12. Understanding and treating photoaging

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INTRODUCTION

Pigment blemishes, wrinkles, and flaccid skin are not just the natural consequence of the passage of time, but the result of excessive time in the sun. Photodamage also develops into actinic keratoses and skin cancer, and because we live in an era of ‘ozone holes’, we can expect the incidence of photodamage and skin cancer to increase dramatically. People are simultaneously living longer than ever before, and more people will want facial rejuvenation, but a facelift carried out with photodamaged skin merely produces a ‘neater’ older person. When we do facelifts on people with healthy, young-looking skin, we actually rejuvenate the face. Therefore, all plastic and cosmetic surgeons should advise their patients on how to achieve healthy skin.

In order to devise a scientifically based strategy for preventing or treating photodamage, we need to understand what sunlight does to the skin. Of course, we all have to go out into the sun because we also need sunlight to be healthy so we have to use science to protect young skin while getting healthy sunlight, yet minimize photoaging.

A tan is the first step in photoaging and since most of our sun exposure occurs before the age of 20, young people already show signs of photoaging.

ETIOLOGY OF PHOTOAGED SKIN

Photons are ‘packets’ of energy that vary according to their wavelength. Most people believe that damage is done only by ultraviolet (UV) light, but in fact even violet, blue and green light can damage cells (‘soft’ green light can stimulate melanogenesis). Essentially, light enters into the skin and photon energy interacts at either the molecular or subatomic level. At the molecular level, photons are absorbed by chromophores in the skin (e.g. vitamin A, nucleic acids, amino acids, urocanic acid and melanin). Some chromophores shield the skin and melanin is the main chromophore that protectively absorbs photons of wavelengths ranging from 350 to 1200 nm.

Retinyl palmitate (a form of vitamin A) is a classical example of an important molecule that is destroyed by the energy of UVA photons (330–334 nm). DNA is destroyed by UVB (250–270 nm), whereas vitamin C (ascorbic acid) is damaged by visible, blue light.

At the subatomic level, the absorption of energy can also result in electron changes with a generation of free radicals. If a photon strikes a ‘vulnerable’ electron in the outer circuit of an oxygen atom, the electron is cast out of its circuit and the molecule, in its quest for another electron to stabilize...
the atomic relationship, now becomes a free radical. This initiates a destructive chain of chemical reactions, involving tens of thousands of molecules in a fraction of a second, which may result in damaged DNA or cell membranes. Furthermore, the displaced electron may be absorbed by another oxygen radical to make a superoxide radical, which is a much more aggressive free radical.

**SIGNS OF PHOTODAMAGE**

In essence, the various signs of photoaging are attributable to structural and DNA damage to the following cells.

The DNA in keratinocytes can be damaged to produce changes in appearance and function. These cells should be removed by the immune system, but they survive because the same irradiation depletes Birbeck granules in Langerhans cells, which lose their dendrites, and fail to recognize and facilitate the destruction of abnormal keratinocytes and melanocytes. Abnormal cells may proliferate and develop into keratoses and/or skin cancer. The epidermis becomes thinner, with flattening of the rete pegs. The stratum corneum thickens but with less cohesion of the cells.

1. Each melanocyte unit involves about 36 keratinocytes and dermal macrophages. If one melanocyte DNA is altered, it slowly forms a clone of hyperactive cells. While the change may only be detectable initially with the Woods light, later on this will manifest as typically mottled aged skin. If there is adequate vitamin A, pigmentation is controlled and melanin is evenly distributed. If the DNA of the melanocyte is damaged by irradiation, excessive amounts of melanin may be produced under lower light fluxes.

2. The fibroblast produces less collagen and glycosaminoglycans so the skin feels drier and wrinkles show up very easily. Ultraviolet irradiation, even at suberythemal doses, induces the release of matrix metalloproteinases (MMPs) that degrade anchoring fibrils, collagen and elastin. Vitamin A inhibits the activation of MMPs, so if the skin is deficient in vitamin A, the skin will age rapidly.

3. Collagen mRNA (messenger ribonucleic acid) is also downregulated, and with increased MMPs, this results in a net loss of collagen, which is itself damaged by UV light. Elastin, by contrast, is formed in greater quantities, but the elastin fibers are thick and do not form a healthy fine mesh to support the skin. Elastin fibers, fractured by UV light, roll up into little balls that result in elastosis.

