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PART I — CORE THESIS, MECHANICS AND ADAPTATION: The Bruce Maneuver as a Falsifiable Upper-Airway Training Hypothesis

Series position, archival boundary, and what TRIE adds

The author-selected architecture remains **INTRO + five numbered Parts**. `00_START_HERE` is an unnumbered prologue, while **PART I —** `01_CORE_THESIS_MECHANICS_AND_ADAPTATION` is the first substantive lane; the archive expressly directs the reader there first to understand the "main intervention and adaptation logic." Its six works are, in order, `01_bruce_maneuver_declaration_and_overview.pdf`, `02_optimized_uninverting_canonical_breathing_truths.pdf`, `03_intrapharyngeal_pressure_and_airway_adaptation.pdf`, `04_law_of_adaptation_source_bundle.pdf`, `05_historical_valsalva_like_techniques.pdf`, and `06_shut_your_mouth_historical_breathing_precedent.pdf`. Nothing in the inspected architecture warrants renumbering INTRO, creating a Part VI, or separating mechanics from Part I. filecite turn2file0

Within that architecture, the **Bruce Maneuver is the central intervention**, not an example of generic "breathwork." The declaration describes it as a "specific breathing training technique," attributes its development to Bruce through three years of personal somatic experimentation, and states that it was not derived from a recorded text. Those statements establish the author's declared provenance. They do not independently establish historical priority, physiological mechanism, safety, or efficacy; each of those requires a different evidentiary test. filecite turn3file0

TRIE 5.4.0 revision 3 contributes something important to Part I, but it does **not** contribute new clinical validation of the Bruce Maneuver. The verified session receipt identifies distribution **5.4.0**, packaging revision **3**, inherited kernel **5.1.1**, and only eight indexed sources; it explicitly records `historical_corpus_included: false`. Its verification scope is stated as delivered-byte integrity rather than "general meaning, truth or author adoption." Revision 3 primarily adds six operator manuals while retaining the revision-2 executable payloads. filecite turn2file1 filecite turn5file4

That is nevertheless directly useful to this paper because TRIE's operator method strengthens **provenance discipline**. `START_HERE.md` instructs the reader to verify the release, reopen the source used for a decision, and change a condition to see whether the reading changes; `TEXT_AND_EVIDENCE.md` instructs the operator to preserve the request in the reader's words and to answer using "a source, an exact readback, and a stated reading." filecite turn5file7 filecite turn5file1 NOCI likewise keeps contributions by named writers distinct and records which inputs a later writer says were used. filecite turn5file9 For Part I, the substantive consequence is methodological: **Bruce's propositions, historical witnesses, later AI synthesis, and independent biomedical evidence must remain separate evidentiary objects**.

The evidence available in this research session contains no exact, current readback of an additional Bruce-Maneuver clinical source from `PROD_TRIE` or `__WORKDEN_TRIE_v5`; therefore no such source is represented here as inspected. The verified TRIE receipt itself lists only the eight sources above and states that the historical corpus was not included. filecite turn2file1 The genuinely relevant later materials found outside the original six are instead identifiable as supplementary corpus records: the FWOCR documentation on automatic sleep-state function, later source audits, and COMINI IV CELS work on longitudinal airway/sleep/adaptation measurement. These are incorporated below only where they sharpen Part I's hypotheses and endpoints, not as new top-level parts. filecite turn7file0 filecite turn7file2 filecite turn6file4

Exegesis of the six Part I works

The first work is both declaration and causal proposal. Its physiological appendix characterizes the Bruce Maneuver as involving pressure and resistance challenges to the respiratory system, including phenomena described as akin to Valsalva-type loading or breath holding. It then proposes strengthening of airway-patency muscles, altered CO_2 tolerance and respiratory control, and eventual improvement in sleep-state airway behavior. Those are **author hypotheses**. The document itself acknowledges that its support includes anecdotal reports and “adjacent scientific literature,” which is a crucial limitation: evidence for another intervention can establish biological plausibility without establishing efficacy of this one. filecite turn3file6 filecite turn3file1

The second work enlarges the claim into a theory of trainable physiology. Its useful core is that respiratory function is adaptive and that repeated stimuli may change future response; it also emphasizes dynamic capacity rather than treating resting state as the whole of health. filecite turn4file0 The stronger rhetorical opposition it draws between “homeostasis” and “allostasis” is not needed to sustain the Part I thesis. Modern physiology can contain both regulated stability and adaptive change; the scientifically consequential proposition is narrower: **a defined respiratory stimulus can be tested for a dose-dependent, persistent adaptation**.

