

CENTRAL APNEA

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TOPICS COVERED

- Definition
- Classification
- Treatment

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DEFINITION

DEFINITION

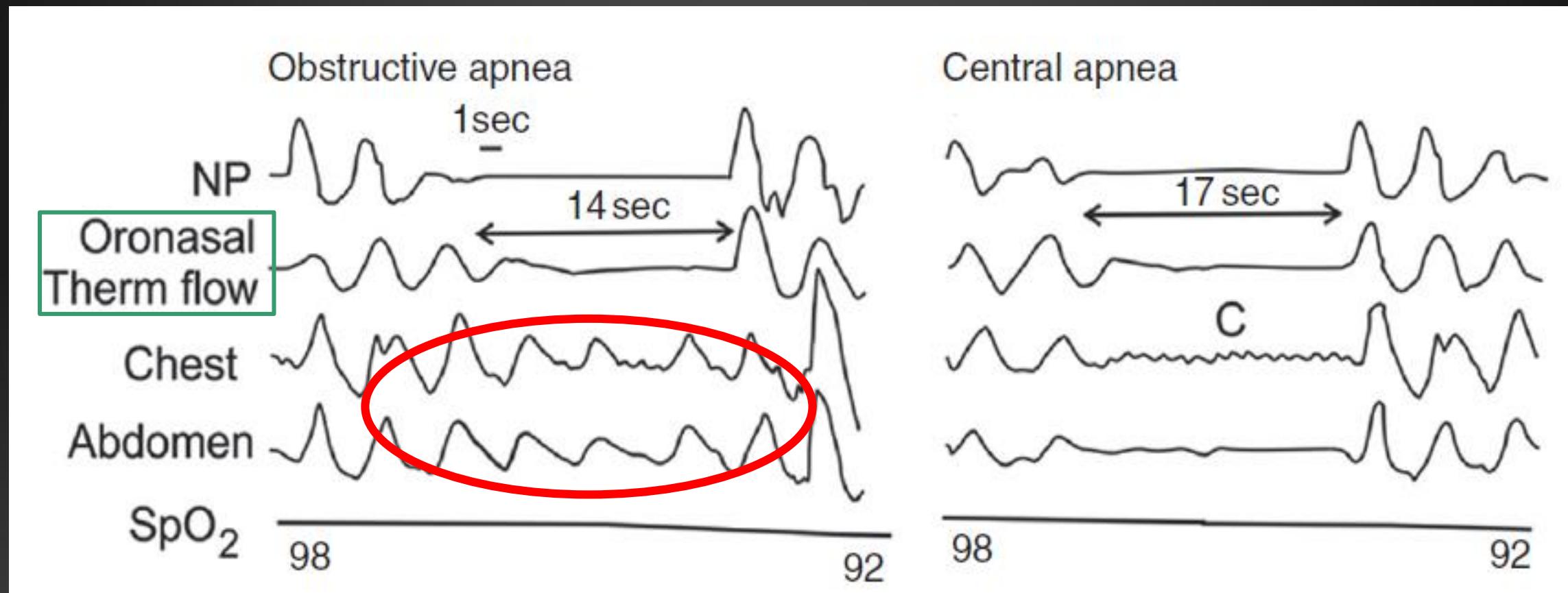
- A) Central Apnea as an EVENT
- B) Central Apnea as a DISORDER
- C) Central Apnea as a SYNDROME

A) CENTRAL APNEA- EVENT

- Apnea:
 - 90- 100% reduction of flow
 - Thermal oronasal resistor/ PAP
 - At least 10 seconds
 - No effort in central



CENTRAL VS OBSTRUCTIVE APNEA

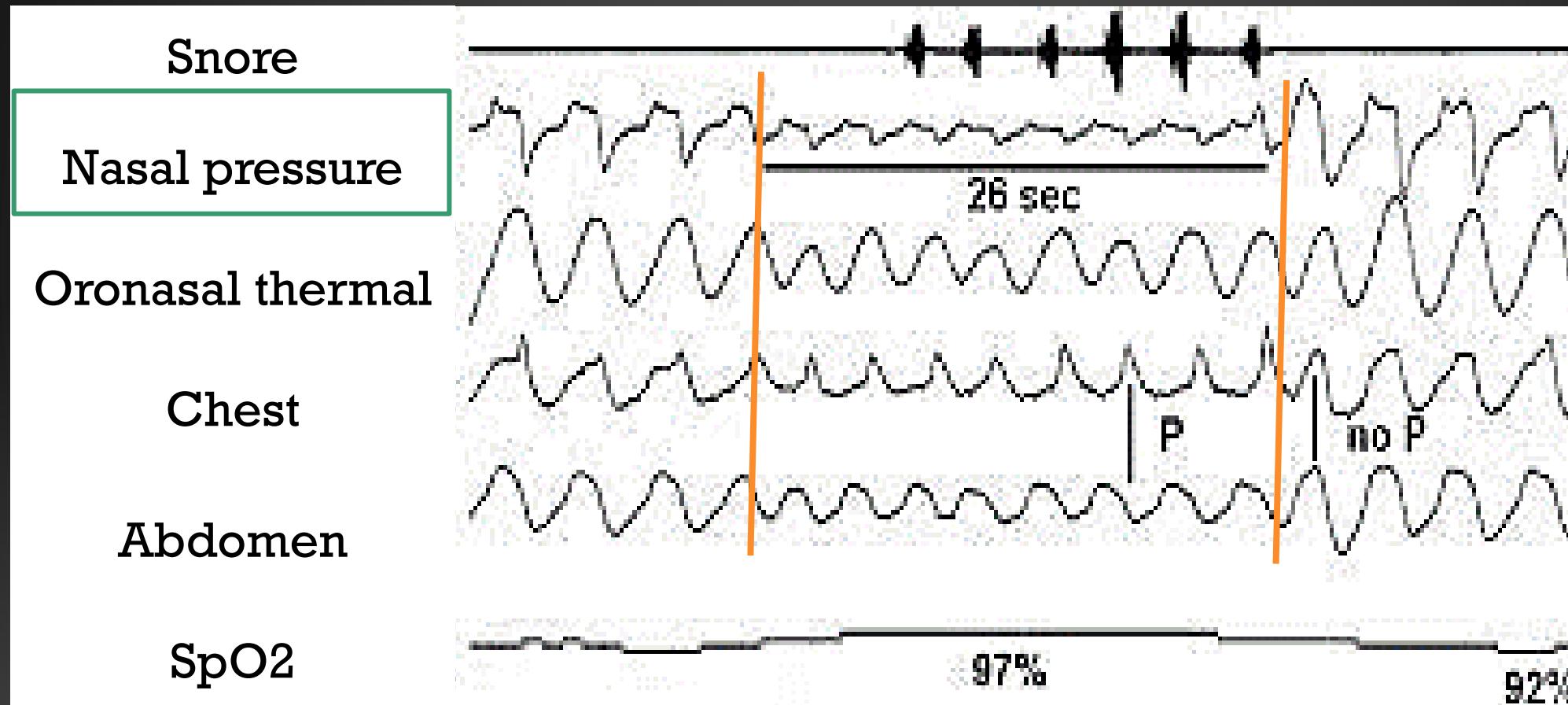


A) CENTRAL APNEA- EVENT

- Hypopnea:
 - 30- 90% reduction of flow + Desaturation/Arousal
 - Nasal pressure/ PAP
 - At least 10 seconds
 - Decreased effort in central



