

The Fine Art of Laser Dentistry with Dr. Glenn van As

The Diode laser in treating Ulcerative Oral lesions.



Introduction:

In this month's edition of Dentistry Today, I look at the role of the diode laser in helping with palliative care in the treatment of ulcerative oral lesions (Recurrent Aphthous Ulcers (RAU or RAS)) and Oral Herpetic lesions (HSV-1)). RAU and HSV-1 are relatively common oral diseases, which can be both painful and be unesthetic for many patients. These diseases have been traditionally treated pharmacologically with steroids (Dexamethasone mouth wash, Kenalog in Orabase gel) for RAU lesions, and expensive antiviral medications (Acyclovir) for HSV-1 outbreaks. Recently alternative therapies such as the use of Low Intensities Laser Therapy (LILT) have been introduced for their abilities to accelerate wound healing and provide almost immediate pain relief.



Recurrent Aphthous Ulcers (RAU)

Commonly known as canker sores, aphthous ulcers (aphthous stomatitis) are a common affliction that affects 10% of the population. The cause of these lesions is not thought to be viral or bacterial in origin but likely an auto-immune mediated condition. Although these lesions can be a byproduct of a systemic illness such as Crohn's disease, HIV, Behcet's syndrome or other autoimmune diseases, the majority of the time the lesions are not part of any systemic illness and are characterized as Minor (3-10mm in size), Major (> 10mm) and Herpetiform (Multiple coalescing lesions). The exact cause of many aphthous ulcers is unknown, but possible etiological factors include: citrus fruits, trauma, stress, lack of sleep, immune system reactions, and deficiency of B12, Iron or Folic Acid. (1-3)

These lesions are found on the "loose" tissues of the mouth including the inner aspects of the lips, buccal mucosa, tongue, soft palate and the floor of the mouth. The ulcers begin with a tingling or burning sensation at the site that progresses after a few days to a yellow or white oval surrounded by an inflamed red border. (Fig.1) The ulcer is painful to palpation, and spicy or acidic foods, and can become secondarily infected at times. Use of a toothpaste containing Sodium Lauryl Sulfate (SLS) can trigger attacks in some patients.

Typically, dentists have looked at using topical agents like Debacterol (chemical cauterizing agent), or topical steroid agents (Kenalog in Orabase). Recently, several studies of using lasers at non ablativ settings (Low Intensity Laser Therapy)(LILT) have shown that settings of 50-500 mw (.05 - 0.5 watts) can help with both a reduction

in pain and provide for quicker healing compared to corticosteroid treatment. In fact, the results from De Souza et al revealed that 75% of the patients reported a reduction in pain in the same session after laser treatment, and total regression of the lesion occurred after 4 days. Total regression in the corticoid group was from 5 to 7 days. (4) Bladowski et al also found that the diode laser used at low levels of energy (200 mw) cut the healing time in half when compared to a pharmaceutical method (Solcolseryl). (5) Khademi et al found similar benefits of quicker healing and reduced pain after using low levels of laser treatment on RAS. (6)

Dr. L.J. Walsh has done a tremendous amount of research on the proposed mechanisms of the action of LLLT on both hard and soft tissues (7-8) and has proposed that cold lasers (LLLT lasers) accelerate wound healing and reduce pain by perhaps “stimulating oxidative phosphorylation in mitochondria and modulating inflammatory responses”. (7-8).

