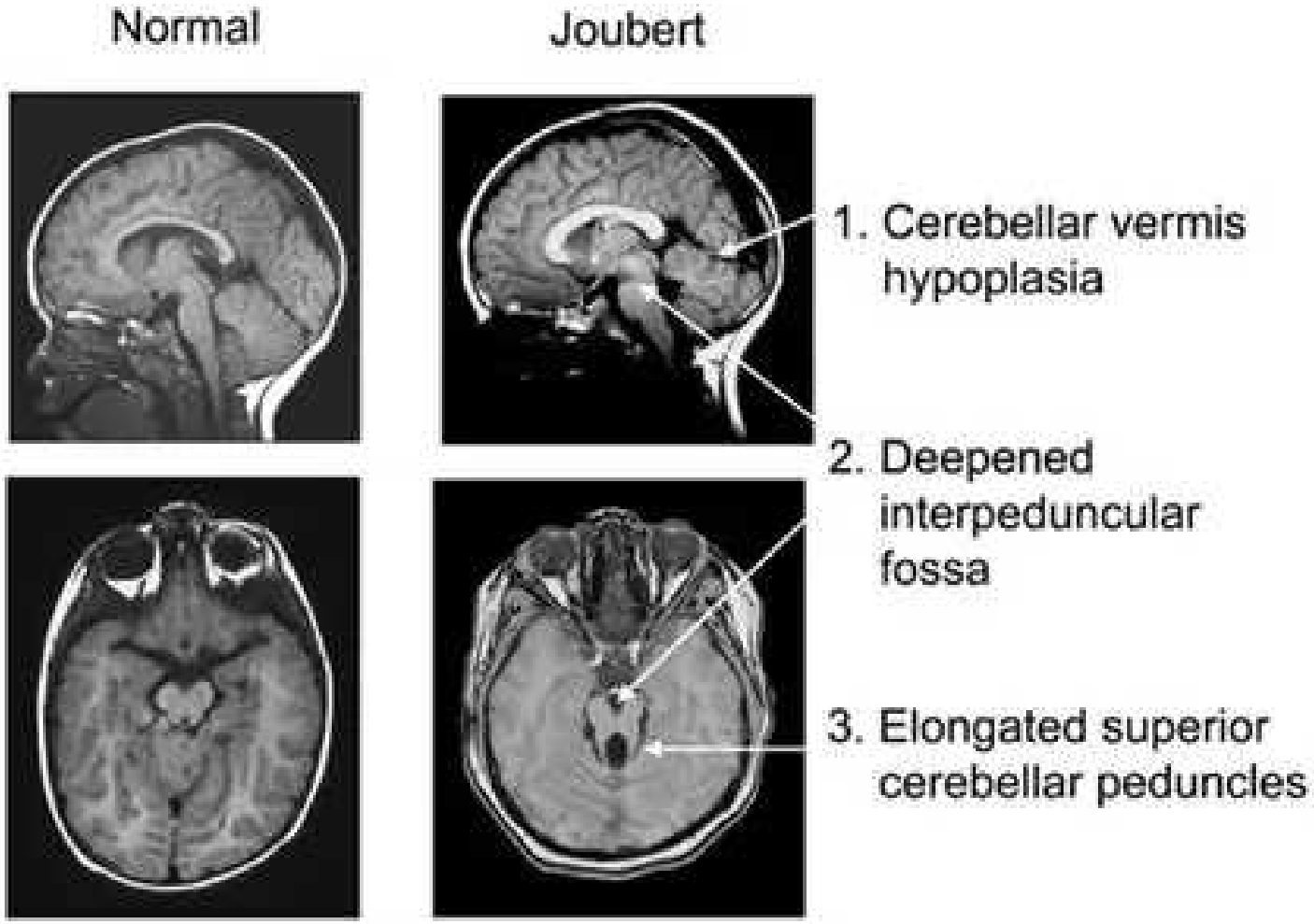
Respiratory evaluation	Findings
AHI Per hour	**38,8**
AHI (Desat.-Cor.)[Per hour]	(15,1)
RDI Per hour	**41,1**
RDI (Desat.-Cor.) [Per hour]	(15,5)
Apnea Index AI [Per hour]	23,3
Hypopnea Index HI [Per hour]	15,5
No. of Apnea [n]	51
Of them Central: [n]	4
Mean duration of apneas [Sec]	13
No. of Hypopnea [n]	34
Total Apnea / Hypopnea time (RDT) [Min]	18:24
Apnea / Hypopnea time per hour [Min Per hour]	8:24
Longest Apnea [Min] (t=22:08:43)	1:53
Longest Hypopnea [Sec] (t=20:32:55)	37
Periodic breathing time per hour [Sec Per hour]	0
Total Periodic breathing time [Sec]	0
Snoring Index SI [Per hour]	0,0
Snoring Index SI irregular [Per hour]	0,0
Total Snoring Time ST [Sec]	0



