

08/23/2026

The Question: Please describe the following in detail:

1. What are the three types of sleep apnea and how are each treated
2. What are the demographics related to sleep apnea i.e. how many people have it and how many are left undiagnosed and once diagnosed that are then left untreated
3. What are the methods of obtaining a diagnosis
4. What are the dangers of having untreated and/or undiagnosed sleep apnea and what are the comorbidities associated with it
5. Describe the following treatment options along with the advantages and disadvantages of each
 - a. CPAP (auto-PAP, BiPap, ASV devices)
 - b. Mandibular Advancement Devices more commonly called Oral Appliance Therapy
 - c. Various surgical procedures including the Inspire Implant system and weight loss surgeries
 - d. Weight loss itself as related to the GLP-1 medications
 - e. Adjunctive therapies such as Positional Therapy and/or Nocturnal Supplemental Oxygen and their indications
6. When planning and managing a case why the following is critical with respect to obtaining a favorable outcome
 - a. Open and transparent Medical-Dental collaboration
 - b. Why physicians that are credentialed-boarded in sleep are critical with respect to outcome
 - c. Knowing that Sleep Disturbed Breathing is a chronic condition that needs “chronic care monitoring”, what and how are the best methods of insuring that takes place and what exactly needs to be monitored and tracked
7. Why dentists that are specifically trained in this space are critical with respect to outcome and why the following experience is particularly important
 - a. Naturally and of course a practice that is limited to sleep

- b. But also, why being a provider that has vast experience in treating TMJ-TMD Cranio-facial and head and neck pain is important

And finally, what makes Dr. John L. Bouzis in Casper Wyoming particularly suited to deliver this form of care and what are the advantages for patients in our community and surrounding area?

The Response:

No doubt this is a large topic, but it is also one where the **clinical model matters as much as the individual treatment**. The central point is that sleep-disordered breathing should not be approached as simply “find an AHI and give the patient a CPAP or appliance.” It is a **chronic, heterogeneous medical disorder** in which the type of apnea, anatomy, physiology, comorbidities, treatment preference, adherence, and longitudinal response all matter.

The epidemiology below has been updated because some of the older “30 million/80% undiagnosed” estimates **are now** substantially underestimating the apparent U.S. burden.

1. The three types of sleep apnea

There are **two fundamental physiologic types** of sleep apnea:

1. **Obstructive sleep apnea (OSA)**
2. **Central sleep apnea (CSA)**

In addition to those that have a combination of OSA and CSA, a third clinically important category is:

3. **Treatment-emergent central sleep apnea (TECSA)** — sometimes called “complex sleep apnea”

It is important to distinguish these because **the treatments are not interchangeable**.

A. Obstructive Sleep Apnea — OSA

What happens?

The patient continues trying to breathe, but the upper airway collapses.

The basic sequence is:

Reduced pharyngeal stability → airway narrowing/collapse → continued respiratory effort → reduced/absent airflow → oxygen desaturation/arousal → airway reopening

The patient may have:

- loud snoring
- choking/gasping
- witnessed apneas
- fragmented sleep
- excessive daytime sleepiness
- morning headaches
- nocturia
- hypertension
- atrial fibrillation
- impaired concentration
- mood changes

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But importantly, **some patients have very few subjective symptoms.**

Major mechanisms

OSA is influenced by:

- craniofacial anatomy
- tongue position
- mandibular position
- soft palate
- lateral pharyngeal wall collapsibility
- obesity/adipose tissue
- nasal resistance
- sleep position
- age
- menopause
- alcohol/sedatives
- neuromuscular control of the airway
- ventilatory control/“loop gain”

Treatment

Depending upon severity and phenotype:

- CPAP/APAP
- oral appliance therapy
- weight reduction
- positional therapy
- nasal/upper-airway treatment

- selected upper-airway surgery
- maxillomandibular advancement
- hypoglossal nerve stimulation/Inspire
- bariatric surgery in appropriate patients
- pharmacologic weight-loss therapy
- combination therapy

B. Central Sleep Apnea — CSA

CSA is fundamentally different.

The airway may be open, but the **brain's respiratory control system temporarily fails to generate adequate respiratory effort**.