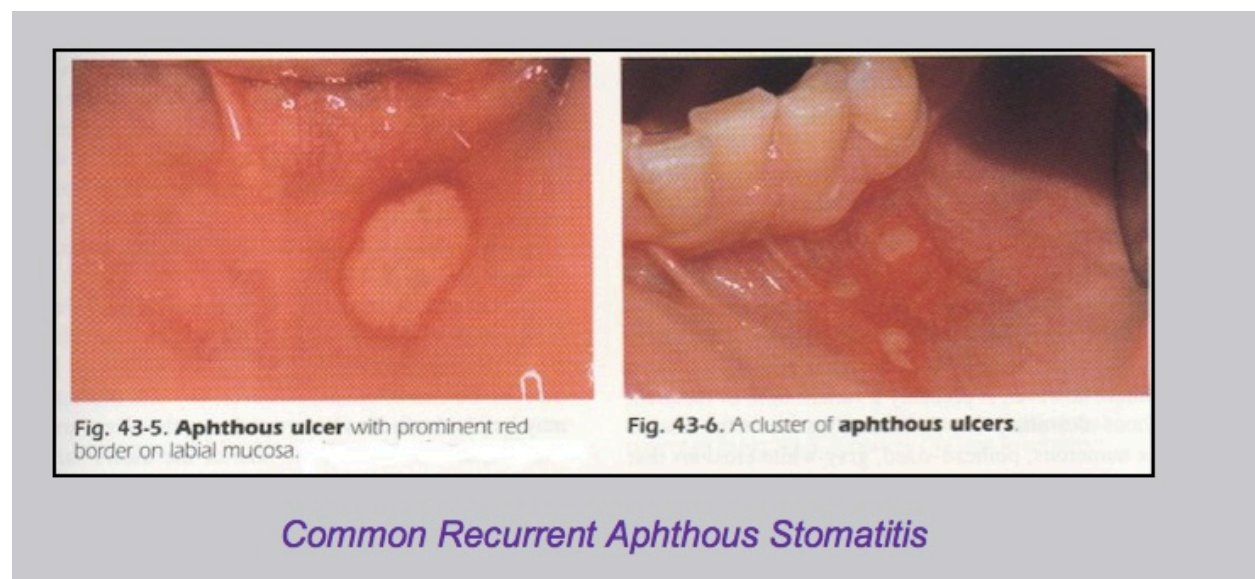


Fig.1 Appearance of Recurrent Aphthous Ulcers.
Photo courtesy of Dr. Steven R. Pohlhaus, DDS, FAGD (Linthicum, Maryland).

Recurrent Herpetic Simplex Lesions

Herpes Simplex is a viral disease caused by the Herpes Simplex Virus. Oral lesions (type 1) are caused by the HSV-1 strain and the genital herpes (type 2) is caused by the HSV-2 strain. Oral herpes are characterized by visible symptoms called fever blisters or cold sores, and although most people are exposed to the HSV-1 virus in childhood, only a small percentage of people suffer from the recurrent form of the disease. (9, 10)

The lesions typically break out in the “non-movable” parts of the mouth including gums, lips and hard palate, and often patients will feel itching or tingling in a prodromal stage

one to two days before the outbreak of the vesicular lesions. These lesions crust over and heal over a period of 10-14 days. (Fig.2) The virus itself has been found to become latent after childhood exposure in the ganglion of the nerves at the primary site. Triggers such as trauma, stress, sunburn, illness, cold weather or systemic illnesses such as AIDs patients, transplant patients, and cognitive illnesses such as Alzheimer's and bipolar disease can bring on the lesions. (11, 12) These lesions are contagious and can be spread to other people or other parts of your body from the very first sign of an outbreak (including the tingling, itching stage) until the area is completely healed again.

Typically, dentists have looked at using pharmaceutical agents like Acyclovir (anti viral drug) in ointments to speed up healing of lesions, and in pill format to help prevent reoccurrence. Various laser wavelengths have been found to be effective in the treatment of Herpes Lesions including HeNe (660nm), Erbium:YAG(2940nm) and other diode wavelengths. (13-14) , Alfonso et al (15) looked at 232 patients using a GaAlAs diode laser (30mW – 40 sec) in the prodromal stage and stage of vesicles; or (20mW – 2 min) in the crust stage and in lesions infected secondarily. In addition, in all these patients the diode laser was also applied radiation among the vertebrae C2-C3 where the resident ganglion of the virus is located during the latent periods (30mW - 30sec). They found **100% of the prodromal stages, 95% of the vesicular ones and 91% of crust stages were able to cure during the first 48 hours.** The patients with lesions infected secondarily needed more than 48 hours to cure, although they never surpassed 5 days. Other studies have confirmed the benefit of Low Level Laser Therapy in decreasing pain, improving wound healing compared to other treatment modalities like acyclovir. (16, 17). Donnarumma et al (18) have postulated a mechanism of action that laser irradiation “acts in the final stage of HSV-1 replication by limiting viral spread from cell to cell and that laser therapy acts also on the host immune response unblocking the suppression of pro-inflammatory mediators induced by accumulation of progeny virus in infected epithelial cells.”

