Bridging Guidelines and Practice: CSA Management in Today's Sleep Labs

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CSA
Common Causes and Treatments

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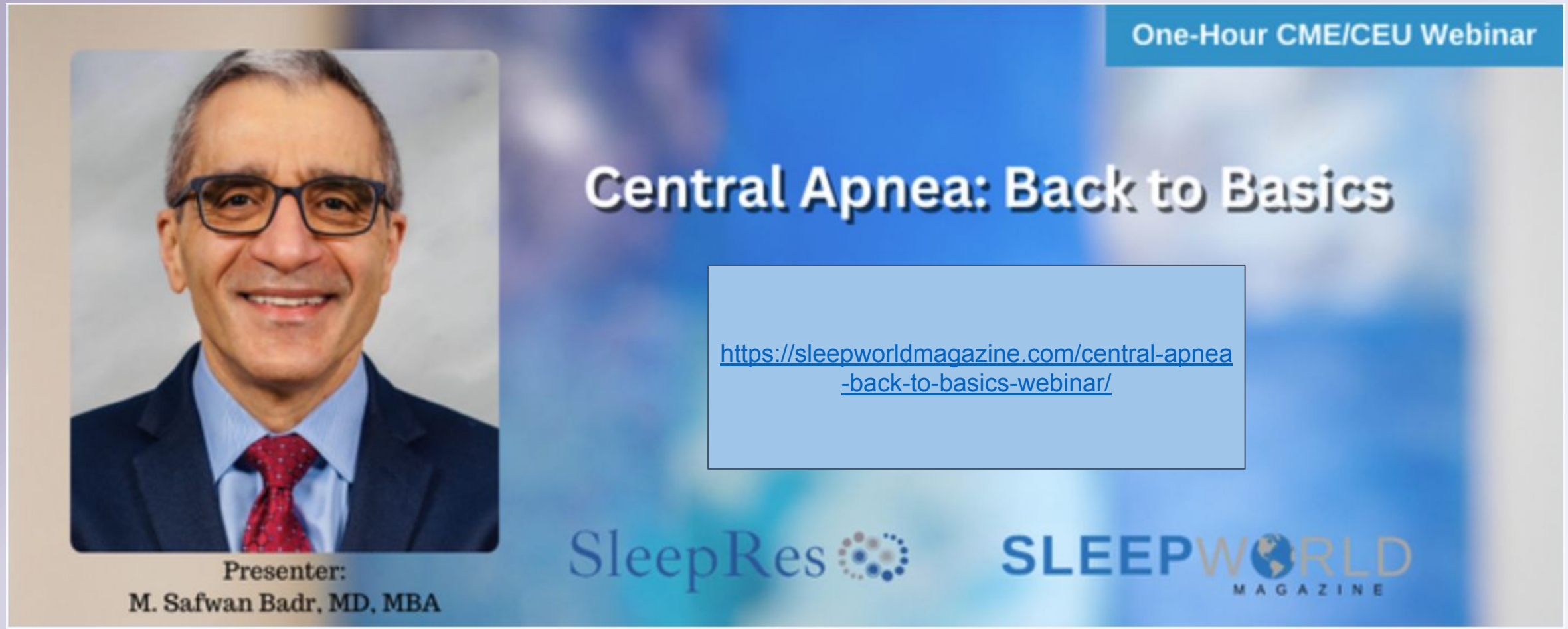
Central Sleep Apnea (CSA): Clinical Foundations

