

Dermatologic Lasers in the Treatment of Aging Skin

Jasna Lipozenčić, Zrinka Bukvić Mokos

University Hospital Center Zagreb, Department of Dermatology and Venereology,
School of Medicine University of Zagreb, Zagreb, Croatia

Corresponding author:

Professor Jasna Lipozenčić, MD, PhD
University Hospital Center Zagreb
Department of Dermatology and Venereology
School of Medicine University of Zagreb
Šalata 4
HR-10000 Zagreb
Croatia
jasna.lipozencic@zg.htnet.hr

Received: June 30, 2010

Accepted: July 18, 2010

SUMMARY Skin aging includes intrinsic aging, a universal and inevitable process attributable to the passage of the time alone; and photoaging, changes attributable to chronic sun exposure, which are neither universal nor inevitable. The major clinical features of aging skin include xerosis, laxity, wrinkles, slackness, and the occurrence of benign neoplasms such as seborrheic keratoses and cherry angiomas. Photoaging is characterized by dryness (roughness), actinic keratoses, irregular pigmentation (freckling, lentigines, guttate hypomelanosis, persistent hyperpigmentation), wrinkling, stellate pseudoscars, elastosis (fine nodularity and/or coarseness), inelasticity, telangiectasia, venous lakes, purpura (easy bruising), comedones (*maladie de Favre et Racouchot*) and sebaceous hyperplasia. Current antiaging therapy consists of lasers, intense pulsed light as well as fillers, neurotoxins, radiofrequency, microdermabrasion and chemical peeling. Over the last 50 years, lasers applications in dermatology have become more specific and often irreplaceable. In this manuscript laser resurfacing and laser therapy of vascular and pigmented lesions of aging skin will be overviewed. Current trends show an increase in the number of nonablative and fractional resurfacing procedures because they are followed by less intense side effects and faster recovery rates compared to ablative laser rejuvenation, although producing mild improvement.

KEY WORDS: aging skin, lasers, corrective procedures

INTRODUCTION

For decades now, we are witnessing a great increase in the number of individuals who visit dermatologists, hoping to reverse the signs of aging.

Skin aging is the result of two distinct phenomena: intrinsic aging, a universal and inevitable process attributable to the passage of time alone; and photoaging, as superposition upon intrinsic aging of changes attributable to chronic sun exposure, which are neither universal nor inevitable.

Intrinsic aging depends primarily on genetic and hormonal factors. Accumulation of reactive oxygen species (ROS) has an important influence on the process of intrinsic aging, producing endogenous damage to cellular membranes, enzymes, and DNA (1). Additionally, wrinkling occurs as the result of different influences: changes in muscles, the loss of subcutaneous fat tissue, gravity forces, and the loss of substance of facial bones and

cartilage (2). The major clinical features of aging skin include xerosis, laxity, wrinkles, slackness, and the occurrence of benign neoplasms such as seborrheic keratoses and cherry angiomas. These manifestations are accompanied by histologic changes such as decreased number of melanocytes and Langerhans cells in epidermis, flattening of the rete ridges with reduced surface contact of the epidermis and dermis at the dermoepidermal junction, accompanied by the loss of dermal volume (3).

Photoaging is not simply 'accelerated aging'; it comprises of specific clinical and histologic skin changes (4). Both UVA (320-400 nm) and UVB (280-320 nm) rays are implicated in causing the changes of actinically damaged skin. UVA rays were originally thought to be an unimportant factor in sun-damaged skin; now, it is believed that UVA plays a substantial role in photoaging. UVA constitutes approximately 90% of total UV radiation and penetrates deeper into the skin, having its greatest effect in the dermis rather than the epidermis. UV radiation that is absorbed by cellular DNA causes a number of cutaneous events: the formation of DNA photoproducts, particularly pyrimidine dimers, the impaired function through mutation of the cutaneous cell cycle-regulating intranuclear p53 protein, and alterations in immune surveillance (5). Clinical features of actinically damaged skin include dryness (roughness), actinic keratoses, irregular pigmentation (freckling, lentiginos, guttate hypomelanosis, persistent hyperpigmentation), wrinkling, stellate pseudoscars, elastosis (fine nodularity and/or coarseness), inelasticity, telangiectasia, venous lakes, purpura (easy bruising), comedones (*maladie de Favre et Racouchot*) and sebaceous hyperplasia. UV radiation is also responsible for the induction of most non-melanoma skin cancers and probably of melanoma as well (6).

The histologic hallmark of actinically damaged skin is elastosis, an overgrowth of abnormal deranged elastic fibers that are deposited in the papillary dermis. A massive loss of collagen is observed. Lymphocytic infiltrate predominates in the dermis, a phenomenon that gave rise to the term 'heliodermatitis' when referring to inflamed photodamaged skin. Small vessels in papillary dermis appear dilated and tortuous and display thickened basement membrane. The dermoepidermal junction is flattened, and the keratinocytes frequently display disorganized maturation as well as some cytologic atypia. There is also a significant decrease in the number of Langerhans cells.

