

ISMC 2026

15th - 18th September | Newcastle



British Sleep Society



UK Multidisciplinary Sleep Professionals

Dental Sleep Medicine

Oral appliance therapy for obstructive sleep apnoea: mechanism, evidence and patient selection by phenotype & endotype

Preparation session for the International Sleep Medicine Course (ISMC) 2026 · British Sleep Society · aligned with the ESRS Sleep Medicine textbook & examination syllabus

Learning objectives

What you should be able to do after this session



Define the field

Describe the scope of dental sleep medicine and the dentist–physician pathway.



Appraise the evidence

Summarise guideline recommendations and the efficacy-vs-adherence trade-off with CPAP.



Manage therapy

Outline titration, objective follow-up, side effects and combination therapy.



Explain the mechanism

Explain how mandibular advancement devices (MADs) stabilise the upper airway.



Select patients

Apply phenotypic and endotypic criteria to predict MAD response.



Pass the exam

Recall the key numbers, names and guideline thresholds examiners ask about.

Obstructive sleep apnoea: the essentials

Recap before we talk about oral appliances

~1 bn

adults worldwide estimated to have OSA
(Benjafield et al., Lancet Respir Med
2019)

≥5 /h

AHI threshold for OSA with symptoms
(AASM scoring; ICSD-3)

15–30

AHI range defining moderate OSA; ≥30 =
severe

50–65%

of unselected patients respond to a
MAD — the reason patient selection
matters

Pathophysiology in one line

- Sleep-onset loss of pharyngeal dilator tone → collapse of an anatomically vulnerable airway (velopharynx, oropharynx, tongue base, epiglottis).
- Repetitive hypopnoeas/apnoeas → intermittent hypoxia, arousals, sympathetic surges → daytime sleepiness, hypertension, cardiometabolic risk.
- Non-anatomical traits (ventilatory control, arousal threshold, muscle responsiveness) modulate severity — the basis of endotyping (Eckert et al., AJRCCM 2013).
- Diagnosis is medical (PSG or home sleep apnoea testing); the dentist provides therapy under a prescription and shares follow-up.

Summary of Sleep Disorders That Can Be Qualified as Dental Sleep Disorders

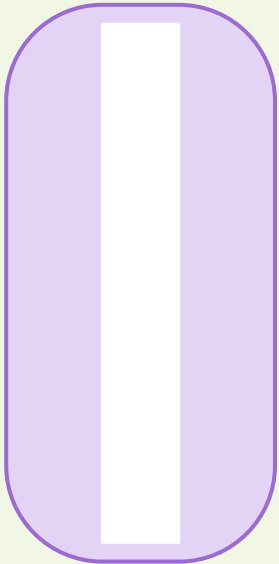
Sleep Disorder	Examples
Sleep-related breathing disorders	Snoring Obstructive sleep apnea
Orofacial pain	Dentoalveolar pain Temporomandibular disorder pain Headaches
Oral moistening disorders	Dry mouth (xerostomia) Hypersalivation
Gastroesophageal reflux disease	Iatrogenic reflux resulting from a weak or damaged stomach valve (lower esophageal sphincter), obesity, pregnancy
Mandibular movement disorders	Sleep bruxism Orofacial dyskinesia Oromandibular dystonia

How mandibular advancement works

More than “pulling the jaw forward”

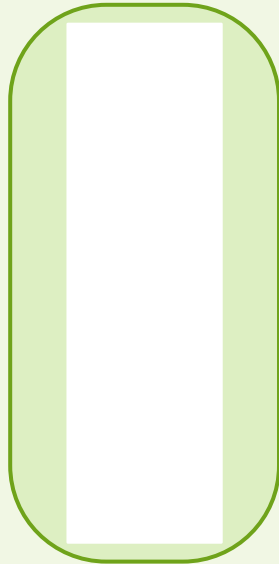
Before vs after advancement

Baseline



narrow

With MAD



widened & stiffened

Schematic cross-section of the pharyngeal lumen



Anterior displacement

Mandible (and hyoid) move forward, carrying the tongue base and lateral pharyngeal walls via the palatoglossal and palatopharyngeal folds.