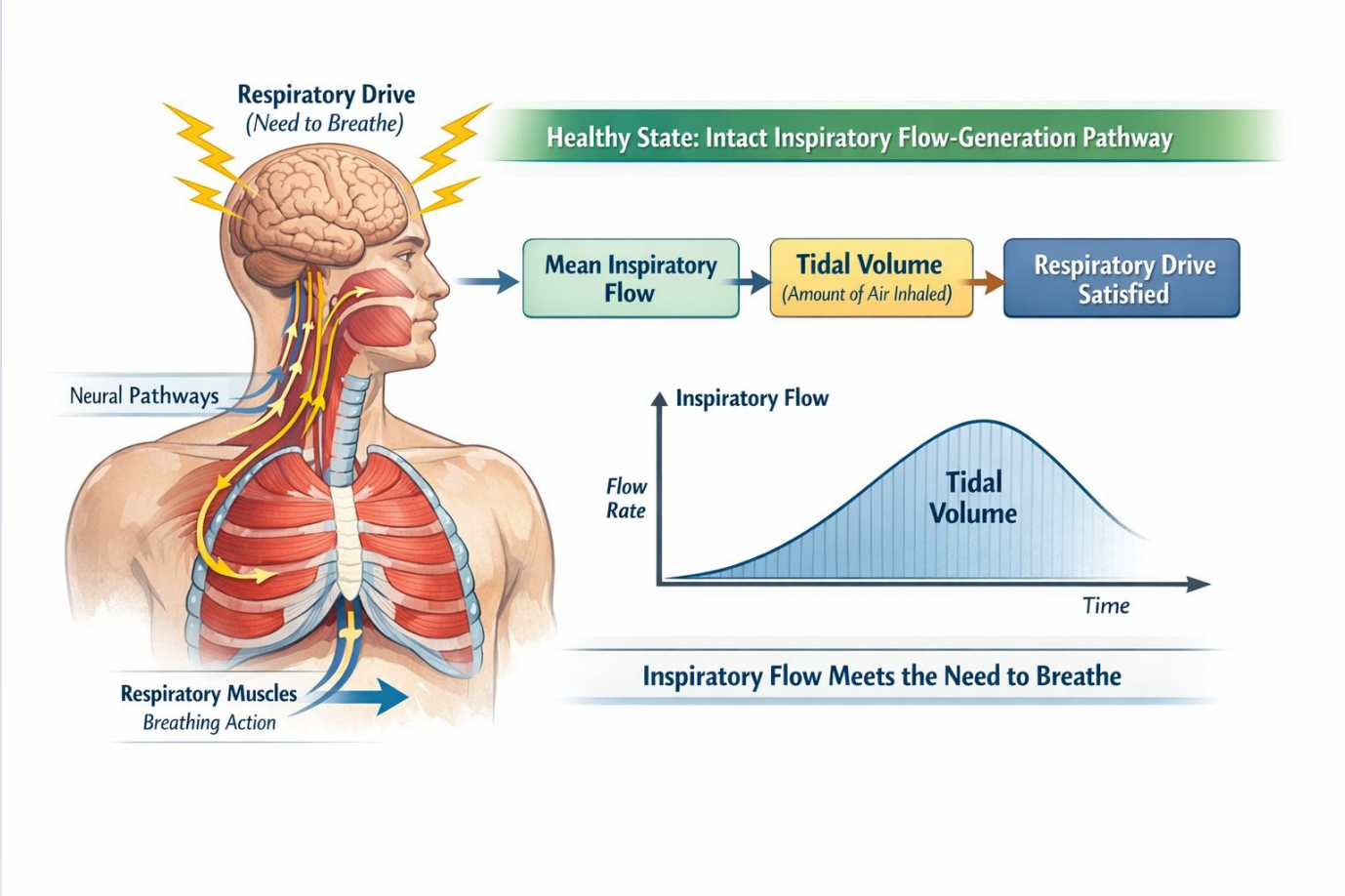
- What is CSA?
- Common etiologies and risk factors
- Evidence-based treatment options and emerging therapies



