# NEEDLE PERFORATION OF ORAL MUCOSA AND LOCAL OCCURRENCE OF APHTHAE: MECHANISM AND APPROACHES

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**ABSTRACT:**

Oral aphthae may be prevented by maintaining the mucosal integrity, and such lesions may occur at the site of penetration of the anesthetic needle. In these cases, a gel may be indicated soon after completion of anesthesia, explaining this approach to the patient. Some individuals with aphthae report previous occurrence of aphthae after anesthesia, and the professional may suggest the patient to carry the gel on the following consultation to apply this "preventive" procedure. For patients with frequent small oral aphthae, the professional may follow the described management protocol, using the gel for local application according to the presented sequence. When aphthae appear, it is very important to identify the associated causes, prescribe local and/or systemic drugs and instruct the patients to reduce their duration and frequency. There are well-defined causes and therapies, yet this requires diagnostic and therapeutic skill of the professional, persistence of all individuals involved, and patient awareness.

**KEYWORDS:**

Aphthae. Small aphthae. Mikulicz aphthae. Oral anesthesia.

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## INTRODUCTION

Patients with sporadic aphthous ulcerations often report that "they appeared at the site perforated by the local anesthesia needle" used by the dentist. Other correlated events are accidental bites or injuries resulting from a hard movement of the toothbrush during oral hygiene.

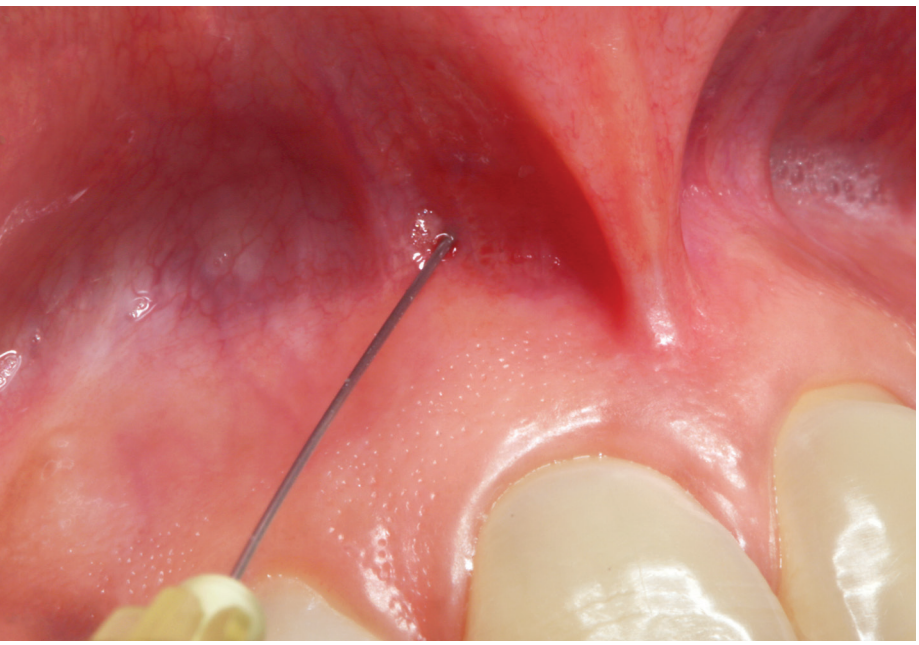

**Figure 1:**
Site of needle penetration, where a microinjury exposes epithelial structure to underlying lamina propria cells.

Originally, the term aphtha literally meant "I inflame", "I fire myself up" or "I burn". First used by Hippocrates, the Father of Medicine who lived between 460-370 BC, it described thrush, or pediatric pseudomembranous candidiasis. Along time, its use was extended to any ulceration of the mucosas. Currently, the word ulcer should be reserved for superficial lesions that do not repair spontaneously after two weeks, and the word ulceration may be applied to lesions that tend to repair spontaneously, including oral aphthous ulcerations.

In several of these clinical events, the patients usually ask why these ulcerations occurs and even what their mechanism of action is. Dentists do not always have a clear and objective explanation for this condition, but sometimes search for and find it in the Internet. This study provides resources for clinical dentists in any specialty to explain to their patients the association of oral mucosa perforations by needles (Fig 1) and the occurrence of an aphthous ulceration at the same site.

