Central Sleep Apnea (CSA)

Full Educational Lecture Script

Good evening.

Today we will discuss **Central Sleep Apnea**, a disorder fundamentally different from obstructive sleep apnea.

This condition is not caused by airway blockage —it is caused by a **failure of the brain to initiate breathing.**

Definition

Central Sleep Apnea

Central Sleep Apnea is characterized by:

- Repeated pauses in breathing during sleep
- **Absence of respiratory effort**
- Reduced or absent airflow

The key feature is:

No effort to breathe

How It Differs from Obstructive Sleep Apnea

Obstructive Sleep Apnea

Central Sleep Apnea:

- No respiratory effort
- Brain does not send signal to breathe

Obstructive Sleep Apnea:

- Respiratory effort present
- Airway is physically blocked

This distinction is critical.

Control of Breathing

Breathing is regulated by:

- Brainstem respiratory centers
- Chemoreceptors detecting CO_2 and oxygen

The primary driver of breathing is:

Carbon dioxide (CO_2)

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What Goes Wrong in CSA

In CSA:

- The brain becomes unstable in regulating breathing
- CO_2 levels fluctuate
- The signal to breathe temporarily stops

This results in:

Central apneas

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Types of Central Sleep Apnea

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1. Primary (Idiopathic) CSA

- No clear cause
- Rare

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2. Cheyne-Stokes Respiration

Common in:

Heart failure

Pattern:

- Cyclic breathing
- Gradual increase and decrease in breathing effort
- Followed by central apnea

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3. Treatment-Emergent CSA

Occurs when:

- CPAP is initiated for OSA
- Central events appear

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4. CSA Due to Medical Conditions

Associated with:

- Stroke
- Brainstem lesions
- Renal failure

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5. Drug-Induced CSA

Commonly caused by:

Opioids

These suppress respiratory drive.

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Cheyne-Stokes Breathing (High Yield)

This pattern is:

- Crescendo–decrescendo breathing
- Followed by apnea

Mechanism:

- Delayed circulation time
- Instability in CO_2 feedback

Highly associated with heart failure.

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Pathophysiology

Key mechanisms include:

- Instability of respiratory control system
- Hypersensitivity to CO_2 changes
- Delayed feedback loop

This creates a cycle of:

Overbreathing → CO_2 drops → apnea → CO_2 rises → breathing resumes

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Symptoms

- Patients may report:
- Fragmented sleep
- Frequent awakenings
- Daytime fatigue
- Insomnia

Bed partners may notice:

- Periods of no breathing
- Irregular breathing patterns

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Physiological Effects

Each apnea leads to:

- Oxygen desaturation
- Arousals
- Sympathetic activation

This stresses the cardiovascular system.

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Diagnosis

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Polysomnography (PSG)

Findings:

- Apneas without respiratory effort
- Reduced airflow
- Oxygen desaturation

Key measurement:

Apnea-Hypopnea Index (AHI)

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When to Suspect CSA

- Known heart failure

- Stroke history
- Opioid use
- Persistent apneas despite CPAP

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Treatment

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Treat Underlying Cause

- Optimize heart failure
- Reduce opioids
- Manage neurological conditions

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Positive Airway Pressure

- CPAP (in some cases)
- BiPAP (with backup rate)

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Advanced Therapy

Adaptive Servo-Ventilation

ASV:

- Adjusts pressure dynamically
- Stabilizes breathing pattern

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Supplemental Oxygen

May help:

- Stabilize oxygen levels
- Reduce central events

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Important Clinical Consideration

ASV is **contraindicated in certain heart failure patients**.

This is a high-yield exam point.

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Prognosis

Depends on:

- Underlying condition
- Severity of CSA

Treating the cause improves outcomes.

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Key Clinical Insight

Central Sleep Apnea is a disorder of: **control, not obstruction**

The airway is open —but the brain fails to signal breathing.

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Summary

Central Sleep Apnea is characterized by:

- Absence of respiratory effort
- Instability in breathing control
- Association with medical conditions

Management focuses on:

- Treating underlying causes
- Stabilizing breathing

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Final Message

- Breathing during sleep is not automatic in all patients.
- When the brain fails to regulate breathing properly, serious consequences can occur.
- Recognizing Central Sleep Apnea is essential for proper diagnosis and treatment.

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