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# Hair Stimulation Following Laser and Intense Pulsed Light Photo-Epilation: Review of 543 Cases and Ways to Manage It

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## BACKGROUND

Stimulation of terminal hair growth following photo-epilation is a poorly understood problem with significant clinical relevance. We have observed this problem in a number of patients in our dermatologic laser practice in Basque Country Spain. To better evaluate the incidence and character of this phenomenon a retrospective chart review was performed on all patients who received laser and intense pulsed light (IPL) photo-epilation at this single center within the 5-year period from December 1998 to December 2003. Patient images before and after treatments were compared (digital images after 1999 and non-digital images before year 1999) and the medical history reviewed.

## METHODS

Five hundred forty-three patients with Fitzpatrick skin types II, III, and IV (13%, 68%, and 19%, respectively) ranging in age from 16 to 52 years received laser and/or IPL hair photo-epilation of the beard, neck and chin areas, excluding the upper lip. The number of treatments received by each patient ranged from 3 to 23. Hair epilation treatments were performed using a long pulsed 755 nm alexandrite laser (Gentlelase, Candela, Wayland, MA) and IPL source (Epilight, Lumenis, Santa Clara, CA) and a 1,064 nm Nd:YAG (Lyra, Laserscope, San Jose, CA). Only 10% of the treatments were performed with the 1,064 nm Nd:YAG laser. Treatments were usually performed every 2–3 months. The alexandrite laser was used in 85% of the treatments, IPL in 10% and Nd:YAG in 5%. The parameters used are listed in Table 1.

## RESULTS

Of the 543 patients who received laser/IPL hair photo-epilation, 57 (10.49%) demonstrated an increase in hair growth compared to baseline. The increased hair growth occurred within the area that was treated and also in the areas bordering the treated area, and appeared thicker and darker than the hairs initially treated (Figs. 1 and 2). An additional 44 (8.10%) patients demonstrated no apparent reduction in hair growth following treatment. Four hundred twenty-four patients (78.08%) demonstrated a decrease in hair growth with ongoing treatments. Only 14 patients (2.5%) were discharged from the clinic due to near complete hair reduction. These results are summarized in Table 2.

The increased terminal hair growth occurred mostly in areas in which fine hair or both fine and coarse hair was present prior to initiation of treatment. Hair growth occurred with greater frequency in patients treated with the Alexandrite and IPL devices compared those treated with the Nd:YAG, however, the later device was used less frequently. Patients that developed terminal hair growth were in the following age groups: 19–31 years, 44 patients; 30–40 years, 8 patients; greater than 40 years, 5 patients. The onset of increased terminal hair growth was noted between the third and tenth treatment in 39 (72.2%) of 57 patients, and 11 (19%) of 57 between the third and fourth treatment. Most patients had a normal hormonal history. Sixteen patients had irregular menses or documented ovarian cysts.

Because the terminal hair growth occurred both within the treated areas and also at the periphery of treated areas it was thought that sub-therapeutic thermal energy delivered to nearby follicles induced terminal hair growth. Subsequent application of cold packs surrounding the treatment area during treatments and treating all patients with two passes has minimized the incidence of terminal hair growth (Fig. 3). Since we have instituted this method in our clinic 2 years ago, we have treated over 200 patients and have not had any patients with hair growth stimulation.

## DISCUSSION

Despite the widespread use of lasers and IPL for hair reduction, the biologic mechanism of photo-epilation is largely unknown. Upon treatment with a laser or IPL device, light is absorbed over millisecond pulse durations by melanin contained within melanosomes in the hair matrix and within keratinocytes in the hair shaft [1,2]. Heat energy is transferred from the follicular matrix to the surrounding non-pigmented follicular epithelium and perifollicular dermis [1–4]. Sufficient thermal injury to the follicle and its surrounding tissue results in miniaturization of follicles such that they become clinically unapparent for a variable duration of time [2]. The precise target of thermal injury from adjacent melanosomes or the

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**TABLE 1. Laser Hair Epilation Devices and Parameters**

| Device | Wavelength (nm) | Fluence (J) | Spot size | Pulse duration (milliseconds) | Cooling |
|---|---|---|---|---|---|
| Alexandrite gentle plus (Candela™) | 755 | 12–18 | 12 and 18 mm | 3 | Cryogen spray 40 milliseconds/ 30 milliseconds |
| IPL (Epilight™) | 645, 690, and 735 | 35–38 | 4.5 cm × 0.5 cm | 3–20 | Cold gel |
| Long pulsed Nd:YAG (Lyra™) | 1,064 | 24–30 | 10 mm | 35–50 | Cold gel |

