Biochemical Mechanisms Linking Mouth Breathing, Airway Obstruction, and Bedwetting

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Airway fragmentation commonly seen as mouth breathing, airway occlusion, apnoea, and hypopnea creates a complex cascade of biochemical and physiological mechanisms that culminate in nocturnal enuresis (bedwetting).

These pathways involve hormonal dysregulation, cardiovascular stress, renal dysfunction, neurological impairment, and metabolic disturbances. Below is a comprehensive description of each mechanism, with emphasis on cortisol, adrenaline, kidney function, and blood pressure regulation mechanisms.

1. Hormonal Pathway: Atrial and Brain Natriuretic Peptide (ANP/BNP) Release

Mechanism

When a child experiences upper airway obstruction during sleep, they generate exaggerated inspiratory efforts against the occluded airway in an effort to maintain oxygen supply. This creates extreme negative intrathoracic pressure (ranging from -15 to -17 mmHg), which dramatically increases venous return to the right side of the heart. The resulting atrial and ventricular wall stretch triggers myocardial cells to release ANP from the atria and BNP from the ventricles.

Effect on Kidneys

ANP and BNP are potent natriuretic hormones that act directly on the kidneys to promote sodium and water excretion. They inhibit the renin-angiotensin-aldosterone system (RAAS), reduce antidiuretic hormone (ADH) release, and increase glomerular filtration while promoting diuresis. This leads to nocturnal polyuria—excessive nighttime urine production that exceeds bladder capacity.

Effect on Blood Pressure

While ANP and BNP have vasodilatory properties, their blood pressure effects are largely overridden by simultaneous sympathetic activation in sleep apnoea. However, they do contribute to pressure-induced natriuresis following the repetitive blood pressure surges that occur at the termination of each apnoeic event.

Evidence

Studies show that ANP levels increase during apnoeic periods in direct correlation with apnoea-hypopnea index (AHI) severity. Morning BNP levels are elevated in children with sleep-disordered breathing (or is it Breathing disordered Sleep?) and enuresis, with odds ratios of 4.6 for severe OSA (AHI ≥30). Importantly, these elevations resolve with effective treatment through adenotonsillectomy or CPAP therapy, and we are starting to see similar results from the release of tethered oral tissues (TOT’s) and orthodontics to rectify airway.

Cardiac stretch mechanism: Airway obstruction causes negative intrathoracic pressure, stretching heart chambers and triggering ANP/BNP release, leading to increased kidney urine production

2. Hormonal Pathway: Antidiuretic Hormone (ADH/Vasopressin) Suppression

Mechanism

Normal sleep triggers increased ADH secretion, which concentrates urine and reduces nighttime urine volume. However, sleep fragmentation from repetitive apnoea's and hypopneas disrupts the normal circadian rhythm of ADH secretion. Additionally, elevated ANP and BNP directly suppress ADH release.

Effect on Kidneys

Reduced ADH levels impair the kidney's ability to reabsorb water in the collecting ducts, resulting in dilute urine and increased urine volume during the night. This nocturnal polyuria contributes significantly to bladder overfilling.

Effect on Blood Pressure

ADH (vasopressin) normally has vasoconstrictive properties, so its suppression may contribute to altered nocturnal blood pressure patterns, though this effect is minor compared to sympathetic activation.

Evidence

Research demonstrates that enuretic children with OSA have blunted nocturnal ADH secretion compared to non-enuretic controls. The ADH circadian rhythm fails to show the normal nighttime peak, contributing to the "non-dipping" blood pressure pattern observed in these patients.

3. Hormonal Pathway: Cortisol and Catecholamines (Stress Response, i.e. The Bear is in the Room to choke me).

Mechanism

The cycle of breathing cessation, hypoxia, and repeated arousals activates both limbs of the stress system: the hypothalamic-pituitary-adrenal (HPA) axis and the sympathoadrenal system. Nocturnal hypoxia and sleep fragmentation serve as potent stressors, triggering pulsatile cortisol release with each arousal. Simultaneously, the adrenal medulla releases epinephrine (adrenaline), while sympathetic nerve terminals release norepinephrine (noradrenaline). My hypothesis is that these are part of the survival response to stiffen and tighten the airway, or to arouse the patient out of the phase of sleep that the airway occlusion/fragmentation occurs in and start breathing again.

Effect on Kidneys

Cortisol: While cortisol's direct renal effects in sleep apnoea are complex and still being elucidated, chronic HPA axis activation may contribute to altered renal function and impaired bladder control mechanisms. Cortisol influences blood sugar balance, insulin, glycation and immune activation as well as fluid balance. While it also can affect the sensitivity of bladder receptors.

