Sun-induced Aging of the Skin: Prevention and Treatment

G. Daniel Schachter, MD, FRCPC, DABD, Consultant Dermatologist, Sunnybrook & Women's College Health Sciences Centre and St. John's Rehabilitation Hospital; Lecturer, University of Toronto, Toronto, ON.

During the past century, the amount of time spent at leisure and exposed to the sun has increased, yet we have also become increasingly aware of the detrimental effects of the sun. The skin ages slowly (intrinsic, chronologic aging), but this process is enhanced or accelerated by sun exposure (extrinsic aging, photoaging). The features of photoaging will be presented, followed by the importance of the prevention of sun damage by sun avoidance and use of sunscreens. Methods of treating or reversing photodamage will be reviewed, including topical agents, chemical peels and use of lasers and other light sources.

Key words: photoaging, ultraviolet radiation, prevention, sunscreen, skin rejuvenation.

With the passage of time, most people will notice changes in their skin associated with aging. Babies have smooth, unwrinkled skin with few imperfections. As we age, some areas of skin that have received little or no sun exposure, such as the buttocks and the inner aspect of the upper arms, will retain their youthful appearance and texture. At the other end of the spectrum, those who have worked or played outdoors for years (e.g., farmers, fishermen, roofers and golfers), who have lived in sunny climates or have lightly pigmented skin, will eventually develop weather-beaten or leathery skin. In this article, the features of aging skin, how it develops, as well as prevention and treatment options will be reviewed.

Photodamage

Factors involved in the aging of the skin can be categorized as intrinsic (chronologic, genetic) or extrinsic (photoaging).[1-5] When not exposed to sun, the skin's aging process is slow and determined genetically. On the other hand, photoaging is related to sun or ultraviolet (UV) exposure over a span of many years. Most sun damage occurs in the first two decades of life when a child or young person spends hours, days or weeks in the sun at leisure (as adults, leisure time becomes intermittent). Photodamage, however, is cumulative over a person's lifetime. Smoking and other external or environmental factors, such as wind and cold, may play a small role in the development of aging, damaged skin. Exposure to ultraviolet radiation in tanning parlors also adds to photodamage and photoaging.

The principle UV radiation that reaches earth includes short ultraviolet B rays (UVB) and long ultraviolet A rays (UVA). UVB rays are filtered and diminished by the ozone layer, whereas UVA rays are unaffected by ozone, clouds or glass. UVB is 90% absorbed by the outer layer of the skin, the stratum corneum, whereas UVA penetrates more deeply into the skin to the dermis. UVB rays produce sunburn, while UVA rays produce a tan. It is now believed that UVA rays contribute to cumulative photodamage as well as aggravate and accelerate changes due to UVB rays.

Sun damage to the skin is either acute (sunburn, tan) or chronic (wrinkles, lentigines, aged skin, skin cancers). The skin acts as a protective cover for the body, blocking entry of chemicals and infectious agents, and protecting against thermal, physical and UV insults. The skin is a complex, multi-layered organ and is part of the immune system (Langerhans cells). Solar radiation can cause a local and a generalized immunosuppressant effect.[5,6]

The skin's barrier function includes complex molecular processes that are altered by chronic sun exposure, with resultant damage to the connective tissue. This damage is initiated by the generation of reactive oxygen species (ROS), leading to chemical modification of cellular elements such as DNA, proteins and lipids. UV radiation is absorbed by chromophores in the skin (DNA and urocanic acids), setting in motion photochemical reactions that eventually result in photoaging and skin cancer.[7-9]

Clinical Features of Photoaged Skin

Photoaged skin is mottled with blotchy hyperpigmentation and patchy loss of pigmentation (guttate hypomelanosis) (Table 1). Both freckling and lentigines (brown patches) may be present (Figure 1). The skin may be pebbly, yellowish and sallow (solar elastosis), sometimes with comedones, thickened and leathery (Figure 2), or thin and transparent with telangiectatic vessels. There is sagging of the skin, with wrinkles and furrows and cross-hatched lines (Figure 3), especially on the posterior aspect of the neck (cutis rhomboidalis nuchae) (Figure 4). There

Table 1
Effects of Sun Exposure on the Skin

Acute
– sunburn
– tan
– freckles, lentigines
Chronic (dermatoheliosis)
– solar elastosis (yellow, pebbly)
– telangiectatic vessels
– mottled, blotchy skin
– comedones
– cutis rhomboidalis nuchae
– poikiloderma of Civatte

