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Vagus Nerve Stimulation as a Therapeutic Strategy for Bronchoconstriction and Airway Hyperreactivity

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Introduction

Bronchoconstriction and Airway Hyperreactivity (AHR) are hallmark features of obstructive airway diseases such as asthma, Chronic Obstructive Pulmonary Disease (COPD) and certain forms of allergic bronchitis. These conditions significantly impact global health, contributing to substantial morbidity, healthcare costs and reduced quality of life. Traditional therapies-bronchodilators, corticosteroids and leukotriene modifiers-while effective in many cases, often fail to provide adequate symptom control in severe or treatment-resistant patients. Furthermore, chronic pharmacologic treatment is often associated with side effects and diminished efficacy over time [1].

The field of bioelectronic medicine is rapidly evolving and several future directions are noteworthy. Integrating genomics, neuroimaging and biosensors to tailor VNS protocols. Potential synergistic effects with bronchodilators or anti-inflammatory drugs. Enabling long-term use and real-time monitoring. Including pulmonary fibrosis, sleep apnea and ventilator-induced lung injury. Advanced techniques to map vagal circuitry and optimize therapeutic targeting [2].

Description

Recent advances in neuroimmunology and bioelectronic medicine have introduced novel therapeutic paradigms targeting neural pathways to modulate immune and inflammatory responses. One of the most promising of these is Vagus Nerve Stimulation (VNS)-an approach traditionally used in the management of epilepsy and depression, but now under investigation for inflammatory and respiratory conditions. Given the vagus nerve's integral role in autonomic control of airway tone, mucus secretion and immune regulation, VNS represents a compelling strategy to address bronchoconstriction and AHR. This explores the anatomical and physiological basis of vagus nerve involvement in respiratory control, reviews preclinical and clinical evidence supporting VNS for airway modulation and discusses potential mechanisms, device technologies and future applications [3].

The vagus nerve, or cranial nerve X, is the principal parasympathetic nerve and a key regulator of visceral function. It comprises both afferent (sensory) and efferent (motor) fibers that innervate the lungs, heart, gastrointestinal tract and other visceral organs. Approximately 80% of vagal fibers are afferent, transmitting sensory signals from peripheral organs to the brainstem, while the remaining efferent fibers control organ function through parasympathetic outputs. Vagal efferents stimulate muscarinic receptors, causing bronchoconstriction. Mediating cough, reflex bronchospasm and inflammation. Vagal sensory fibers respond to chemical (e.g., histamine, prostaglandins) and mechanical stimuli (e.g., stretch), initiating reflex arcs that modulate bronchomotor tone and inflammation. Bronchoconstriction refers to the narrowing of airways due to smooth muscle contraction, often accompanied by increased mucus secretion and edema. In asthma and related conditions, hyperresponsive airways react excessively to triggers such as allergens, cold air, pollutants, or exercise [4].

These alterations create a self-reinforcing loop of inflammation, neural activation and bronchomotor dysfunction. VNS involves delivering low-voltage electrical impulses to the vagus nerve using implantable or transcutaneous devices. The proposed mechanisms by which VNS ameliorates bronchoconstriction and AHR include, activation of the cholinergic anti-inflammatory pathway (CAP), wherein acetylcholine released from vagal efferents binds to alpha-7 nicotinic acetylcholine receptors (α7nAChRs) on macrophages, suppressing pro-inflammatory cytokine release (e.g., TNF-α, IL-1β). Although vagal stimulation classically causes bronchoconstriction, low-frequency or selective stimulation may paradoxically reduce bronchomotor tone by modulating afferent signaling and central reflex circuits. Inhibiting exaggerated vagal sensory responses may attenuate cough, dyspnea and reflex bronchospasm. Chronic VNS may induce neuroplastic changes in brainstem nuclei (e.g., nucleus tractus solitarius) involved in autonomic and respiratory control. Numerous animal studies have demonstrated the efficacy of VNS in reducing airway inflammation and hyperreactivity. VNS reduced eosinophilic infiltration, airway hyperresponsiveness and Th2 cytokines (IL-4, IL-5, IL-13). Selective vagal stimulation attenuated methacholine-induced bronchoconstriction. VNS suppressed LPS-induced lung injury and inflammation via α7nAChRs. These studies highlight both the anti-inflammatory and neuromodulatory potential of VNS in respiratory disease contexts. Pilot studies using transcutaneous auricular VNS (taVNS) have shown improvements in lung function (FEV1), reduced dyspnea scores and decreased inflammatory biomarkers. Implanted VNS devices are under investigation in severe asthma patients resistant to pharmacotherapy [5].

Conclusion

Vagus nerve stimulation represents a groundbreaking approach in the management of bronchoconstriction and airway hyperreactivity. By modulating both neural and immune pathways, VNS offers a dual-action strategy that addresses the complex pathophysiology of obstructive airway diseases. Early evidence from preclinical and clinical studies supports its efficacy and safety, particularly when traditional therapies fall short. While challenges remain in terms of device optimization, patient stratification and long-term efficacy, the convergence of neuroscience, immunology and biomedical engineering heralds a new era in respiratory therapeutics. With continued research and clinical validation, VNS may soon become a valuable adjunct or alternative to conventional treatments in asthma, COPD and beyond.

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Conflict of Interest

None.

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