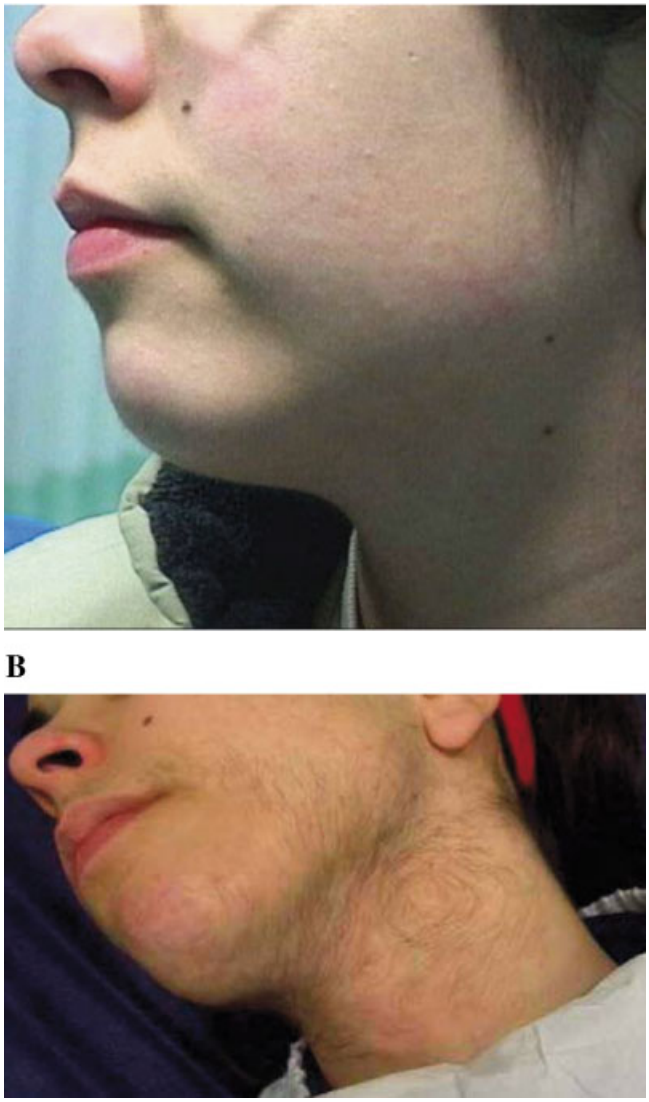


Fig. 1. **A**: Before photo-epilation. **B**: Terminal hair growth stimulation within and around the photo-epilated area after 12 sessions. [Figure can be viewed in color online via www.interscience.wiley.com.]

subsequent biologic events that lead to clinical hair reduction is not understood. Potential targets include cells critical for follicular cycling, including the follicular stem cells located within the bulge area of the outer root sheath and cells of the follicular papilla [5]. This may include cell populations necessary for communication during follicular cycling as well as cell populations essential for follicular morphogenesis itself.

Histological studies have shown that within a photo-epilated area not all of the follicles are thermally injured

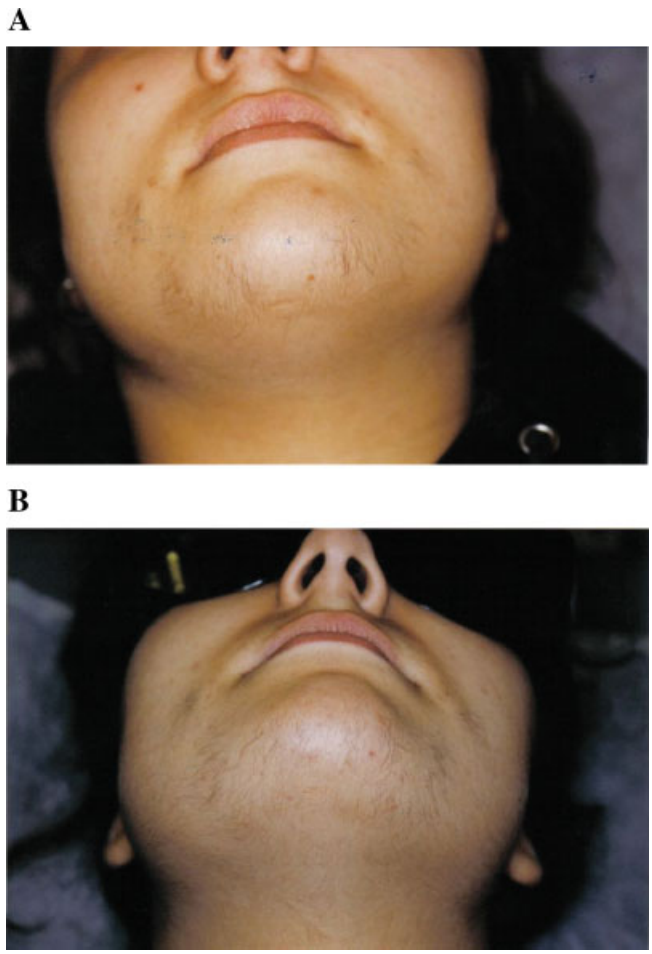


Fig. 2. **A**: Before photo-epilation. **B**: Terminal hair growth stimulation within and around photo-epilated area after five sessions. [Figure can be viewed in color online via www.interscience.wiley.com.]