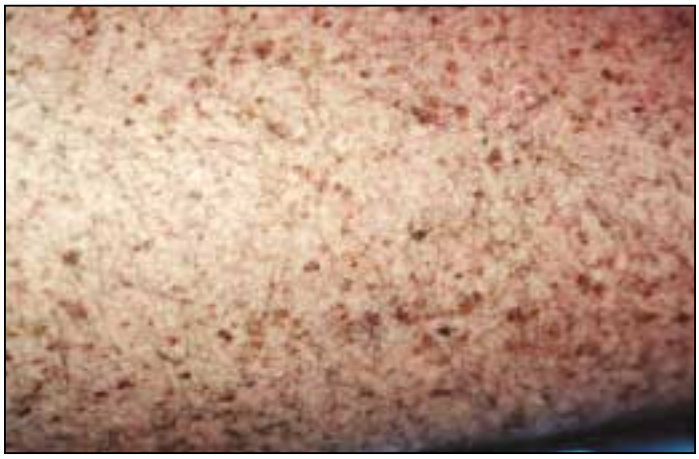

Figure 1: Severe photodamage with lentigines on the arm.

may be red, scaly, tender patches (actinic keratoses) (Figure 5),[10] or warty brown papules (seborrheic keratoses). Skin cancers such as basal cell carcinoma, squamous cell carcinoma and lentigo maligna melanoma may be seen on sundamaged, aged skin (Figure 6).

Prevention of Photoaging

At present there are no proven strategies to prevent intrinsic aging, but there are strategies to protect against photoaging. The most important aspect of prevention is sun avoidance. If possible, children, adolescents and adults should try to work and play in the shade. Outdoor activities should be scheduled to avoid the middle of the day (10 am to 4 pm) when UV radiation is at its peak. Wearing a wide-brimmed hat (5cm brim) and tightly woven clothing, such as t-shirts, is recommended. Little children should wear sun protective clothing and swim wear with a tight weave and coverage for the trunk and proximal limbs to optimize sun protection.

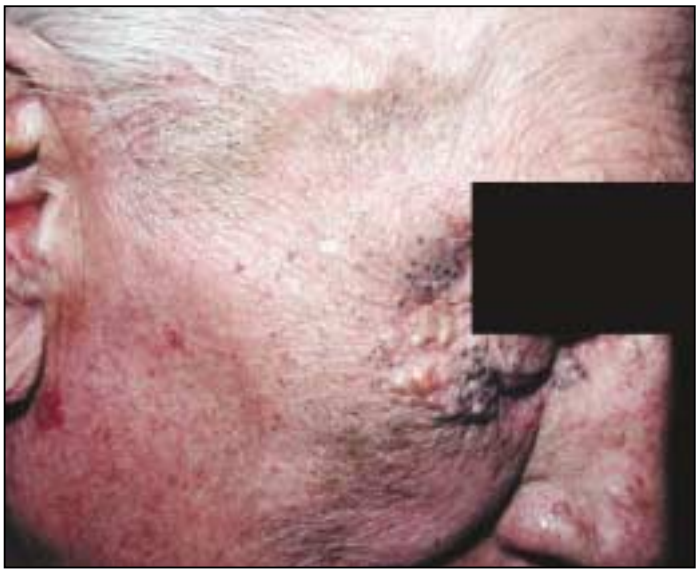

Figure 2: Severe photodamage with comedones and cysts.

Sunscreens are topical agents in cream, lotion, gel or spray forms that are applied to the entire skin before going outdoors and are re-applied after swimming, sweating and bathing.[11] Sunscreens should be applied before dressing (clothes or bathing suits), and are most important for areas of skin that are not covered by clothing. They should not be applied to the skin of an infant younger than six months, and should be used on cloudy as well as sunny days, since 60% of UV rays will penetrate the clouds. Sunscreens are needed at any time of the year when there is sun exposure, including winter when there is reflection of UV rays off the snow.

The Sun Protection Factor (SPF) number is a measure of the suncreen's ability to protect against erythema or UVB rays (250–320nm).[5,12,13] It is the ratio of the amount of UVB radiation required to produce erythema on sunscreen-protected skin to that required on unprotected skin. SPF numbers range up to 60 in North America, and higher in Europe. One must apply a sunscreen with an SPF of 15 or higher. Paraminobenzoic acid (PABA) and PABA esters (seldom used today), salicylates and cinnamates protect against or absorb UVB rays, while anthranilates, benzophenones, dibenzoylmethanes (Parsol 1789) and Mexoryl-SX protect against UVA rays (320–400nm).[12] The physical blockers, such as zinc oxide and titanium dioxide, block both UVB and UVA rays and visible light.