<u>Catecholamines</u>: Noradrenaline (norepinephrine) and adrenaline (epinephrine) cause renal vasoconstriction, alter glomerular filtration rate, enhance proximal tubular sodium reabsorption, and stimulate renin secretion. Studies show that 24-hour urinary noradrenaline levels are elevated approximately 45% in children with sleep apnoea (58.2 ng vs. 40.2 ng in controls), with levels correlating directly with AHI severity.

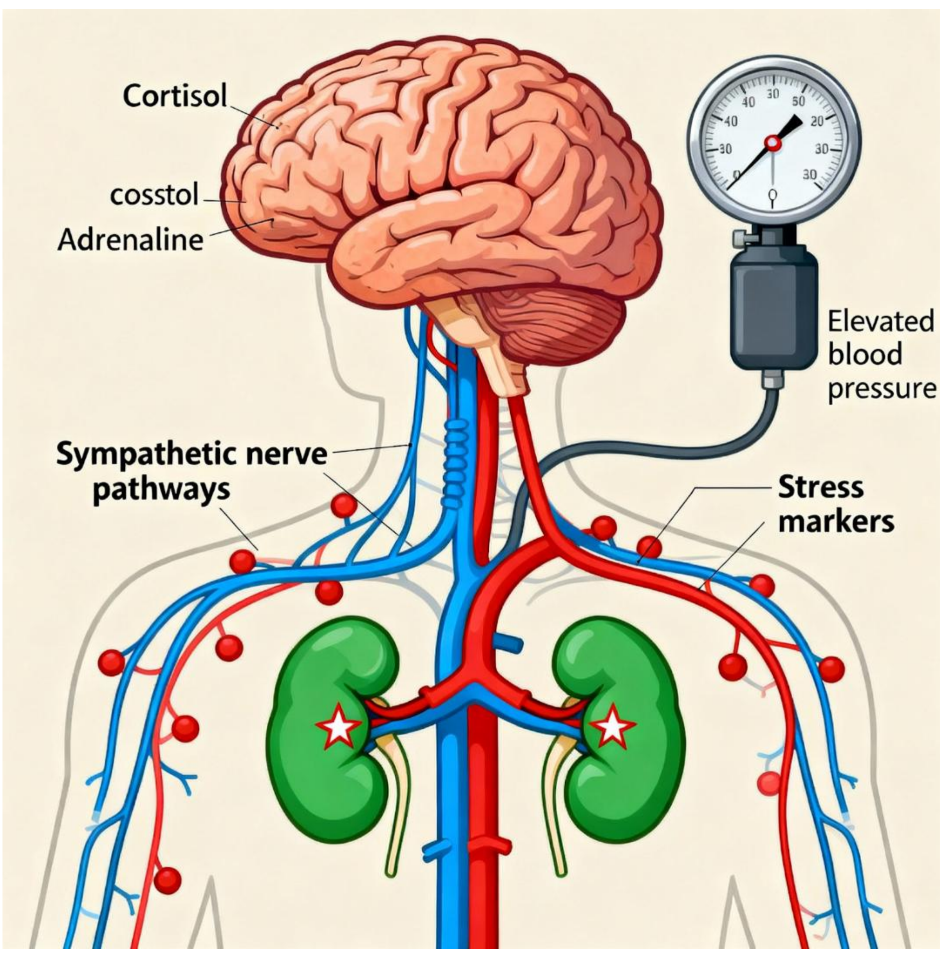
Effect on Blood Pressure

This is where the stress hormones have their most dramatic effect. Catecholamines cause systemic vasoconstriction, increasing peripheral vascular resistance and heart rate. Elevated plasma and urinary catecholamines contribute to sustained daytime hypertension in OSA patients, with systolic blood pressure elevated by 5.87 mmHg and diastolic by 3.40 mmHg compared to controls. Enhanced chemoreceptor sensitivity from chronic intermittent hypoxia perpetuates excessive sympathetic vasoconstrictor activity even during daytime.

Evidence

Muscle sympathetic nerve activity (MSNA) measurements confirm elevated sympathetic discharge in OSA patients. Urinary catecholamine measurements show significant correlations between adrenaline/noradrenaline levels and obstructive apnoea-hypopnea indices, particularly with the degree of nocturnal hypoxemia. These markers decrease following CPAP treatment.

Stress hormone pathway: Hypoxia activates HPA axis and sympathetic nervous system, releasing cortisol and catecholamines that affect kidney function and blood pressure regulation.



4. Cardiovascular Pathway: Negative Intrathoracic Pressure Effects

Mechanism

Obstructive apnoea's create a unique cardiovascular challenge. During futile inspiratory efforts against the occluded pharyngeal tissues, extreme negative intrathoracic pressure develops. This immediately increases left ventricular (LV) transmural pressure, this being the difference between intracardiac and extracardiac pressure. This is a key component of LV afterload.