The loss of immune responsiveness appears to be important in the development of skin malignancies (7).

The acquisition of new scientific concepts has been paralleled by novel scientific developments resulting in new therapeutic methods in corrective dermatology. In the 1997-2007 period, the American Society for Aesthetic Plastic Surgery (ASAPS) noticed a 446% increase of corrective procedures, including a 747% increase of non-surgical and 98% increase of surgical procedures. A variety of anti-aging products and medical interventions have been developed to improve the individual's appearance: topical agents (all-trans-retinoic acids, alpha-hydroxy acids), chemical peels (alpha-hydroxy acids, trichloroacetic acid, Jessner's, phenol), hormone replacement therapy, injectable agents, surgical therapy, dermabrasion, lasers, intense pulsed light, radiofrequency, etc. However, laser therapy is now considered first-line treatment for most of the clinical signs of aging skin.

LASER HISTORY

The term laser is an acronym for 'light amplification by stimulated emission of radiation'. Laser light has unique properties that account for its therapeutic activity: monochromaticity (the emitted light is of a single, discrete wavelength), coherence (laser light travels in phase with respect to both time and space) and collimation (emission of a narrow, intense beam of light in parallel fashion). Thus, laser light can be focused onto small spot sizes allowing for precise tissue destruction (8).

The first laser was developed by Theodore Maiman in 1960, using for the first time monochromatic 694 nm ruby light. In 1962, J. Robiex at Compagnie Générale d'Electricité (CGE), later CILAS Company, produced neodymium-glass lasers delivering nanosecond pulses of energy. In 1963, Dr Leon Goldman, a dermatologist, initiated ruby laser treatment for a variety of cutaneous pathologies. Later, advances in laser technology have progressed rapidly, resulting in development of new laser systems with better therapeutic results and low risk of adverse effects (9). In the 1960s, the emphasis was on the mode locking of flashlamp-pumped ruby, neodymium-glass and organic dye lasers; in the 1970s and 1980s there were major developments in the generation of sub-picosecond and femtosecond pulses from continuous-wave organic dye lasers; whereas in the 1980s and early 1990s there were concerted efforts in the generation of femtosecond pulses in

the near-infrared spectral region. Cutaneous laser surgery was revolutionized in 1983, when Anderson and Parish proposed a theory of selective photothermolysis. According to this theory, controlled destruction of targeted lesion with minimal damage to the adjacent tissues can be achieved under following conditions: first, the emitted wavelength must be absorbed preferentially by the intended tissue target or chromophore (molecules with unique absorption spectra, which are responsible for imparting color to substances); second, the energies produced by laser systems should be sufficiently high to inflict thermal damage to the target; and third, the time of tissue exposure to the laser must be shorter than thermal relaxation time (defined as the time required for the targeted site to cool to one half to its peak temperature immediately after laser irradiation). On the basis of these principles, laser parameters can be tailored to effect destruction of the tissue confined to microscopic sites of selective light absorption in the skin, such as blood cells and pigmented cells, with minimal collateral thermal damage (10).

PREFERABLE LASER TREATMENT FOR VASCULAR LESIONS OF AGING SKIN

Vascular lesions that are most frequently observed on aging skin include telangiectasia, cherry angiomas, venous lakes, and rosacea. The most commonly used lasers in treating these lesions include pulsed-dye laser (585-595 nm), KTP or diode (532 nm), and Nd:YAG (532 and 1064 nm) laser (11). Their use is based on the evidence that peak absorption of light by oxyhemoglobin occurs within the yellow portion of the electromagnetic spectrum (12).

Pulsed-dye laser (PDL) device is nowadays considered the treatment of choice for port wine stains, hemangiomas and telangiectasias. Beside these, other vascular lesions amenable to ablation with PDL are: cherry angiomas, venous lakes, nevus simplex, arteriovenous malformations, and lymphatic malformations (13). Preoperatively, especially when larger lesions are treated, topical application of eutectic mixture of 2.5% lidocaine and 2.5% prilocaine can be used. Postoperatively cool gel packs should be applied to provide temporary pain control and decrease swelling. Erythema and swelling usually last for about 1-2 hours post treatment. If crusting occurs, neutral ointment should be used. Treatment is usually repeated every 4-6 weeks. Complications from PDL are uncommon.

Very rarely, cutaneous depressions have occurred 1-2 months after the treatment with PDL. They are temporary and usually resolve in 3-18 months (14).