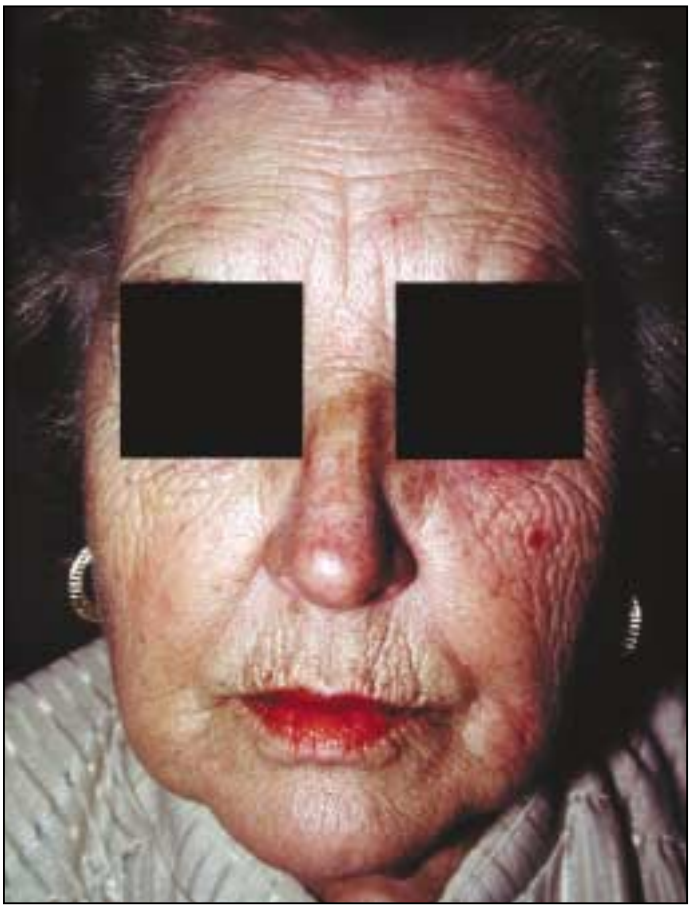

Figure 3: Severe photodamage with marked wrinkling.

Sunscreens have been shown to decrease the incidence of actinic keratoses—the precursors of squamous cell carcinomas. However, this protective effect has not been shown for basal cell carcinomas or melanomas.[14] Because sunscreens prevent sunburns, there is concern that people will get far more UV exposure since they will not burn, and UVA damage, deeper in the skin, will be greater.[1,12,14] If one must have a tan, use of a self-tanning lotion is safe and effective, but one must learn how to apply such a lotion properly.

Treatment of Photoaged Skin

Our society yearns for a youthful appearance. To achieve this, there are many options for rejuvenating the skin.[1] Surgical redraping of the skin by plastic surgery is the most aggressive and dramatic treatment. I will concentrate on other, more conservative approaches to treatment of photoaged skin.

Topical Agents

There are several topical agents that have been shown to improve photodamage. These include alpha hydroxy acids (AHAs), such as glycolic acid, as well as vitamin C and tretinoin. The initial work on AHAs by Van Scott, *et al.* suggested effects on cellular adhesion in the outer layers of the epidermis.[15] Bergfeld, *et al.* later showed that AHAs could improve photodamage, texture and wrinkling.[16]

Topical vitamin C (ascorbic acid) is an antioxidant that neutralizes free radicals and regenerates vitamin E.

