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## Review Article

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## Nasal Therapy with Lidocaine for the Prevention of Inflammation Due to Coronavirus (Covid-19)

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### Abstract

Nasal therapy with lidocaine for the prevention of inflammation due to coronavirus (Covid-19). The purpose of this document is to propose and explain nasal therapy as a therapeutic proposal for the preventive management of inflammation of the airways caused by the response to coronavirus infection (Covid-19) by using the anti-inflammatory and membrane stabilizing properties of the lidocaine hydrochloride and procaine hydrochloride. Explain the mechanism of action of nasal therapy with local anesthetics in the prevention of inflammation by Covid-19.

**Keywords:** Anti-inflammatory; Coronavirus (Covid-19); Nasal therapy

### Introduction

**Nasal therapy:** A therapeutic proposal developed by the author of this article, uses the anti-inflammatory and membrane and epithelial stabilizing properties of local anesthetics such as lidocaine and procaine, providing an anti-inflammatory effect, and a more lasting stabilization of epithelia than that provided by aines and steroids. Nasal therapy is a non-invasive, easy to apply procedure that, as its name implies, uses the mucosa of the upper respiratory tract to induce an anti-inflammatory and stabilizing effect on respiratory epithelia.

**Pathophysiology:** In acute respiratory disease, either viral or bacterial, a combination of factors occurs, including mucosal edema, increased mucous secretion, infiltration by inflammatory cells (eosinophils, mast cells, T lymphocytes).

**Mast cells:** They are found in the vicinity of blood or lymphatic vessels, in the vicinity of nervous structures, in the dermis or under the mucosa of the respiratory or digestive tract. Through the process of degranulation (breakdown of the mast cell), the release of cytokines, preformed inflammatory mediators such as

histamine, and Newly formed mediators such as leukotrienes, thromboxane's and prostaglandins are generated as a consequence of the metabolism of arachidonic acid from membrane lipids, and also proteoglycans, trypase, neutral proteases, acid hydrolases, and carboxypeptidase. A Mast cells produce a wide range of cytokines such as IL-1, IL-3, IL-4, IL-5, IL-6, INFgamma, and TNF alfa, granulocyte-macrophage colony stimulating factor and certain chemokines; SRS-A (slow-acting anaphylactic substance). Such processes tend to promote and perpetuate the inflammatory response.

**Eosinophils:** Granulocytes derived from the same stem cells as mast cells, neutrophils, and basophils. The circulating quantity is small and most are found in the tissues. Granulocytes present in their membrane leukotriene B and several cytokines in addition to receptors for Ig G, A and E, in this way it intervenes in allergic reactions and other immunological processes. They release both preformed mediators and lipid derivatives and cytokines.

The specific granules contain lysosomal hydrolases and cationic peptides (major basic protein, cationic eosinophil protein, eosinophil-derived neurotoxin, and eosinophilic peroxidase). These molecules have toxic properties for normal cells, and therefore are considered the main mediators of tissue damage. Among the main lipid derivatives are platelet activating factor and leukotrienes C4,

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D4, and E4, which are involved in the processes of chemotaxis, increased vascular permeability, and tissue edema.

Eosinophils are one of the main sources of cytokines. They produce IL-3, IL-5, and GM-CSF, which promote the proliferation, migration, and activation of new eosinophils, release tumor growth factor and other interleukins involved in the inflammatory response, fibrosis, and tissue repair and possess molecules receptors for derivatives of the complement cascade. They modulate immediate hypersensitivity reactions by degrading or inactivating the mediators released by mast cells (histamine, leukotrienes, phospholipids, and heparin).

**T lymphocytes:** T Lymphocytes constitute 70-80% of the circulating lymphocytes. They come from stem cells of the bone marrow that give rise to precursor cells that are located in the thymus, where CD4 (helpers) or CD8 (cytotoxic) lymphocytes mature and originate. Allergenic molecules are recognized, phagocytosed, and processed by the macrophage, B lymphocyte, or other antigen presenting cells, and are then expressed on the membrane along with the major type II histocompatibility complex.