Therefore:

No respiratory effort → no airflow

rather than:

Respiratory effort → obstructed airflow

Causes include:

- heart failure
- opioid medications
- high altitude
- certain neurologic disorders
- treatment-emergent CSA
- idiopathic/primary CSA
- certain medications/substances

The new 2025 AASM CSA guideline specifically recognizes several etiologic categories and recommends that treatment be tailored to the cause.

Treatment

Depending upon cause:

- treating the underlying disease
- CPAP in selected patients
- BPAP with a backup rate
- ASV

- supplemental oxygen in selected CSA populations
- medications such as acetazolamide in selected circumstances
- phrenic nerve stimulation in selected patients

Importantly, a **mandibular advancement device is not a treatment for primary central apnea** because the fundamental problem is not upper-airway obstruction.

C. Treatment-Emergent Central Sleep Apnea

This is particularly important in PAP management.

A patient begins with predominantly obstructive apnea.

PAP opens the airway.

But after obstruction is removed, the patient develops repeated central apneas.

This can occur because of alterations in:

- CO₂ reserve
- ventilatory control
- loop gain
- arousal threshold
- respiratory stability

Some cases resolve with continued CPAP therapy. Others persist.

Persistent clinically important treatment-emergent CSA may require:

- optimization of CPAP
- addressing medications/opioids
- BPAP with backup rate in selected patients
- ASV in appropriate patients

The current AASM guideline supports ASV for several CSA etiologies, including treatment-emergent CSA, with special caution and experienced-center management in patients with heart failure with reduced ejection fraction.

This is one reason why simply saying “the patient failed CPAP” is inadequate. You need to know *why* the patient failed.

2. How many people have sleep apnea?

This is where the magnitude of the problem becomes striking.

Worldwide

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A major Lancet Respiratory Medicine analysis estimated:

- approximately **936 million adults age 30–69** have OSA with $AHI \geq 5$
- approximately **425 million** have moderate-to-severe OSA with $AHI \geq 15$.

That study is older, but it remains one of the most widely cited global prevalence estimates.

United States

More recent modeling suggests the burden is substantially larger than the traditional “25–30 million” figure.

A 2025 analysis estimated approximately:

83.7 million U.S. adults with OSA

or approximately **32.4% of adults ≥ 20 years old.**

Estimated prevalence was:

- **39.1% in men**
- **26.0% in women**

with approximately:

- 52% mild
- 30% moderate
- 18% severe.

Another 2025 analysis estimated approximately **80.6 million** U.S. adults with OSA.

The exact number varies because prevalence depends heavily on:

- AHI definition
- oxygen-desaturation criteria
- age
- sex
- obesity prevalence

- whether symptoms are required
- study methodology

But the overall conclusion is clear:

OSA is vastly more prevalent than the number of diagnosed patients suggests.

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3. How many are undiagnosed and untreated?

A recent U.S. estimate presented at SLEEP 2025 estimated:

- up to **85.6 million adults** with OSA
- approximately **68.5 million (80%) undiagnosed**
- approximately **17.1 million diagnosed**
- approximately **4.5 million of those diagnosed (26%) untreated**

And there is another major problem:

Among people who initiate PAP, approximately **63% discontinued treatment or failed Medicare adherence criteria** in the analysis.

So the problem isn't simply:

“How many people have OSA?”

It is:

How many have it → how many are diagnosed → how many start therapy → how many actually use it → how many achieve adequate physiologic control?

That is a much more clinically meaningful continuum.

4. How is sleep apnea diagnosed?

There are two principal objective diagnostic approaches.

A. Home Sleep Apnea Testing — HSAT

A properly selected patient can undergo a home sleep apnea test.

Typical measurements may include:

- airflow
- respiratory effort
- oxygen saturation
- pulse
- snoring
- body position

Advantages:

- convenient
- inexpensive relative to PSG
- occurs in the patient's normal environment
- easier access
- can dramatically increase diagnostic capacity

The limitation is that HSAT generally does **not measure sleep directly with EEG**, so respiratory indices are often calculated using recording time rather than true sleep time.

Therefore, HSAT can underestimate severity, particularly when the patient spends substantial time awake.

AASM resources emphasize that HSAT is appropriate primarily for **uncomplicated adults with suspected OSA**, whereas polysomnography remains necessary in many more complicated situations.