The negative pressure also increases venous return to the right ventricle, leading to its distension. The resulting leftward shift of the interventricular septum impedes LV diastolic filling. This combination of increased LV afterload and reduced LV preload causes progressive reduction in stroke volume and cardiac output during obstructive events.

Effect on Kidneys

The increased venous return and atrial stretch directly trigger ANP and BNP release, as described in mechanism 1 above. The cardiac chambers experience acute volume overload with each apnoeic event, promoting continuous natriuretic peptide secretion that drives excessive urinary sodium and water excretion.

Effect on Blood Pressure

During the initial phase of an obstructive apnoea, blood pressure may actually drop due to reduced stroke volume. However, toward the end of apnoea event and immediately following their termination, blood pressure surges occur due to sympathetic activation and the sudden restoration of cardiac output. These repetitive nocturnal blood pressure spikes contribute to sustained daytime hypertension and a "non-dipping" pattern where blood pressure fails to decrease normally during sleep.

Evidence

Hemodynamic studies document intrathoracic pressure swings reaching -15 to -17 mmHg during obstructive events, with corresponding increases in right atrial transmural pressure. These pressure changes correlate with increases in ANP secretion and urinary cGMP (a marker of ANP activity).

5. Renal Pathway: Renin-Angiotensin-Aldosterone System (RAAS) Activation

Mechanism

Chronic intermittent hypoxia and sustained sympathetic activation stimulate renin secretion from the juxtaglomerular cells of the kidney. Renin catalyses the conversion of angiotensinogen to angiotensin I, which is then converted to angiotensin II.

Angiotensin II stimulates aldosterone release from the adrenal cortex, completing the RAAS cascade.

Effect on Kidneys

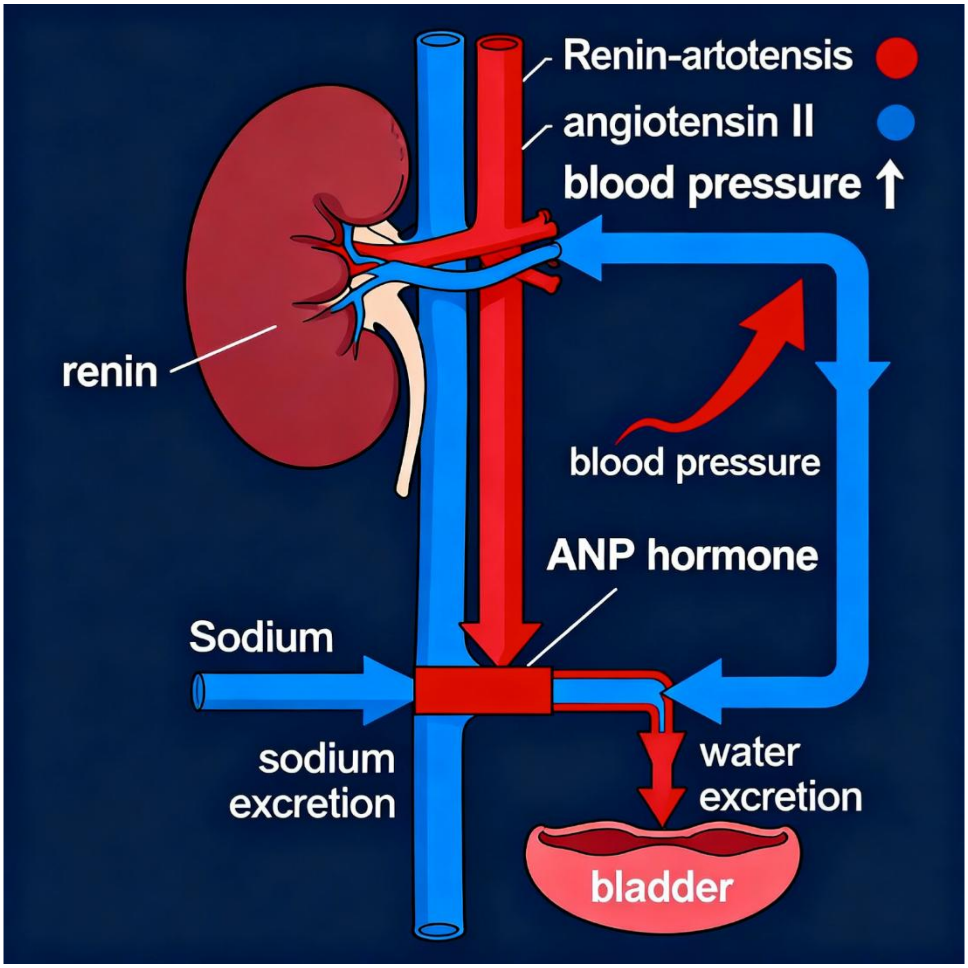
Under normal circumstances, angiotensin II and aldosterone promote sodium and water retention, which should reduce urine output. However, in sleep apnoea, the simultaneous elevation of ANP and BNP overwhelms this compensatory mechanism, and the natriuretic effects predominate. The fractional excretion of sodium increases in parallel with SDB severity.