4. Damage to vascular loops results in sallow skin and the loss of supporting fibers leads to telangiectasia.

These changes happen because free radicals and photons damage essential molecules normally found in skin. They happen particularly because vitamin A is damaged and hence is unable to prevent some important chemical responses that lead to photoaged skin. The antioxidant vitamins, C and E, and carotenoids are required in high doses to minimize free radical activity.

**VITAMIN A**

For decades it has been known that vitamin A (retinyl palmitate) is vital for healthy skin. Wise and Sulzberger worked with vitamin A and realized that it was extremely unstable in light and in 1938 they suggested that there is localized hypovitaminosis A in wrinkled skin. We now know that UVA rays are responsible for photodecomposition of retinyl palmitate, which is the major form of vitamin A in the skin. Ultraviolet-A rays are ubiquitous and plentiful and can penetrate through clouds and windowpanes. Tanning may lower the epidermal vitamin A (as retinyl palmitate) by 70–90%. Once the skin retinoids are depleted after heavy exposure to sunlight, it takes several days before diet alone can restore the normal cutaneous retinoid levels. On the other hand, application of an effective vitamin A cream can restore the normal levels within hours.

We also know from experience that topical vitamin A (retinoic acid) reverses almost all of the changes of photoaging. Retinoic acid is the one key signaling hormone for normal growth and differentiation that regulates about 350 genes and
modulates photoaging.\textsuperscript{7} It is essential for normal function of all the important cells of the skin: \textsuperscript{8} keratinocytes, \textsuperscript{9} melanocytes, \textsuperscript{10} Langerhans cells, \textsuperscript{11} and fibroblasts.\textsuperscript{12}

Cluver and Politzer recognized that vitamin A played an essential role in counteracting sun damage. They showed that every time we go out into sunlight, the photosensitive vitamin A molecule is denatured not merely in the skin, but also in the blood.\textsuperscript{13} With time, investigations have demonstrated that vitamin A is not merely beneficial for aging skin,\textsuperscript{14} but is essential.\textsuperscript{15} Women have an added disadvantage because blood levels of vitamin A drop when they menstruate.\textsuperscript{16,17} This means that during menstruation their skin is more vulnerable to photodamage.\textsuperscript{18}

Vitamin A has a vast array of physiological actions on the cells of the skin but is not (for practical purposes) an antioxidant, whereas vitamin C has a physiological role in the DNA of fibroblasts\textsuperscript{19} and melanocytes but is primarily an important antioxidant. Vitamin C is crucial for the reactivation of the tocopheryl radical that results from quenching another lipophilic free radical.\textsuperscript{20} Vitamin E, which is also denatured by light, has no metabolic action at all and is simply a lipophilic antioxidant.

This localized deficiency of vitamins A and C and skin antioxidants is insidious. Every time we go out into sunlight, even for only a few minutes, we destroy some cutaneous vitamin A. This happens day after day, year after year, and results in photoaging.

Ascorbic acid is water-soluble and is not stored in cells, so the loss of vitamin C has to be replaced daily by the blood supply. Deficiencies of vitamin C immediately permit more free radical damage and defective collagen is formed.

**TREATMENT OF PHOTOAGED SKIN**

Chronic deficiency of vitamin A lies at the heart of photoaging.\textsuperscript{21} We also know that vitamin C deficiency aggravates the effects of vitamin A depletion as far as collagen and melanin are concerned. We can also deduce that the antioxidants in skin are stressed by exposure to sunlight.\textsuperscript{22} There are other photosensitive molecules in the skin, but vitamins A, C, and E primarily determine the development or repair of actinic damage. Therefore, the treatment of photoaged skin has to include topical replenishment of these vitamins every day.