## THE STRUCTURE OF THE ORAL MUCOSA IS HIDDEN!

The oral mucosa has two main components: the stratified squamous epithelial lining, and the papillary fibrous connective tissue between the epithelial crests, called lamina propria.

In the skin, similar papillary connective tissue is called dermis. The oral mucosa is composed of the epithelium and the lamina propria, whereas the skin has the epithelium and the dermis. The oral mucosa epithelium has about 15 to 30 layers of epithelial cells.

In the basal layer, closer to the papillary fibrous connective tissue, the long axis of cells is vertical and, as new cells are created by constant mitosis, they migrate toward the surface and gradually begin to flatten and become horizontally oriented. From an oval shape in the basal layer, they reach the surface as lamellar squamous cells that are shed or exfoliated into the oral environment.

**VERY IMPORTANT: THERE ARE TWO TYPES OF KERATINIZATION IN THE ORAL MUCOSA!**

When the cells are very close to the surface, epithelial cells form and deposit protein granules, called keratohyalin granules, which converge and fuse until all the leaf-shaped cells transform into a keratin plaque. It is next to the surface layer and is called the granular layer of the lining epithelium. These combined layers and keratin squamous cells line and protect the surface of the oral mucosa on the dorsal surface of the tongue, hard palate and attached gingiva. Keratin formation from granules and complete disappearance of cell nuclei in the exfoliated cells characterize orthokeratinized tissue.

> "Oral aphthae may be prevented by maintaining the mucosal integrity, and such lesions may occur at the site of penetration of the anesthetic needle."

Diseases of the oral mucosa are induced by the invasion of chemicals or the action of physical agents, and hardly ever occur in the keratinized areas. When affected, these areas are attacked from outside to inside, as in the case of recurrent herpes simplex, which occurs as the virus leaves peripheral nerves, and in diseases on the walls of periodontal pockets.

Orthokeratinized tissue is an efficient protection mechanism for underlying epithelial cells. Mouth cancer, particularly squamous cell carcinoma, rarely originates in the orthokeratinized areas, probably because they are better protected from carcinogenic chemicals and physical agents.

In all the other sites, except the dorsal surface of the tongue, the hard palate and the attached gingiva, the oral mucosa is thinner

and its surface also sheds flat and squamous cells into the oral environment, but before they are fully keratinized, because they have not had time yet to lose their cell nuclei.

Under the microscope, no granules can be seen, only horizontal cells with persistent nuclei. The incomplete keratin formation on the surface layer is called parakeratinization. Ortho- and parakeratinization are protective mechanisms of the oral mucosa, and the orthokeratinized mucosa is better protected than the parakeratinized mucosa.

## EPITHELIUM AND LAMINA PROPRIA ARE SEPARATED BY THE BASEMENT MEMBRANE

The lamina propria is formed by lamellar connective tissue of variable thickness and papillary morphology that supports the oral mucosa epithelium both physically and nutritionally. The papillae of the lamina propria increase the area of contact and imbricate with the oral mucosa, and simultaneously facilitate the exchanges of nutrients, as epithelia have no blood vessels.

The connective tissue of the lamina propria at its deepest point naturally extends into the submucosa, which corresponds to the dermis in the skin. However, the lamina propria may be directly united to the periosteum of the alveolar bone, or be lining the submucosa, as we see in different regions of the mouth, such as the soft palate and the floor of the mouth.

The basement membrane is between the oral mucosal epithelium and the lamina propria, and it is a 1-to 4-µm thick connective tissue with no cells composed of glycosaminoglycan, glycoproteins and anchorage fibers and fibrils. The basement membrane can be visualized under light microscopy. Wounds due to local trauma that result in ulcerations expose the epithelium of the oral mucosa to underlying tissues, such as the connective tissue of the lamina propria.

## WHERE AND WHY ORAL APHTHAE APPEAR

Aphthae do not affect thick orthokeratinized oral mucosa, such as the dorsal surface of the tongue, hard palate and attached gingiva, because microinjuries are less frequent in these sites than in non-orthokeratinized areas.