The third work supplies the most important candidate mechanism. It locates the proposed training effect in **intrapharyngeal pressure, local mechanosensation, upper-airway motor recruitment, and subsequent adaptation**. It correctly points toward pressure-sensitive upper-airway control: experiments in humans show that brief negative airway pressure can rapidly increase genioglossus activity, demonstrating a mechanically responsive upper-airway reflex. [1] Upper-airway surface mechanoreceptors materially contribute to this response; topical anesthesia reduces genioglossus reflex excitation to negative-pressure pulses. [2] The Part I source then extrapolates from such phenomena toward repeated pressure loading as training. filecite turn4file2 That extrapolation is the central unresolved bridge.

The polarity of the pressure stimulus matters. Classic physiological work reports increased genioglossus activity with **negative** airway pressure but decreased activity with **positive** pressure under the conditions tested. [3] Thus, “airway pressure activates the airway” is too coarse a model. A Bruce-specific theory must identify pressure sign, anatomical compartment, timing within the respiratory cycle, glottic state, tissue deformation, muscular co-contraction, and whether the principal training signal arises from local pressure, active muscle force, strain, sensory feedback, altered flow, or some combination. The original source's use of “intrapharyngeal pressure” is therefore scientifically more valuable than collapsing the maneuver into a generic Valsalva analogy. filecite turn4file2

The fourth work functions as the archive's adaptation foundation. Its source bundle ranges across adaptation, hormesis, tissue-loading ideas, thermoregulation, and related response models. filecite turn0file17 Its strongest role in Part I is not to prove a universal “law” from which Bruce-Maneuver efficacy can be deduced; rather, it supplies the general proposition that **stimulus, response, recovery, and repeated exposure must be treated as a time-dependent system**. This distinction becomes decisive because adaptation can be beneficial, neutral, or maladaptive depending on tissue, dose, recovery, disease state, and endpoint.

The fifth and sixth works provide genealogy rather than intervention efficacy. Historical Valsalva-like practices demonstrate that humans have long used closed-airway strain or breath-hold pressure for particular purposes; the archive's Sushruta example is one such retrospective comparison. filecite turn4file3 Catlin's nineteenth-century *Shut Your Mouth and Save Your Life* provides a documented historical precedent for advocacy of closed-mouth/nasal respiration, with multiple nineteenth-century editions preserved in library scans. filecite turn4file1 Neither historical resemblance establishes that those practices are mechanically identical to the Bruce Maneuver, that Bruce derived his intervention from them, or that the historical claims were clinically correct. Their proper Part I role is **contextual precedent**.

The resulting source hierarchy is therefore clear: **the first work defines the intervention and its claimed effects; the second supplies the adaptive worldview; the third supplies the candidate local mechanism; the fourth supplies general adaptation concepts; the fifth and sixth establish historical analogies and antecedent respiratory thought.** That ordering preserves the Bruce Maneuver as the object under test rather than allowing historical or generic breathing material to replace it. filecite turn2file0

A falsifiable mechanistic model from pressure to retained sleep-state function

A doctoral formulation needs to convert the archive's narrative into a causal sequence whose links can fail independently. The minimal model is:

$$\text{defined Bruce stimulus} \rightarrow \text{local pressure/strain} + \text{active motor loading} \rightarrow \text{acute sensory and motor response} \rightarrow \text{rec}$$

The first link is not yet operationally established in the corpus strongly enough to invent a research protocol. The author must supply a frozen, reproducible specification of the maneuver before intervention trials can legitimately begin. That specification would need to define the maneuver without the investigator silently substituting ordinary Valsalva, Müller maneuver, inspiratory muscle training, myofunctional therapy, or another exercise. This is exactly the sort of object-preservation problem TRIE 5.4.0 is designed to expose: source identity and operation identity must survive the transformation from author description to experimental implementation. filecite turn5file1

At the acute-response level, the literature establishes that upper-airway pressure perturbations can alter pharyngeal dilator activity. In awake humans, brief negative-pressure stimuli cause rapid genioglossus activation. [1] In OSA, upper-airway collapsibility and genioglossus reflex behavior vary across the respiratory cycle, making timing an experimentally meaningful variable rather than a nuisance. [4] Regional responses are also heterogeneous: one study found different excitation/inhibition patterns across

genioglossus regions in a substantial subset of participants, underscoring that “the genioglossus” is not necessarily a single uniform actuator. [5]