Because prevention is better than cure, we should start by wearing protective clothing and applying sun protection creams. Because we do not know the long-term effects of organic sunscreens, we should rather depend on safer inorganic reflecting molecules, such as titanium dioxide or zinc oxide. Since no sun protection cream is 100\% effective, we should also enrich the skin with antioxidants, such as vitamins C and E and carotenoids, every day to minimize the free radical damage. We should use topical vitamin A and the associated antioxidants very soon after we are first exposed to sunlight, and do that for the rest of our lives. That means that the skin will never suffer from transient deficiencies of vitamins A and C, etc. As a result, the skin will become more resistant to the development of skin cancer.\textsuperscript{23} This could also apply to melanoma. In view of the near epidemic of skin cancer today, everyone who ventures into sunlight should use topical vitamin A and the antioxidants every day.

**Which vitamin A?**

The medical literature about retinoid replenishment is virtually confined to retinoic acid, which has rather harsh topical effects. Fortunately, there are a number of other effective forms of vitamin A. They only differ in the atomic arrangements at the terminal carbon atom of the vitamin A molecule. All-trans-retinoic acid and its isomers are the actual active molecules that interact with DNA, but retinoic acid is an intracellular, not an extracellular, molecule and is rather irritant and normally makes up only a tiny fraction of the vitamin A in the skin. Topical applications of retinoic acid do raise the levels of retinoic acid in the skin, but at the same time retinyl palmitate levels are also increased. Retinoic acid irritates skin and causes a marked retinoid reaction, especially if there are inadequate retinoid receptors on the cell walls. Retinol in similar international unit (iu) doses also irritates the skin. Retinyl palmitate in the same iu doses, however, is very much less likely to cause a retinoid reaction.
Retinol is normally found as free retinol only in tiny doses in the skin. Research shows that enzymes in the skin convert topically applied retinol into retinyl palmitate and other esters of vitamin A.\textsuperscript{24} Virtually all the retinol applied to skin becomes retinyl palmitate and the stores of vitamin A in the skin cells are increased whereas the retinol levels remain the same (see Fig. 12.1).

Retinaldehyde is only one step away from retinoic acid, but, once again, enzymes convert virtually all topically applied retinaldehyde into retinyl palmitate and only a tiny fraction actually gets converted into retinoic acid.\textsuperscript{25} The esters of vitamin A (e.g. retinyl palmitate or acetate) are the liver-storage forms and are milder, active and more easily tolerated by the skin. Retinyl acetate is similar but more effective than topical retinoic acid and has fewer side-effects.\textsuperscript{26} Retinyl acetate also passes through the horny layer at a faster rate than retinoic acid.\textsuperscript{27}

We should use retinyl palmitate in the initial stages, because it is effective and will provide all the effects of retinoic acid provided it is used in adequate concentrations (see Figs 12.2 and 12.3).\textsuperscript{28}

For more intense treatment in patients whose skin has adapted to vitamin A, we can use retinyl acetate or retinol. Patients are generally reluctant to continue using retinoic acid, whereas with retinyl acetate or retinyl palmitate there is no problem in using high dosage for several years. Retinyl palmitate is destroyed every day, so that is the molecule that has to be replaced daily.\textsuperscript{29}

Vitamin A should commence at a concentration of about a 1000 IU g% used initially once a day, but better effects will be achieved using it twice a day. Gradually work up to concentrations of 10 000 IU g% and even, for more intense effects, to 25 000–50 000 IU g%.

**WHICH VITAMIN C?**

The ideal skin-care regime includes vitamin C (ascorbic acid), but it is unstable and rapidly decomposes. The stronger the concentration and the lower the pH, the more stable it is. Ascorbic acid solutions are virtually colorless and become brownish yellow as they degrade; they remain at
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stable than conventional ascorbic acid and can last up to 200 days before there is any appreciable loss of activity. Lower concentrations (compared to ascorbic acid) are required to get the same amount of ascorbic acid into the cell itself.

It is important to understand that a single molecule is never going to be adequate for rejuvenation of the skin. That is why vitamins C and A should always be combined with other antioxidants, such as vitamin E, carotenoids, etc. Vitamin C cannot substitute for the actions of vitamins A, E, etc. They have to work in concert. Clinical results of using VCP Mg in conjunction with retinyl acetate and antioxidants show significant rejuvenation of skin (see Figs 12.4 and 12.5). These effects are dramatically shown where continuous use of retinyl

their intended concentration for a maximum of three months. Therefore, a solution of ascorbic acid is best sold unmixed to your patients. The pH is naturally low and ascorbic acid acts like an alpha-hydroxy acid, which increases the penetration of vitamin C into the deeper layers of the skin. However, ascorbic acid is not easily taken through the horny layer and passes with difficulty into the cell wall because it is a water-soluble molecule.