Effect on Blood Pressure

RAAS activation is a major contributor to hypertension in OSA. Meta-analyses show that patients with OSA have significantly higher levels of plasma renin activity (pooled mean difference +0.25), plasma aldosterone (+30.79 ng/dL), and angiotensin II (+5.19 ng/L) compared to controls. Angiotensin II causes potent vasoconstriction, while aldosterone promotes fluid retention, both are mechanisms that elevate blood pressure. This pathway may be particularly important in OSA-related resistant hypertension.

Evidence

Studies demonstrate that RAAS hormone elevations are especially pronounced in hypertensive OSA patients compared to normotensive OSA patients. Treatment with CPAP can reduce RAAS activation, though the effect is variable. Currently there is a paucity of evidence for CPAP treatment in bed wetting children, however we might expect similar improvement to the RAAS hormonal system from TOT release and airway expansion orthodontics.



Renal hormonal pathway: RAAS activation increases blood pressure while ANP/BNP override this system, promoting excessive sodium and water excretion at night

6. Renal Pathway: Direct Hypoxic Effects on Kidney Function

Mechanism

Intermittent hypoxia from repeated apnoea's and hypopneas causes direct oxidative stress and inflammatory damage to renal tissues which are notably fragile. Hypoxia activates hypoxia-inducible factors (HIFs) and triggers production of reactive oxygen species (ROS), inflammatory cytokines, and fibrogenic factors.

Effect on Kidneys

Chronic renal hypoxia leads to tubular epithelial cell dysfunction, impaired tubular reabsorption, reduced urine concentrating ability, and progressive tubulointerstitial damage. The proximal tubule, which is packed with mitochondria and dependent on oxidative phosphorylation, is particularly vulnerable. Sympathetic renal nerve activation during apnoeic events further reduces renal blood flow, exacerbating hypoxic damage.

Effect on Blood Pressure

Renal hypoxia contributes to hypertension through multiple mechanisms: endothelial dysfunction, impaired pressure-natriuresis relationships, enhanced sympathetic outflow from kidney to central nervous system, and progression of chronic kidney disease. The kidney's role as an oxygen sensor may link renal hypoxia directly to increased sympathetic drive and elevated blood pressure.

Evidence

Experimental studies using lower body negative pressure to simulate sympathetic activation show decreased renal blood flow and reduced renal perfusion, though direct measurement of renal tissue oxygenation in human OSA remains challenging, few seem excited to have an experimental sensor placed in the kidney to directly measure the O2 level.

7. Neurological Pathway: Elevated Arousal Threshold

Mechanism

Paradoxically, while OSA causes sleep fragmentation through repeated brief arousals, chronic sleep disruption leads to increased sleep pressure and an elevated arousal threshold. Children with enuresis experience more sleep fragmentation in their habitual environment, making them more sleep-deprived and creating a higher threshold for awakening, (it's harder to stimulate the child to wake).

Polysomnographic studies show that enuretic children have increased slow-wave activity (indicated by higher cyclic alternating pattern rates in stage N3 sleep) but decreased phase A2 and A3 indexes, which reflect high-power arousals. This means they have difficulty transitioning from deep sleep even when bladder signals are present.