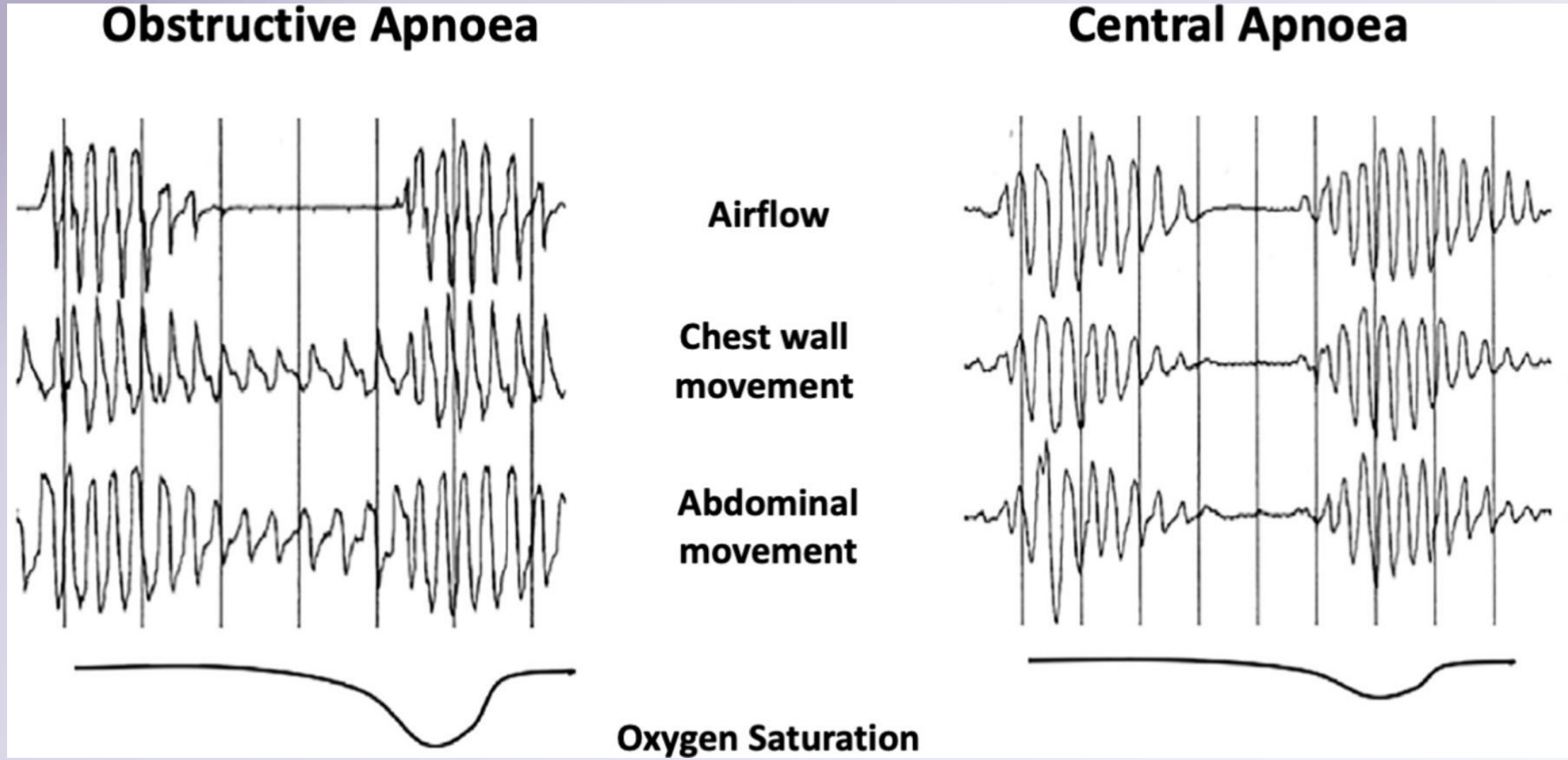
Central Apnea: Back to Basics

Respiratory Drive: ‘Normal’ Breathing’

- Respiratory drive (need to breathe) travels through neural pathways to respiratory muscles, which execute the breathing process and generate inspiratory flow (inspiratory flow-generation pathway).
- In a healthy state, inspiratory flow-generation pathway is intact, and thus respiratory drive is satisfied by the rate of volume increase, expressed by mean inspiratory flow, which in turn determines tidal volume