Ascorbic acid can be combined with other molecules that are selectively taken into the cell. An example is magnesium-L-ascorbyl-2-phosphate (VCP Mg), which is also water-soluble but is taken up into cells much more effectively, and easily converted inside the cell to L-ascorbic acid, phosphate and magnesium.30 These solutions are also more

Fig. 12.2 50-year-old female with typical photoaging.

Fig. 12.3 Result after using retinyl palmitate and antioxidants in a cream for 5 months both morning and evening. This is similar to results seen after using topical retinoic acid.

Fig. 12.4 70-year-old female with typical photoaging that has progressed to solar keratoses and also rosacea. Treated with retinyl acetate and magnesium ascorbyl phosphate in a gel both morning and evening for 3 months.

Fig. 12.5 Significant changes are noted with high dose vitamin C in conjunction with vitamin A.
Acetate and VCPMg for 5 years has resulted in fewer wrinkles than before. This is contrary to nature but exactly what a scientific skin-care product is supposed to do. Neck skin responds well to retinyl acetate and VCPMg (see Figs 12.6 and 12.7).

Ascorbic acid is too aggressive for people with sensitive skin, whereas they can use VCPMg products without any difficulty—and they will get more vitamin C into their cells if the correct dose is used. Patients with pigmentation problems should avoid any product that peels the skin significantly, because they need the horny layer to be as thick as it can be to protect the melanocytes and avoid aggravating pigmentation. Ascorbic acid has an exfoliant property so patients with melasma or other pigmentation problems should use VCPMg products instead (see Figs 12.8 and 12.9).

Even better results come from using a fat-soluble complex of ascorbic acid, ascorbyl-4-isopalmitate (VCIP), which is extremely stable. Four molecules of palmitic acid are attached to the ascorbic acid that comprises less than a fifth of this large molecule. However, this fat-soluble form passes more easily through the horny layer than L-ascorbic acid, enters the cell wall with great ease and achieves up to 10 times more active L-ascorbic acid inside the cell itself than ascorbic acid. This leads to more effective control of melanin formation, enhanced collagen deposition and greater antioxidant protection. Of course, it should also be combined with a wide antioxidant brigade and effective UVA protection. VCIP should not to be confused with ascorbyl...
palmitate or ascorbyl dipalmitate, which are much less effective. Because of the fundamental importance of vitamins A, C, E and other antioxidants, if a skin-care range does not include them, it cannot claim to be true skin care. We need affordable products that will be used regularly by young children, teenagers and adults. If we really want to preserve young skin, then we have to start when the skin is young. The burgeoning problem of atmospheric ozone depletion means that virtually everyone who ventures out into sunlight will get more complete destruction of cutaneous vitamin A and therefore be at greater risk of photodamage. Everyone on earth, under ideal circumstances, will need to apply vitamin A and the antioxidants every day to maintain skin health and we should use better protective clothing to shield ourselves from the sun.

As a footnote, we should remember alpha-hydroxy acids. They do have a role in acne and also as penetration enhancers, but basically their function of smoothing skin is more physical than physiological. Lactic acid has great value in enhancing natural moisturizing factors and is much kinder to skin than glycolic acid. Their role is more as an adjuvant than an actual therapeutic agent.

In women, the advantages are known of using estrogen to recondition and thicken the skin during the menopause. Vitamins cannot play the role of hormones and growth factors, so we have to use hormones when they are required, and it is hoped that future skin-care creams will also include essential growth factors.

**INTENSIVE TREATMENTS FOR PHOTOAGED SKIN**

While it is axiomatic that it is easier, and better, to preserve young skin, most patients arrive at the plastic surgeon’s office with established photoaging. They really need more than a simple home-care system to get the best rejuvenation.