The topical retinoids, especially tretinoin, have been studied extensively and have been shown to reverse several features of photoaging. The classic paper by Weiss, *et al.* showed that tretinoin reversed photoaging, both histologically and clinically, in a vehicle-controlled, double-blind study.[17] Clinical improvements are due to tretinoin's effects on the dermis and epidermis and include less mottled pigmentation (especially hyperpigmentation), less skin fragility, increased smoothness of the skin, decreased sallowness and improvement in fine wrinkles. Tretinoin acts as a hormone by binding to the retinoic receptors in the skin. Histologically, there are increases in dermal collagen and mucin, and reductions in abnormal elastin and melanin. The findings by Weiss, *et al.* have been reproduced in multiple publications since their initial report. The results of long-term topical tretinoin usage have been shown to reverse photoaging after four to 10 months, and the results may be long lasting or persistent.[18]

Recently, Phillips, *et al.* have reported that tazarotene cream 0.1%, another topical retinoid, can similarly reduce signs of photoaging.[19] Kligman and colleagues have shown that histologically, tretinoin cream can improve intrinsically aged (non-sun-exposed) skin.[20] Although the histologic changes were marked, the effects were not seen clinically.[21,22]

Resurfacing Chemical Peels

Chemical peels are classified by the depth of damage induced, from superficial (glycolic acid), to medium (trichloroacetic acid), to deep (phenol) peeling agents. Both glycolic acid (20–70%) and trichloroacetic acid (25–50+%) can be applied in varying strengths. The effects of a superficial peel are limited, and include exfoliation, mild erythema and perhaps a mild change in pigmentation, with repeated peels. There may be a greater effect on pigmentation and texture with medium-depth peels. The deep peels, which can affect the deep dermis, can have marked clinical effects with reversal of photoaging (improved pigmentation and texture, loss of wrinkles and tightening of the skin).

Figure 4: Severe photodamage with cutis rhomboidalis nuchae on the neck.

Microdermabrasion

This procedure, which fires microcrystals of aluminum or salt at the skin, is a mild abrasion or exfoliation of the skin's surface cells performed in a series of treatments.

Although the skin feels smooth and looks fresher after repeated treatments, the clinical improvement is not marked. Pigmentation may be improved, especially when microdermabrasion is carried out in association with non-ablative resurfacing.[23,24]

Laser Resurfacing and Dermabrasion

Both of these techniques produce a controlled depth injury to the skin. The injury can be limited to the epidermis, or the

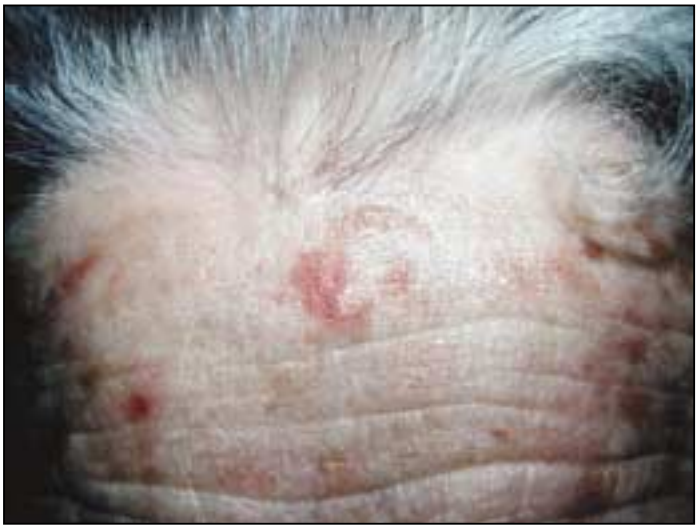

Figure 5: Actinic keratoses on the forehead.

upper, mid or deep dermis with resulting improvement clinically and histologically in photoaged skin. Dermabrasion has become less popular with the advent of lasers. It is a bloody procedure with the worry of HIV exposure, and there is a significant risk of scarring and pigmentary change. Laser resurfacing using the CO_2 or Erbium:YAG lasers was very popular in the 1990s until reports of scarring and late-onset loss of pigmentation became increasingly frequent. For this reason, the procedure has lost much of its glamour and appeal. In experienced hands, laser resurfacing can produce marked improvement in photoaged skin with changes in texture, pigmentation and wrinkling with tightening of the skin.[25,26]

Non-ablative Resurfacing

Since the mid-1990s, it has been noted that various non-invasive procedures with different lasers or other light sources (e.g., intense pulsed light [IPL]) produce remodeling changes in the skin with increased collagen and elastin production and clinical improvements in pigmentary and vascular features of photoaging. This procedure selectively produces thermal damage to the dermis while sparing the epidermis.[27,28]

The non-ablative procedures produce increasing improvement with multiple treatments. Clinically, one sees improvements in pigmentation (mottling, erythema, lentigines), pore size, texture and fine wrinkles. Non-ablative photorejuvenation is safe with few side effects, no down time, and little interference with lifestyle. One can return to work immediately after the procedure.