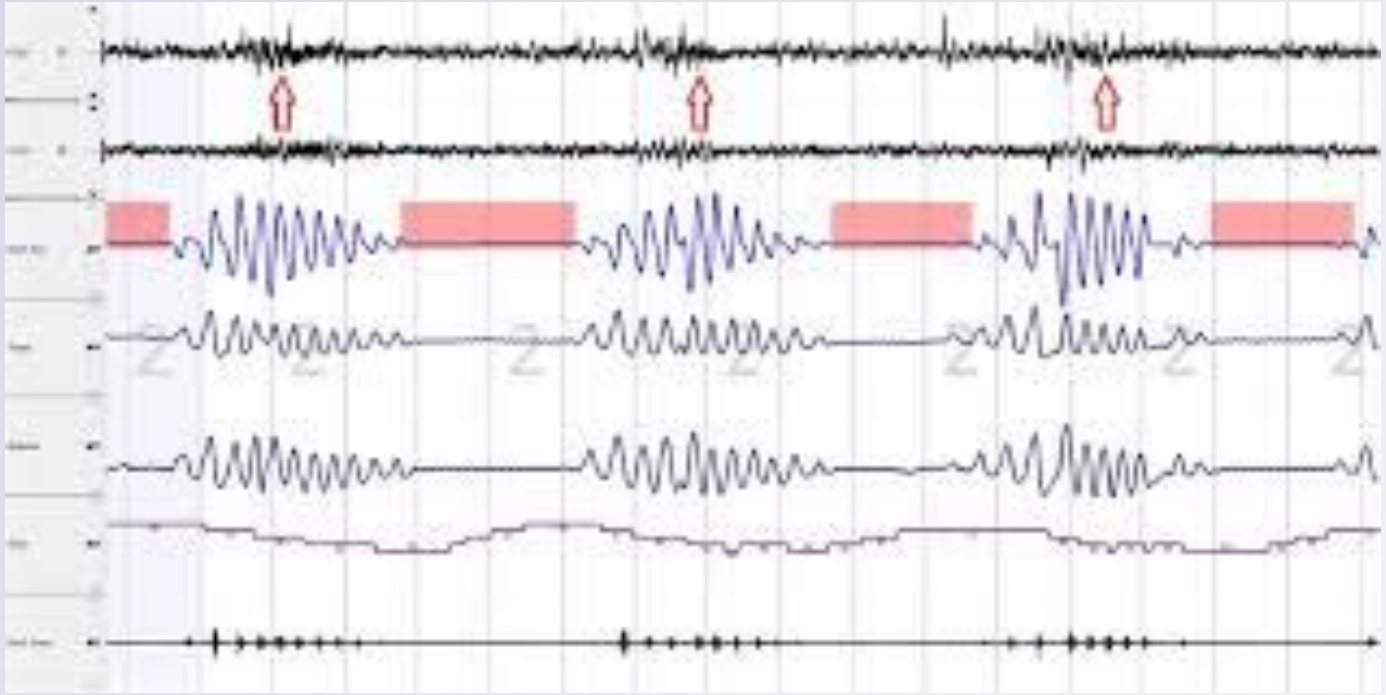


Central Sleep Apnea (CSA): Clinical Foundations