Effect on Kidneys and Bladder

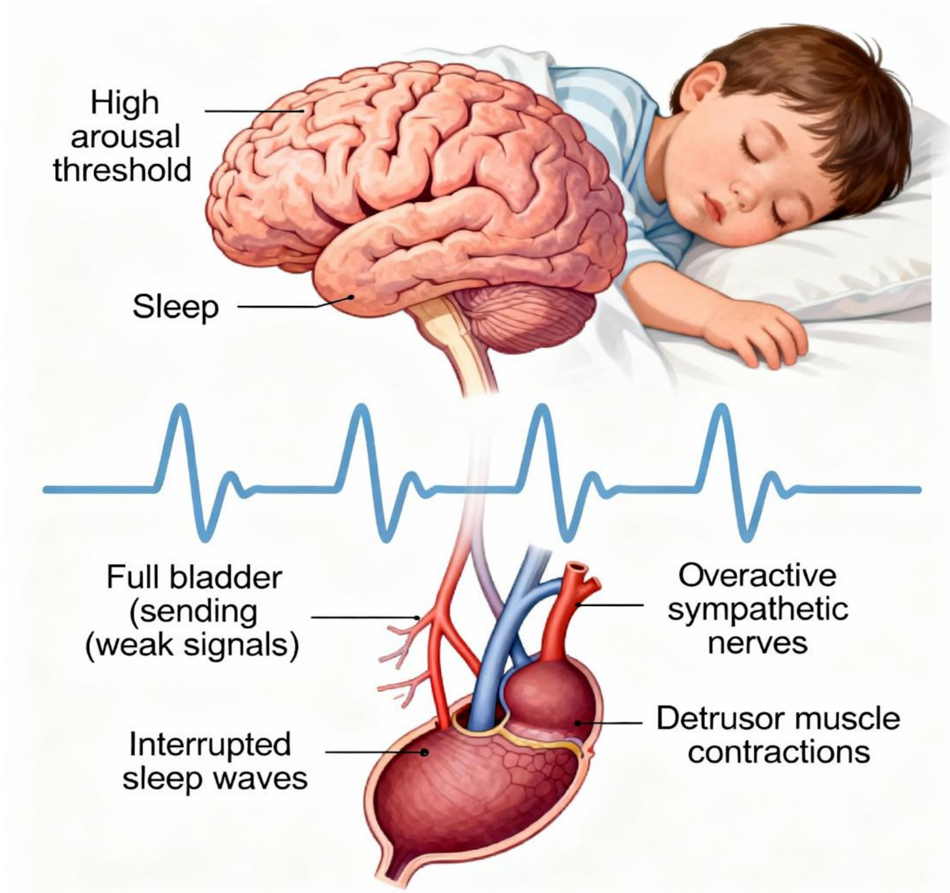
Children with elevated arousal thresholds have decreased sensitivity to bladder filling or detrusor contraction, the signals that normally trigger the urge to void. When the bladder reaches capacity, the usual arousal mechanisms fail, and the child remains in deep sleep.

Effect on Blood Pressure

The arousal threshold elevation itself doesn't directly affect blood pressure, but the autonomic dysfunction associated with sleep fragmentation contributes to the overall cardiovascular dysregulation.

Evidence

Sleep studies demonstrate that enuresis episodes typically occur during non-REM sleep transitions (N3→N2 or N2→N1), when arousal signals are present but insufficient to wake the child. The arousal threshold is demonstrably higher in enuretic children with OSA compared to non-enuretic controls.



Neurological arousal mechanism: Sleep fragmentation elevates arousal threshold while sympathetic overactivation causes bladder overactivity, preventing the child from waking to bladder signals

8. Neurological Pathway: Sympathetic Nervous System Overactivation

Mechanism

As detailed in mechanism 3 above, intermittent hypoxia, increased respiratory effort, and repeated arousals drive sustained sympathetic nervous system overactivation. This represents a fundamental autonomic imbalance in children with sleep-disordered breathing (breathing-disordered Sleep) and enuresis.

Effect on Kidneys and Bladder

Sympathetic activation has direct effects on both renal and bladder function. In the kidneys, increased sympathetic nerve activity alters renal blood flow, modulates renin secretion, and enhances sodium reabsorption. In the bladder, chronic sympathetic overactivation contributes to detrusor overactivity and bladder instability.

Animal models and human studies show that OSA-related sympathetic activation can cause oxidative stress in bladder tissue, leading to ultrastructural changes in the detrusor muscle and increased bladder contractility.

Effect on Blood Pressure

Sympathetic overactivation is the primary driver of hypertension in OSA. Elevated MSNA persists even during daytime wakefulness, causing sustained vasoconstriction, increased peripheral vascular resistance, and elevated heart rate. This contributes to the characteristic "non-dipping" blood pressure pattern where nighttime blood pressure fails to decrease normally.

Evidence

Microneurography studies directly measure elevated MSNA in OSA patients compared to controls. Twenty-four-hour blood pressure monitoring reveals non-dipping patterns in children with enuresis and OSA. Bladder studies demonstrate associations between OSA severity, oxygen desaturation, and overactive bladder symptoms.

9. Bladder Pathway: Detrusor Overactivity and Instability

Mechanism

Chronic intermittent hypoxia causes oxidative stress directly in bladder tissues. Animal models of sleep apnoea demonstrate that sustained intermittent hypoxia produces ultrastructural changes in bladder smooth muscle cells, including mitochondrial dysfunction, increased reactive oxygen species production, and altered calcium handling.

Effect on Bladder Function

These cellular changes manifest as detrusor overactivity, premature, involuntary contractions of the bladder muscle during filling. The functional bladder capacity is thus reduced, and the bladder becomes unstable, contracting before it reaches normal volume. Studies show a direct correlation between bladder wall thickness (a marker of chronic detrusor hypertrophy) and overactive bladder symptom scores in OSA patients.