At the chronic-adaptation level, a plausible—but still indirect—biological bridge exists. Skeletal muscle converts mechanical deformation into intracellular signals through mechanosensitive structures; cyclic stretch in skeletal myocytes has been shown to increase β1D-integrin and activate focal adhesion kinase and RhoA, while human loading studies demonstrate load-sensitive mechanosensor and focal-adhesion responses. [6] These findings make mechanotransduction a legitimate candidate mechanism. They do **not** show that intrapharyngeal pressure during the Bruce Maneuver generates a sufficient strain field in human pharyngeal dilators, connective tissue, mucosa, or sensory structures to produce beneficial remodeling. That particular chain remains unmeasured.

The sleep-state endpoint is especially important because later Bruce documentation sharpens the intended target. It distinguishes **posture**—a voluntarily maintained configuration—from **position**, meaning a state that continues when voluntary attention is unavailable. The same document states that the desired endpoint is a system that continues working while the person is not supervising it and treats sleep as a consequential test condition. It simultaneously labels itself a source record, not a validated training manual. filecite turn7file0 This is a genuinely useful later addition to Part I because it turns an imprecise claim about “better breathing” into a falsifiable endpoint: **does a daytime training exposure produce a persistent change in upper-airway function during sleep when the subject is no longer actively performing the maneuver?**

Existing physiology makes that question nontrivial. Negative-pressure genioglossus reflex activation is attenuated during non-REM sleep in several human studies. [7] Other work shows that the sleep effect is more complex than a simple on/off suppression, including inhibitory components and respiratory-phase dependence. [8] The hypothesis therefore cannot simply be “train the waking reflex and the same reflex remains stronger asleep.” A more defensible prediction is that repeated training might alter one or more persistent properties—muscle capacity, coordination, airway stiffness, geometry, sensory gain, motor recruitment threshold, or recovery kinetics—and those changes might then modify sleep-state airway stability.

FWOCR is relevant here only as a boundary check. The later FWOCR work distinguishes ordinary respiratory-muscle mechanical/metabolic work from the much broader proposed nostril-to-cell-and-back systems boundary and explicitly warns against treating a measurement of one component as a measurement of the entire system. filecite turn7file2 filecite turn7file4 For Part I, this means a reduction in airway effort, pressure requirement, or apnea burden must not automatically be converted into a claim about total cellular energy savings. COMINI IV CELS has a similarly limited but useful role: its later framework separates an **author proposition**, an **externally supported association**, a **mechanistically plausible inference**, and an **unvalidated prediction**. filecite turn6file4 Applied here, the Bruce Maneuver is an author-defined intervention; pressure-sensitive airway control is externally supported; chronic Bruce-specific adaptation is mechanistically plausible but unvalidated; and any longevity effect remains a separate, downstream prediction.

Independent evidence, adjacent efficacy, and results that constrain the thesis

Independent intervention research supports the broad proposition that **upper-airway function is trainable**, but it does not yet validate the Bruce Maneuver. In the 2009 randomized trial by Guimarães and colleagues, 31 adults with moderate OSA received either three months of daily oropharyngeal exercises or sham therapy; the active program targeted the tongue, soft palate, and lateral pharyngeal wall and improved objective and subjective outcomes. [9] A separate randomized didgeridoo trial also found improvement in sleep-apnea outcomes after sustained training. [10] Both studies are particularly relevant to Bruce's sleep-state endpoint because the training occurred while awake while outcomes were measured during later sleep; they therefore demonstrate that **daytime training can, in some interventions, change nocturnal function**. They do not identify intrapharyngeal pressure as the active ingredient and do not test the Bruce Maneuver.

The newest broad synthesis located for this review, a 2026 systematic review and meta-analysis of 13 randomized trials totaling 429 participants, found that oropharyngeal training reduced AHI by a pooled mean of 7.55 events/hour and respiratory-muscle training by 3.73 events/hour; resistance training alone did not produce a statistically significant AHI change, and daytime sleepiness did not improve consistently. The authors characterized oropharyngeal and respiratory-muscle training as **adjunctive options** and emphasized heterogeneous protocols and the need for standardized methods and longer follow-up. [11] This is important contemporary evidence for trainability but simultaneously argues against transferring efficacy indiscriminately from “muscle training” to any particular respiratory maneuver.