Aphthae occur in places that have previously suffered a microinjury that may progress into an aphthous ulceration in two days. This may happen when the oral mucosa is perforated or punctured with a local anesthetic needle. It may also occur because of accidental bites, brushing that hurts the mucosa, mastication of fruit fragments, such as pineapples, and the placement of an orthodontic appliance. In the latter two situations, there are multiple microinjuries and, consequently, the possibility of equally multiple aphthous ulcerations.

If the epithelium of the oral mucosa is separated from the connective tissue of the underlying lamina propria, why may an aphthous ulceration occur when this organized structure is injured? Why does it affect only some people?

To answer these questions, we discuss three important factors that, although apparently independent, are eventually interconnected.

***1. Integrity and maintenance of the oral mucosa structure***

Most immunological components of the organism do not directly interact with the epithelial tissue, except the intraepithelial macrophages known as Langerhans cells. When the oral mucosa is hurt, the epithelial cells, called keratinocytes, are directly exposed to the components of the underlying connective tissues, which include the cells responsible for immunological responses.

In smokers, there is an increase in the epithelial thickness of the oral mucosa and of its keratinization in all regions of the mouth. Smokers do not have oral aphthous ulcerations because of this functional adaptation induced by tobacco. When patients that smoke quit the addiction, they may have aphthous ulcerations and, because of that, may relapse.

Patients with nutritional vitamin B and iron deficiency have anemia, as these nutrients are important cofactors in the proliferation of bone marrow cells that produce red blood cells, leukocytes and platelets. Together with the bone marrow, the epithelial lining of the mouth is one of the tissues with the greatest proliferation and renewal in the organism, particularly the dorsal surface of the tongue. Nutritional deficits of these two components affect the bone marrow and induce anemia, and the oral mucosa of these patients becomes thin and less keratinized. Several patients with anemia also have multiple oral aphthous ulcerations, because microinjuries are more frequent when the mucosal surface is fragile.

***2. Heredity and oral aphthous ulcerations***

The possibility of inheriting the potentiality to develop oral aphthous ulcerations is greater, or almost absolute, if both parents had aphthous ulcerations along their lives. This risk remains high, at about 50%, if one of the parents had this potentiality. Chances are substantially lower, or very much reduced, in children of parents that did not have these ulcerations, because the molecular characteristics of the epithelial cells in the oral mucosa membrane are inherited.

***3. Stress and oral aphthous ulcerations***

People under stress do no eat well, but may grind or clench their teeth everyday, have gastritis and a deregulated immunosystem. The adrenal glands are overloaded and release a very high amount of hormones from its cortex into the organism. These cortical hormones are also produced in laboratories, in which case they are called corticosteroids.

The hormones released in excess by the adrenal cortex deregulate and depress the immunosystem, particularly of patients exposed to stress everyday. In these cases, the incorrect identification of foreign proteins or even those that are part of the patient's organism may have a broader effect and increase the risk of autoimmune diseases, such as aphthous ulcerations.

Gastrointestinal disorders are also common in these patients. Maybe because of that, some professionals, as well as laypeople, may associate aphthous ulcerations with gastric disorders, which is not true, nor supported by any clinical or methodological evidence. Aphthous ulcerations are not associated with gastric disorders in any way.

> "When the cells are very close to the surface, epithelial cells form and deposit protein granules, called keratohyalin granules."

### *4. Oral aphthous ulcerations due to placement of appliances, prosthesis and restorations*

Orthodontic brackets and archwires always produce microinjuries in the lips, cheeks, lateral tongue margins and other sites lined with parakeratinized epithelial cells. The multiplicity and continuity of microinjuries of the same delicate oral mucosa in an organism with a potential to develop oral aphthous ulcerations because of heredity, bacterial or autoimmune factors, as explained above, may induce the occurrence of aphthous ulcerations after the placement of orthodontic appliances.

After the appliance has been in place for some weeks, there is a structural adaptation of the oral mucosa, which increases its epithelial thickness and keratinization. Aphthous ulcerations decrease, then, and may even disappear completely because of this adaptation, but before that, may be a great clinical and personal nuisance, especially for adults.