Enlargement of the lumen

Cross-sectional area increases mainly in the velopharynx and oropharynx (imaging and DISE studies), lateral dimension more than anteroposterior.



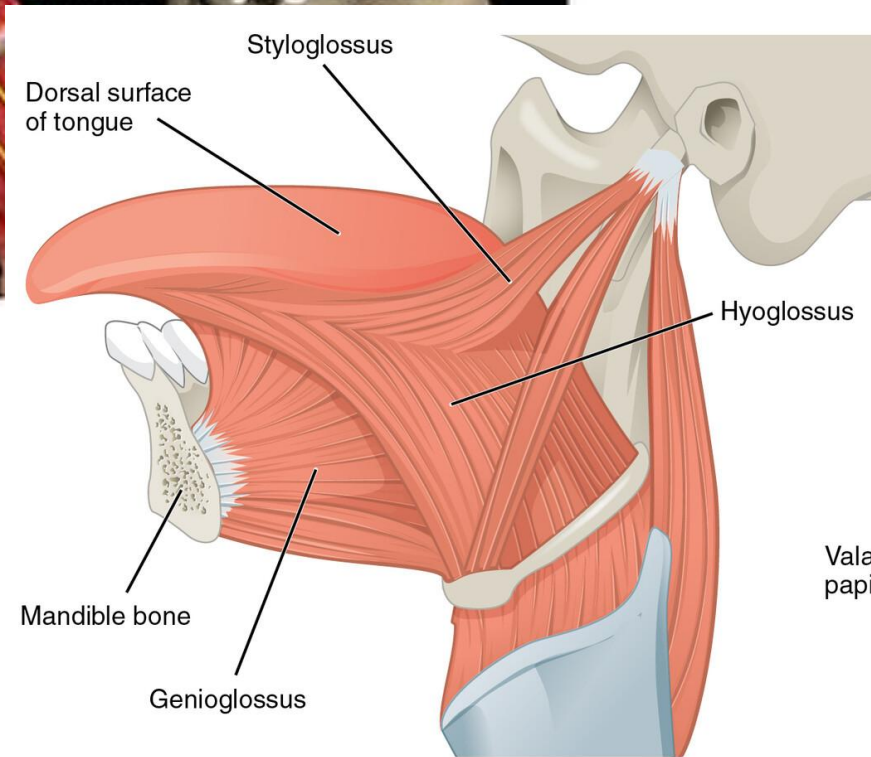
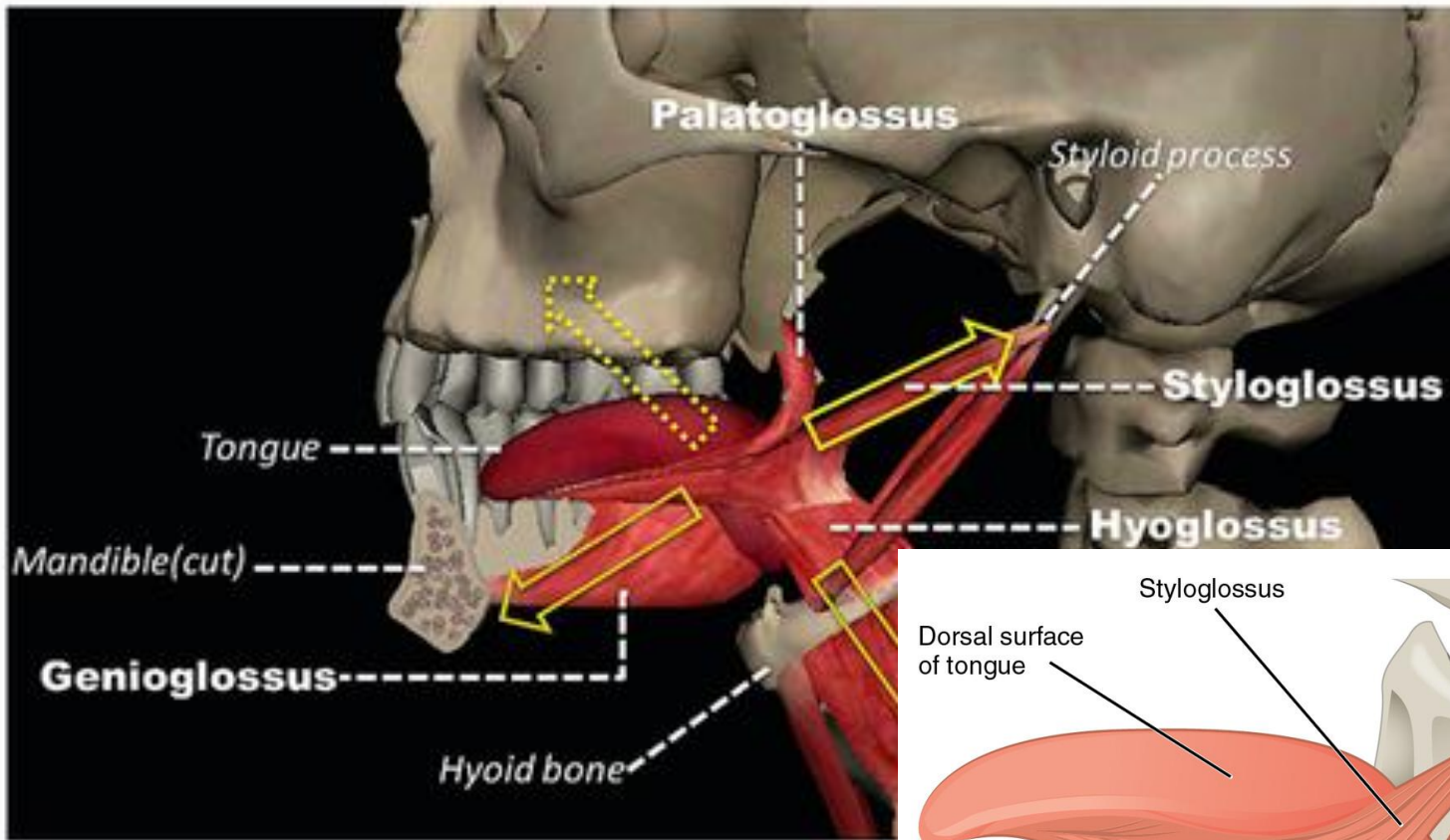
Reduced collapsibility