**TABLE 2. Results**

| Subjects (%) | Response |
|---|---|
| 57 (10.49) | Increased terminal hair growth (equally distributed in all skin types) |
| 44 (8.10) | No change in hair growth |
| 424 (70.08) | Reduced hair growth (continued treatment) |
| 14 (2.5) | Persisting reduction in hair growth (discharged from clinic) |

[1,6], which suggests that some follicles are more susceptible to photo-thermal injury than others. Differences in the content of melanin associated with differing stages in the hair cycle may account for this, since melanogenesis ceases during catagen and telogen, commencing again during anagen. Follicles in early anagen have been thought to be most susceptible to photo-epilation since they contain melanin which is located high in the dermis relative to late anagen follicles residing deep in the subcutaneous tissue beyond the optical penetration of red and near infrared wavelengths [1,6]. Although the degree of melanin within growing hairs is associated with efficacy of treatment [1,2], the degree to which melanin in the shaft versus the matrix plays a role in affecting efficacy depends upon whether the target of thermal injury is primarily in the follicular stem cells located in the bulge or the follicular papilla; heat transferred from the shaft may heat primarily the follicular infundibulum, whereas heat transferred from the matrix may affect primarily the lower segment of the follicle. Clearly, the efficacy of laser hair epilation is fluence dependent, with the greatest amount of hair loss occurring with higher fluences [1,2]. Our observations that stimulation of hair growth occurred at the periphery of treated areas and that such hair growth could be minimized in the same patients by applying cold packs to the surrounding area and by using double passes with each treatment suggests that sub-therapeutic fluences at the periphery of treated areas induce terminal differentiation of hair growth rather than miniaturization. In other words, instead of inducing miniaturization with a subsequent prolonged telogen phase, follicles are instead shifted towards terminal anagen hair growth.

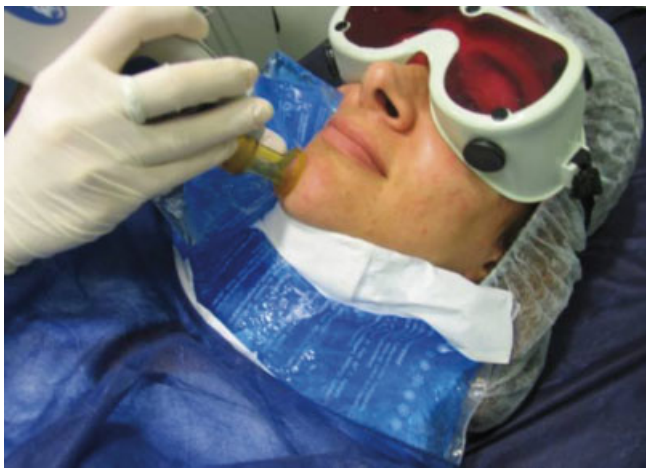

Fig. 3. The use of cold packs to prevent peripheral stimulation of hair growth. [Figure can be viewed in color online via www.interscience.wiley.com.]

Acquired localized hypertrichosis has been described in various other settings of dermal injury, including terminal hair growth at the periphery of a burn [7], transient limb hypertrichosis associated with casting [8,9] peri-incisional hypertrichosis following knee surgery or fracture sites [10,11], distal hair growth following lymphadenectomy [12], terminal hair growth at the site of bug bites [13] and verruca vulgaris [14], local reactions to measles [15], smallpox [16] and other vaccines [17], and chronic rubbing, scratching, and biting associated with various clinical situations [18,19]. Common to these widely varied clinical presentations is the hypothesis that local hyperemia or inflammation may lead to localized terminal hair growth.

Consonant with this idea, in addition to the complex orchestrated events of follicular cycling that lead to transformation of the follicle proper, the surrounding follicular epithelium, associated dermal components, and follicular vasculature undergo spectacular morphogenesis with each growth cycle [5]. A pronounced increase in follicular vascularization occurs during anagen that is accompanied by the upregulation of vascular endothelial growth factor (VEGF) in outer root sheath keratinocytes [20]. This is followed by a rapid regression in perifollicular vessels during catagen. Indeed growing follicles have much higher perfusion requirements than resting follicles. These accounts taken together with our observations raise the compelling possibility that sub-therapeutic thermal injury to the follicular vasculature may affect follicular cycling in such a way to induce terminal hair growth rather than miniaturization. Alternate hypotheses include the possibility that sub-therapeutic injury to the follicle may result in the release of factors that alter follicular angiogenesis and influence hair cycling. Additionally, both ultrastructural and light microscopic studies have demonstrated the uniform induction of perifollicular inflammation associated with photo-epilation that persists for up to 2 weeks. Thus, it follows that this local inflammatory response may also affect follicular cycling in such a way to induce terminal hair growth. While feasible, this idea does not explain why some follicles react in this way and others do not, since inflammation is not selective and thus not limited to less thermally injured areas.