### *5. Bacteria and oral aphthous ulcerations*

Bacteria of the streptococcus and staphylococcus morphotypes may have protein components in their membrane that are structurally similar to the proteins of epithelial cells of the oral mucosa in some people. Moreover, the molecular characteristics of epithelial cells are inherited.

Everyday the immunosystem produces mediators, antibodies and sensitized cells against bacterial components, and this may be detected by measuring the plasma levels of immunoglobulins and antibodies against microorganisms. In summary, our organism has cellular and biochemical tools to eliminate streptococcus and staphylococcus that may sporadically invade our connective tissues.

## THIS IS THE MECHANISM OF ORAL APHTHOUS ULCERATIONS AUTOIMMUNITY AND CROSS-REACTIVITY

The microulcerations of the oral mucosa induced by perforations, such as those made by needles, food, instruments, brackets and toothbrushes, result from the direct exposure of epithelial cells, or keratinocytes, to the components of the underlying connective tissue, which includes those that take part in immunological responses. The patient may have epithelial cells that are structurally similar to the proteins of streptococci or staphylococci, and the immunosystem components are ready to act against these bacteria of the oral microbiota. In these cases, a cross-reactive autoimmune response is triggered because of the similar proteins on the surface of epithelial cells of the oral mucosa, which are exposed to the connective tissue by microinjuries. Epithelial cells in the areas of microinjuries die, and the ulceration expands, forming an oral aphthous ulceration. What was a mere perforation by a local anesthetic needle, a pineapple fragment, or an accidental bite may progress to an oral aphthous ulceration. The process ends when the immunosystem refines its molecular recognition after some days, and understands that, in fact, the epithelial cells of the oral mucosa are similar, but not identical, to those bacterial components. After three to five days, the aphthous ulcerations start their reparation process. The time from the event that triggers this reaction to full recovery is about five to ten days.

## AN APHTHOUS ULCERATION APPEARED AFTER A NEEDLE PERFORATION: WHAT CAN BE DONE? HOW TO TREAT IT?

The ideal plan for these cases is to take into consideration the history of the oral aphthous ulceration, which should be taken during the first visit. If the patient reports sporadic or frequent aphthous ulcerations, the chances of ulcerations at the site of needle perforation (Fig 1) should be clarified, and if it occurs, guidelines as the ones described below should be followed.

There are multiple treatments[1,2,3], some popular and mostly empirical, for oral aphthous ulcerations. Clinical observations and the study of the composition of topic medicines applied to oral aphthous ulcerations led us to develop a gel that follows these determinant principles:

» Efficiency in the rapid relief of symptoms; and

» Few iatrogenic effects and greater patient comfort due to ease of application.

Immediate relief of local symptoms is very important, and we developed a simple protocol to help patients and dentists:

1. Gently clean the yellowish or whitish material on the wound bed using a cotton bud with peroxide, physiological solution, or even water. This pseudomembrane, not always present, is an obstacle to the penetration of the medication and should be removed only for the first gel application in most cases.

2. Use the gel five times a day. It should be dispensed by a trustworthy pharmacist and prescribed as follows:

Topical use

Components:

* *Betamethasone*.....*0.1%*
* *Acetylsalicylic acid*.....*2.0%*
* *Hydroxypropyl cellulose gel*.....*50g*

Apply it up to five times a day, when necessary, using fingertips or mildly wet cotton buds.

3. Make it clear to patient the purpose of applying the gel: it should only alleviate symptoms and improve patient comfort.

This gel should be purchased in small amounts so that it does not lose its effect, which may be assessed by observing if there is loss of transparency, color change, or thickening. The components of this medication are common in the market, and its cost is affordable for people in general. It is easily applied and may be used alone or in association with other topical or systemic treatments. Systemic treatments may be sporadically administered to increase patient resistance to aphthous ulcerations or correct predisposing factors, such as in the case of anemia, stress and use of medications to quit smoking.

The principal recommendations are limited to the usual contraindications for the use of topical corticoids, such as for patients with diabetes, and to the guidelines for the patient not to apply an excessive amount of it. The three components of the gel are prescribed to promote anti-inflammatory action, act as a local painkiller and promote the formation of a temporary film to retain the medication for some minutes, or hours, on the wound bed, thus alleviating symptoms and reducing the time to recovery.