CENTRAL VS OBSTRUCTIVE HYPOPNEA

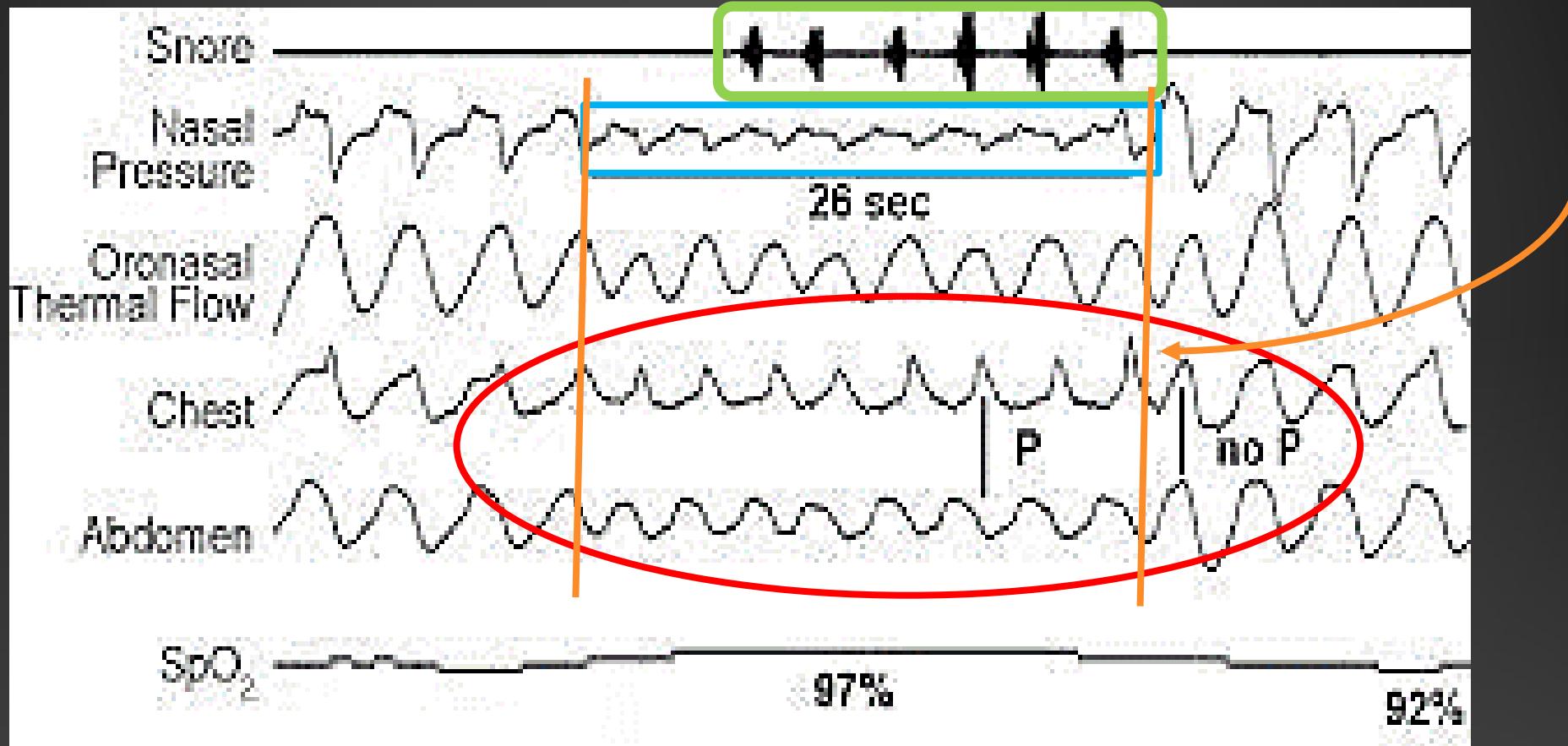


CENTRAL VS OBSTRUCTIVE HYPOPNEA

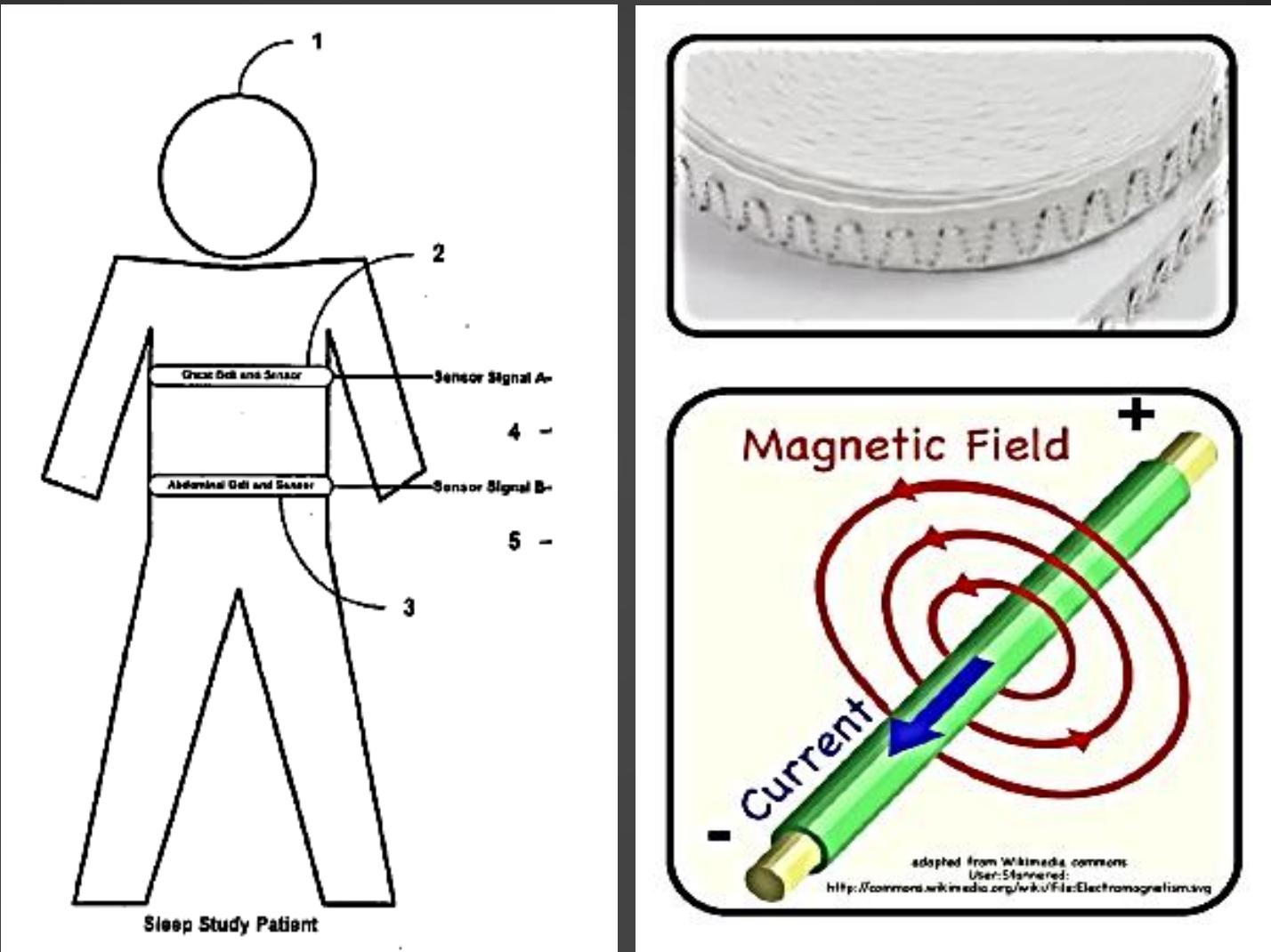
- Diagnosed by Lack of
 - Snoring
 - Flow limitation
 - Thoracoabdominal paradox