B. In-Laboratory Polysomnography — PSG

PSG remains the most comprehensive diagnostic study.

It can measure:

- EEG
- EOG
- chin EMG
- leg EMG
- ECG
- airflow
- respiratory effort
- oxygen saturation

- body position
- snoring

It can therefore determine:

- actual sleep time
- sleep stages
- arousals
- REM vs NREM effects
- positional effects
- central vs obstructive events
- periodic breathing
- limb movements
- sleep architecture

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PSG is particularly important when there is:

- suspected central apnea
- significant cardiopulmonary disease
- neuromuscular disease
- unexplained hypoxemia
- significant insomnia
- parasomnia
- narcolepsy concern
- inconclusive HSAT
- unusual or complex sleep-disordered breathing

5. Why isn't one night always enough

This is particularly relevant to mild OSA.

OSA can vary substantially between nights because of:

- sleep position
- REM percentage
- alcohol
- nasal congestion
- weight
- medications
- sleep deprivation
- altitude
- illness

Consequently, a single negative or borderline night does not always establish that clinically important OSA is absent.

This is one reason a **multi-night home monitoring strategy** can be particularly valuable in borderline cases.

6. What happens if OSA remains untreated?

The danger isn't simply “the patient is tired.”

The fundamental pathophysiology is:

airway obstruction → hypoxia → reoxygenation → sympathetic activation → blood-pressure surges → intrathoracic pressure swings → endothelial stress → inflammation/metabolic dysfunction → sleep fragmentation

OSA is strongly associated with:

Cardiovascular disease

- hypertension
- resistant hypertension
- coronary artery disease
- myocardial infarction
- heart failure
- atrial fibrillation
- other arrhythmias
- pulmonary hypertension
- stroke
- cardiovascular mortality

The American Heart Association notes that OSA prevalence may reach **40–80% among patients with hypertension, heart failure, coronary artery disease, pulmonary hypertension, atrial fibrillation and stroke.**

Metabolic disease

- insulin resistance
- type 2 diabetes
- metabolic syndrome
- obesity
- dyslipidemia
- fatty liver disease

Neurologic/cognitive

- excessive daytime sleepiness
- impaired concentration
- memory impairment
- reduced executive function
- mood disturbance
- depression/anxiety associations

Safety

Perhaps one of the most immediate consequences is:

increased motor-vehicle and occupational accident risk from sleepiness and impaired vigilance.

Reviews of OSA complications include cardiovascular, metabolic, neuropsychiatric and accident-related consequences.

7. Treatment options

A. CPAP

How it works

CPAP produces positive airway pressure that acts as a **pneumatic splint** to prevent upper-airway collapse.

It is exceptionally effective at reducing:

- apneas
- hypopneas
- oxygen desaturation
- arousals

AASM recommends PAP therapy for OSA and recommends either CPAP or APAP for ongoing routine treatment.

Advantages

- highly effective
- works across a broad spectrum of OSA severity

- particularly powerful in moderate/severe disease
- excellent physiologic control
- can be objectively monitored
- rapidly reduces respiratory events

Disadvantages

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- mask discomfort
- nasal congestion
- dry mouth
- leaks
- pressure intolerance
- claustrophobia
- aerophagia
- noise
- inconvenience
- adherence problems

And this is the key distinction:

CPAP efficacy is excellent; CPAP effectiveness depends upon actual use.

8. APAP — Auto-PAP

APAP automatically changes pressure according to detected respiratory obstruction.

Advantages

- adapts to changing pressure requirements
- may improve comfort
- can often be initiated without laboratory titration in uncomplicated patients
- provides substantial data for longitudinal monitoring

AASM specifically supports either home APAP initiation or laboratory titration in appropriate adults without significant comorbidities.

Disadvantages

- pressure changes can disturb some patients
- not appropriate for every phenotype
- can be problematic in certain complex respiratory disorders
- requires interpretation of downloaded data

9. BiPAP/BPAP

BPAP provides:

- higher inspiratory pressure
- lower expiratory pressure

It is particularly useful when ventilatory assistance or pressure support is needed.

However, **BPAP is not simply “better CPAP.”**

For routine uncomplicated OSA, AASM suggests CPAP/APAP rather than BPAP.