Other ointments, creams and pastes with similar compositions may be used according to the same protocol, but their application is usually more difficult for the patient because of their formulation. Special equipment and specialized operators are required for the use of lasetherapy[2,3], and the patient does not always have access to this type of treatment, which, if adopted, does not rule out the use of the gel recommended here.

Patients with oral ulcerations known as major aphthous disease, also called Sutton's aphthae, or with herpetiform ulcerations may have other factors involved, and a more complex stomatologic assessment should be made. These factors include other associated diseases, viruses, autoimmune diseases and even chemotherapy and radiotherapy. These cases should be referred to a clinical dentist specialized in Stomatology. The gel indicated here may also be applied in these cases, but the resolution will depend more on the control of the causes of that specific clinical condition.

Immediately after an anesthetic procedure is completed, the gel may be applied to the site, or sites, of needle perforation to prevent the development of an aphthous ulceration, and the instructions to apply it should be explained to the patient. For the patient that reports previous episodes of aphthous ulcerations after anesthetic injections, the dentist may recommend bringing the gel to the next visit, so that this preventive measure can be taken.

## ORAL APHTHOUS ULCERATIONS: PROTOCOL WITH GUIDELINES AND PROCEDURES FOR CURRENT AND RECURRENT EVENTS

The patient should receive instructions about how to avoid microinjuries during:

1. **eating:** avoid solid perforating foods, such as pineapple fragments, bread crusts and popcorn. If they want to eat these fruit, tell them that they should be ingested as juices or creams, to avoid microinjuries. Moreover, very acid or astringent foods may enlarge microinjuries of the oral mucosa. Explain that aphthous ulcerations are not an allergic reaction to any food.

2. **oral hygiene:** avoid the contact of the bristles with the oral mucosa and hard brushing movements.

3. **mouthguards:** give the patient instructions about the use of mouthguards over provisional restorations, implants, prosthesis, brackets, archwires and other appliances placed in the mouth to avoid biting the mucosa while sleeping or when clenching teeth, and also if the patient has bruxism; there are products made of soft and flexible materials that dentists and patients may adapt efficiently to serve as mouthguards, so that they appliances are "hidden" from the oral

mucosa, protect it from microinjuries and prevent oral aphthous ulcerations.

4. **time:** ensure patient that after some weeks using the prosthesis, archwires or brackets, the oral mucosa will adapt and increase keratinization, and that the aphthous ulcerations will be fewer and may even not occur again.

One of the points that requires special attention is the evaluation of whether the patient that present with more frequent or numerous aphthous ulcerations has discolored or yellowish mucosas, gets tired easily or has a history of deficiency anemias. These conditions suggest anemia and, if there is any question about the diagnosis, the patient should be referred to a family doctor or another doctor of the patient's choice for evaluation. Multiple aphthous ulcerations are often associated with anemia, and the correction of this hematologic deficiency improves the oral condition or completely resolves them.

It is also important to ask if the patient that presents with multiple and persistent aphthous ulcerations has quit smoking after the beginning of the dental treatment. This event may complicate the occurrence of oral aphthous ulcerations because the thickness of the mucosa gradually goes back to normal, as tobacco induces hyperplasia and epithelial hyperkeratinization. These factors should be explained to the patient.

## FINAL CONSIDERATIONS

The secret to avoid aphthous ulcerations is mucosal integrity. Therefore, in some cases, these lesions may appear at the site of penetration of a local anesthetic needle.

In these cases, the gel indicated in this text may be applied immediately after the procedure is completed, and its application should be explained to the patient. Patients may report previous episodes of aphthous ulcerations after anesthetic injections, and the dentist may recommend bringing the gel to the next visit as a preventive measure.

For patients with smaller frequent oral aphthous ulcerations, the dentist may follow the protocol and the instructions described here, and the gel may be applied topically.

The associated causes of aphthous ulcerations have to be identified, so that topic and systemic medications can be prescribed, and the patient should receive instructions to abbreviate duration and frequency of ulcerations. There are clearly defined causes and treatments, but their definition depends on the dentist's diagnostic and therapeutic expertise, persistence of all those involved, and patient awareness of the importance of the treatment.

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» Patients displayed in this article previously approved the use of their facial and intraoral photographs.