We should rehabilitate skin in a very gentle manner. The epidermis is an extremely complex, ultra-thin layer of cells, only about 0.2 mm thick, that has the sole responsibility of protecting our bodies from the environment. Stem cell keratinocytes must continue to survive, but differentiating keratinocytes are the only cells in the body that fulfill their destiny by dying. That is how the protective nature of the granular and horny layer develops. This dead lipophilic layer provides the waterproofing character of the epidermis and is the real guardian of our bodies, yet it is only 0.01-0.02 mm thick— but so many people regard it as disposable.

It is sad to reflect that today most plastic surgeons will consider procedures that torture the epidermis in order to obtain smooth skin. Deep peeling, dermabrasion and laser resurfacing spring to mind. Once the normal epidermis has been destroyed it never returns. The resultant skin might be smooth, but it is not young, healthy skin.

It is better to resort to physical science and physiology to rehabilitate photoaged cells and achieve young skin. We use physics to enhance penetration and physiological ingredients to activate cells back into normal patterns. Light repetitive peeling has been in vogue for a long time and should not be ignored. It has value in speeding up the rejuvenation of skin, as well as helping to rehabilitate severe actinic damage (see Figs 12.10 and 12.11). However, much better results have been achieved with iontophoresis, sonophoresis, physically enhanced skin penetration and percutaneous collagen induction (PCI).

**Light repetitive peeling**

Repetitive light peeling can be used on thick, rough, sun-damaged skins to gradually remove the excessive horny layer and allow greater penetration of active ingredients into the deeper parts of the epidermis. Peeling should not be carried out for pigmentation problems. The success of peeling for pigmentation problems is largely due to the hydroquinone that is used at the same time. However, hydroquinone is a cytotoxic agent and should be avoided. The horny layer is the best natural defense against light, so this should be made denser and preserved as much as possible to minimize stimulation of melanocytes.

Rosacea and acne are two excellent indications for peeling. The skin should always be prepared
with vitamin A to promote rapid healing (it is not an exfoliant). Light peeling has the advantage of sterilizing the acne eruption while not damaging the surrounding skin.

Light peeling can be achieved with trichloracetic acid gels or creams at a concentration of 1–5%. While this may sound ridiculously low, prolonged contact with the skin leads to a safe and controlled peel that will enhance penetration while still preserving the epidermis. Light peeling can even be done on the upper eyelid to rejuvenate eyelid skin in conjunction with vitamin A.

**Iontophoresis**

There has been a resurgence of interest in iontophoresis in the medical profession, which largely ignored the first demonstration of iontophoresis almost a hundred years ago. The principle of iontophoresis is that charged ions in a water-based product will be repelled into the skin when a similar charge is applied to the skin. The vehicle has to be water-based so that it can conduct electricity. Properly executed, iontophoresis can get up to 400% better penetration of a selected ion than a simple topical application. The process is easily carried out with a galvanic machine, or preferably a special pulsed-electrical-current unit, and can be done in the consulting room or esthetician’s salon. The skin has to be cleaned thoroughly and then covered with the specially formulated treatment gel containing vitamins A and C. Either electrodes or a mask is used to apply the electrical impulse to the area. Then the appropriate charged current is switched on. A mask of a highly conductive material will permit treatment to the whole face for the full treatment period of 20–30 minutes. A pulsed current is not merely safer, but also more effective.
The current does not have to exceed 2 mA to obtain a good result. One advantage for the cosmetic surgeon is that iontophoresis treatments on negative charge seem to improve lymphatic drainage and thus are very useful in reducing edema soon after facial surgery. When treating photoaging or acne scars, the treatments should continue for at least 20 minutes, twice a week for a minimum of 24 treatments which means that the full treatment period takes at least 3 months.

Modern iontophoresis has been used to minimize scars, reduce wrinkles eliminate pigment blemishes, and tighten facial skin (see Figs 12.12 and 12.13). However, this only happens when the gels used contain the right ingredients, at the right pH, with the right concentration, and the patient is treated with the correct current properties for the correct amount of time. Change only one of these important points and the treatment simply becomes a complex manipulation of skin and the patient’s purse.

Retinol, retinoic acid, ascorbic acid, VCP Mg and estriol are some of the agents suitable for iontophoresis. Retinol, retinoic acid, ascorbic acid, VCP Mg and estriol are some of the agents suitable for iontophoresis. There are probably many more that will no doubt become known, but at this stage we only have experience with the essential vitamins for the skin.