Baby Y

Evaluation of SpO2 / Pulse	Findings
Desaturation-Index ODI [Per hour]	**36,2**
No. of desaturations [n]	68
No. of desaturations < 90%: [n]	3
Total time [Min]	9:34
Time per hour [Min Per hour]	5:06
Lowest Desaturation [%] (22:08:30)	87
Longest Desaturation [Sec] (21:12:33)	31
Mean Duration [Sec]	8
Mean Desaturation [%]	93,5
Mean Saturation [%]	97,5
Max. Saturation [%] (20:33:07)	100
Min. Saturation [%] (20:32:00)	80
t90 [%]	0,4
Min. Puls (20:40:04) [1/min]	54
Max. Puls (22:26:55) [1/min]	139
Mean Pulse [1/min]	99
Pulse variances [n]	96
Pulse variance index [Per hour]	51,3




Normal
Joubert
1. Cerebellar vermis hypoplasia
2. Deepened interpeduncular fossa
3. Elongated superior cerebellar peduncles

Rx OSA **first 2 yrs** (ERS statement 2017)

- Obvious clinical manifestations of upper airway obstruction
- SDB symptoms/signs AND obstructive AHI > 1 event / hr.
- Complex disorder associated with SDB and obstructive AHI > 1 event /hr

OSA Rx first two years – ERS 2022

Table 1. A stepwise treatment approach for OSAS in the first 2 years of life

1) OSA and gastro-oesophageal reflux: efficacy of antireflux medication is controversial
2) Adenoidal with/without tonsillar hypertrophy: adenoidectomy with/without tonsillectomy
3) CPAP or NIPPV if OSA is moderate-to-severe and there is: laryngomalacia; midface or mandibular hypoplasia; cerebral palsy; SMA; achondroplasia; mucopolysaccharidoses; or Trisomy 21
4) Supraglottoplasty for laryngomalacia; surgical tongue advancement (*i.e.* glossopexy *via* tongue–lip adhesion) or mandibular distraction osteogenesis for mandibular hypoplasia; midface advancement for midface hypoplasia
5) Tracheostomy if treatment modalities 1–4 are not adequate or in order to secure the upper airway while awaiting surgical intervention

Nocturnal PSG, polygraphy or oximetry should be used at each step to move onto the next treatment step if residual OSA is present.

Indications for Rx of OSA **ages 2 -18 yr** - ERS 2016

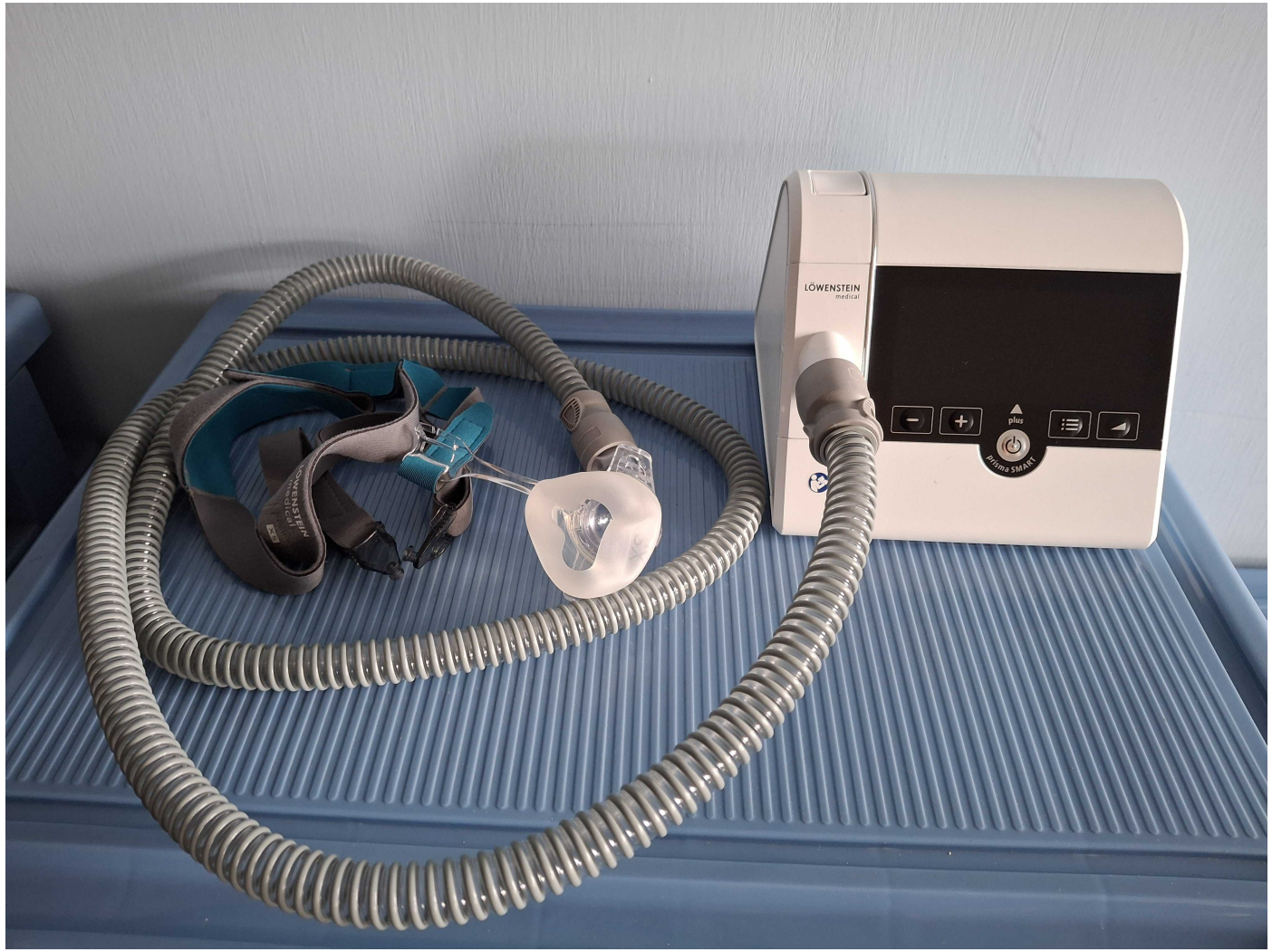
- Moderate-to-severe OSA (AHI of >5 events·h^{-1}) irrespective of the presence of morbidity.
- Mild OSA (AHI of 1–5 events·h^{-1}) with OSA-related morbidity (*e.g.* enuresis, inadequate somatic growth, poor academic performance, inattention, hyperactivity, PH, or SBP or DBP >95th percentile for sex, age and height) or a decrease in quality of life.
- Mild OSA in combination with risk factors for SDB persistence (male sex, obesity, increasing BMI percentile, tonsillar hypertrophy, narrow mandible).
- OSA treatment is a priority in the presence of major craniofacial abnormalities, neuromuscular disorders, achondroplasia, Chiari malformation, Trisomy 21, mucopolysaccharidoses and Prader–Willi syndrome. Patients with these conditions may be at risk of developing PH and it is unlikely that SDB will resolve spontaneously.