Effect on Bedwetting

Detrusor overactivity combines with the elevated arousal threshold (mechanism 7 above) to create the perfect storm for bedwetting. The bladder contracts prematurely while the child (and potentially the elderly) is unable to wake in response to these contractions, resulting in involuntary voiding during sleep.

Evidence

Clinical studies demonstrate that OSA is associated with significantly higher overactive bladder symptom scores. The severity of urgency (a key Over Active Bladder symptom) correlates with the degree of nocturnal hypoxemia, specifically, lower minimum oxygen saturation and longer time spent below 90% oxygen saturation. Animal models confirm that intermittent hypoxia produces measurable detrusor instability and nocturia-like voiding patterns.

10. Metabolic Pathway: Respiratory Alkalosis from Mouth Breathing

Mechanism

Mouth breathing, particularly chronic mouth breathing, often involves elements of hyperventilation that lead to excessive CO_2 loss. Normal nasal breathing allows for optimal CO_2/O_2 exchange, but mouth breathing disrupts this balance. The reduction in arterial CO_2 (hypocapnia) decreases carbonic acid (H_2CO_3) formation, which shifts blood pH toward alkaline (>7.45).

Effect on Kidneys

To compensate for respiratory alkalosis, the kidneys excrete bicarbonate in an attempt to restore normal blood pH. However, this compensatory mechanism has negative consequences: the kidneys must also release essential minerals including magnesium, sodium, phosphorus, and calcium into the urine. These minerals are critical for cellular energy production (ATP synthesis) and tissue mineralization.

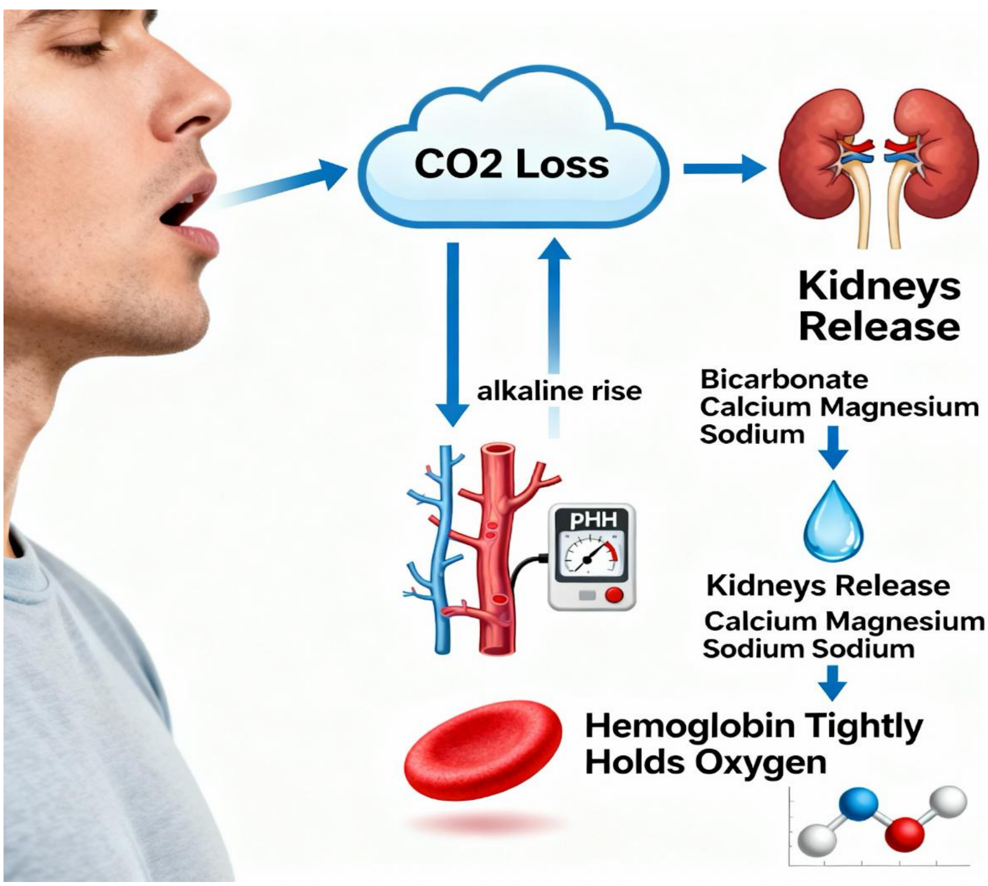
Additionally, the more alkaline blood pH creates a stronger bond between haemoglobin and oxygen in red blood cells. This means that even though oxygen levels may appear normal, the red blood cells cannot easily release oxygen to tissues, nor can they efficiently capture CO2 from cells. This creates a paradoxical tissue hypoxia despite adequate oxygen saturation.