For central apnea, the newer AASM guideline distinguishes BPAP **with a backup rate** from BPAP without one. It recommends against BPAP without a backup rate for several CSA etiologies because excessive pressure support can actually promote central apnea.

10. ASV — Adaptive Servo-Ventilation

ASV is a sophisticated form of PAP designed to stabilize ventilation.

It continually responds to the patient's breathing pattern and adjusts pressure support.

It is particularly relevant to:

- treatment-emergent CSA
- certain forms of primary CSA
- medication/opioid-associated CSA
- selected heart-failure-associated CSA

The current AASM guideline supports ASV in several CSA categories but emphasizes special caution and experienced-center management in patients with HFrEF.

Advantages

- highly sophisticated control of unstable breathing
- excellent for selected central-apnea phenotypes
- can address periodic breathing

Disadvantages

- expensive
- more complex
- requires appropriate diagnosis
- not appropriate for every patient
- heart-failure phenotype must be carefully evaluated

11. Mandibular Advancement Devices / Oral Appliance Therapy

For appropriately selected OSA patients, a **custom, titratable mandibular advancement device** moves the mandible forward, increasing upper-airway stability.

The AASM/AADSM guideline recommends oral appliance therapy for adults with OSA who are intolerant of CPAP or prefer an alternative, and specifically recommends a **custom, titratable appliance** rather than a non-custom device.

Advantages

- small
- silent
- portable
- no mask
- no hose
- no electricity
- generally easy to travel with
- often better accepted
- frequently high nightly use
- useful for positional/mild-to-moderate OSA
- useful for CPAP-intolerant patients
- can be combined with PAP in selected cases

This is where an important clinical concept arises:

Efficacy versus effectiveness

CPAP generally reduces AHI more.

But if:

CPAP reduces AHI by 90% but is worn 4 hours

while

MAD reduces AHI by 60% but is worn 7 hours

the real-world therapeutic comparison becomes much more interesting.

Clinical trials have repeatedly demonstrated that CPAP generally produces greater reduction in respiratory events while MAD adherence can be greater.

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Recent evidence also suggests that MAD therapy can provide clinically meaningful blood-pressure effects, including in patients with hypertension and elevated cardiovascular risk.

Disadvantages

- may not completely eliminate OSA
- titration required
- dental discomfort
- tooth movement
- occlusal changes
- TMJ symptoms in susceptible patients
- salivation/dry mouth
- limited suitability with inadequate dentition
- requires ongoing dental surveillance
- objective follow-up testing is essential

The AASM/AADSM guideline specifically recommends dentist follow-up for dental side effects/occlusal changes and follow-up sleep testing to confirm efficacy.

12. Surgery

Surgery should not be viewed as one treatment.

It is a **family of treatments directed at different anatomic problems.**

Examples include:

- tonsillectomy
- adenoidectomy
- nasal surgery
- turbinate surgery
- palatal procedures
- UPPP
- tongue-base procedures
- epiglottic procedures
- maxillomandibular advancement

- hypoglossal nerve stimulation
- bariatric surgery

AASM recommends discussion of surgical consultation for appropriately selected adults who are intolerant or unaccepting of PAP and emphasizes collaborative care between sleep clinicians and surgeons.

13. Inspire — hypoglossal nerve stimulation

Inspire is fundamentally different from PAP.

It electrically stimulates the hypoglossal nerve during inspiration, helping maintain tongue-base airway patency.

Current FDA labeling has expanded the AHI range up to **100**, with BMI labeling/warnings extending to **40**.

It is intended for appropriately selected patients with:

- moderate/severe OSA
- inability to use or benefit adequately from PAP
- appropriate upper-airway anatomy

Complete concentric collapse at the soft palate is an important exclusion, and significant central/mixed apnea is also a contraindication.

Advantages

- no mask
- no external PAP equipment during sleep
- excellent adherence in many patients
- physiological stimulation rather than pneumatic splinting

Disadvantages

- surgery required
- implanted device
- battery replacement eventually
- surgical/device complications
- cost
- patient selection limitations
- not appropriate for central apnea
- requires ongoing follow-up

14. Bariatric surgery

For patients with:

- obesity
- significant OSA
- particularly BMI ≥ 35

bariatric surgery can produce substantial weight reduction and frequently improves OSA.