CENTRAL VS OBSTRUCTIVE HYPOPNEA

Obstructive hypopnea



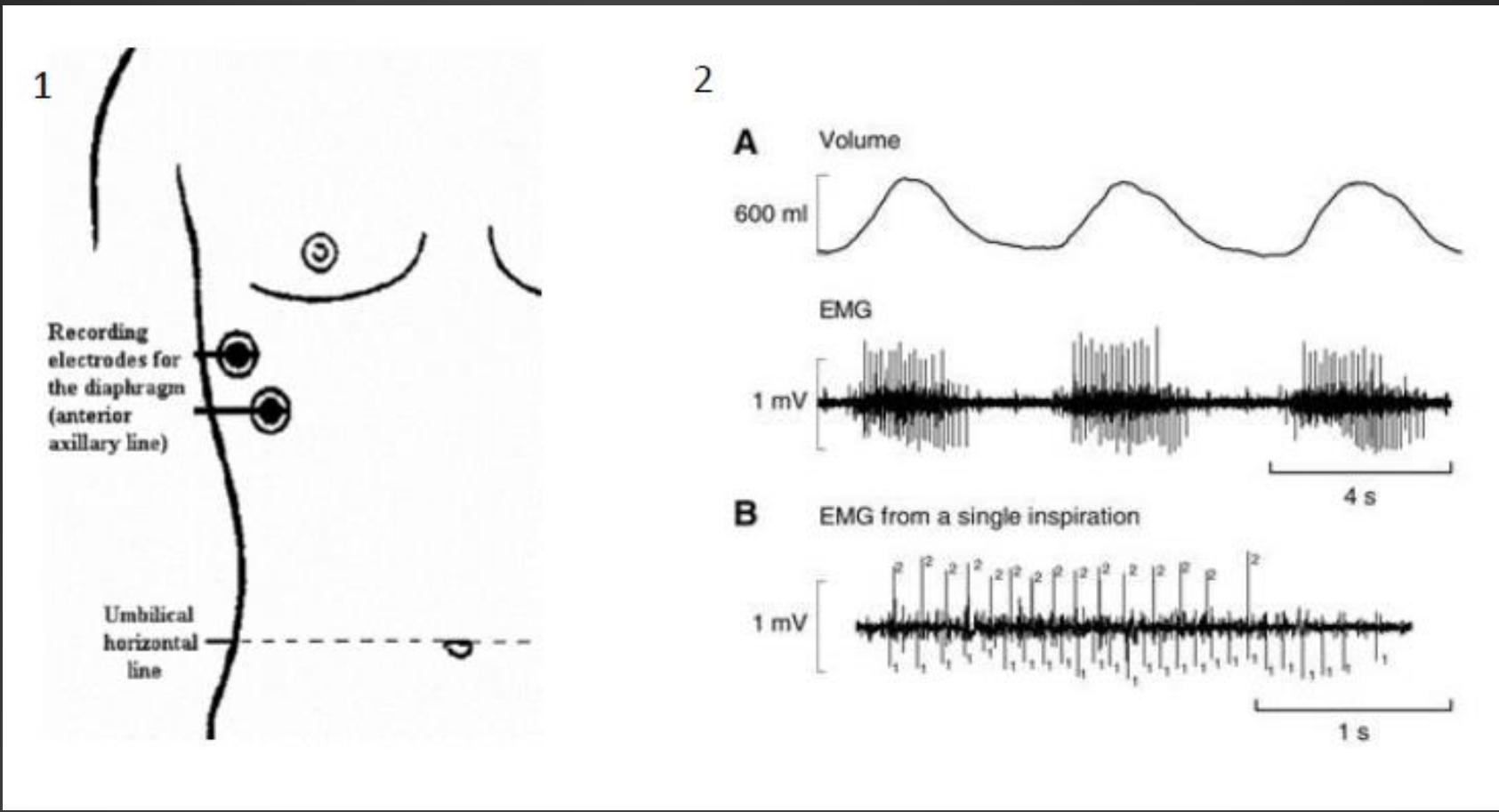
RIP



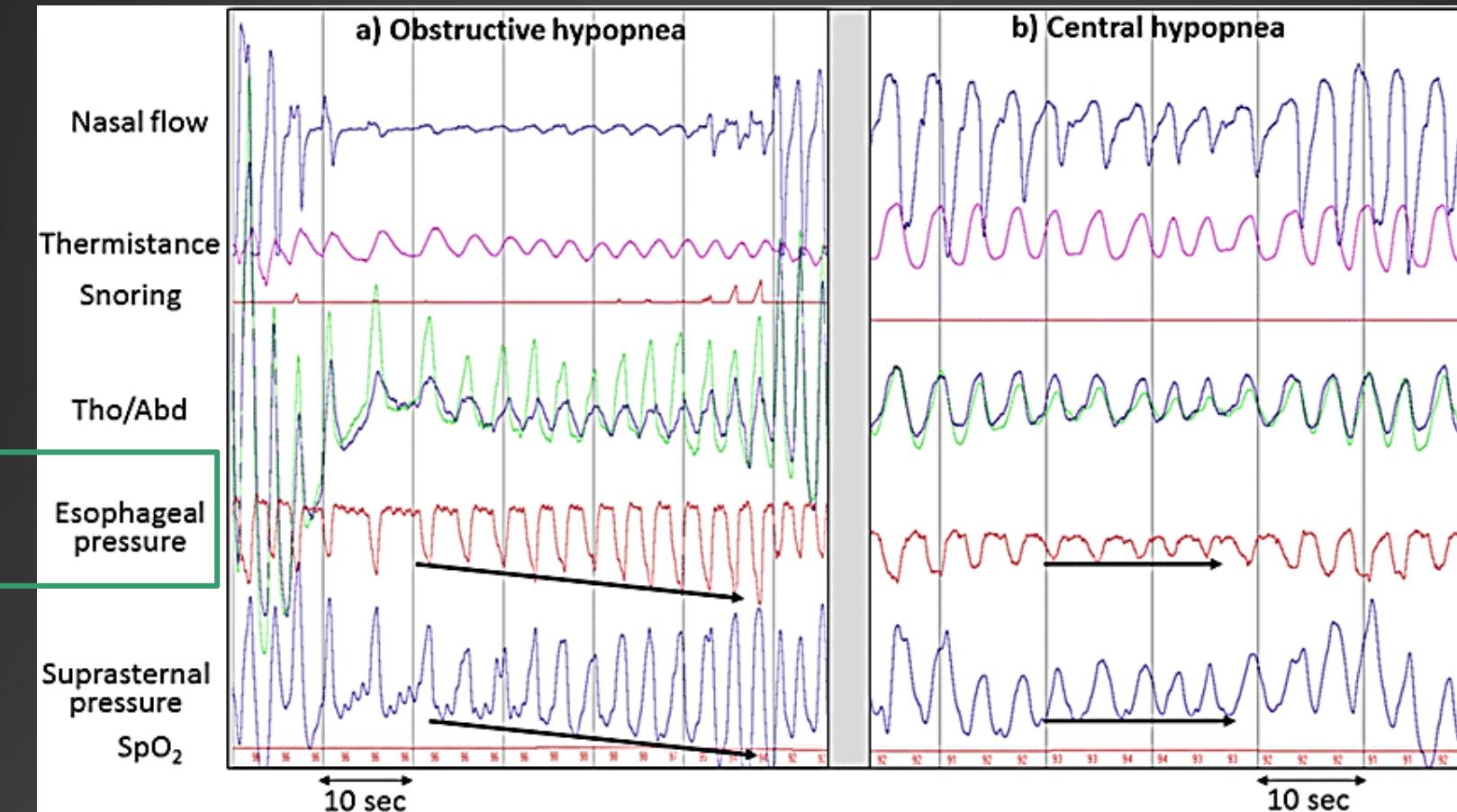
PVDF



DIAPHRAGM EMG



ESOPHAGEAL PRESSURE



B) CENTRAL SLEEP APNEA- DISORDER

- More than $\frac{1}{2}$ of total AHI is central

AHI	Rating
<5	Normal (no Sleep Apnea)
5-15	Mild Sleep Apnea
15-30	Moderate Sleep Apnea
>30	Severe Sleep Apnea

C) CENTRAL SLEEP APNEA- SYNDROME

- CSA disorder + Symptoms
 - Snoring
 - Restless sleep
 - Nocturnal awakening
 - Morning headache
 - Insomnia
 - Daytime sleepiness

CLASSIFICATION

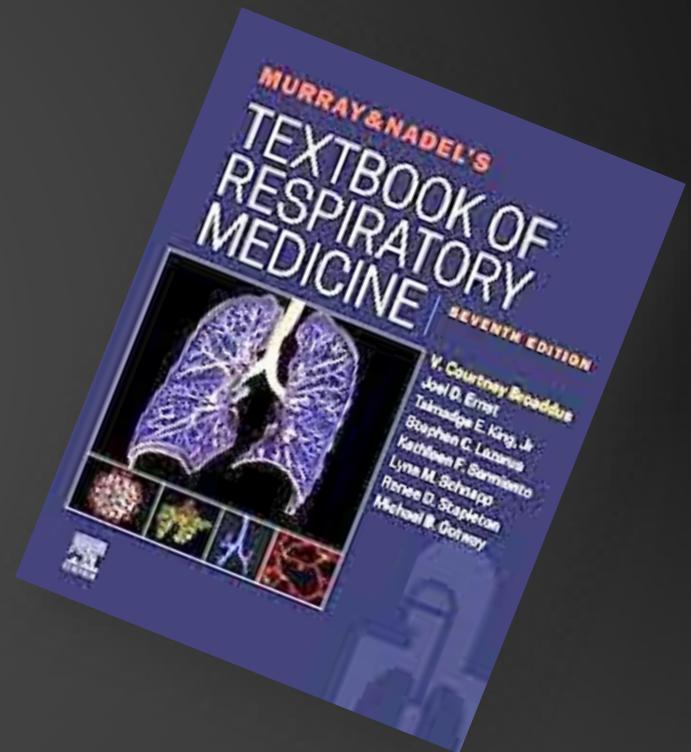
CLASSIFICATION

1. Cheyne stokes Respiration
2. Medical disorders
3. Medication/ Opioid
4. High Altitude
5. TE-CSA
6. ICSA



CLASSIFICATION

- Hypercapnic CSA
 - DRIVE pathology
- Non Hypercapnic CSA
 - Apnea threshold pathophysiology