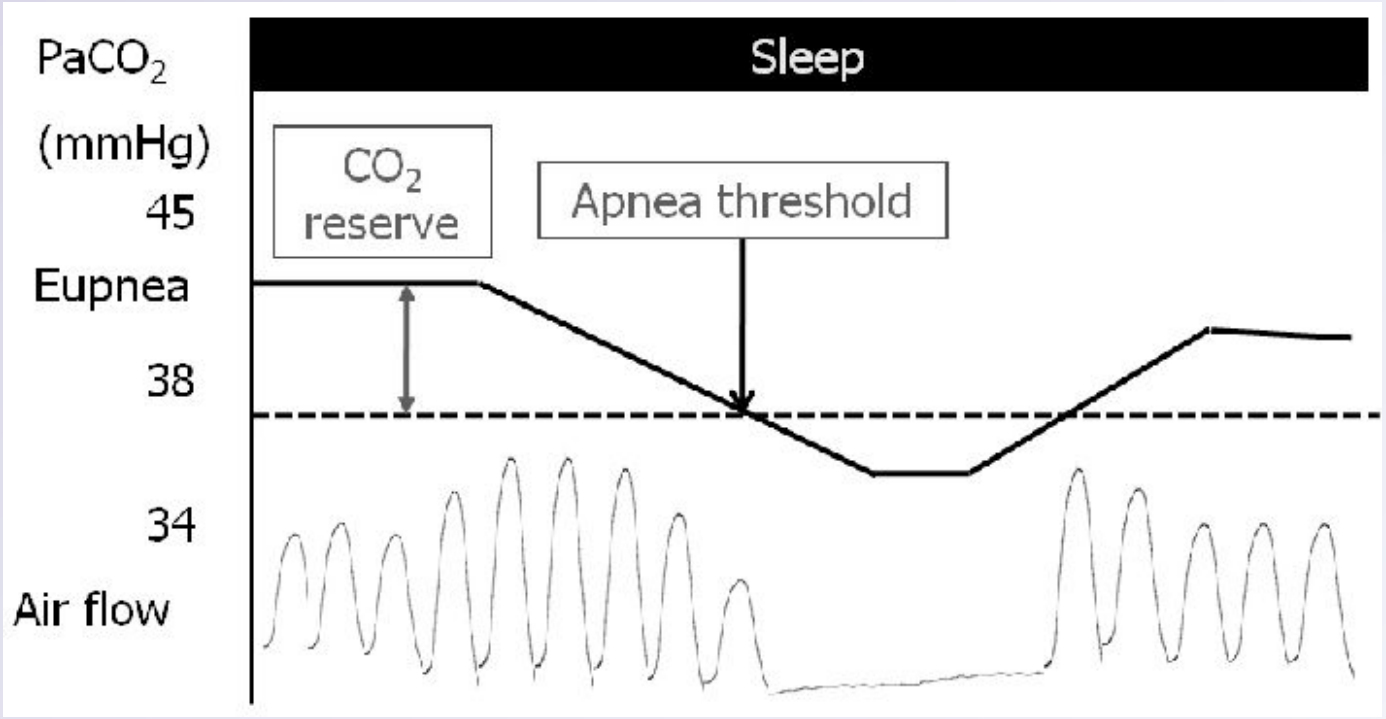
Central Sleep Apnea (CSA): Clinical Foundations