AASM specifically recommends discussion of bariatric surgical referral for adults with OSA and BMI ≥ 35 who are intolerant or unaccepting of PAP.

But:

Improvement is not synonymous with cure.

A patient should be retested after substantial weight loss rather than simply assuming the OSA has disappeared.

15. Weight loss and GLP-1/GIP medications

This is one of the most important recent changes in OSA management.

The FDA approved **tirzepatide (Zepbound)** in December 2024 for moderate-to-severe OSA in adults with obesity, combined with reduced-calorie diet and increased physical activity. It became the first FDA-approved medication specifically for OSA.

The mechanism is primarily:

reduced appetite → weight loss → reduced upper-airway adiposity/collapsibility → improved OSA

This does **not** mean GLP-1/GIP medication replaces PAP or oral appliance therapy in every patient.

It should instead be viewed as another component of a potentially comprehensive OSA treatment strategy.

For example:

Weight loss + MAD

or

Weight loss + CPAP

or

Weight loss + positional therapy

may be considerably more powerful than relying on one intervention alone.

16. Positional therapy

Some patients have dramatically worse OSA when supine.

For these patients:

back sleeping → more airway collapse

while:

lateral sleeping → less obstruction

Positional therapy can therefore be highly useful when the patient's OSA is genuinely positional.

Advantages

- inexpensive
- noninvasive
- no mask
- no medication
- can be combined with other treatments

Disadvantages

- only works if OSA is positional
 - patients can adapt to devices
 - patients may return to supine sleep
 - does not address non-positional OSA
-

17. Supplemental nocturnal oxygen

This requires considerable caution.

Oxygen can improve:

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- oxygen saturation
- hypoxemic burden

But oxygen **does not necessarily eliminate the airway obstruction.**

A patient can continue having:

- apneas
- hypopneas
- arousals
- intrathoracic pressure swings
- sympathetic activation

while the oxygen saturation looks better.

Therefore:

Oxygen can improve the oximeter while the underlying obstructive disease continues.

It should generally **not be considered a substitute for PAP or an effective oral appliance in uncomplicated OSA.**

It has much more specific roles in selected patients with:

- persistent hypoxemia
- certain cardiopulmonary diseases
- selected CSA phenotypes
- high-altitude CSA

The 2025 AASM CSA guideline, for example, conditionally supports low-flow oxygen for CSA associated with high altitude.

This is especially important when considering combination therapy such as:

MAD + oxygen

because the MAD addresses the obstruction while oxygen addresses hypoxemia—but the combination still needs objective follow-up.

18. Why medical-dental collaboration is so important

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This may be one of the most important principles in modern dental sleep medicine.

OSA is a **medical disease involving an anatomic structure that happens to be within the dentist's area of expertise.**

The dentist understands:

- teeth
- occlusion
- mandible
- TMJ
- craniofacial anatomy
- oral appliance mechanics

The sleep physician understands:

- diagnosis
- respiratory physiology
- cardiovascular consequences
- central apnea
- PAP
- medications
- comorbidities
- differential diagnosis

The best model combines those skill sets.

The AASM/AADSM guideline explicitly emphasizes collaboration between sleep physicians and dentists.

19. Why a board-certified sleep physician matters

This is not simply about having an MD.

Sleep medicine is a multidisciplinary specialty requiring understanding of:

- respiratory physiology
- cardiology
- neurology
- pulmonology
- psychiatry
- sleep architecture
- PAP
- central apnea
- medications
- complex sleep disorders

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A board-certified sleep physician is particularly valuable when:

- the diagnosis is uncertain
- central apnea is present
- significant cardiovascular disease exists
- opioids are involved
- PAP has failed
- ASV is being considered
- oxygen is being considered
- surgery is being considered
- there is unexplained residual sleepiness

In other words:

The more complicated the patient, the more important specialized sleep-medicine oversight becomes.

20. Chronic-care monitoring

I strongly agree with your premise here.

OSA should be treated as a **chronic disease requiring longitudinal management**, not:

“Here is your CPAP.”

or

“Here is your appliance. See you next year.”

The AASM PAP guideline specifically calls for adequate follow-up after PAP initiation and during treatment.

For oral appliances, AASM/AADSM specifically recommend periodic physician/dentist follow-up and objective sleep testing to confirm treatment efficacy.