Effect on Blood Pressure and Overall Function

The impaired oxygen delivery to tissues perpetuates cellular dysfunction throughout the body. The mineral depletion affects cardiovascular function, neuromuscular function, and renal concentrating ability.

Evidence

Studies demonstrate that healthy subjects experience a 42% increase in net water and energy loss when switching from nasal to oral breathing. Mouth breathing is associated with reduced oxygen absorption and increased CO2 concentration, with related acid-base disturbances.



Respiratory alkalosis mechanism: Mouth breathing causes CO2 loss and alkaline blood pH, triggering renal compensation with mineral loss and impaired oxygen delivery to tissues

Integrated Cascade: How All Mechanisms Converge

These mechanisms don't operate in isolation, they form an interconnected cascade where each pathway amplifies the others:

- Airway obstruction → Negative intrathoracic pressure + Hypoxia
- Negative pressure → Cardiac stretch → ANP/BNP release → Renal natriuresis
- Hypoxia + Arousals → HPA axis activation → Cortisol + Catecholamines → Vasoconstriction + Hypertension + Renal dysfunction
- Sympathetic activation → RAAS stimulation → Angiotensin II + Aldosterone (but overwhelmed by ANP)
- Intermittent hypoxia → Direct renal hypoxic damage + Bladder oxidative stress → Detrusor overactivity
- Sleep fragmentation → Elevated arousal threshold + Disrupted ADH secretion
- Mouth breathing → Respiratory alkalosis → Renal mineral loss + Impaired tissue oxygenation
- Combined effects → Nocturnal polyuria + Bladder overactivity + Failure to wake → Bedwetting

Complete integrated pathway: All five major mechanisms (cardiac, stress, renal-hormonal, neurological, and metabolic) work together to cause nocturnal enuresis in children with airway obstruction

Clinical Implications

Understanding these mechanisms has important therapeutic implications. Treatment of the underlying airway obstruction through adenotonsillectomy resolves bedwetting in 68-85% of affected children. CPAP therapy in adults similarly improves or eliminates nocturia and enuresis by:

Normalizing intrathoracic pressure (reducing ANP/BNP release)

Eliminating hypoxic episodes (reducing sympathetic activation and oxidative stress)

Restoring normal sleep architecture (improving arousal mechanisms and ADH secretion)

Reducing blood pressure and RAAS activation

Allowing bladder tissue to recover from oxidative damage

The effectiveness of these treatments provides strong evidence that bedwetting in the context of sleep-disordered breathing is not a behavioural or maturational issue, but rather a consequence of specific, measurable biochemical and physiological derangements.

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References:

Hormonal & Endocrine Mechanisms:

Association between primary nocturnal enuresis and obstructive sleep apnoea - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC3400898/

Relationship between enuresis and obstructive sleep apnoea - PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC7727060/

Nocturnal enuresis is associated with moderate-to-severe obstructive sleep apnoea - Nature
https://www.nature.com/articles/pr2014137

Enuresis and upper airway obstruction: BNP and ADH levels - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC9747030/

Relationship Between Obstructive Sleep Apnoea and Enuresis in Children - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC11804225/

Mechanism of atrial natriuretic peptide release with increased intrathoracic pressure - PubMed
https://pubmed.ncbi.nlm.nih.gov/8783640/

Vasopressin as a Possible Link between Sleep-Disturbances and Cardiovascular Disease - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC9778878/

Changes of circulating atrial natriuretic peptide and antidiuretic hormone - PubMed
https://pubmed.ncbi.nlm.nih.gov/1439228/

Association between Obstructive Sleep Apnoea and Elevated Levels of Circulating BNP - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC3466799/

Stress Response & HPA Axis:

Sleep Apnoea and its association with the Stress System - PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC2128620/

Cortisol and Sleep: Can It Cause Insomnia? - Healthline https://www.healthline.com/health/cortisol-and-sleep

Hypothalamic-Pituitary-Adrenal (HPA) Axis - Cleveland Clinic
https://my.clevelandclinic.org/health/body/hypothalamic-pituitary-adrenal-hpa-axis

Sympathetic Nervous System & Catecholamines:

Impact of Obstructive Sleep Apnoea and Sympathetic Nervous System - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC11277108/

Association between nocturnal activity of the sympathetic nervous system and enuresis - Nature
https://www.nature.com/articles/s41598-021-91329-6

Sympathetic and Catecholaminergic Alterations in Sleep Apnoea - Frontiers
https://www.frontiersin.org/journals/neurology/articles/10.3389/fneur.2012.00007/full

The Effect of Sleep Apnoea on Plasma and Urinary Catecholamines - Academic OUP
https://academic.oup.com/sleep/article-pdf/18/5/377/8698442/sleep-18-5-377.pdf