- The vast majority of CSA is post-hyperventilation hypocapnia.
  - Hypocapnia - a state of reduced carbon dioxide in the blood, usually resulting from deep or rapid breathing (hyperventilation).
  - Can also be hypoventilation (breathing too slow or shallow)



Central Sleep Apnea (CSA): Clinical Foundations

- Occurs most often in NREM
- The removal of the wakefulness “drive to breathe” during NREM sleep renders respiration critically dependent on chemical influences, especially PCO2.
- NREM sleep unmasks the apneic threshold, a phenomenon that is sleep state-dependent
- Central sleep apnea occurs if arterial PCO2 drops below a highly sensitive “apneic threshold”



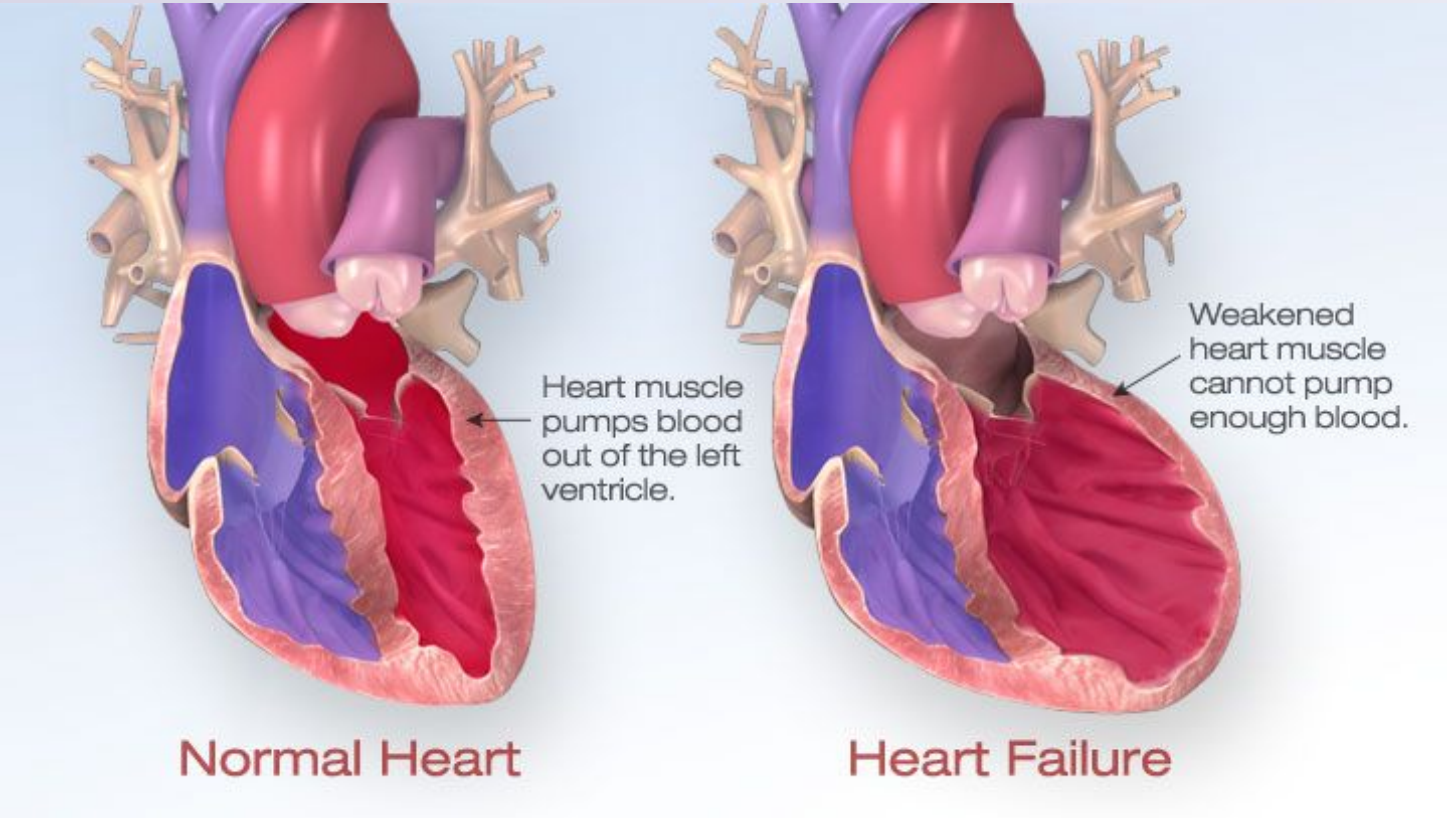
Common Etiologies of CSA

- **Heart failure (Cheyne–Stokes respiration)**
  - 51% of male patients with heart failure had sleep disordered breathing.
    - central apnea (40%) and the remainder (11%) had obstructive apnea
- **Stroke or brain stem disease**
  - 40% of the patients demonstrate central apnea after a CVA
  - the brain injury disrupts the neural pathways responsible for controlling automatic breathing during sleep

- **Chronic opioid or sedative use**
  - suppressing the central nervous system, which reduces the body's responsiveness to carbon dioxide and slows the respiratory drive.
- **High-altitude exposure**
  - at altitudes above 5,000–6,000 feet, low oxygen forces rapid breathing (hyperventilation), which reduces carbon dioxide in the blood too much
- **Idiopathic CSA**
- **Treatment Emergent Central Sleep Apnea (TESCA)**

Key Risk Factors

- **Reduced LVEF (left ventricular ejection fraction)**
  - Reduced LVEF slows circulation and destabilizes CO_2 feedback to the brain, leading to over- and under-ventilation cycles that trigger central apneas (often seen as Cheyne–Stokes respiration)
  - An ejection fraction (EF) of x% means your heart's main pumping chamber (left ventricle) is pumping out x% of the total blood volume inside it with each beat



Key Risk Factors

- **Advanced age**
  - 1.7% in the middle-aged group vs. 12.1% in the older adults group
  - Older adults have a narrower gap between their normal, resting CO2 levels and the threshold at which the brain triggers breathing, making them more prone to apnea when CO2 drops slightly.
- **Male sex**
  - Prevalence of central apnea in women of 0.3%, compared with 7.8% in men
  - Hormonal differences - Testosterone is linked to higher loop gain (a unstable respiratory control system) and can elevate the apneic threshold, meaning men require less of a drop in CO2 to stop breathing during sleep.
  - More likely to have heart failure