Ferreira et al demonstrated that among treatment options, low-level laser therapy (LLLT) has shown promising clinical results as a longer-lasting suppression therapy. Patients were symptom free for 17 months following initial treatment for recurrent simplex lesions with the laser. (19) Schindl and Neumann (13) and found similar results in their LLLT study for the laser in delaying reoccurrence of the herpetic lesion outbreaks. **The median recurrence-free interval in the laser-treated group was 37.5 wk (range: 2–52 wk) and in the placebo group 3 wk (range: 1–20 wk).** Vélez-Gonzalez et al (16) also discovered delays in reoccurrence of lesions after laser therapy.

Laser technology at low intensities has been shown in addition to be helpful for the treatment of Chemotherapy Induced Mucositis where a diode laser (150mw, 2.5 times per week) was shown to reduce the severity and duration of mucositis and to relieve pain significantly.(20). Other studies have demonstrated that LLLT can be of benefit towards reducing symptoms and size of Erosive Lichen Planus lesions, where typically corticosteroids have been used without the adverse effects associated with steroid treatment.(21, 22)



Fig. 2 Appearance of Herpetic Lesion.

Image: Courtesy of Dr. Herrmann/ Centers for Disease Control and Prevention (CDC)

Technique:

When considering using the diode laser for the treatment of oral lesions, the clinician must consider several factors. Diode lasers, when used for the treatment of oral lesions are used in NON-CONTACT mode. The tip of the laser is therefore not initiated as the clinician wants the energy to penetrate into the lesion, and therefore so initiation of the tip on articulating paper is needed. It is important to realize that energies used for these treatments should be below ablative potential. Therefore typically the energy used should be under one watt. In most cases the energy should be between 150-800mw. Very little difference in the appearance of the lesion will occur, other than at times when the diode is used in the vesicular stage of herpetic lesions the dentist will note that the lesion “dries up” with laser therapy.

In general when treating oral lesions it is best to treat these lesions in the early stages such as the first 48 hours (Aphthous ulcers) or even during the prodromal stage (Herpes lesions). A 400 micron tip is chosen for smaller lesions, but a LLLT tip (8mm) or even the bleaching handpiece can be used for larger areas. If the bleaching tip is used the energy used should be **double** that of the other tips because energy is lost due to the lower fluency (power density) of the larger bleaching handpiece tip. The laser is started defocussed from the lesion (5-8mm) and advanced slowly towards the area ending up 2-3mm away. Continual movement from the periphery of the lesion to the center “painting “ the entire area and moving away from the lesion if the patient feels warmth. The setting is initially put at 0.6w CW (1.2w pulsed) for 30-45 seconds. A refractory period of 15-20 seconds between laser “passes” allows the tissue to cool down, and the clinician can “rub” the area with a wet gloved finger to determine if a decrease in pain is felt by the patient. Likely a 2nd , and 3rd pass with the laser will be needed to decrease the pain of palpating the area. A 2nd pass is done with the setting of 0.7w CW (1.4w pulsed) for 30-45 seconds, and a third and final pass is completed with 0.8w CW (1.6w Pulsed) for a similar period of time. After each pass, the area is

checked with palpation, but a maximum time of 2 minutes total of laser energy is permitted.

Table 1 - Clinical Procedure for Frenectomy

Step	Procedure
1	Pick a 400 micron- 5mm 90 degree tip , or 8mm LLLT tip for small to moderate sized lesions. 0.5-2.5 cm.
2	Pick a bleaching handpiece for larger lesions. (over 2-3cm)
3	Do not initiate the tip, as the energy must penetrate the lesion. Start defocussed (5-8mm) and advance towards the lesion (2-3mm away) painting the entire surface of the area.
4	Energy should be 0.6 watt CW (1.2 watts pulsed) for the first pass for 30-45 seconds . Rest phase is 15-20 seconds. Rub the lesion to see if pain is less.
5	Energy should be 0.7 watt CW (1.4 watts pulsed) for the 2nd pass for 30-45 seconds . Rest phase is 15-20 seconds. Rub the lesion to see if pain is less.
6	Energy should be 0.8 watt CW (1.6 watts pulsed) for the 3rd and FINAL pass for 30-45 seconds . Rub the lesion to see if pain is less.
7	Use double the power settings if using the bleaching handpiece.

Typically, the area will tingle immediately after the laser is used, and if treated early in the emergence of the lesion, the area will heal much faster than the traditional period of 10-14 days. In addition, in the authors experience, the laser treated area will be less likely to reappear with another lesion in the same area once treated 1-2 times. (Fig 3-4).



