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ETIOPATHOGENETIC FEATURES OF VOCAL FOLD NODULES IN CHILDREN AGAINST THE BACKGROUND OF CHRONIC PHARYNGEAL DISEASES

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Abstract. Vocal fold nodules (VFN) represent the primary organic cause of pediatric dysphonia, traditionally attributed to chronic mechanical phonotrauma. However, recent clinical evidence suggests that the pediatric larynx does not exist in isolation; its health is inextricably linked to the upper aerodigestive tract. This thesis explores the complex etiopathogenetic synergy between VFN and chronic pharyngeal diseases, such as chronic tonsillitis and pharyngitis. It posits that chronic pharyngeal inflammation serves as a "pro-inflammatory substrate" that lowers the threshold for mechanical injury, stabilizes the presence of nodules, and prevents spontaneous regression. By synthesizing statistical data, acoustic analysis, and histological markers, this work argues for a holistic diagnostic paradigm in pediatric laryngology.

Keywords: Pediatric Dysphonia, Vocal Fold Nodules (VFN), Chronic Pharyngitis, Chronic Tonsillitis, Etiopathogenesis, Phonotrauma, Laryngopharyngeal Reflux (LPR), Mucosal Remodeling.

1. **Introduction**. Pediatric dysphonia is a significant clinical concern, with prevalence rates estimated between 6% and 23% in school-aged children. Among these cases, vocal fold nodules—bilateral, symmetrical thickenings of the superficial lamina propria—account for 38% to 60% of all diagnosed organic lesions (Martins et al., 2016). Historically, these were labeled "Screamer's Nodules," reflecting a purely behavioral etiology focused on vocal abuse, such as yelling, shouting, or excessive crying.

However, the "behavioral-only" model fails to explain why many children with high vocal loads never develop nodules, while others with moderate use suffer from persistent lesions. Modern research highlights a multifactorial etiopathogenesis where chronic pharyngeal diseases act as a "descending" inflammatory stimulus. In these cases, the larynx is subjected to constant irritation from pharyngeal secretions, resulting in secondary laryngeal edema. This edema necessitates increased subglottic pressure to maintain phonation, thereby creating a vicious cycle of mechanical trauma and biological inflammation.

2. Statistical Framework and Epidemiological Correlations

The correlation between chronic upper respiratory inflammation and the development of VFN is supported by robust clinical statistics.

2.1. Prevalence of Pharyngeal Comorbidities

In a comprehensive longitudinal study of 420 children diagnosed with VFN, researchers identified a remarkably high incidence of comorbid pharyngeal pathology (Martins et al., 2016, p. 17):

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- Chronic Tonsillitis and Adenoiditis: Present in 42.3% of cases. The mechanical obstruction and chronic infection of the Waldeyer's ring alter the resonance and necessitate compensatory vocal strain.
- Chronic Pharyngitis: Observed in 31.5% of patients. This is often exacerbated by mouth breathing, which bypasses the nasal humidification system and leaves the laryngeal mucosa vulnerable to drying and irritation (Akçay et al., 2019).
- Laryngopharyngeal Reflux (LPR): Often co-occurring with chronic pharyngeal irritation, LPR is diagnosed in 54% to 68% of pediatric patients with persistent nodules (Block & Bauman, 2002, p. 46).

2.2. Quantitative Acoustic Divergence

The presence of pharyngeal disease significantly alters the acoustic profile of the child's voice. When chronic pharyngeal inflammation is present, the vocal fold cover becomes heavier and less pliable.

- Jitter (Frequency Perturbation): In children with isolated VFN, jitter levels typically hover around 0.88%. In the presence of chronic pharyngeal disease, this increases significantly to 1.42% ($p < 0.05$), indicating a high degree of aperiodicity in vocal fold vibration (Akçay et al., 2019, p. 114).
- Shimmer (Amplitude Perturbation): Mean levels increase from roughly 3.2% in healthy controls to over 5.11% in children with comorbid pharyngeal irritation, reflecting the instability of the glottal cycle due to inflammatory edema (Akçay et al., 2019).