Table 2. A stepwise management approach for 2–18-year-old children with OSAS who have indications for treatment

1) Weight loss if the child is overweight or obese
2) Nasal corticosteroids for 4–12 weeks and/or montelukast for 16 weeks, if adenoidal hypertrophy is present and the AHI is 1–10 events·h^{-1}; this may also be used for residual OSA following adenotonsillectomy
3) Adenotonsillectomy for adenotonsillar hypertrophy
4) Orthodontic devices for craniofacial abnormalities (*e.g.* oral appliances for retrognathia or malocclusion and rapid maxillary expansion for maxillary constriction); orthodontic treatment may be used for residual OSA after adenotonsillectomy (AHI of >5 events·h^{-1})
5) Nasal CPAP if the AHI is >5 events·h^{-1} and:
There is residual OSA after adenotonsillectomy
OSA is related to obesity
OSA is related to craniofacial abnormalities
OSA is associated with major craniofacial abnormalities while awaiting a craniofacial procedure
If OSA is accompanied by nocturnal hypoventilation as in neuromuscular disorders, NIPPV is preferred
6) Craniofacial surgery if the treatment modalities above are not adequate for OSA resolution; craniofacial procedures include mandibular distraction osteogenesis for micrognathia and midface advancement for midface hypoplasia
7) Tracheostomy if treatment modalities 1–6 are not adequate for OSA resolution or to secure the upper airway while awaiting surgical intervention

Nocturnal PSG, polygraphy or oximetry should be used at each step to move onto the next treatment step if residual OSA is present. Reproduced and modified from Simonds *et al.* (2012) with permission.

CPAP device and Mask



Benefits of CPAP

- Better QOL
- Appreciable growth
- Reduced daytime somnolence, irritability , hyperactivity
- Enuresis cured or diminished
- Reduction in systemic BP, PAP and Cor Pulmonale

Key points

- Adenotonsillectomy is first-line treatment for OSAS in infancy. Other treatments (orthodontic appliances, CPAP and tracheostomy) may be indicated in selected patients.
- Adenotonsillar hypertrophy is a frequent cause of OSAS in otherwise healthy children, but <30% of them achieve a normal AHI (<1 episode·h^{-1}) after adenotonsillectomy.
- Oral appliances and functional orthopaedic devices are effective in cases of maxillary constriction or mandibular retrusion with associated OSAS.
- Long-term home ventilation is required for children with central hypoventilation.
- NIPPV in neuromuscular disorders should be initiated when nocturnal hypoventilation develops.
- NIPPV improves quality of life, morbidity and mortality in many stable or slowly progressive neuromuscular disorders, and may be considered to palliate symptoms in other more progressive conditions.

Childhood OSA is Under recognised

- Think of it !
- Listen to parents – they are observers of their child's sleep
- If unsure, ask for advice .