Fig.3
Appearance of
Recurrent
Aphthous
Ulcers.
Photo courtesy
of Dr. Steven R.
Pohlhaus, DDS,
FAGD
(Linthicum,
Maryland)

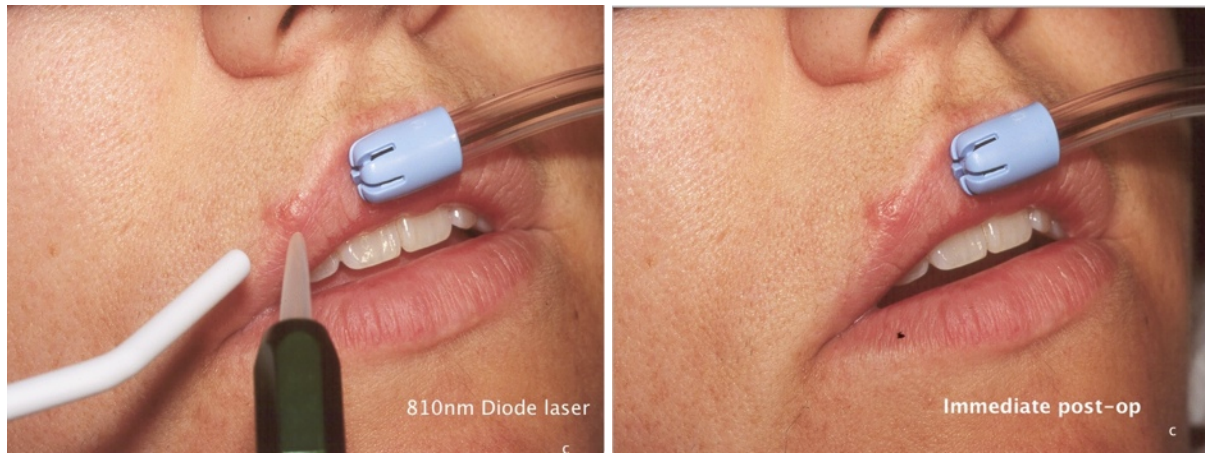


Fig.4 Appearance of Herpetic Lesion treated with Diode Laser.
Photo courtesy of Dr. Donald Coluzzi (Redwood City, California).

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[Int J Immunopathol Pharmacol.](#) 2010 Oct-Dec;23(4):1167-76.

Inhibition of HSV-1 replication by laser diode-irradiation: possible mechanism of action.

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Source

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Abstract

Herpes labialis are the most frequent clinical manifestations of HSV-1 infection. Epithelial cells are able to respond to HSV-1 presence inducing the expression of IL-6, IL-1, TNF- α and IL-8. These proinflammatory cytokines have a function in the acute-phase response mediation, chemotaxis, inflammatory cell activation and antigen-presenting cells. In the human epithelial cell models, it has been demonstrated that, after an early induction of proinflammatory host response, HSV-1 down-modulates the proinflammatory cytokine production through the accumulation of two viral proteins, ICP4 and ICP27, whose transcription is induced by tegument protein VP16. These viral proteins, through the decreasing of stabilizing the mRNAs of proinflammatory genes, delay cytokine production to an extent that allows the virus to replicate. Moreover, viral transactivating proteins, ICP-0 and VP-16 induce IL-10 expression. The conventional treatment of herpes labialis involves the topical and systemic use of antiviral drugs but it is necessary to find new therapies that can act in a selective and non-cytotoxic manner in viral infection. Laser diode therapy has been considered as a non-invasive alternative treatment to the conventional treatment of herpes labialis in pain therapy, in modulation of inflammation and in wound healing. This study aims to report a possible mechanism of action of laser diode irradiation in prevention and reduction of severity of labial manifestations of herpes labialis virus. We investigated, in an in vitro model of epithelial cells HaCat, the laser-effect on HSV-1 replication and we evaluated the modulation of expression of certain proinflammatory cytokines (TNF- α , IL-1 β and IL-6), antimicrobial peptide HBD2, chemokine IL-8 and the immunosuppressive cytokine, IL-10. Our results lead us to hypothesize that LD-irradiation acts in the final stage of HSV-1 replication by limiting viral spread from cell to cell and that laser therapy acts also on the host immune response unblocking the suppression of proinflammatory mediators induced by accumulation of progeny virus in infected epithelial cells.