Level of urinary catecholamine in children with Sleep Disordered Breathing - PubMed
https://pubmed.ncbi.nlm.nih.gov/36327585/

Cardiovascular Mechanisms:

Sleep Apnoea and Cardiovascular Disease - American Thoracic Society
https://www.atsjournals.org/doi/10.1164/ajrccm.164.12.2107045

Intrathoracic pressure swings induced by simulated obstructive apnoea - Europace
https://academic.oup.com/europace/article/18/1/64/2398588

Sleep Apnoea and Cardiovascular Disease - Circulation AHA
https://www.ahajournals.org/doi/10.1161/circulationaha.111.070813

Renin-Angiotensin-Aldosterone System (RAAS):

Influence and implications of the renin-angiotensin-aldosterone system - Monash Research
https://research.monash.edu/en/publications/influence-and-implications-of-the-reninangiotensinaldosterone-sys

Influence and implications of the renin-angiotensin-aldosterone system - PubMed
https://pubmed.ncbi.nlm.nih.gov/36104933/

Meta-analysis of effects of obstructive sleep apnoea on the renin-angiotensin-aldosterone system - PMC
https://pmc.ncbi.nlm.nih.gov/articles/PMC4921546/

Renal Function & Kidney Effects:

Sympathetic activation by lower body negative pressure decreases renal perfusion - PubMed
https://pubmed.ncbi.nlm.nih.gov/30390156/

Hypoxia and chronic kidney disease - PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC8921539/

Editorial: Hypoxia in Kidney Disease - Frontiers
https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2018.00485/full

Glomerular haemodynamics, the renal sympathetic nervous system and hypertension - NDT
https://academic.oup.com/ndt/article/29/12/2178/1851067

Neurological & Sleep Architecture:

Sleep Monitoring of Children With Nocturnal Enuresis - Frontiers
https://www.frontiersin.org/journals/pediatrics/articles/10.3389/fped.2021.701251/full

The role of the autonomic nervous system in nocturnal enuresis - Science Direct
https://www.sciencedirect.com/science/article/abs/pii/S1477513122004132

Non-REM Sleep Instability in Children With Primary Nocturnal Enuresis - JCSM
https://jcsm.aasm.org/doi/10.5664/jcsm.6762

Sleep and Sleepiness in Children with Nocturnal Enuresis - Academic OUP
https://academic.oup.com/sleep/article/34/2/191/2433803

Enuresis - StatPearls - NCBI Bookshelf https://www.ncbi.nlm.nih.gov/books/NBK545181/

Bladder Function & Detrusor Activity:

Bladder Oxidative Stress in Sleep Apnoea Contributes to Detrusor Instability - Science Direct https://www.sciencedirect.com/science/article/abs/pii/S0022534714049015

Obstructive Sleep Apnoea Syndrome Is Associated with Overactive Bladder - PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC2635592/

Overactive bladder syndrome and bladder wall thickness in patients with OSA - SciELO https://www.scielo.br/j/ibju/a/CLjyyVkdPqg6knyJLCqztwS/?lang=en

Mouth Breathing & Respiratory Alkalosis:

Understanding the Pathophysiology of an oral habit - ES Med https://esmed.org/MRA/mra/article/download/3478/99193546630

Mouth breathing and its relationship to some oral and medical conditions - Dialnet https://dialnet.unirioja.es/descarga/articulo/7000577.pdf

Respiratory Alkalosis: Symptoms, Treatments, and Prevention - Healthline https://www.healthline.com/health/respiratory-alkalosis

Respiratory Alkalosis: Causes, Symptoms & Treatment - Cleveland Clinic https://my.clevelandclinic.org/health/diseases/21657-respiratory-alkalosis

General Pathophysiology & Reviews:

Pathophysiology of sleep apnoea - PubMed https://pubmed.ncbi.nlm.nih.gov/20086074/

The Link Between Bedwetting and Sleep-Disordered Breathing - Future Dental https://www.futuredental.com.au/understanding-the-link-between-bedwetting-and-sleep-disordered-breathing/

How are Sleep Apnoea and Bedwetting Connected? - Sleepless in Arizona https://sleeplessinarizona.com/how-are-sleep-apnoea-and-bedwetting-connected/

Nocturia - a guide to assessment and management - RACGP https://www.racgp.org.au/afp/2012/june/nocturia-a-guide-to-assessment-and-management

Sleep Apnoea and Bed Wetting: What to Know - SleepQuest https://www.sleepquest.com/sleep-apnoea-and-bed-wetting/

Adenotonsillectomy can decrease enuresis - Science Direct https://www.sciencedirect.com/science/article/abs/pii/S147751311630331X

The Connection Between Sleep Apnoea and Nocturia - Alliance Urology https://allianceurology.com/connection-between-sleep-apnoea-and-nocturia/