A 2021 pilot randomized trial specifically targeting genioglossus strengthening found no significant change in AHI. [12] That negative result is theoretically valuable: even if a target muscle becomes stronger, the clinically important sleep phenotype need not improve. Upper-airway obstruction is governed by interacting anatomy, neuromuscular compensation, ventilatory control, arousal characteristics, sleep stage, body position, and other person-specific factors; a single “strength” variable cannot be assumed to dominate the system. The pressure-reflex literature itself shows substantial within-person and regional complexity. [13]

Another constraint comes from pressure polarity. Negative pressure can excite genioglossus activity, whereas positive pressure has been reported to reduce it in acute experiments. [3] That does not refute the Bruce hypothesis, because the proposed maneuver may create active co-contraction, localized pressure gradients, strain, or a training load unlike externally applied positive pressure. It does mean that the third archival paper's move from “pressure-sensitive system” to “repeated positive pressure strengthens the airway” cannot be accepted without direct measurement. filecite turn4file2

The historical evidence is similarly bounded. Ancient or premodern closed-glottis strain shows that humans recognized useful effects of internal pressure long before modern respiratory physiology. filecite turn4file3 Catlin demonstrates sustained nineteenth-century advocacy of closed-mouth breathing. filecite turn4file1 Neither line of history determines the efficacy of Bruce's specific intervention. Historical recurrence increases the interest of the question; it does not answer it.

Current clinical standards reinforce this evidentiary distinction. Positive-airway-pressure treatment remains an evidence-based treatment for appropriately diagnosed adult OSA in American Academy of Sleep Medicine guidance, and a 2025/2026 AASM guideline for hospitalized adults likewise incorporates diagnosis

and PAP into the management pathway. [14] Nothing located in the biomedical literature for this review identifies the Bruce Maneuver as a validated substitute. That absence is not evidence of ineffectiveness; it establishes the present state of validation.

Objective measurement and decisive experimental design

A strong Part I research program would test **identity, mechanism, dose, persistence, specificity, clinical effect, and safety separately**. The protocol cannot responsibly begin by inventing the maneuver from analogies in the archive. The initial research object must be a Bruce-authored, frozen operational specification, preserved with version and provenance. TRIE's exact-readback discipline is particularly apt here because an intervention trial becomes uninterpretable if the tested maneuver drifts into an investigator-created version. filecite turn5file1

The first experimental stage would be mechanistic rather than therapeutic. In screened adults, an acute laboratory study could measure the maneuver's actual physical exposure: pharyngeal or epiglottic pressure where ethically and technically appropriate, mouth pressure, airflow, respiratory timing, thoracoabdominal movement, surface or intramuscular genioglossus EMG in a specialist laboratory, blood pressure, ECG/heart rate, oxygen saturation, and end-tidal or transcutaneous CO_2. Upper-airway collapsibility can be quantified using established physiological constructs such as pharyngeal critical closing pressure, while brief pressure-perturbation paradigms provide a way to quantify reflex responsiveness. [15] The experiment's purpose would not be to demonstrate clinical benefit; it would determine **what the Bruce Maneuver physically does**.

The central dose variable needs to be measured rather than inferred. A useful abstract representation is

$$D = \int_0^T f\Big(P_{\text{pharynx}}, \Delta P, \dot{V}, EMG, t_{\text{cycle}}, \text{anatomy}\Big)\, dt,$$

where D is the candidate training dose and the terms represent local pressure, pressure gradient, airflow, muscle recruitment, respiratory-cycle timing, and participant anatomy. This is deliberately not a maneuver instruction. Its purpose is to prevent “minutes practiced” from being mistaken for a physiologically uniform dose.

A subsequent adaptation study would repeatedly measure the same mechanistic variables before training, during the training interval, after recovery periods, and after cessation. The core prediction is not simply improvement over time. It is a **stimulus–response–recovery relation**: exposure of a defined magnitude produces an acute response; sufficient recovery permits a persistent change; excessive or poorly recovered loading may fail to improve or may worsen relevant measures. A scientific law of adaptation must be able to represent both adaptation and maladaptation.