Passive critical closing pressure (Pcrit) falls — typically by 2–4 cmH₂O; larger drops in responders (Bamagoos et al., 2019).

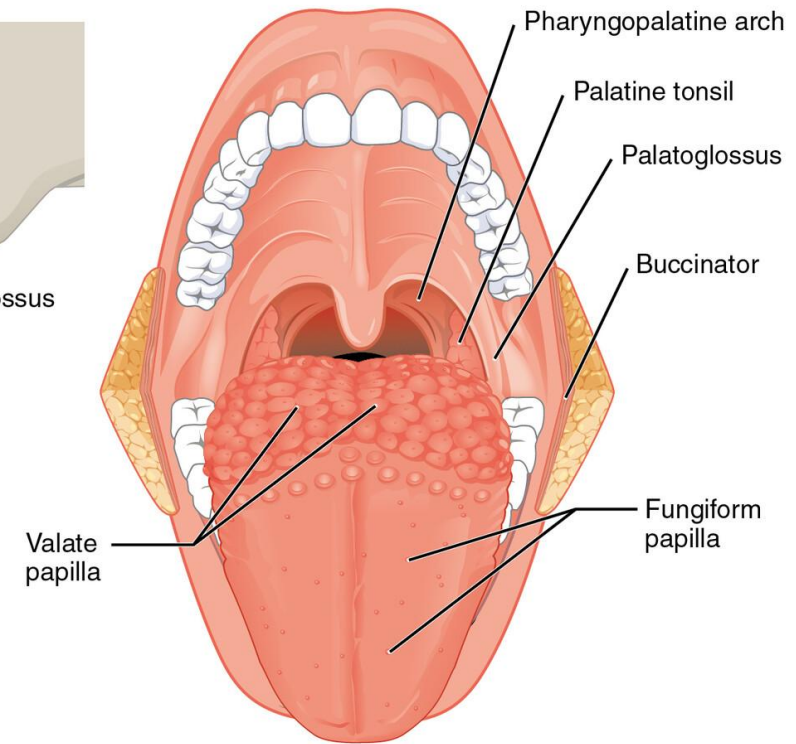


Stabilisation

Mouth opening is limited, preventing posterior rotation of the mandible; some designs also improve genioglossus efficiency.



(a) Extrinsic tongue muscles



(b) Palatoglossus and surface of tongue

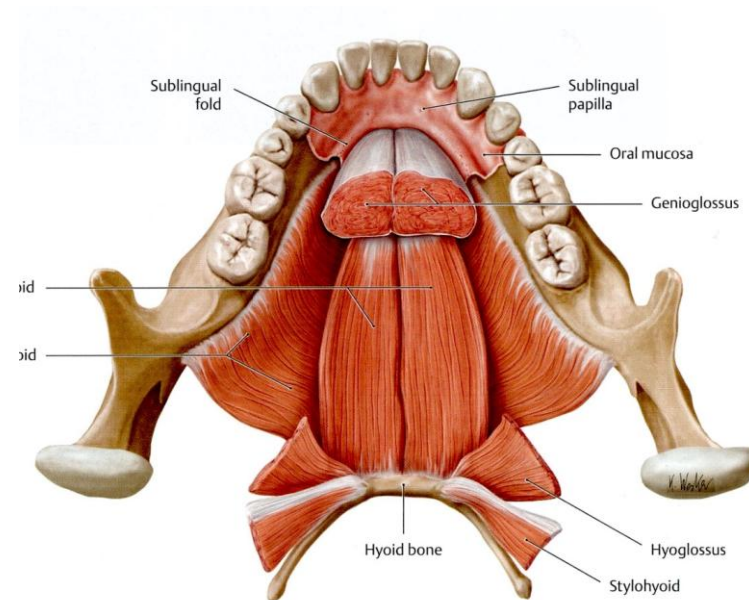
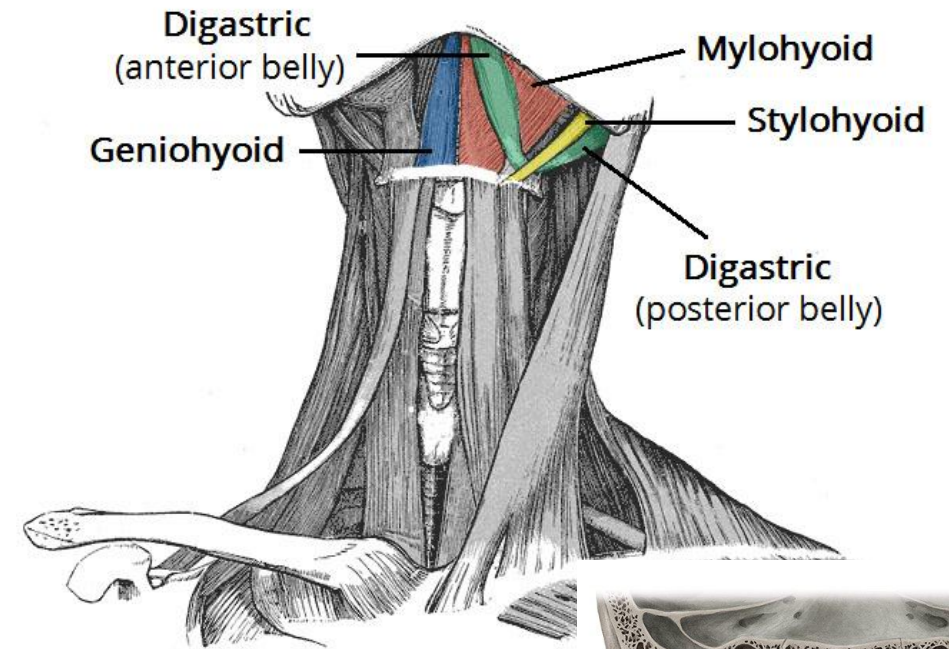
Anatomical considerations