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HYPERCAPNIC CSA

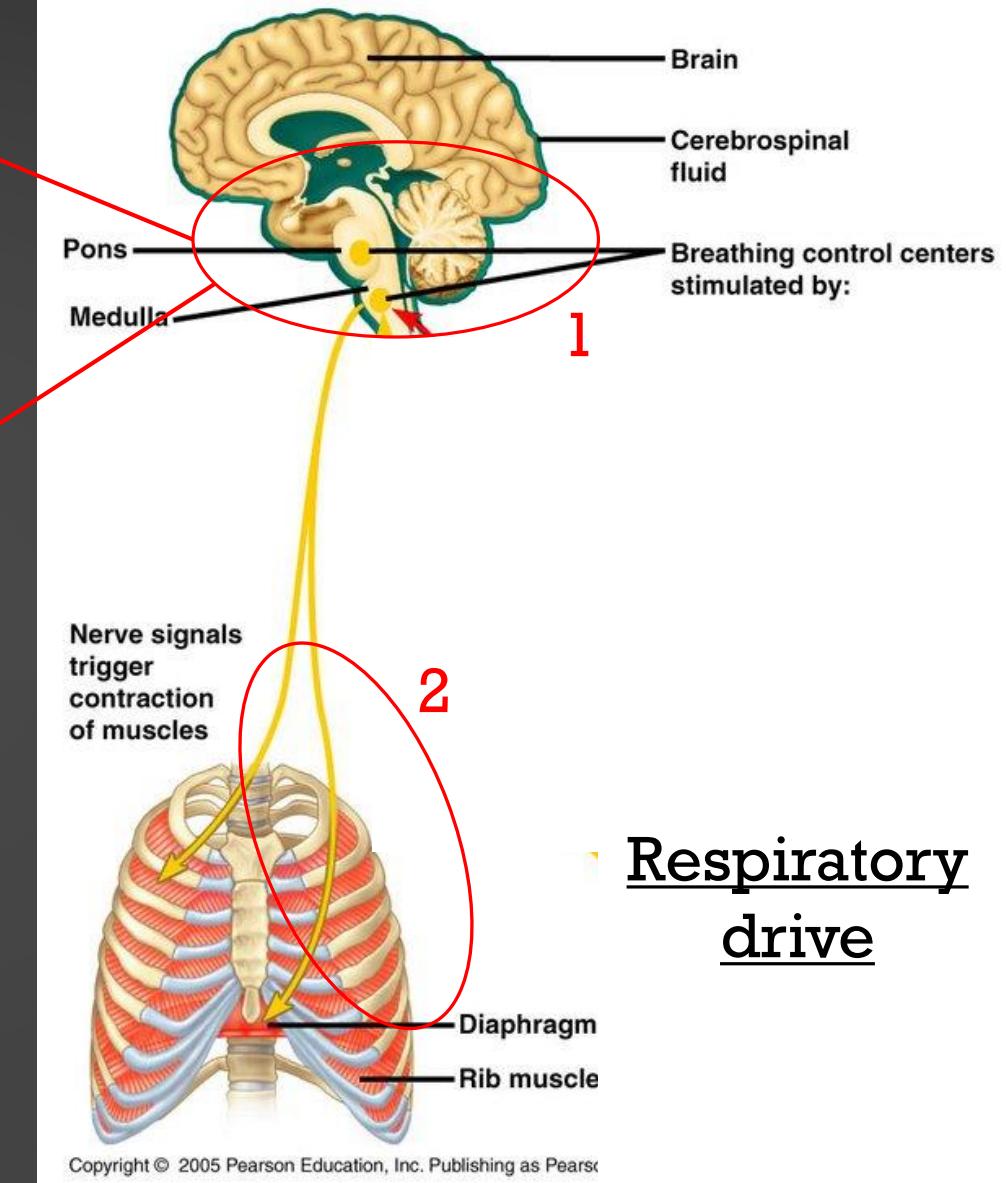
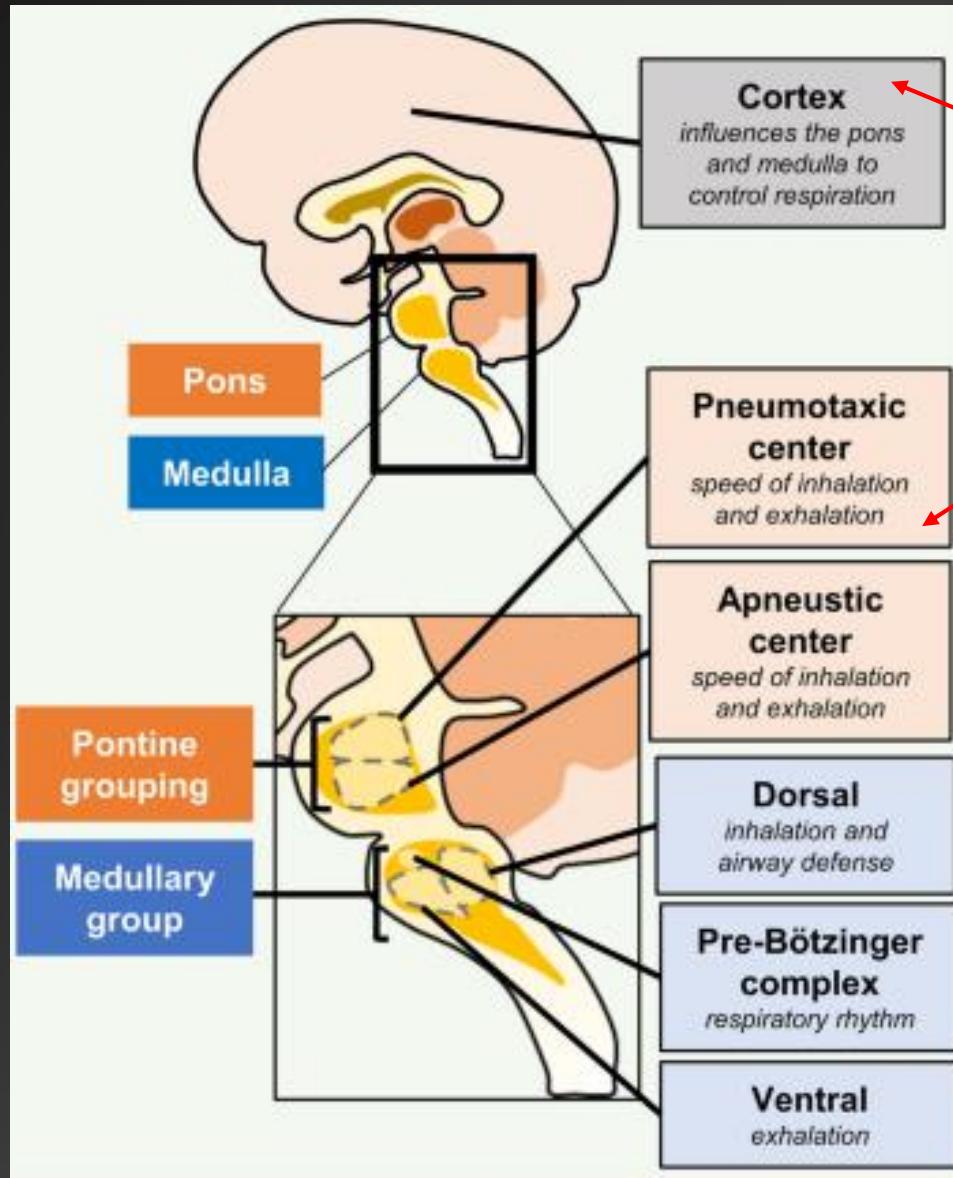
HYPERCAPNIC CSA

Won't breath

- Central Drive pathology
 - Chiari
 - Opioid
 - Degenerative
 - Genetic

Can't breath

- Motor neuron pathology
 - MSA
 - ALS
 - MG
 - Kyphoscoliosis



Respiratory drive

HYPERCAPNIC CSA

- Damage to
 - Cortex
 - Brainstem
 - Spinal cord
 - Muscles
 - Motor neurons
- Causes Hypoventilation

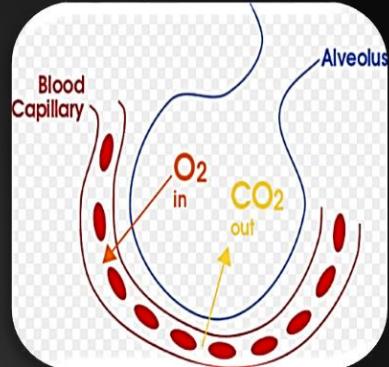


MINUTE VENTILATION

Minute ventilation = Tidal volume × Rate

Hypoventilation = ↓TV/ ↓Rate (Central apnea)

PCO₂/ APNEA

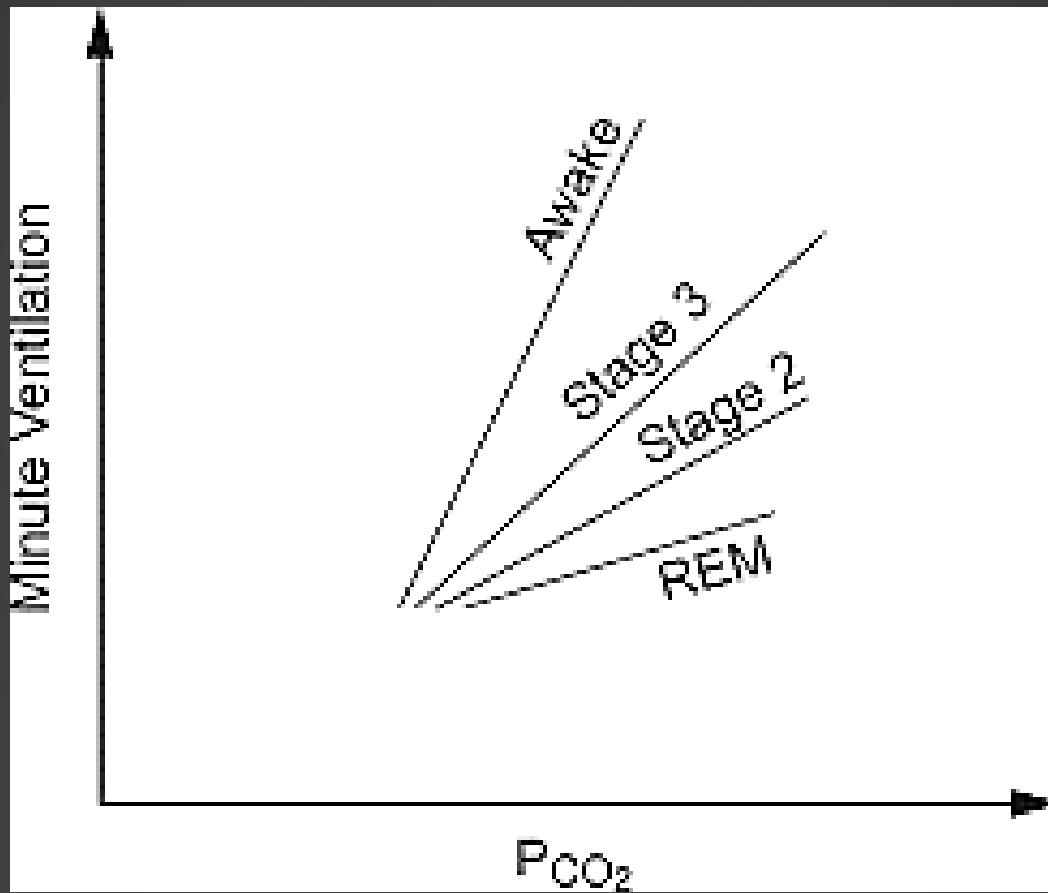


- Respiratory control system
 - tightly control PaCO₂ around 35-45 mmHg
 - Ventilation increase → Hypocapnia
 - Ventilation decrease → Hypercapnia

PRESENTATION OF HYPERCAPNIC CSA

- Almost always presents as
 - Chronic hypoventilation syndrome
- More pronounced during sleep
 - REM > NREM > Wake

VENTILATION IN WAKE VS SLEEP



HYPERCAPNIC CSA

- Clinical presentation
 - Hypercapnia
 - Hypoxemia
 - Right heart failure
 - Cyanosis
- +/_ Sleep related symptoms