Evidence-Based Treatment Options

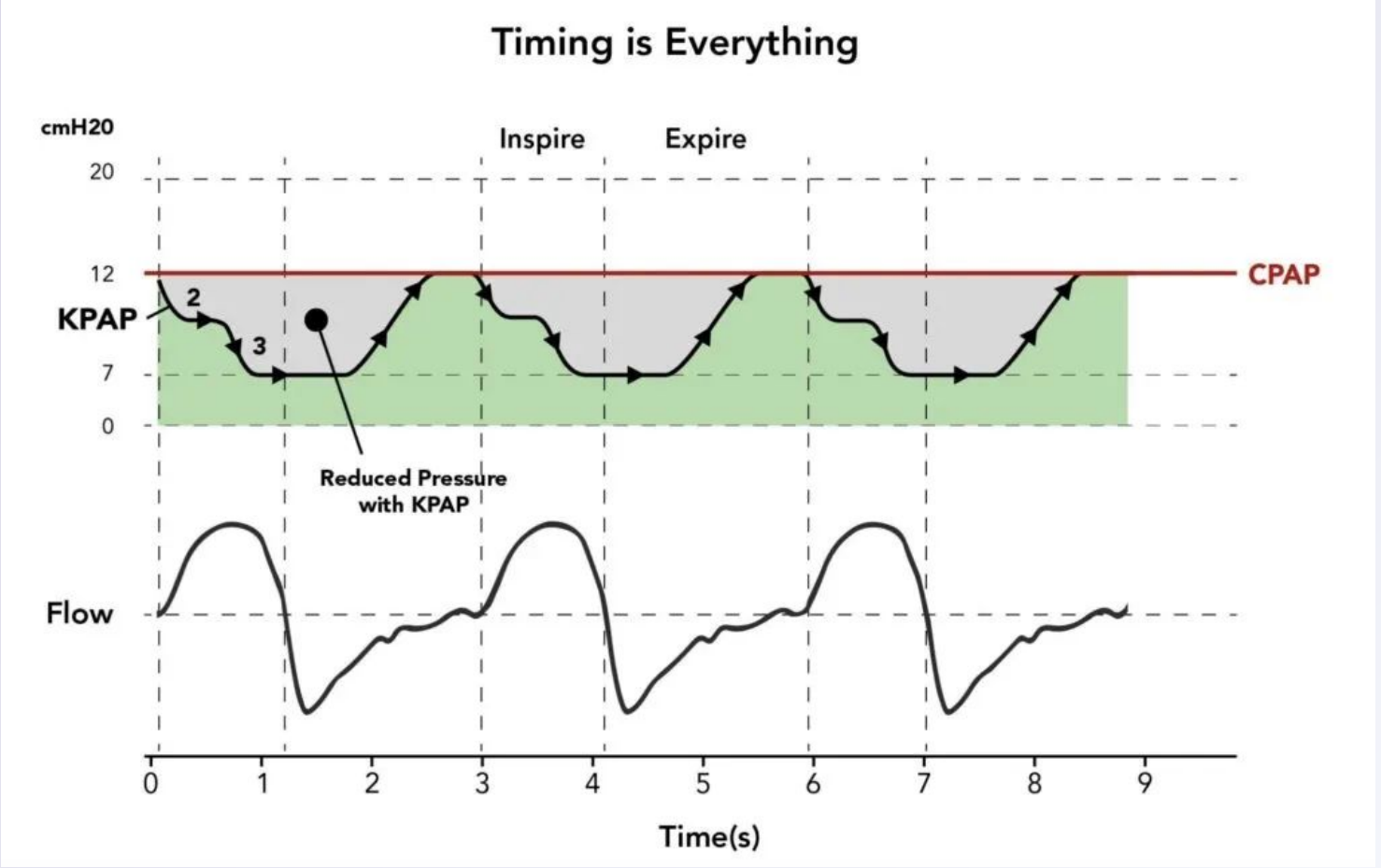
- **Positive Airway Pressure**
  - **CPAP** - estimated to work in 50% of patients
    - preventing pharyngeal narrowing during central apnea and dampening the ensuing ventilatory overshoot
    - co-occurrence of central and obstructive apnea
  - **B-PAP with backup rate** (not to be used without backup rate)
    - effective in restoring alveolar ventilation during sleep
    - Clinical indications include nocturnal ventilatory failure and central apnea secondary to hypoventilation
  - **ASV (with guidelines)** - 2016 recommended against after SERVE-HF
    - provides varying amount of ventilatory support, against a background of positive end expiratory pressure (EPAP)
    - Contrary to bi-level, pressure support devices, changes in respiratory effort results in reciprocal anticyclical changes in the magnitude of ventilatory support.