Anterior movement of the Suprahyoid and Genioglossal muscles

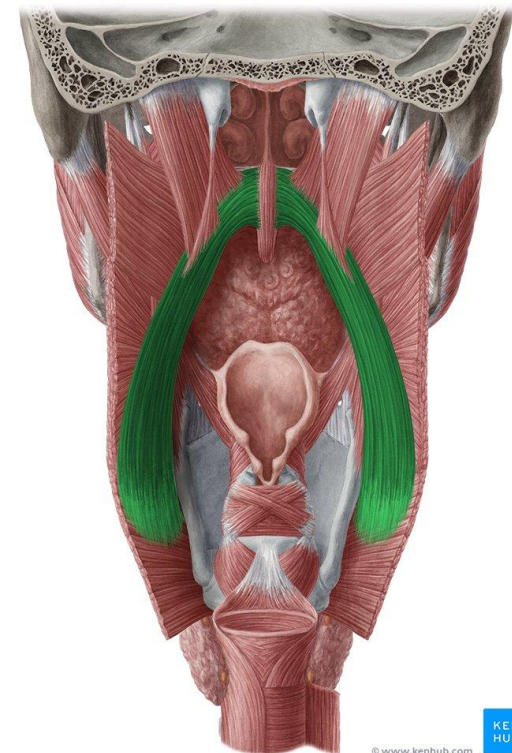
Decreasing gravitational effects on the tongue

Pal. Glossal and Pal. Pharyngeal stretch

Mandible – Hyoid stabilisation



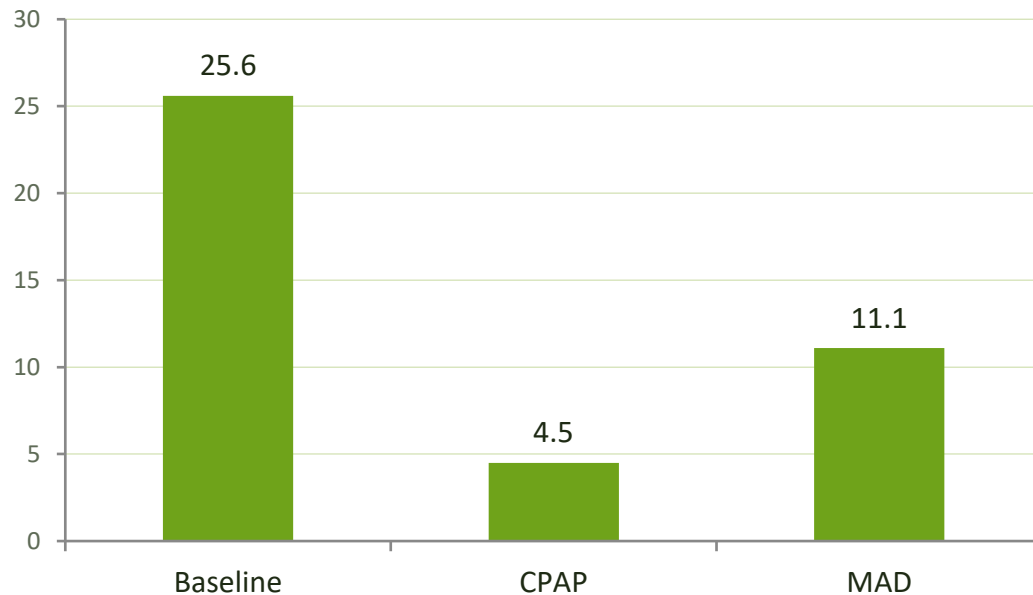
D Superior view of the mandible and hyoid bone.