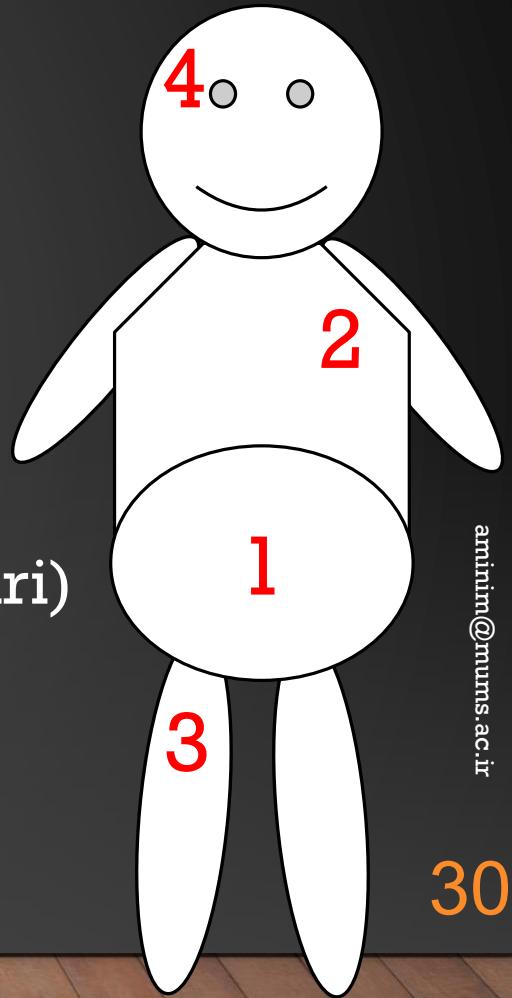
NOTE! CONSIDER CSA

- In every patient with Hypoventilation
- In every patient with Sleep complaints (even snoring)

APPROACH TO HYPOVENTILATION

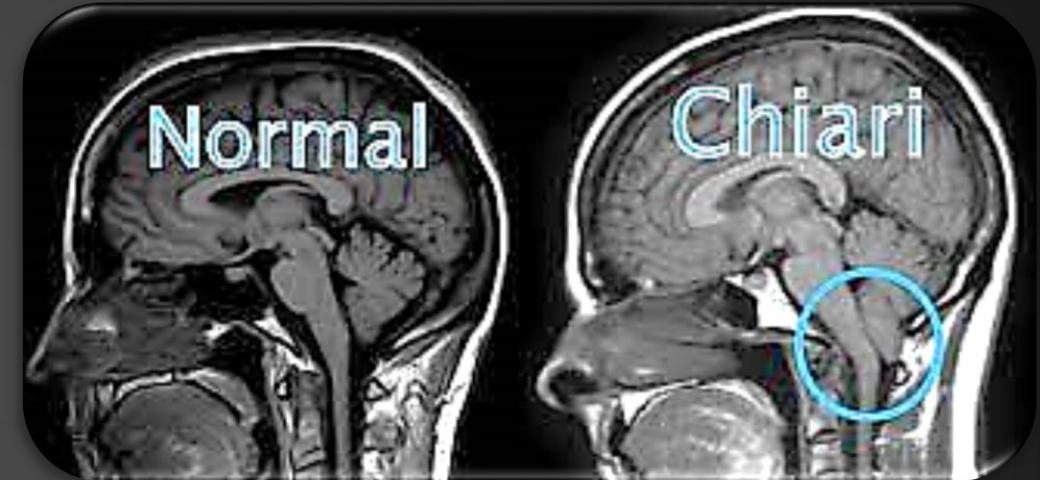
Hypoventilation = Daytime hypercapnia

1. Obesity hypoventilation syndrome
2. Thoracic (COPD, IPF, Kyphoscoliosis)
3. Neuromuscular disorders
4. Central drive (Opioid, Hypothyroidism, CCHS, Chiari)



CAUSES OF HYPERCAPNIC CSA

- Congenital
 - Chiari malformation
 - CCHS (Ondine curse)
- Acquired
 - Neurodegenerative
 - Leigh syndrome, Parkinson, MSA)
 - Tumors
 - CVA
 - Neuromuscular
 - Duchene, Myotonic dystrophy, MG, MS



TREATMENT OF HYPERCAPNIC CSA

- According to underlying cause
 - Tumor resection,
 - Oxygen with careful monitoring of PaCO₂,
 - Watchful waiting for stroke, ...
- NIV

TREATMENT OF HYPERCAPNIC CSA

- NIV
 - When above mentioned treatment ineffective
 - Goal: $\text{PaCO}_2 \text{ sleep} < 50$, $\text{PaCO}_2 \text{ wake} < 45$, $\text{O}_2\text{Sat} > 90$
- The best way is POLYSOMNOGRAPHY
PAP trial with transcutaneous CO_2 monitoring

Sleep lab



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NON HYPERCAPNIC CSA

CAUSES OF NON HYPERCAPNIC CSA

Shouldn't breath

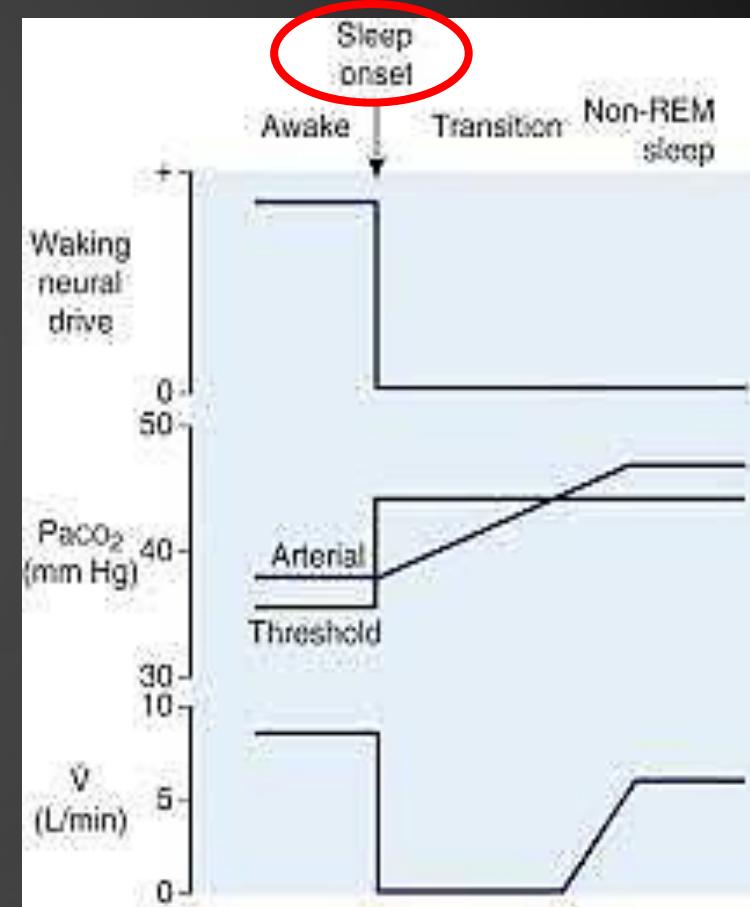
- Wake sleep transition
- Heart failure
- AF
- CVA
- Renal failure
- Acromegaly
- High altitude
- Opium
- TE-CSA
(Complex sleep apnea)

NON HYPERCAPNIC CSA

- Low or normal PaCO₂
- PaCO₂ falls below apneic threshold
- Characteristically associated with periodic breathing

APNEA THRESHOLD

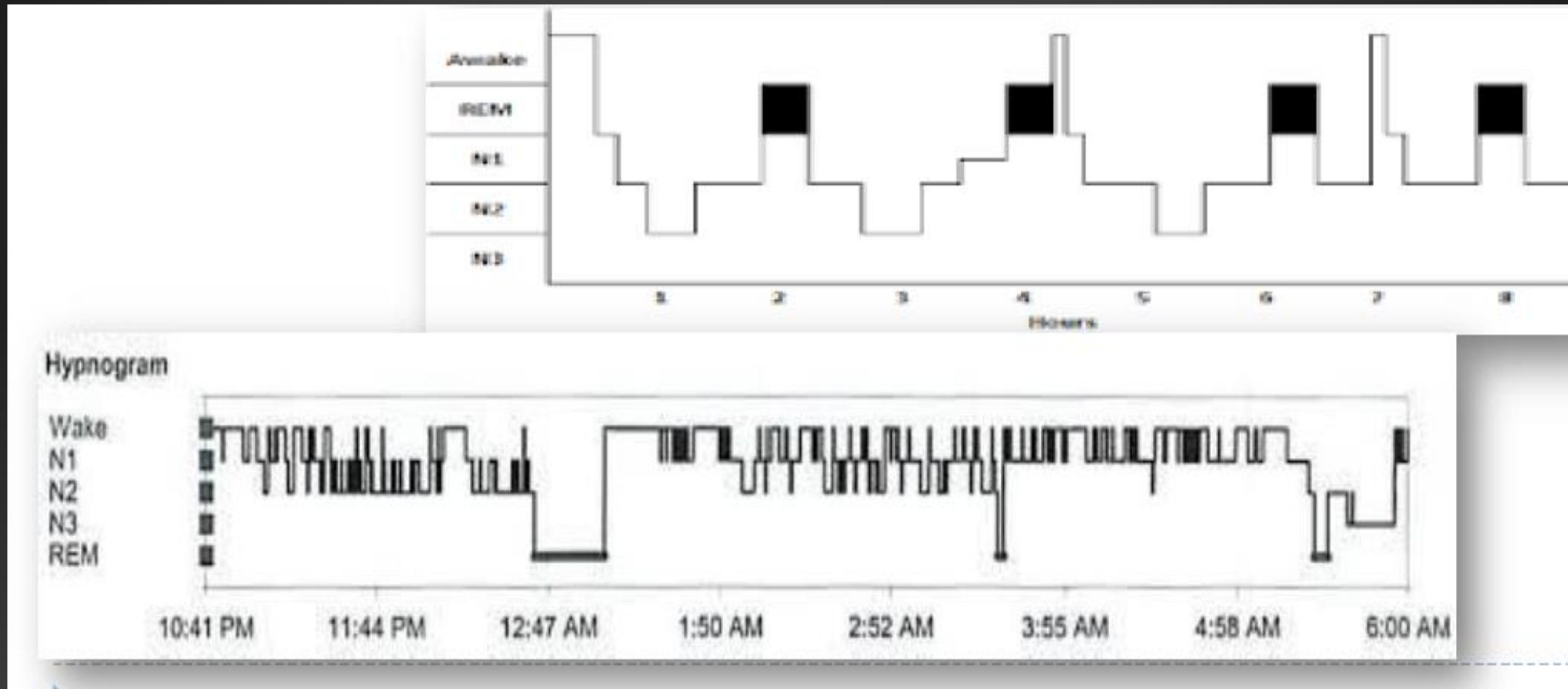
- Normally PaCO₂ is higher in SLEEP
 - by 3 to 5 mmHg