Coblation

Coblation is a resurfacing technique that uses an electrical device to remove damaged tissue; an electric current breaks the bond between the cells in the skin so the top layer can be wiped away. The procedure disintegrates damaged tissue layer by layer, giving the surgeon precise control and the ability to remove and sculpt tissue while causing minimal damage to adjacent healthy tissue. At present, coblation is not in widespread use. There have been few studies presenting data on this technology, and little interest has been shown.[1,29]

Radiofrequency Device

The Thermacool/TC system (Thermage Inc.) uses radiofrequency technology applied by electrodes, while the Aurora system (Syneron) combines radiofrequency and IPL technologies to produce non-ablative photorejuvenation.

Non-ablative photorejuvenation produces subtle clinical changes with increasing improvement in the skin with additional treatments. Improvement also has been proven histologically.[30]

Botox (Botulinum Toxin-A)

The treatment of wrinkles varies according to whether they are static or dynamic; static are treated with fillers, whereas Botox is used for dynamic. Botox is a potent neurotoxin that acts by inhibiting acetylcholine release at motor end plates to relax dynamic wrinkles caused by muscle action. Its greatest use is for glabellar frown lines, crowsfeet or laugh lines, and forehead lines. It is used for the upper face.[31]

Alone or in combination with dermal fillers, Botox can produce a more relaxed, youthful face.

Skin Fillers/Tissue Augmentation

Several substances can be injected into the skin to fill in or plump up static wrinkles and folds due to photoaging.[32] Filling agents include fat (microlipoinjection), collagen, hyaluronic acid products (Hylaform, Restylane, Perlane) and the permanent fillers, silicone and Artecoll (which contains tiny plexiglas polymethyl methacrylate beads, suspended in collagen). Most dermal fillers are injected at intervals, and the correction of wrinkles and folds can be maintained. The dermal filling agents will plump up or fill wrinkles and folds or scars

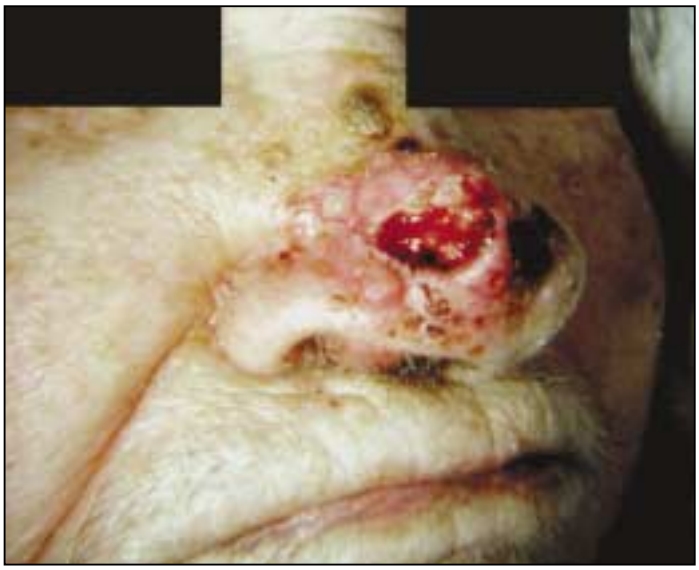

Figure 6: Basal cell carcinoma on the nose.

for varying lengths of time. The skin will turn over or metabolize collagen hyaluronic acid agents, whereas the more permanent agents will persist.

Non-facial Photoaged Skin

Photodamaged skin on the neck can be treated with Botox and non-ablative photorejuvenation. Skin on the dorsum of the hand and arms can be bleached or treated with tretinoin, superficial chemical peels or with non-ablative photorejuvenation. Fat injections into the dorsa of the hands also can produce younger looking hands.

Summary

Photoaged skin demonstrates cumulative changes in the skin due to sun exposure. These age-related changes can be improved or reversed. Prevention remains the most important concept for photoaging of the skin. ◆

No competing financial interests declared.