Evidence and guidelines

Efficacy favours CPAP; effectiveness is often equivalent

Illustrative: efficacy on AHI (Phillips et al., AJRCCM 2013)



CPAP lowers AHI more, but similar improvements in sleepiness, quality of life and blood pressure — because MAD adherence is higher (crossover RCT, n=126).

AASM / AADSM 2015 (Ramar et al.)

Recommend OAT rather than no treatment for adults with OSA who are intolerant of CPAP or prefer an alternative; use custom, titratable devices; dentist-delivered with physician follow-up.

ERS 2021 (Randerath et al.)

MAD suggested as a first-line alternative in mild–moderate OSA and for CPAP-intolerant patients across severities; emphasis on objective follow-up.

NICE NG202 (2021, UK)

Offer a custom MAD to adults with mild OSA and symptoms, and to those with moderate–severe OSA who cannot tolerate or decline CPAP.

Cochrane review (2006, updated evidence)

MADs improve symptoms and AHI vs placebo; CPAP superior on AHI; patient preference often favours MAD.

Device example: ProSomnus® EVO & Tens more!

A precision-milled, custom mandibular advancement device





PART 2

Patient selection

Who responds to a mandibular advancement device? Two complementary lenses: the clinical phenotype and the physiological endotype.

Selection by phenotype

Observable clinical, anatomical and dental characteristics



Favours MAD response

- Mild–moderate OSA (AHI 5–30)
- Supine-predominant / positional OSA
- Lower BMI (<30 kg/m²) and smaller neck
- Younger age; female sex
- Retrognathic mandible, smaller oropharynx
- Lower baseline AHI and less severe desaturation
- Good protrusive range (≥6–7 mm)



Weights against

- Severe OSA (AHI >30), esp. with marked hypoxaemia
- Obesity (BMI >35)
- Central or mixed apnoea predominance
- Large tonsils / nasal obstruction as main site
- Complete concentric collapse of the velum on DISE
- Bruxism with severe TMD symptoms
- Fewer than ~8–10 sound teeth per arch, active periodontitis

Drug-induced sleep endoscopy (DISE) with a mandibular protrusion manoeuvre or a simulation bite can predict response; anteroposterior collapse at the velum and tongue base responds best, lateral-wall and concentric collapse least (Op de Beeck et al., 2019; Vroegop et al., 2013).

Selection by endotype

The four pathophysiological traits (PALM model, Eckert et al., 2013)



Pcrit — anatomical collapsibility

Passive critical closing pressure. MADs lower Pcrit; responders tend to have mild–moderate collapsibility (Pcrit $\approx$ -5 to 0 cmH₂O) rather than a highly collapsible airway.

MAD-favourable: mild–moderate



Arousal threshold

Propensity to wake at a given respiratory drive. A low threshold destabilises breathing; a higher threshold has been associated with better MAD outcomes in some cohorts.

MAD-favourable: not low



Loop gain — ventilatory control

Sensitivity of the chemoreflex feedback loop. High loop gain (unstable control) predicts MAD failure; low loop gain is one of the strongest positive predictors.

MAD-favourable: low



Muscle responsiveness

Genioglossus compensation during sleep. Poor responders to MAD show greater dependence on muscle compensation; MAD may partly substitute for weak compensation.

MAD-favourable: less critical

Sources: Edwards et al., AJRCCM 2016; Bamagoos et al., Ann Am Thorac Soc 2019. Traits can now be estimated from routine PSG (“polysomnographic endotyping”), making this clinically feasible.