21. What should actually be monitored?

I would divide it into **six domains**.

1. Disease control

Track:

- AHI
- ODI
- oxygen nadir
- time below 90%
- hypoxic burden when available
- central apnea index
- obstructive apnea index
- RDI/RERAs when available
- positional dependence
- REM dependence
- heart-rate response

2. Treatment adherence

For PAP:

- nights used
- hours/night
- percentage ≥ 4 hours
- percentage ≥ 6 hours
- leak
- pressure
- residual AHI
- central events
- mask problems

For OAT:

- nightly use

- advancement
- symptoms
- appliance integrity
- dental changes
- occlusal changes
- TMJ symptoms

3. Patient outcomes

Track:

- Epworth Sleepiness Scale
- fatigue
- morning headache
- cognition
- mood
- quality of life
- snoring
- bed-partner observations

4. Cardiometabolic outcomes

Track or coordinate with the medical team regarding:

- blood pressure
- weight
- BMI
- glucose
- HbA1c
- lipids
- atrial fibrillation
- cardiovascular disease
- medication requirements

5. Oxygenation

Particularly:

- overnight SpO₂
- ODI
- oxygen nadir
- cumulative hypoxemia
- persistent hypoxemia despite control of obstruction

6. Anatomy

For oral-appliance patients:

- range of motion
- TMJ symptoms
- occlusion
- morning bite
- tooth mobility
- periodontal status
- appliance fit
- advancement

This is where a dentist with substantial TMD experience becomes extremely valuable.

22. Why a dentist specifically trained in dental sleep medicine matters

There is an enormous difference between:

a dentist who owns an oral appliance

and

a dentist who understands sleep medicine and manages oral-appliance therapy as part of a medical treatment plan.

The latter needs to understand:

- sleep studies
- AHI
- RDI
- ODI
- oxygen burden
- central apnea
- PAP
- ASV
- positional OSA
- hypoxemia
- cardiovascular risk
- oral-appliance mechanics
- mandibular advancement
- titration
- treatment failure

- combination therapy
- follow-up testing

The AASM/AADSM guideline specifically calls for qualified dentists to use custom titratable appliances and monitor dental side effects and occlusal changes.

23. Why a practice focused on sleep can be advantageous

A dentist who treats 5 oral-appliance patients per year has a fundamentally different experience from someone who has treated hundreds or thousands.

A sleep-focused practice develops experience with:

- different appliances
- different mandibular advancement requirements
- titration
- difficult patients
- CPAP failures
- combination therapy
- treatment failures
- appliance side effects
- follow-up sleep testing
- interpretation of longitudinal data

There is also an important systems advantage:

The practice develops a treatment pathway rather than simply a treatment.

Screen → diagnose → phenotype → select therapy → titrate → objectively verify → monitor → adjust

That is a fundamentally better model.

24. Why TMD, craniofacial pain and head/neck experience matters

This is particularly important for oral appliance therapy.

You are deliberately changing mandibular position night after night.

That means you are interacting with:

- TMJ loading
- muscles of mastication
- occlusion
- mandibular posture
- periodontal structures
- cervical musculature
- craniofacial biomechanics

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A dentist who does not understand these systems may successfully reduce an AHI while creating a new problem:

the patient's airway is better, but their jaw hurts.

An experienced TMD clinician can recognize:

- joint instability
- disc displacement
- limited opening
- muscle pain
- parafunction
- occlusal instability
- anterior open bite
- posterior interferences
- asymmetric mandibular movement

and determine whether the patient is an appropriate appliance candidate and how aggressively the mandible should be advanced.

This is one of the major advantages of combining **sleep medicine + dental sleep medicine + TMD/craniofacial expertise.**

25. How I would characterize Dr. John L. Bouzis

Based on publicly available information, there are several aspects of Dr. Bouzis's background that are particularly relevant.

He graduated from Creighton University Dental School in **1975**, meaning he has approximately five decades of dental experience. His practice identifies a particular focus on sleep apnea and TMJ/craniofacial conditions.

His practice reports:

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- advanced training in TMD
- craniofacial pain
- headaches
- bite and jaw disorders
- sleep medicine
- OSA treatment
- CPAP/APAP
- oral appliances
- collaboration with medical providers.