NON HYPERCAPNIC CSA

- Transient fluctuation/ Sleep state instability
 - Intact control system

SLEEP STATE INSTABILITY



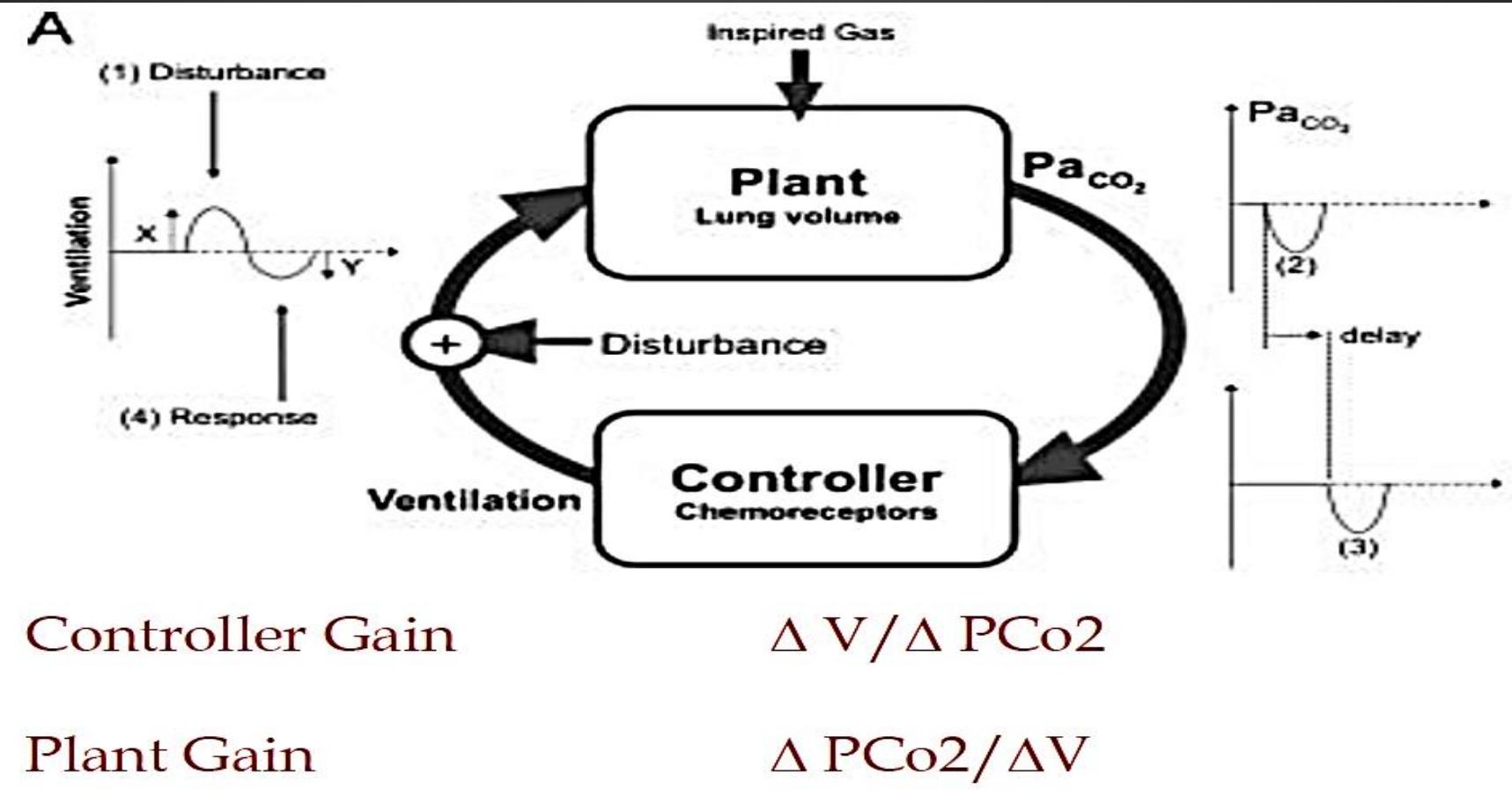
SLEEP STATE INSTABILITY

- Wake to constant sleep > 10 min
- Sleep efficiency <70%
- SWS > 1 Hz
- N1 of titration part >10%

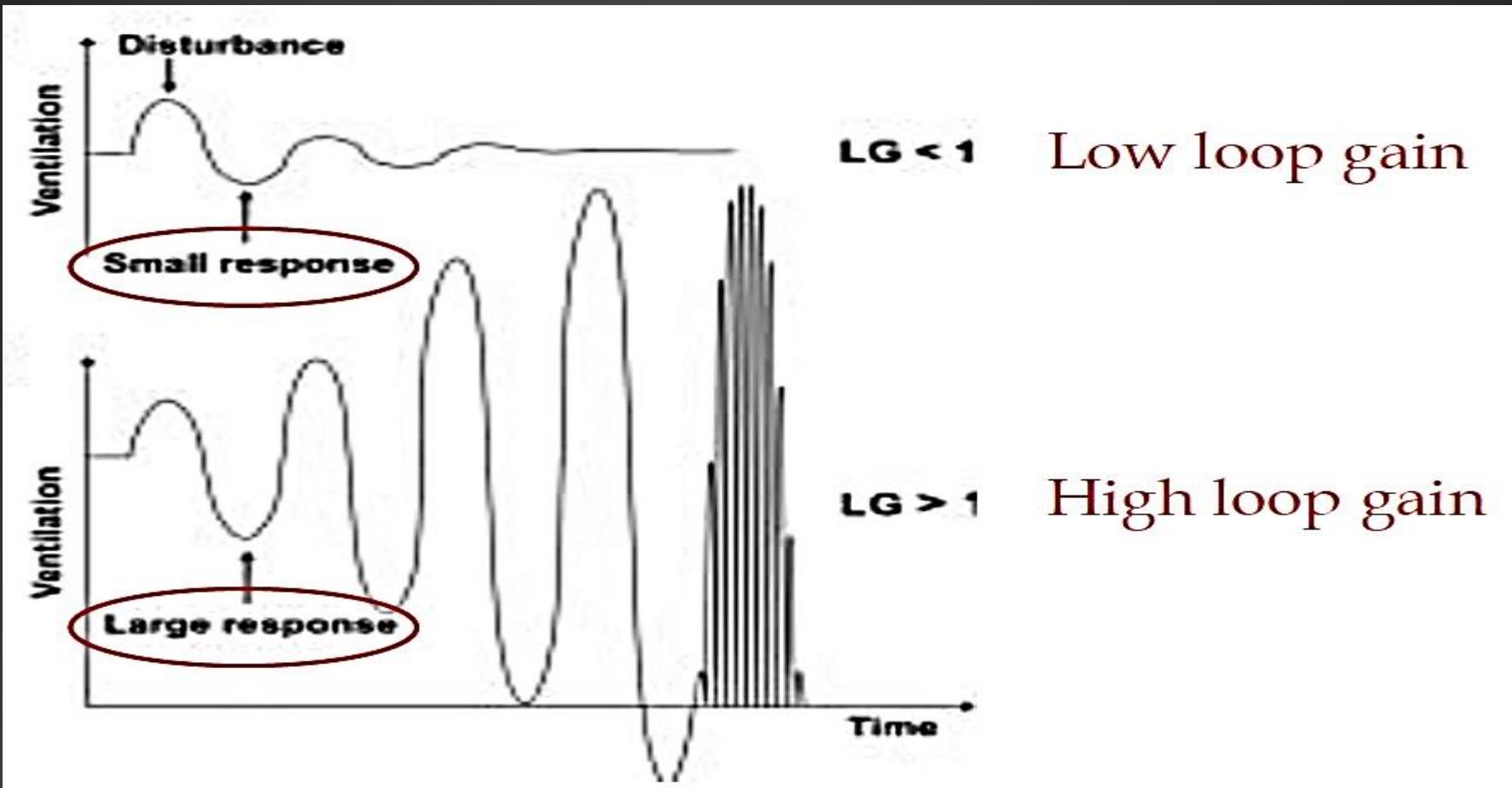
WAKE SLEEP TRANSITION

- The most common
- Stops if sleep firmly established
- Periodic breathing if repeated change between “wake” and “sleep”
- Loop gain
 - Plant gain
 - Controller gain