According to our own experience, Nd:YVO4 (diode pumped frequency doubled solid state) 532 nm laser has been successful in treating telangiectasia and cherry angiomas. The procedure is well tolerated, producing minimal discomfort. The use of ice-packs for cooling before and during laser treatment protects the epidermis from potential overheating, permitting higher fluences to be safely applied to the skin and reducing the risk of dyspigmentation. The most common side effects include mild erythema, edema and transient crusting. This laser is suitable for the treatment of superficial vascular lesions and does not produce significant postoperative purpura.

LASER MANAGEMENT FOR PIGMENTED LESIONS OF AGING SKIN

Irregular pigmentation (freckling, lentigines, persistent hyperpigmentation) are the most common hypermelanotic lesions of aging skin. To obtain selective photothermolysis of melanin, these lesions should be treated with laser light having a wavelength appropriate to absorption characteristics of melanin. A variety of different laser systems, which emit wavelengths of 500-1100 nm, may be used to remove melanin from the skin: pigmented lesion pulsed dye laser (510 nm), Q-switched (QS) ruby laser (694 nm), the Q-switched alexandrite laser (755 nm) and the Q-switched Nd:YAG laser (1064 nm), which can be frequency-doubled to produce visible green light with a wavelength of 532 nm (15). Green light lasers with shorter wavelengths penetrate optically very little into skin layers and produce excellent results in treating epidermal lesions. Lasers which emit red light with longer wavelengths are more successful in treating dermal lesions.

The pigmented lesion dye 510 nm laser is effective in the treatment of epidermal pigmented lesions. The Q-switched ruby 694 nm laser emits visible red light that is strongly absorbed by superficial melanin, which can lead to permanent hypopigmentation and depigmentation in darker skin types. QS alexandrite 755 nm laser light penetrates deeper in tissue and less often produces hypopigmentation compared with the Q-switched ruby laser. QS Nd:YAG laser emits 1064 nm light, but it can also be frequency-doubled using a potassium diphosphate crystal to produce visible

green light with a wavelength of 532 nm. Green light of 532 nm is strongly absorbed by melanin, while red light of 1064 nm penetrates deeper in the dermis but with lower melanin absorption coefficient.

After most of the above mentioned laser treatments, the crust is formed at the site of laser irradiation and peels off after several days (16).

CUTANEOUS LASER RESURFACING

Cutaneous laser resurfacing represents the major advance in the treatment of severely photodamaged facial skin, photoinduced facial rhytides, dyschromias, and atrophic scars. Ablative laser resurfacing was introduced in the 1980s with continuous-wave CO₂ lasers, but their use was limited because of delayed healing and scarring. High-energy, pulsed, and scanned CO₂ and Er:YAG lasers have been in widespread use since the mid-1990s for ablative resurfacing. The Er:YAG laser has a higher absorption coefficient in water-containing tissue; because 90% of the epidermis is composed of water, most of the energy of the erbium laser is superficially absorbed. With Er:YAG laser photomechanical tissue effect results, whereas a photothermal tissue reaction is primarily effected by CO₂ treatment. However, the prolonged recovery time and significant risk of postoperative hyper- and hypopigmentations, scars and other complications, prompted to development of nonablative and fractional resurfacing. Nonablative laser systems produce stimulation of collagen production and remodeling while preserving epidermis. This results in little or no healing time and less patient discomfort. The most commonly used lasers for nonablative resurfacing include pulsed dye laser (585-595 nm), Nd:YAG (1064 nm, 1320 nm), diode (1450 nm) and Er:glass (1540 nm) laser. Fractional resurfacing thermally ablates microscopic columns of epidermal and dermal tissue in regularly spaced arrays over a fraction of skin surface. Each column is approximately 70-150 microns in width and 400-700 microns in depth. Intervening areas of normal skin are left untouched, which rapidly repopulate the ablated columns of skin. This concept increases efficacy as compared with nonablative resurfacing but with faster recovery as compared with ablative resurfacing. For these reasons, fractional skin resurfacing is nowadays the most popular method of laser rejuvenation (17).

Proper patient selection for laser resurfacing is essential for obtaining successful procedure. Pa-

tients should be evaluated for their skin type according to the Fitzpatrick system; lighter skin types (I-II) are at a lower risk to develop post-inflammatory hyperpigmentation, whereas skin types IV-VI more often develop dyschromia. Postoperative scarring usually develops in patients with impaired wound healing from an underlying collagen vascular disease, immunodeficiency, or history of isotretinoin use within the past year. Absolute contraindications for laser resurfacing include isotretinoin use within 1 year of the procedure, concurrent bacterial or viral infection and ectropion. Patients with unrealistic expectations should be discouraged. Relative contraindications for laser resurfacing include history of dyschromias, ongoing UV exposure, hyperelastic or keloidal skin, history of radiation therapy, collagen vascular disease and prior cosmetic surgery (chemical peels, dermabrasion, laser surgery, and blepharoplasty). Preoperative measures are performed to maximize the result of laser resurfacing and to avoid the possible unwanted effects. In order to provide a more homogeneous wound during resurfacing, expedite re-epithelialization and minimize postoperative hyperpigmentations, topical hydroquinone, alpha-hydroxy acids and tretinoin are introduced. Also, the standard of practice is that all laser resurfacing patients need to be started on antiviral therapy 2 days prior to the procedure (continued for 10-14 days until complete re-epithelialization).