References

1. Lawrence N. New and emerging treatments for photoaging. Dermatologic Clinics 2000;18:99-112.
2. Yaar M, Eller MS, Gilchrest BA. Fifty years of skin aging. JID Symposium Proceedings 2002;7:51-8.
3. Gilchrest BA. Skin aging and photoaging: an overview. J Am Acad Dermatol 1989;21:610-3.
4. Guinot C, Malvy DJ, Ambriosine L, et al. Relative contribution of intrinsic vs. extrinsic factors to skin aging as determined by a validated skin age score. Arch Dermatol 2002;138:1454-60.
5. Rougier A. Are UV rays dangerous? Protection of the skin against ultraviolet radiation; John Libbey Eurotext 1998:1-9.
6. Uitto J, Brown DB, Gasparro FP, et al. Molecular aspects of photoaging protection of the skin against ultraviolet radiation. John Libbey Eurotext 1998:37-45.
7. Trautinger F. Mechanisms of photodamage of the skin and its functional consequences for skin ageing. Clin Exp Dermatol 2001;26:573-7.
8. Pinnell S. Cutaneous photodamage, oxidative stress and topical antioxidant protection. J Am Acad Dermatol 2003;48:1-19.
9. Fisher GJ, Kang S, Varani J, et al. Mechanisms of photoaging and chronological skin aging. Arch Dermatol 2002;138:1462-70.
10. Monheit GD. Consultation for photoaging skin. Dermatol Clin 2001;19:401-3.
11. Rosen CF. Photoprotection. Semin Cutan Med Surg 1999;18:307-14.
12. Debuys HV, Levy S, Murray JC, et al. Modern approaches to photoprotection. Dermatol Clin 2000;18:577-90.
13. McLean DI, Gallagher R. Sunscreens use and misuse. Dermatol Clin 1998;16:219-26.
14. Almahroos M, Yaar M, Phillips TJ, et al. Effect of sunscreen application on UV-induced thymine dimers. Arch Dermatol 2002;138:1480-5.
15. Van Scott EJ, Yu RJ. Hyperkeratinization, corneocyte cohesion and alpha hydroxy acids. J Am Acad Dermatol 1984;11:867-79.
16. Bergfeld W, Jung R, Vidimos A, et al. Improving the cosmetic appearance of photoaged skin with glycolic acid. J Am Acad Dermatol 1997;36:1011-3.
17. Weiss JS, Ellis CN, Headington JT, et al. Topical tretinoin improves photoaged skin: a double-blind vehicle-controlled study. JAMA 1988;259:527-32.
18. Kang S. Photoaging and tretinoin. Dermatol Clin 1998;16:357-64.
19. Phillips TJ, Gottlieb AB, Leyden JL, et al. Efficacy of 0.1% tazarotene cream for the treatment of photodamage. Arch Dermatol 2002;138:1486-93.
20. Kligman AM, Dogadkinn D, Lavker RM. Effects of tretinoin on non-sun-exposed protected skin of the elderly. J Am Acad Dermatol 1993;29:25-33.
21. Griffiths CEM. Retinoids: renaissance and reformation. Clin Exp Dermatol 1999;24:329-35.
22. Griffiths CEM. The roles of retinoids in the prevention and repair of aged and photoaged skin. Clin Exp Dermatol 2001;26:613-8.
23. Alster T. Microdermabrasion— "A Step Up." Peels, Skin and Allergy News 1999;30:9.
24. Alam M, Omura NE, Dover JS, et al. Glycolic acid peels compared to microdermabrasion : A right-left controlled trial of efficacy and patient satisfaction. Dermatol Surg 2002;28:475-9.
25. Fitzpatrick RE, Goldman MP, editors. Skin resurfacing with carbon dioxide and erbium lasers. Cutaneous Laser Surgery, 2nd edition. St. Louis, MD: Mosby, 1999.
26. Matarasso SL, Hanke W, Alster TS. Cutaneous resurfacing. Dermatol Clinic 1997;15:569-82.
27. Nelson JS, Majaron B, Kelly KM. What is non-ablative photorejuvenation of human skin? Sem Cutaneous Med Surg 2002;21: 238-50.
28. Sadick NS, Weiss R. Intense-pulsed light photorejuvenation. Sem Cutaneous Med Surg 2002;21:280-7.
29. Carruthers A, Langtry J, Burns R, et al. Thermal effects of coblation in dermal tissue. Research Outcomes Cosmetic Reconstruct Surgery 1998;1:1-4.
30. Goldberg DJ. New collagen formation after dermal remodeling with an intense pulsed light source. J Cutan Laser Ther 2000;2:59-61.
31. Carruthers A, Carruthers J. Clinical indications and injection technique for the cosmetic use of botulinum A exotoxin. Dermatol Surg 1998;24:1189-94.
32. Klein AW. Skin filling. Collagen and other injectables of the skin. Dermatol Clin 2001;19:491-508.