LOOP GAIN



LOOP GAIN



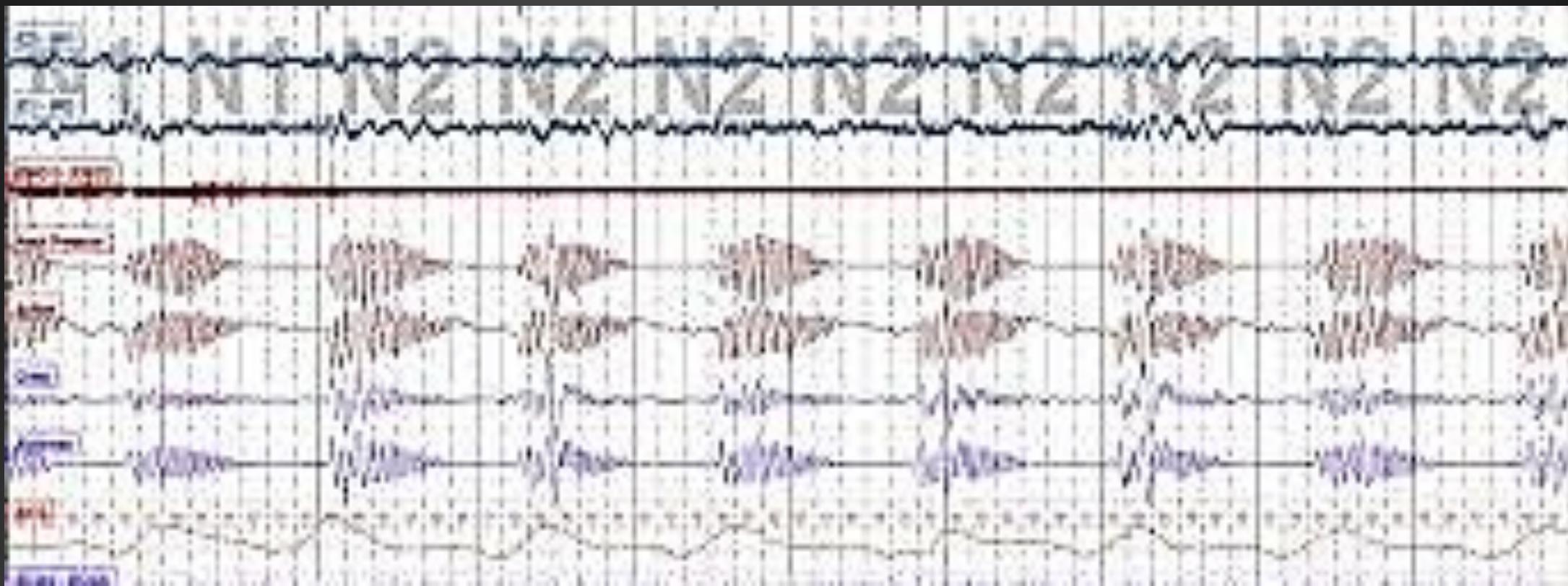
HEART FAILURE

- Periodic breathing
- Cheyne stokes Respiration/ Breathing
- CSR-CSA

CSR-CSA

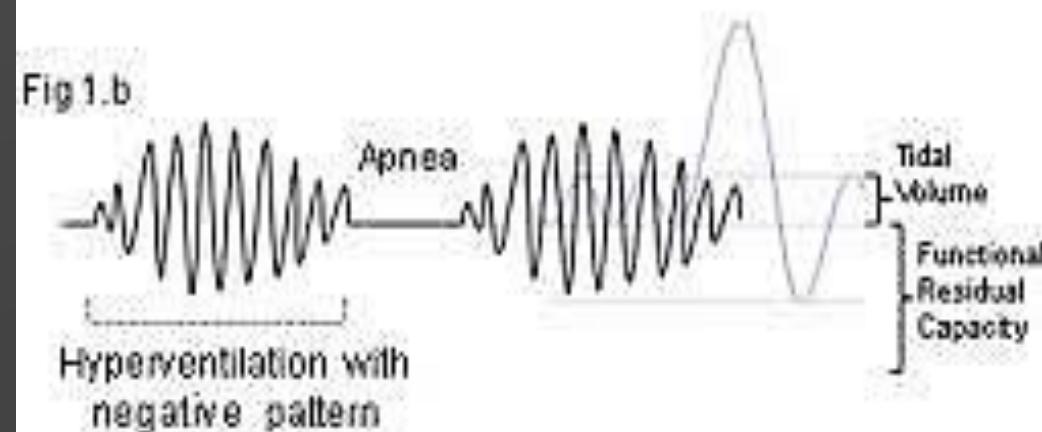
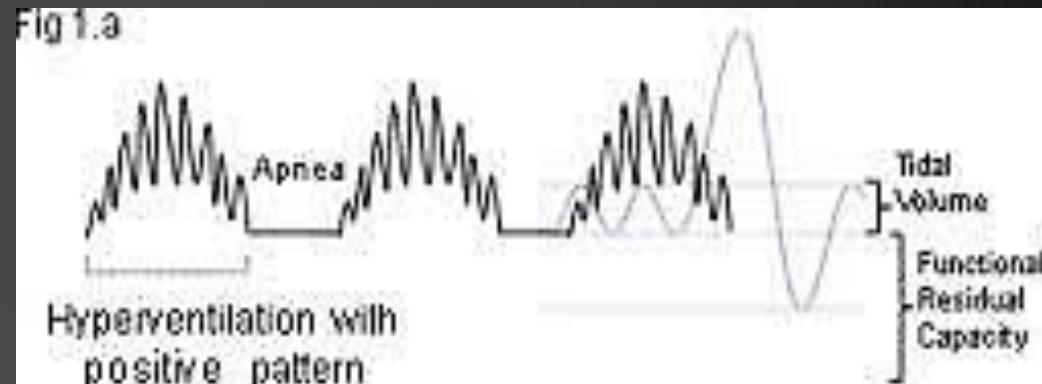
- Periodic/ Cyclic breathing
 - Crescendo- Decrescendo pattern
- Interspersed with central Apneas/ Hypopneas
 - Positive pattern
 - Negative pattern

CSR-CSA



CSR-CSA

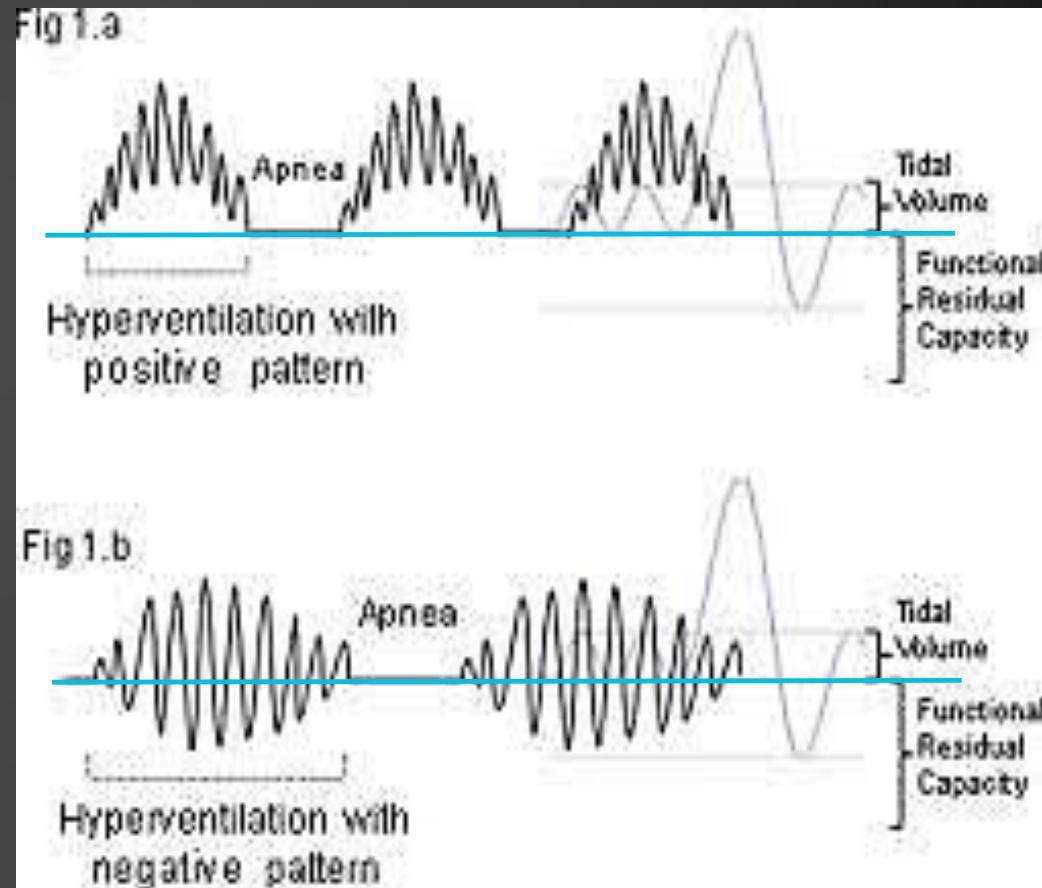
- Positive pattern
- Negative pattern



CSR-CSA

- Positive pattern
EELV remains at or above FRC

- Negative pattern
EELV falls below FRC



NEGATIVE PATTERN OF CSR-CSA

- longer hyperpnea
- Longer cycle durations
- Lower cardiac output
- Higher NT-proBNP
- Greater left ventricular wall tension

CVA

- 10-15% prevalence
 - No relation with vascular territory
- More relevant to left sided heart failure
 - Echo is recommended for CSR-CSA in a stroke patient