The decisive sleep test would occur **without performing the maneuver during sleep**. Full polysomnography before and after training would measure AHI, obstructive-event burden, oxygen-desaturation index, hypoxic burden or time below a prespecified saturation threshold, arousals, sleep architecture, snoring/flow limitation, body position, and ideally sleep-stage-specific airway behavior. Daytime measurements of airway pressure response and muscle recruitment would be linked prospectively to nighttime outcomes. This directly tests Bruce's “position rather than posture” endpoint: whether function remains altered when voluntary supervision is absent. filecite turn7file0

For clinical efficacy, the strongest design is a preregistered, assessor-blinded randomized trial with at least a Bruce-Maneuver arm and a time/attention-matched control; a validated oropharyngeal-training comparator would be scientifically valuable because it distinguishes **Bruce-specific efficacy** from the already supported proposition that some upper-airway exercises work. Existing trials show that exercise modality matters. [16] Primary and secondary outcomes, clinically meaningful thresholds, missing-data handling, adherence measures, subgroup hypotheses, and adverse-event definitions need to be fixed before unblinding.

Three causal tests are especially discriminating. First, **specificity**: if the hypothesized active ingredient is component-specific intrapharyngeal loading, measured exposure to that component ought to predict adaptation better than practice time alone. Second, **mediation**: changes in local mechanics or neuromuscular response ought to statistically precede or mediate clinical sleep improvement if they are genuinely causal. Third, **retention**: if the intervention builds an automatic function rather than a temporary performed state, gains ought to persist for a prespecified interval after the last training exposure. Failure of any of these tests would narrow or falsify part of the current mechanistic model rather than merely being classified as a “nonresponder.”

COMINI IV CELS can contribute exploratory longitudinal variables—particularly airway integrity, sleep resilience, recovery kinetics, and adaptive reserve—but it cannot serve as a validated outcome score at present. Its own doctoral formulation explicitly describes the composite as investigational and requiring prospective validation. FWOCR can likewise motivate energy-accounting questions, but its present archive explicitly does not establish a validated aggregate energy fraction or Bruce-specific energy saving. filecite turn7file2 Those adjacent frameworks therefore belong in exploratory analyses, not in the primary proof of Part I.

Safety, contraindication logic, and evidentiary conclusion

The original declaration's safety argument is not adequate for clinical research. It suggests that excessive breath holding is intrinsically self-limiting because loss of consciousness will allow breathing to resume. filecite turn3file4 Syncope is itself an adverse event, and Valsalva-type strain can produce pronounced phase-dependent cardiovascular and cerebral-perfusion changes. In healthy men performing graded standing Valsalva maneuvers, release can produce hypotension sufficient to cause syncope, with effects dependent on maneuver intensity and duration. [17] In pulmonary arterial hypertension, a case-control study found greater susceptibility to Valsalva-associated syncope because of impaired autonomic compensation and cerebral hypoperfusion. [18] Thus, “the person passes out and breathing resumes” cannot be treated as a safety mechanism.

This does **not** establish that the Bruce Maneuver has the same risk profile as a conventional Valsalva maneuver; the maneuver has not yet been sufficiently operationalized and tested to make that inference. It establishes instead that any intervention capable of producing substantial expiratory strain, breath holding, or intrathoracic/intrapharyngeal pressure must measure cardiovascular consequences rather than assume benignity. [19]

Early mechanistic studies would therefore require conservative screening and independent safety oversight. Until direct safety data exist, investigators would have strong reason to exclude or separately study people with conditions in which large pressure or hemodynamic excursions may be consequential—for example pulmonary arterial hypertension, recurrent syncope, significant unstable cardiovascular disease or arrhythmia, and other conditions identified by the study physician after the maneuver's actual pressure

profile is known. The PAH evidence provides a concrete example of why disease-specific susceptibility matters. [18] Pregnancy, pediatric populations, severe OSA, ocular vascular vulnerability, and other high-consequence groups belong in separate protocols only after the basic physiological exposure and risk profile have been established; Part I's archival claims about broad safety do not supply those data. filecite turn3file4

The resulting doctoral judgment is deliberately narrower—and stronger—than either uncritical acceptance or dismissal. **The Bruce Maneuver is an author-originated, central training intervention with a falsifiable mechanistic hypothesis.** Its proposed foundation in component-specific upper-airway loading is biologically plausible because human upper-airway muscles respond to mechanical pressure perturbation, skeletal muscle possesses well-characterized mechanotransduction machinery, and independently studied upper-airway training programs can modify sleep-apnea outcomes. [20]

