

Restoration of Healthy Natural Nasal Breathing as Therapy of Sleep Apnea

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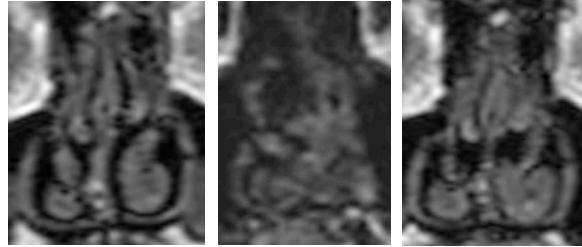
Convenience translation of original poster in German language at DGSM 2019 Congress, Hamburg, Germany

The role of impaired nasal breathing as a cause of sleep apnea is increasingly recognized. Fluid mechanical constrictions in the nasal passages may represent a root cause for obstructions in the pharynx. Highest flow velocity and highest amount of soft tissue (throat walls, soft palate) are present in the naso- and velopharynx. That makes this region prone to occurrence of suction phenomena. Furthermore, the receptors for control of pharyngeal muscle tone and breathing control are located in the nasal passages (e.g. Baraniuk and Merck 2008, Poirrier et al. 2013).

Moreover, the highest amount of nitric oxide (NO) is produced in the sinuses (up to 1.000x more than in vessel endothelium) and transported from there into the lung.

Some of the essential functions of NO in the body:

- Vasodilator (1998 Nobel Prize)
- Neurotransmitter
- Redox balance: strong antioxidant
- Regulation of diverse critical metabolic pathways (inflammatory, antioxidants, cancer)
- Respiratory cycle (prerequisite for O₂ release)
- Control of circulatory system and muscle function



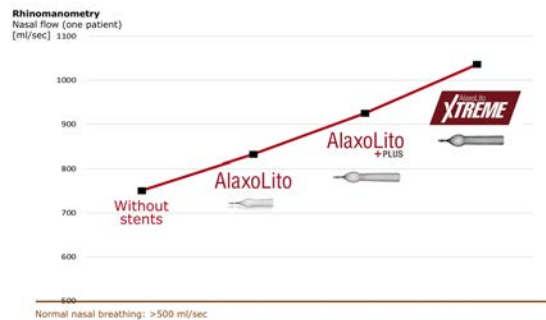
MRI: left normal, middle very narrow, right stent-optimized nasal passages

Cause	Effect
Narrowed nasal passages	Increased breathing resistance, reduced flow volume, increased flow velocity, „jet stream“ and turbulent flow (e.g. Porrier et al. 2013; numerous CFD studies)
Increased flow velocity	Relaxation of airflow in nasopharynx → naso- and velopharyngeal negative pressure, turbulences (numerous CFD studies)
„Sucking“ by lung against nasal stenosis	Negative pressure in pharynx
Pharyngeal negative pressure	Vibrations, suction phenomena → obstructions (hypopneas, apneas), particularly in region of velum → high incidence of concentric and antero-posterior collapse
Decreased nasal breathing	Instable mouth breathing, reduced nasal-ventilatory reflexes (breathing receptors are not sufficiently triggered) → decoupling of pharyngeal and thoracic muscles, reduced muscle tone in pharynx → promotes obstructions → important role in emergence of OSA and fragmented sleep (Poirrier et al. 2013, Torre et al. 2017)
Reduced nasal flow	NO deprivation in lung, circulatory system, organs and muscles → reduced O ₂ -release from hemoglobin

- CPAP increases the diameter of narrowed nasal passage but concurrently reduces that of the wider nasal passage (tested in healthy volunteers) (White et al. 2016) → nasal breathing is not improved
 - CPAP leads to pressure-induced inflammation in the nose and systemically in the entire body by triggering of cytokines (tested in healthy volunteers) (AlAhmari et al. 2012) → inflamed nasal mucosa leads to decreased nasal breathing
 - In case of nasal obstruction CPAP with 10 mbar leads to increased flow velocity (jet stream) and higher pressure drop in the nasopharynx than in OSA patients with normal nasal passages (Wakayama et al. 2016) → high pressure required which, however, is not delivered to the pharynx
 - NO controls (increases) mucociliary beat frequency; CPAP reduces mucociliary beat frequency and entrainment of NO from the sinuses (Whittington et al. 2018); plasma NO level lower than normal (Schulz et al. 2000)
 - NO as a neurotransmitter is strongly involved in regulation of sleep, sleep stages and sleep quality (Cespuglio et al. 2012)
- In the future long term effects of reduced NO supply during CPAP therapy must be examined, only maintaining the airway patent is not sufficient (White et al. 2016)

Mechanical splinting of the nasal airway leads to improved nasal flow and optimized fluid dynamics in the nose (Zhang and Kotecha 2019). Nasal breathing optimized by the AlaxoLito Plus Nasal Stent leads to higher NO transport from the sinuses into the lung, optimized microcirculation (Bizjak et al. 2019) and increased parasympathetic activity (Pyschny 2017; Lellau 2017).

Natural nasal breathing can be restored and occurrence of obstructions can be counteracted by mechanical splinting of the nasal airway. A normal nasal flow is required to ensure a sufficient transport of NO from the sinuses into the lung and to achieve good sleep quality.



The dynamic understanding of the fluid mechanical situation for the individual patient is absolutely necessary for a sleep apnea therapy according to the state of the art and with high therapeutic efficiency as well as high compliance. CPAP users with decreased nasal breathing may benefit from the use of a nasal stent during CPAP therapy. It has to be investigated for which OSA patients use of a nasal stent alone may reduce or totally eliminate obstructions by optimized nasal breathing and fluid mechanics.

Conflict of interest:

The author is shareholder and CEO of Alaxo GmbH which develops, manufactures and sells nasal stent.

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