His Sleep Certified profile identifies him as a **Level 7 Sleep Certified Advanced Trainer**.

He is also listed by the Wyoming Telehealth Network as a **DDS/Sleep Medicine provider in Casper**.

His practice describes its model as combining **medical providers, sleep physicians, PAP/DME resources and a specifically trained dentist**, rather than treating the dental appliance as an isolated dental procedure.





26. What makes that combination potentially valuable to a patient?

I would summarize the potential advantage as:

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He sits at the intersection of three disciplines.

Sleep medicine

→ understanding the disease and objective testing

Dental sleep medicine

→ understanding mandibular advancement and oral-appliance treatment

TMD/craniofacial medicine

→ understanding the consequences of moving the mandible and managing the patient's jaw and occlusion

That is a particularly useful combination because the difficult patient is rarely the straightforward patient.

The difficult patient is:

“I have OSA, I hate CPAP, my jaw hurts, I have an old bite problem, I snore primarily on my back, I have hypertension, my oxygen drops at night, and my first appliance didn't work.”

That patient doesn't need someone who merely knows how to fabricate an appliance.

They need someone who can ask:

What is actually causing the patient's sleep-disordered breathing?

What phenotype does the patient have?

What treatment is most likely to work?

Can the patient tolerate it?

Is the treatment actually controlling the disease?

What is happening to the patient's jaw and occlusion?

What happens if the treatment doesn't work?

What is the next treatment?

That is the difference between **device delivery** and **disease management**.

27. The model I believe represents the future of sleep-apnea care

For a sophisticated practice, I would envision the pathway as:

IDENTIFY

↓

Screen for risk

↓

DIAGNOSE

HSAT / PSG

↓

PHENOTYPE

OSA vs CSA vs TECSA

Severity

Position

REM

Oxygenation

Anatomy

Comorbidities

↓

TREATMENT PLAN

CPAP/APAP

OAT

ASV/BPAP

Weight loss

GLP-1/GIP therapy
Positional therapy
Surgery/Inspire
Combination therapy

↓

OBJECTIVELY VERIFY

Repeat HSAT/PSG
Oximetry
PAP download
Appliance assessment

↓

CHRONIC MONITORING

Symptoms
AHI/RDI
ODI
oxygen burden
BP
weight
cardiovascular status
dental/TMJ status
adherence

↓

ADJUST

Advance appliance
Change appliance
Modify PAP
Add positional therapy
Address weight
Add oxygen when indicated
Refer for surgery
Consider combination therapy

↓

RETEST

Because **feeling better is not the same thing as having the disease controlled.**

The most important conclusion

The biggest mistake in sleep apnea treatment is thinking the objective is:

“Get the patient a treatment.”

The objective is:

“Get the patient’s sleep-disordered breathing under adequate physiologic control with a treatment the patient will actually use, while protecting and improving their overall health over the long term.”

That requires **diagnosis + phenotyping + treatment selection + adherence + objective verification + chronic monitoring.**

And that is precisely why the strongest model is not **physician vs dentist** or **CPAP vs oral appliance.**

It is:

Sleep physician + appropriately trained dentist + patient + objective data + longitudinal follow-up.

The AASM's own guidance supports this collaborative approach, particularly for oral-appliance therapy and surgical alternatives.

For Dr. Bouzis specifically, the publicly documented combination of long-standing dental experience, TMD/craniofacial training, dedicated sleep-apnea focus, oral-appliance experience, PAP/APAP involvement, and collaboration with medical providers gives him a **distinctive clinical positioning in Casper.**

[Dr. John L. Bouzis / RestfulSleep](#)

One qualification is important: It would be inappropriate to represent Dr. Bouzis as a *board-certified physician in sleep medicine*—he is a DDS. His role is the **dental/sleep-appliance side of the multidisciplinary model**, and physician diagnosis/medical management should remain appropriately integrated into the patient's care. That distinction is important both clinically and legally.

This material is intended for **patient education, as well as to provide an understanding and acknowledgment of the importance of physician-dentist oversight, their mutual referral relationship, and how critically important this is for patient care.** It is equally important to **separate** the above *evidence-based medical claims* from any *practice-specific claims* when

evaluating and documenting each practice's credentials, their insurance/DME status, and/or their scope-of-practice.