RENAL FAILURE

- Rostral fluid shift
- Treated with nocturnal dialysis

OPIOID INDUCED CSA

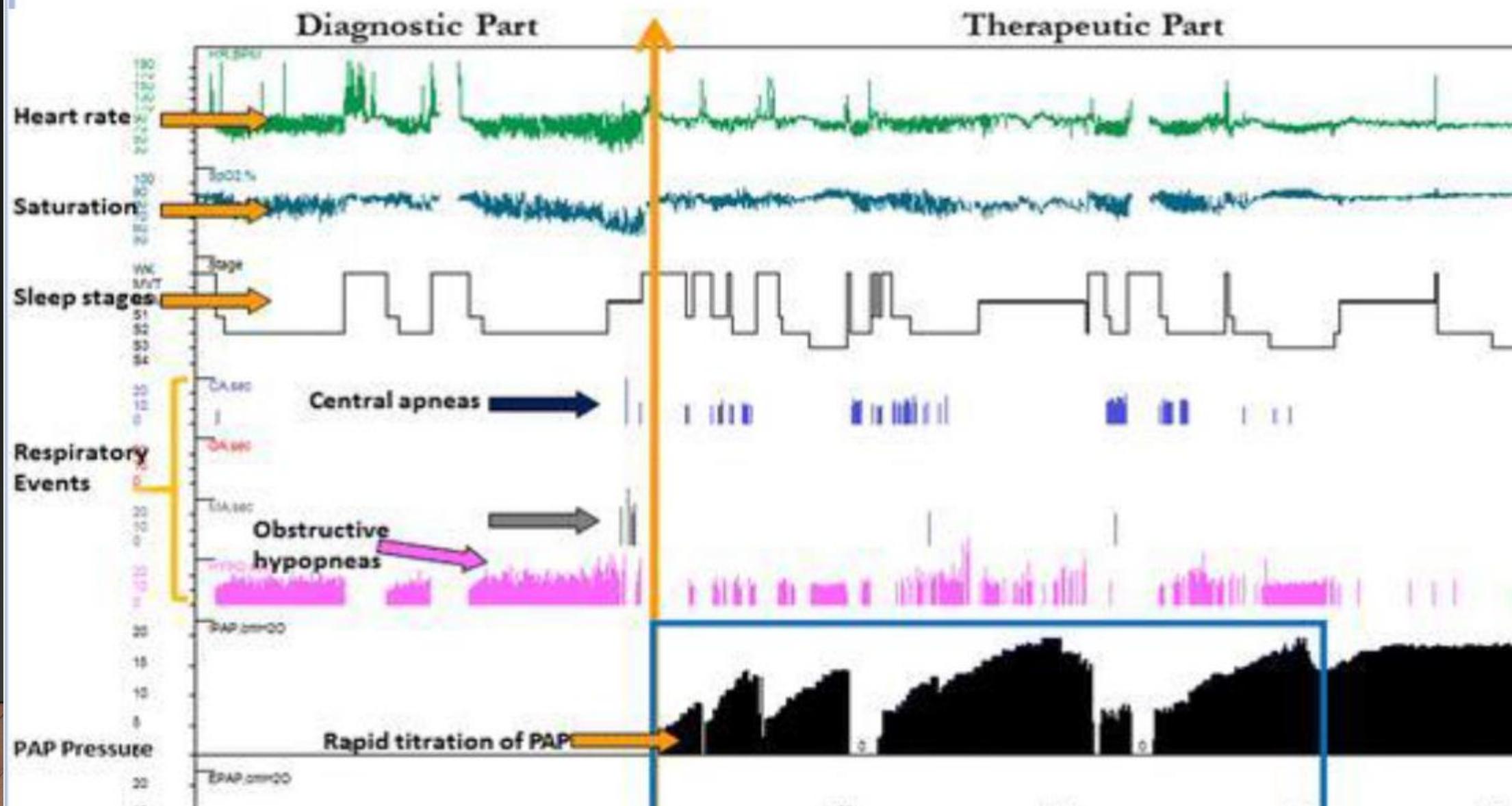
- Cheyne stokes respiration
- Central sleep apnea
- Obstructive sleep apnea
- Sleep related hypoventilation

TREATMENT EMERGENT CSA

- predominant OSA in diagnostic part
- Resolved with PAP/therapy
- CSA emerges then



TE-CSA ON SPLIT-NIGHT



TE-CSA IN HSAT

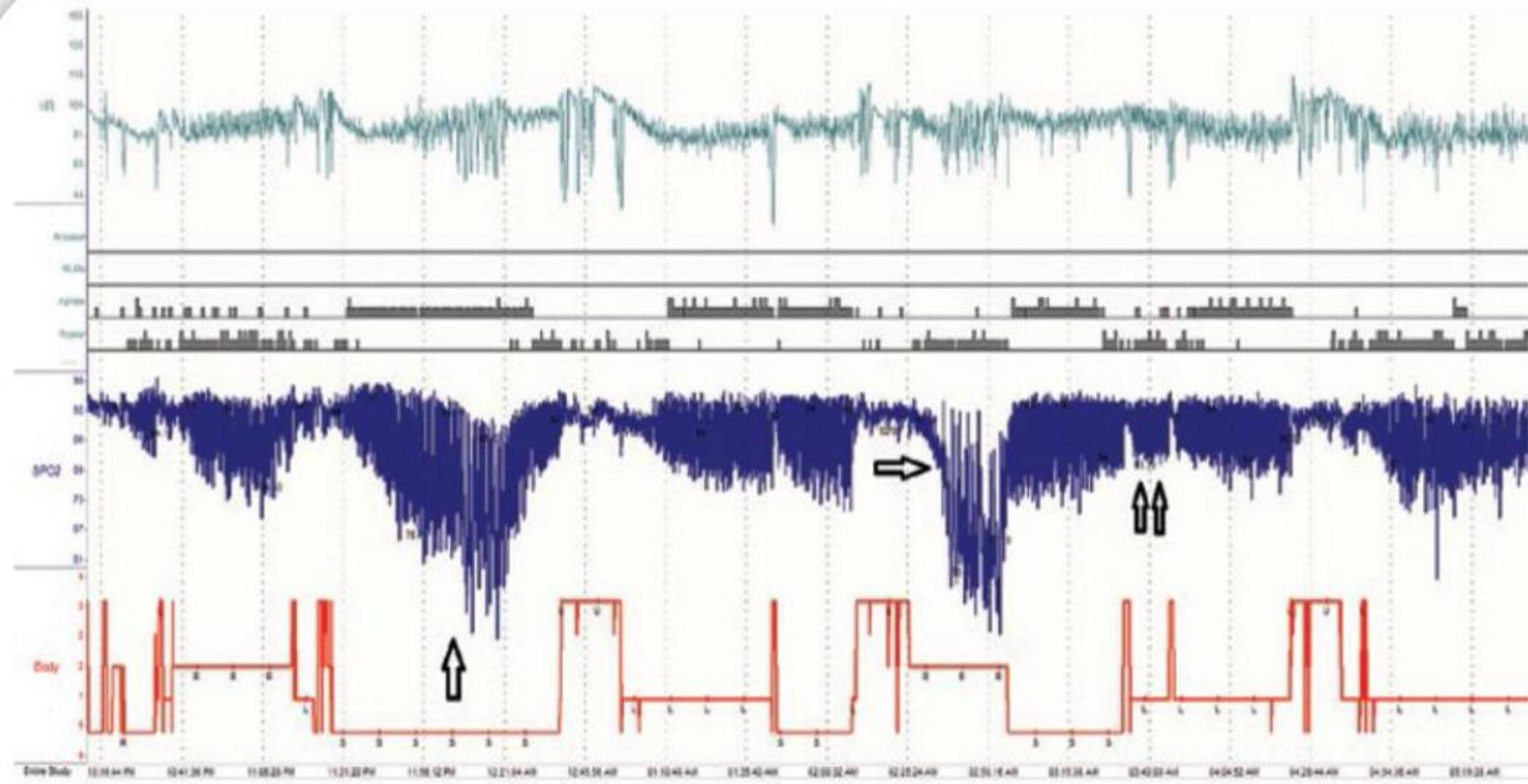


Table 132.4 Common Symptoms and Data Patterns That Support Adjustment

Physiology	Symptoms	Data	Adjustment
Residual REM-related events	Awakenings 2 h or later in the night Nocturia Short use around 2–3 h Intolerance often with difficulty tolerating pressure or feeling short of breath or not enough air in middle of night	Residual AHI often normal if only REM-related events 95%/max pressure often several cm/H ₂ O higher than median pressure Detailed data shows clustered events with REM timing Waveform data may not have classic obstructive flow limitation	Increase EPAP or EPAPmin to ~95% pressure
Treatment-emergent central apnea/periodic breathing	Residual daytime symptoms Short use Intolerance often with difficulty tolerating pressure at sleep onset	Residual AHI often elevated but may be <5 Residual events often have more central apneas than obstructive apneas, but most events may be hypopneas representing periodic breathing Detailed data may show events throughout night that stop with REM pattern or events at sleep onset Waveform data may show periodic breathing or recurrent central apnea pattern	Make sure no leak issues Change to fixed pressure Lower pressure unless patient is having treatment onset events and says feeling in need of more air Turn off EPR Lower PS or change BPAP to CPAP Consider titration with advanced PAP therapy or other treatments discussed in Chapter 125
High leak with auto-on/off	Patient says they have the machine on much longer than the data reports Complains of mask leak	Night summary data shows machine turning on and off repetitively. High mask leak	Mask fitting Lower maximum pressures
Comorbid hypoxia or hypoventilation	Suboptimal clinical response that could include morning headaches, fatigue, awakenings at night	Residual AHI often normal Overnight oximetry on treatment with low baseline oxygen saturations Elevated bicarbonate levels ≥27 or elevated ABG PCO ₂ or home TCco ₂ monitoring despite treatment	Titration study using TCco ₂ monitoring If already on BPAP or volume-assured pressure support, increase pressure support or goal volumes Consider supplemental oxygen after hypoventilation is treated

TAKE HOME MESSAGE

- Consider CSA in
 - Hypoventilation syndromes
 - Sleep complains especially insomnia
- Notice to Hypopnea scoring
especially in titration part of the study
- Approach to Drive and PaCo₂ can be helpful

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THANK YOU