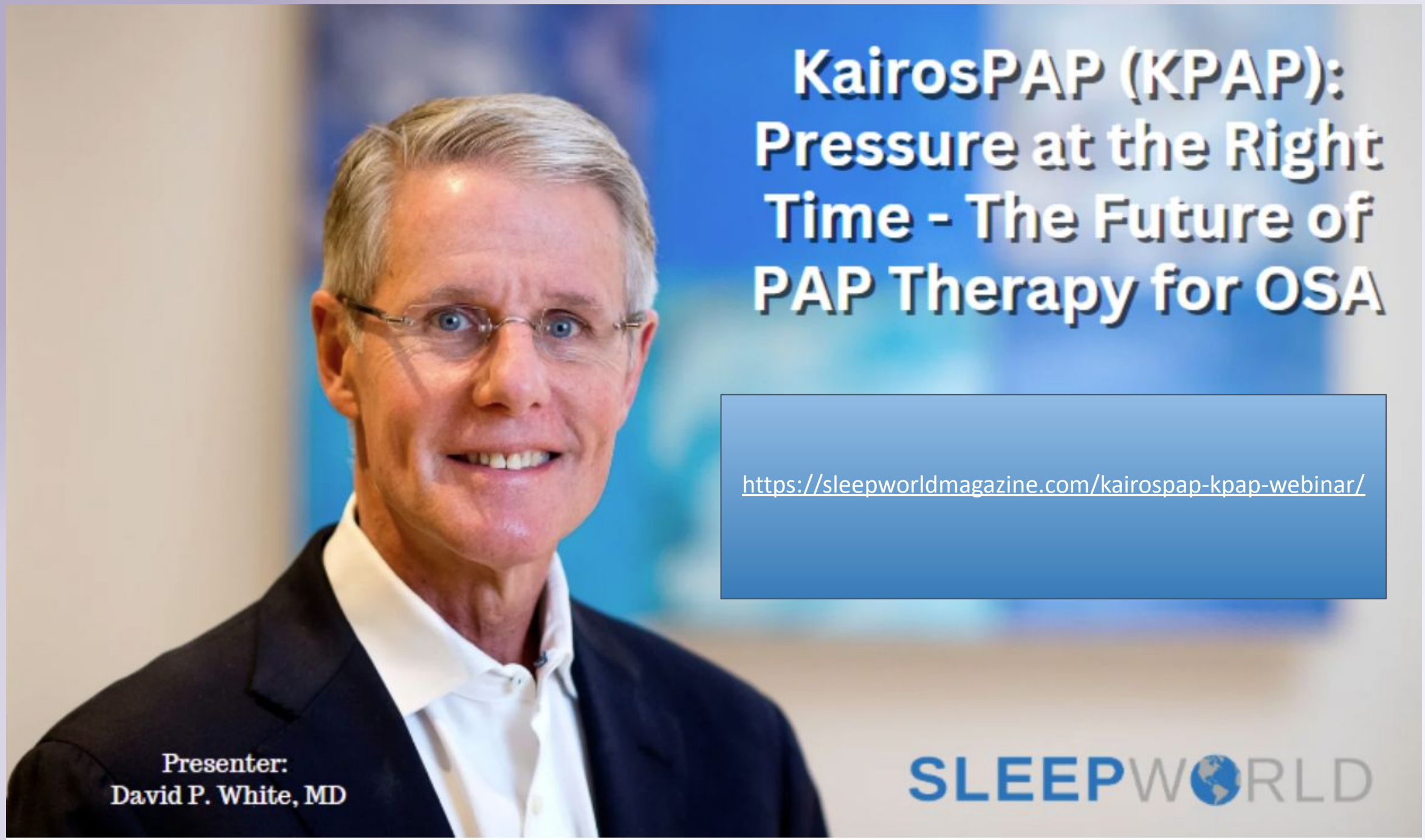
Evidence-Based Treatment Options - Emerging

- **Supplemental oxygen**
  - mitigates the magnitude of hypoxemia and dampens the magnitude of post-apneic ventilatory overshoot
  - most likely explanation is increased cerebral PCO2 by the displacement of carbon dioxide from hemoglobin by the increased oxygen level (Haldane effect)
- **Acetazolamide**
  - a weak diuretic that causes mild metabolic acidosis has been shown in several studies to decrease the severity of central apnea
- **Transvenous Phrenic Nerve Stimulation** - (New in guidelines)
  - Implantable, pacemaker-like device that monitors breathing and stimulates the phrenic nerve to activate the diaphragm, bypassing the brain's failure to send breathing signals.

Evidence-Based Treatment Options - Emerging

- **Kairos Positive Airway Pressure (KPAP)**
  - Variable pressure, reducing inspiratory pressure while maintaining expiratory pressure.
  - Substantially reduced airway pressure during both inspiration and much of expiration, only returning pressure to the optimal treatment level towards the end of expiration. Could help TESCA.




KairosPAP (KPAP): Pressure at the Right Time - The Future of PAP Therapy for OSA
https://sleepworldmagazine.com/kairospap-kpap-webinar/
Presenter:
David P. White, MD
SLEEPWORLD

Why CSA Matters: Patient Outcomes

- Increased morbidity and mortality
- Reduced sleep quality and daytime function
- Higher cardiovascular risk
- Early identification improves outcomes

Why CSA Matters: Sleep Lab Impact

- Accurate event identification
- Appropriate titration strategies
- Guideline-aligned protocols
- Stronger interdisciplinary communication