What has **not** yet been established is the crucial intervention-specific chain: that the Bruce Maneuver produces a defined local intrapharyngeal stimulus; that this stimulus causes beneficial rather than neutral or maladaptive chronic remodeling; that the resulting adaptation survives the transition from waking voluntary performance to automatic function during sleep; and that those changes produce clinically meaningful benefits beyond expectancy, generic respiratory practice, or already validated upper-airway exercise. The negative genioglossus-strengthening trial, pressure-polarity findings, sleep-related attenuation of airway reflexes, and heterogeneous 2026 training meta-analysis make these genuine empirical questions rather than formalities. [21]

TRIE 5.4.0 revision 3 strengthens this Part not by declaring any of those propositions true, but by enforcing the correct epistemic architecture: preserve Bruce's intervention as Bruce defined it; preserve the exact source behind each claim; keep later writer contributions distinct; distinguish exact readback from interpretation; and do not promote package integrity into semantic or clinical certification. filecite turn5file1 filecite turn5file9 filecite turn5file4 That is the appropriate foundation for **PART I — CORE THESIS, MECHANICS AND ADAPTATION**: the Bruce Maneuver remains central, but its strongest scholarly form is now a precisely attributable, experimentally decidable theory of **upper-airway stimulus → mechanotransduction → recovery-dependent adaptation → unsupervised sleep-state function**, with clinical efficacy and safety reserved for direct measurement rather than assumed from analogy.

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[1] [20] Evidence for reflex upper airway dilator muscle activation by sudden negative airway pressure in man
https://pubmed.ncbi.nlm.nih.gov/2061830/?utm_source=chatgpt.com

[2] Mechanisms contributing to the response of upper-airway muscles to changes in airway pressure
https://pubmed.ncbi.nlm.nih.gov/25749447/?utm_source=chatgpt.com

[3] Influence of upper airway pressure changes on genioglossus muscle respiratory activity.
https://pubmed.ncbi.nlm.nih.gov/7037716/?utm_source=chatgpt.com

[4] Changes in pharyngeal collapsibility and genioglossus reflex responses to negative pressure during the respiratory cycle in obstructive sleep apnoea
https://pubmed.ncbi.nlm.nih.gov/31782971/?utm_source=chatgpt.com

5 13 Regional genioglossus reflex responses to negative pressure pulses in people with obstructive sleep apnea.
https://pubmed.ncbi.nlm.nih.gov/35771222/?utm_source=chatgpt.com

6 Effect of cyclic stretch on beta1D-integrin expression and activation of FAK and RhoA
https://pubmed.ncbi.nlm.nih.gov/17267546/?utm_source=chatgpt.com

7 The effect of sleep on reflex genioglossus muscle activation by stimuli of negative airway pressure in humans.
https://pubmed.ncbi.nlm.nih.gov/8046629/?utm_source=chatgpt.com

8 Genioglossus reflex inhibition to upper-airway negative-pressure stimuli during wakefulness and sleep in healthy males
https://pubmed.ncbi.nlm.nih.gov/17395627/?utm_source=chatgpt.com

9 Effects of oropharyngeal exercises on patients with moderate obstructive sleep apnea syndrome
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10 Didgeridoo playing as alternative treatment for obstructive sleep apnoea syndrome: randomised controlled trial.
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11 16 Efficacy of therapies based on muscle strength training among individual with obstructive sleep apnea - a systematic review and meta-analysis - PubMed
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12 21 A Pilot Randomized Controlled Trial of Effect of Genioglossus Muscle Strengthening on Obstructive Sleep Apnea Outcomes.
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14 Guidelines at-a-Glance
https://aasm.org/wp-content/uploads/2019/02/TreatmentOSAwithPAP-Guideline-at-a-Glance.pdf?utm_source=chatgpt.com

15 Upper airway collapsibility measured using a simple wakefulness test closely relates to the pharyngeal critical closing pressure during sleep in obstructive sleep apnea
https://pubmed.ncbi.nlm.nih.gov/30946461/?utm_source=chatgpt.com

17 19 The cerebrovascular response to graded Valsalva maneuvers while standing.
https://pubmed.ncbi.nlm.nih.gov/24744902/?utm_source=chatgpt.com

18 Valsalva Maneuver in Pulmonary Arterial Hypertension: Susceptibility to Syncope and Autonomic Dysfunction - PubMed
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