Putting it together: a selection matrix

Phenotype × endotype — where MAD is most likely to succeed

	Ideal candidate	Consider — with caveats	Unlikely to respond
OSA severity	Mild–moderate; positional	Severe but CPAP-intolerant; combine with positional therapy or CPAP	Severe with marked hypoxaemia; central apnoea
Anatomy	Retrognathia; small oropharynx; AP collapse at velum/tongue base on DISE	Obesity BMI 30–35; mixed collapse patterns	Complete concentric velum collapse; large tonsils/nasal obstruction dominant
Endotype	Mild–moderate collapsibility, low loop gain	Low collapsibility but high loop gain → consider adjunct (e.g., O ₂ , acetazolamide trial)	Highly collapsible airway (Pcrit >0) + high loop gain
Dental	≥8–10 sound teeth/arch, healthy periodontium, protrusion ≥6 mm, no TMD	Partial dentition, stable implants, mild TMD (monitor)	Edentulous (consider TRD), active periodontitis, severe TMD
Patient factors	Prefers OAT; travels; CPAP non-adherent	Ambivalent; needs objective adherence monitoring	Unable to insert/remove device; uncontrolled gag reflex

“Response” definitions vary: ≥50% AHI reduction, AHI <10, or AHI <5 — always state which one when quoting success rates.

Titration, verification and combination therapy

Getting the most from the device once it is fitted



Titration

Start at ~50–70% of maximal protrusion; advance in 0.5–1 mm steps guided by symptoms and partner-reported snoring over 4–12 weeks. Remotely-controlled mandibular positioners during PSG can identify the target position in a single night.



Verify objectively

Repeat PSG or HSAT with the device in situ once titrated (guideline requirement). Assess residual AHI, oxygen nadir and symptoms; re-titrate or escalate if inadequate.



Monitor adherence

Embedded temperature-sensing microsensors give objective wear-time; typical use ≈6–7 h/night, higher than CPAP. Self-report over-estimates use.

Combination strategies

MAD + positional therapy

Additive effect in residual supine-dependent OSA (Dieltjens et al., 2015).

MAD + CPAP

Lower pressures and improved tolerance in CPAP-intolerant patients; hybrid interfaces exist.

MAD + weight loss / myofunctional therapy

Address anatomy and muscle responsiveness; useful for partial responders.

MAD + DISE-guided surgery

Nasal or tonsil surgery may convert non-responders; DISE with the device in situ localises residual collapse.

Side effects and long-term follow-up

Mostly minor and transient — but occlusal change is progressive



Short-term (weeks)

- Hypersalivation or dry mouth
- Tooth and gum tenderness
- TMJ/masticatory muscle discomfort on waking
- Transient bite change in the morning (resolves with exercises)



Long-term (years)

- Reduced overjet and overbite (~1 mm over 5 years, continues with use)
- Posterior open bite; proclination of lower incisors
- Progressive with duration of use, protrusion amount and hours worn
- Rarely a reason to stop if patient informed and monitored



Follow-up schedule

- Dentist: review at 1 week, 1 month, then every 6 months in year 1
- Annually thereafter: occlusion, TMJ, device integrity, adherence
- Physician: repeat sleep test after titration and if symptoms recur
- Device lifespan ~3–5 years; weight change or dental work may need a remake

Informed consent should explicitly cover permanent occlusal change; document baseline occlusion (photos, models/scans).

Ten exam pearls

1

OSA is diagnosed by the physician; the dentist provides and maintains the device.

2

Custom, titratable duobloc MADs outperform prefabricated (“boil-and-bite”) devices.

3

MADs act mainly by reducing pharyngeal collapsibility (P_{crit}) and enlarging the velo/oropharynx.

4

CPAP wins on AHI; MAD often equals it on outcomes because of adherence (Phillips 2013).

5

Guidelines: AASM/AADSM 2015, ERS 2021, NICE NG202 — mild OSA or CPAP-intolerant/refusing.

6

Best phenotype: mild–moderate, positional, lower BMI, younger, retrognathic, good protrusion.

7

Best endotype: mild–moderate collapsibility + low loop gain (Edwards 2016; Bamagoos 2019).

8

DISE: AP collapse responds; complete concentric velum collapse does not.

9

Always confirm efficacy objectively (PSG/HSAT) after titration and monitor adherence.

10

Occlusal change is the key long-term side effect — consent, baseline records, annual review.

Key references

Verify details and check for updates before the course

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