Anesthesia varies from simple local application of EMLA cream (a eutectic mixture of lidocaine and prilocaine) through local infiltration with 1% lidocaine to regional anesthesia with nerve blocks to general anesthesia.

In order to minimize the risk of post-inflammatory hyperpigmentation, sun avoidance is recommended for 2 to 3 months postoperatively, and the use of broad-spectrum sunscreens is obligatory (17,18).

CONCLUSION

Nowadays, nonablative and fractional resurfacing is more frequently performed compared to ablative methods because of minimal down time after procedure and minimal risk of unwanted effects and complications. Proper choice of high quality treatments is the responsibility of dermatologist specialized in the field of corrective dermatology, especially in the light of the great number of new therapeutic options offered by fast technological advancement. Unfortunately, these demanding corrective treatments are ever more frequently

performed at non-medical institutions and by incompetent persons. Properly trained and experienced physician is fully aware of the crucial role of appropriate assessment of the patient including patient's expectations. He will explain to the patient the details concerning corrective procedure, the potential risks and the expected outcome. A predictable lack of patient's compliance during the postoperative period and unrealistic expectations are considered absolute contraindications for any laser treatment.

References

1. Yaar M, Gilchrest BA. Aging of skin. In: Freedberg IM, Eisen AZ, Wolf K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's dermatology in general medicine. New York: McGraw-Hill; 2003. pp.1386-98.
2. Pierard GE, Uhoda I, Pierard-Franchimont C. From skin microrelief to wrinkles. An area ripe for investigation. *J Cosmet Dermatol* 2003;2:21-8.
3. Lavker RM, Zheng PS, Dong G. Aged skin: a study by light, transmission electron, and scanning electron microscopy. *J Invest Dermatol* 1987;88:44s-51s.
4. Gilchrest BA. A review of skin ageing and its medical therapy. *Br J Dermatol* 1996;135:867-75.
5. Pinell SR. Cutaneous photodamage, oxidative stress, and topical antioxidant protection. *J Am Acad Dermatol* 2003;48:1-19.
6. Taylor CR, Stern RS, Leyden JJ, Gilchrest BA. Photoaging/photodamage and photoprotection. *J Am Acad Dermatol* 1990;22:1-15.
7. Leyden J. What is photoaged skin? *Eur J Dermatol* 2001;11:165-7.
8. Anderson RR, Ross E. Laser-tissue interactions. In: Fitzpatrick RE, Goldman MP, editors. *Cosmetic laser surgery*. Mosby: St Louis; 2000. pp. 1-30.
9. Tanzi EL, Lupton JR, Alster TS. Lasers in dermatology: four decades of progress. *J Am Acad Dermatol* 2003;49:1-31.
10. Anderson RR, Parrish J. Selective photothermolysis: precise microsurgery by selective absorption of pulsed radiation. *Science* 1983;220:524-7.
11. Stratigos AJ, Dover JS. Laser therapy for vascular lesions. In: Ferguson J, Dover JS, editors. *Photodermatology*. London: Manson Publishing Ltd; 2006. pp. 141-5.
12. Loo WJ, Lanigan SW. Recent advances in laser therapy for the treatment of cutaneous vascular disorders. *Laser Med Sci* 2002;17:9-12.
13. Laube S, Tibjee S, Lanigan SW. Treatment of resistant port wine stains with the V beam pulsed dye laser. *Laser Surg Med* 2003;33:282-7.
14. Garden JM, Bakus AD, Paller AS. Treatment of cutaneous hemangiomas by flashlamp pumped-dye laser; prospective analysis. *J Pediatr* 1992;120:550-60.
15. Kilmer SL. Laser eradication of pigmented lesions and tattoos. *Dermatol Clin* 2002;20:37-53.
16. Stratigos AJ, Dover JS, Arndt KA. Laser treatment of pigmented lesions – 2000. *Arch Dermatol* 2000;136:915-21.
17. Dover JS, Hruza GJ, Arndt KA. Lasers in skin resurfacing. *Semin Cutan Med Surg* 2000;19:207-20.
18. Alexiades-Armenakas MR, Dover JS, Arndt KA. The spectrum of laser skin resurfacing: nonablative, fractional, and ablative laser resurfacing. *J Am Acad Dermatol* 2008;58:719-37.