These fragments are presented to the T lymphocyte that identifies the antigen through specific receptors, and thus it is activated to produce proinflammatory cytokines, which induce IgE synthesis. Undifferentiated CD4 or CD8 T cells produce interleukin 2 (IL-2) until they are activated, and by the action of IL-12 produced by macrophages, TH1 lymphocytes differentiate. These lymphocytes produce large amounts of IL-2, gamma interferon, and tumor necrosis factor Beta. Undifferentiated T lymphocytes exposed to IL4 originate TH2 cells, which are involved in IgE-mediated allergic phenomena, by releasing IL-4, IL-5, IL-9, IL-10, and IL-13.

**Epithelium:** Epithelium of the airways is more than just a physical barrier that protects the underlying tissue from environmental damage. The entire system, from the trachea to the bronchi, is covered by an epithelium that rests on a thin basement membrane. Epithelial cells play an important role in the initiation of inflammatory and infectious disease of the airways and in the maintenance of chronic inflammation in prolonged disease.

Epithelial cells release pro-inflammatory cytokines and mediators, which in turn attract eosinophils, establishing a cycle of events that perpetuate inflammation. Neuroepithelial bodies are clusters (10 to 30) that contain serotonin, are highly innervated, and may be involved in maintaining the caliber of the airways. The epithelium houses mast cells and eosinophils in the submucosa where they are attacked by IgE and by factors derived from the complement cascade. After the interaction of the intraluminal mast cells and basophils and superficial mucosa, with the specific agents or other degranulatory agents, an alteration of the respiratory epithelial barrier is caused, allowing the passage of inhaled material through the most deeply located cells, producing again the cycle of inflammatory phenomena in depth.

**Cell membranes:** Cell membranes are primarily made up of a bimolecular lipid matrix that contains mainly cholesterol and phospholipids. And globular protein macromolecules of variable volume and composition that participate in transport and with receptor function. The lipids of the membrane provide stability to

the membrane and determine its permeability characteristics. The lipid portion of the membrane and the lipophilic properties of some substances constitute an important part within the pathogenesis and in the proposed treatment. The medication must possess anti-inflammatory qualities within the airway. The efficacy, ease of administration, reduction or elimination of side effects must be taken into account.

## Local anesthetics

Local anesthetics have long been used through their topical application to the nasal mucosa for a large number of otorhinolaryngological procedures.

### A. Chemistry

- Group 1. Linked to an ester
- Group 2. Linked to an amide

### B. Molecule conformation

- An aromatic lipophilic chain
- An intermediate chain
- An amino hydrophilic group

**Procaine:** Procaine, (P-amino benzoyl-diethyl-amino-ethanol hydrochloride), was the first synthetic local anesthetic. It is probably the least toxic of all local anesthetics, with a rapid onset of action and short duration, it is rapidly destroyed by the liver upon reaching the circulation.

**Lidocaine:** Lidocaine is an amide-type local anesthetic, it is currently the most widely used local anesthetic, fairly fast onset of action, fairly long duration of action, with a power and toxicity 2 or 3 times greater than procaine. 4% lidocaine solutions are available for topical use for anesthesia of the nose, oropharynx, and tracheobronchial tree.

## Mechanism of action of local anesthetics in the prevention of inflammation in the respiratory system

**Anti-inflammatory action:** The anti-inflammatory action of local anesthetics occurs at the level of the airway epithelium through the following mechanisms:

- Stabilization of the cell membrane by the aromatic lipophilic chain of the anesthetic that adheres to the lipid portion of the membrane.
- This stabilization of the cell membrane of the airway epithelium prevents the release of cytokines and neuropeptides.
- Stabilization of the cell membrane of mast cells and eosinophils also occurs by the same process, blocking the release of histamine and leukotrienes and other inflammatory mediators.
- The occupation of the lipid portion of the cell membrane prevents the action of CAM (complement membrane attack complex), thus also preventing the release of inflammatory mediators.
- Membrane stabilization also blocks the action of IgE.

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