In our experience, there are five key factors associated with failure to epilate and risk of hair stimulation:

(1) The thickness of treated hair: thicker hair is easier to heat because the follicle reaches a high enough temperature to destroy the cells critical for follicular cycling; thinner hairs derived from follicles with less chromophore absorb light energy less efficiently. This explains the failure to epilate fine hair that occurred on areas on the face with fine hair growth

and at other sites such as the abdomen, linea alba, and back and shoulders in men.

(2) The color of treated hair: melanin within melanocytes of the follicular matrix and shaft is the chromophore absorbed during photo-epilation, thus darker hair is more efficiently heated.

(3) The depth of treated hair: optical penetration of light may not be deep enough to adequately thermally injure deeply growing anagen hairs in some areas. This is only a hypothesis, however, since we do not have in vivo real time sequences of lasers reaching the target cells. It is possible that telogen or late anagen follicles that are located in the dermis may be more susceptible to hair growth stimulation.

Based on these observations, our current photo-epilation protocol includes the use of ice packs for all patients with fine hair growth on facial or body areas. In addition, we have observed in side to side studies that two passes with a long pulsed 755 nm Alexandrite laser using an 18 mm spot size is more effective than a single pass (unpublished work by N.L.). Our current technique is to use 12–14 J/cm$^2$ followed 1 minute later by a second pass using 8–10 J/cm$^2$. Therefore, the amount of energy delivered is important in definitive destruction of the key cells responsible of normal follicular cycling. Sub-optimal energies seem to stimulate these cells and induce longer thicker hairs as a consequence of accelerating the transition from vellus to terminal follicles. This phenomenon by which heat induces hair growth has been previously observed [9,10].

In our cases, the presence of fine hair prior to treatment appeared to be the most important factor for increased risk of paradoxical terminal hair growth. In addition, some areas appeared to be at higher risk: terminal hair growth occurred most often in the low maxillary or "beard" area, neck, lateral cheeks and chin areas in young women with either skin types II, III, or IV. Patients with hair stimulation were found in all skin types. Differences in skin type likely matters only in that lower energies may be used in patients with darker skin types, and thus it was probably easier to deliver sub-optimal energies. In these darker skinned patients, the use of double passes at lower energies should be attempted. Alternatively, a single treatment using one pass followed by a second single pass treatment 1 week later may also be tried. Terminal hair growth was mostly noted between third and fourth treatment, but also occurred as late as after the tenth treatment. The application of cold packs surrounding the treatment area during treatments and use of double pass technique appears to minimize the incidence of terminal hair growth within and around photo-epilated areas. Referral to an electrologist may be considered for patients who are at high risk.

Previous descriptions of hair stimulation have occurred with the use of various devices, including 694 nm Ruby, 755 nm Alexandrite, and 810 nm Diode lasers in addition to IPL sources [21–26]. It is unclear if hair removal with the 1,064 nm Nd:YAG laser is less inclined to cause hair stimulation or if it is simply used less often as is the case in our practice. The more common occurrence of hair stimulation on the lower face in females observed in this study is also consistent with other reports, however reports on the back of men have also been described. Females with vellus hair on facial "beard area" should be anticipated a chronic treatment with on multiple going sessions for years.

Although the majority of women in this study had no history of hormonal abnormalities, the true hormonal status cannot be certain from historical data. Thus, hormonal abnormalities may or may not be directly involved hair growth stimulation. Nevertheless, hirsute individuals may be at an increased risk regardless of hormonal status. It is our observation that the amount of vellus facial hair may change with different hormonal cycles or may be induced by laser epilation during different treatment sessions. In our experience photo-epilation should be performed more frequently and with higher energies to optimize efficacy in these patients.

Previous reports of paradoxical hair growth associated with laser-IPL photoepilation suggest the incidence is uncommon [21–26]. In addition, because the majority of the reported cases occurred in individuals of Fitzpatrick skin types III–V it has been felt that these patients are at greatest risk. Our observations suggest that hair growth stimulation following laser/IPL photo-epilation may be more common than previously recognized and that individuals of Fitzpatrick skin type II are also susceptible.

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