3. Deep Dive into Etiopathogenetic Mechanisms

The interaction between the pharynx and larynx is both biochemical and mechanical.

3.1. The "Descending" Secretory Irritation

Chronic pharyngeal diseases, particularly chronic tonsillitis, produce a persistent flow of mucoid or purulent secretions. During sleep, these secretions migrate inferiorly and pool in the post-cricoid area and the laryngeal vestibule. This "inflammatory bath" induces a state of chronic subepithelial edema in the vocal folds. Edematous tissue is more susceptible to mechanical shearing forces; thus, even "normal" vocal volumes can cause the micro-trauma necessary to trigger nodule formation.

3.2. The Globus Reflex and Throat Clearing

Chronic pharyngitis often leads to a "globus" sensation—the feeling of a foreign body in the throat. To alleviate this, children develop a habit of frequent, forceful throat clearing. Mechanically, throat clearing is one of the most traumatic maneuvers for the vocal folds, as they are slammed together with up to 10 times the force of regular conversational speech (Behlau et al., 2014, p. 282). This repetitive high-impact collision is a primary driver in transitioning soft, edematous nodules into hard, fibrous ones.

3.3. Compensatory Glottic Hyperfunction

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Children with pharyngeal discomfort often suffer from "muffled" vocal resonance. To be heard over the "biological noise" of an inflamed pharynx, the child instinctively increases subglottic pressure. This leads to the Hard Glottal Attack, where the vocal folds are tightly adducted before phonation begins, resulting in significant mechanical stress at the junction of the anterior and middle thirds of the folds—the classic site for VFN development.

4. Histological and Molecular Remodeling

At the microscopic level, VFNs in children with chronic pharyngeal backgrounds exhibit specific markers of pathological tissue remodeling.

4.1. Basement Membrane Zone (BMZ) Reduplication

The most characteristic feature of VFN is the thickening or "reduplication" of the Basement Membrane Zone. This serves as a biological "callus." In children with chronic pharyngeal inflammation, the BMZ is not only thicker but also shows signs of disorganized collagen deposition (Martins et al., 2016). This structural change reduces the pliability of the vocal fold cover, further impairing the mucosal wave.

4.2. Cytokine-Mediated Inflammation

Laryngeal secretions in these children show elevated levels of pro-inflammatory cytokines, specifically Interleukin-1α (IL-1α) and Tumor Necrosis Factor-alpha (TNF-α). These molecules act as signals for fibroblasts in the superficial lamina propria to increase the production of fibronectin. Increased fibronectin levels are associated with "interrupted" wound healing, where the tissue remains in a permanent state of inflammation rather than resolving, thus stabilizing the nodule (Martins et al., 2016, p. 18).

5. Therapeutic Implications and Conclusion

The etiopathogenesis of pediatric VFN is a synergistic process. While phonotrauma is the *initiator*, chronic pharyngeal disease is the *stabilizer*.

5.1. The "Triple-Track" Treatment Model

Based on this etiopathogenetic understanding, treatment cannot be limited to voice therapy alone. It must address:

1. Mechanical Reduction: Voice therapy to eliminate the "Hard Glottal Attack" and reduce vocal load.
2. Inflammatory Control: Management of LPR and pharyngeal irritants through diet and medication.
3. Foci Eradication: Surgical or medical resolution of chronic tonsillitis and adenoiditis to remove the source of descending secretions.

Conclusion. Vocal fold nodules in children should no longer be viewed as an isolated consequence of shouting. Statistics and histological markers confirm that over 50% of persistent VFN cases are sustained by a comorbid pharyngeal inflammatory environment. Effective clinical outcomes require a diagnostic approach that evaluates the entire upper respiratory tract, acknowledging that the resolution of the nodule often depends as much on the health of the pharynx as it does on vocal behavior.

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