**Sonophoresis**

Low frequency sonophoresis at about 20 kHz has become one of the most exciting methods for transdermal drug delivery and can give up to 400% better penetration than simple topical application. Sonophoresis at 20 kHz induces cavitation in the lipid bilayers of the lamellar bodies in between the corneocytes, and rapid transdermal enhancement of penetration of up to 1000 times higher than therapeutic ultrasound (1–3 MHz). With a larger influx of vitamins C and A we can expect quite dramatic changes in skin when combined with iontophoresis (See Figs 12.14–12.16).

From a practical point of view, sonophoresis is easy to perform in the consulting room or esthetician’s salon. A sonophoresis probe with a special resonator is applied to the treatment gel that is on the skin, and gently maneuvered around the treatment area. Within a short time, cavitation of the skin occurs if the sound has the right intensity, and then ‘pores’ open up in membranes and especially in the lipid bilayers between the corneocytes of the stratum corneum of the epidermis. Whole molecules, even of fairly large peptides, can penetrate easily through the stratum corneum. This occurs within 3 minutes and the effects last for more than 3 hours, so products that are placed on the skin after sonophoresis treatment will also penetrate the stratum corneum more efficiently. A treatment for the whole face can be carried out in 20–25 minutes with 24 treatments required over 12 weeks.

**Fig. 12.12** Iontophoresis gives up to 400% better penetration and as a result collagen can be stimulated very effectively. Both vitamin A and C promote collagen formation.

**Fig. 12.13** The effects of increased collagen formation from iontophoresis of vitamin A and C are seen quite clearly in this case where the jowls have clearly been tightened in 3 months of treatment, to give a fresher and younger appearance. The result would be more dramatic if a facelift had been done.
We are merely at the very beginning of an exciting phase of percutaneous treatments that will help us to make even more dramatic advances in the treatment of aging skin. Haematoxylin and eosin staining, magnification ×100.

Physically enhanced skin penetration

Electricity and sound are not the only ways to improve penetration through skin. A much simpler and easier method is to punch a multitude of holes into the stratum corneum. Microneedles have been developed that prick through the horny layer only and cannot be felt by the patient, and as a result of this, the permeability may be increased up to 10,000 times in the prepared area. The patient is taught this procedure by the beautician. Of course, the important point to bear in mind is that the creams or gels applied after needling the skin have to be effective and tolerated by the skin. It is easy to learn how to use this tool and experience has shown that daily treatment, done for as little as 3–5 minutes, will lead to skin thickening and tightening. After about 6–9 months, the skin takes on a younger appearance. Patients should prepare their skin for surgery using this procedure in order to obtain optimal results.

Percutaneous collagen induction

Percutaneous collagen induction (PCI) by multiple fine needle puncturing (as close together as possible to a depth of about 1.5–2 mm) of the skin employs the natural inflammatory response to trauma and creates more collagen (see Figs. 12.17 and 12.18). The epidermis is not destroyed but is simply left with a small hole. Blood vessels in the dermis are punctured and this inevitably leads to bleeding and...
clot formation. Platelets release various growth factors and chemotactic agents that lead to greater numbers of fibroblasts, neutrophils and monocytes, which also augment the growth factors, for example, epidermal growth factor, TGF\(_\alpha\) and TGF\(_\beta\), fibroblast growth factor, and connective tissue-activating peptides among many others. These growth factors lead to thickening of the epidermis and increased production of collagen, elastin and glycosaminoglycans.

Laser resurfacing needs to be replaced by a more physiological procedure that does not destroy tissue and which can be used on areas other than the face. To a great extent percutaneous induction of collagen answers that need (see Figs 12.19 and 12.20). Laser resurfacing makes the epidermis thinner whereas PCI results in a thicker epidermis, tighter and natural, younger-looking skin. However, vitamins A and C are necessary cofactors in assisting adequate collagen deposition.

The treatment is ideally carried out using a local anesthetic and sedation or a general anesthetic for extensive areas. The patient has bruises for a few days. Note that the inflammatory response should not be dampened by using non-steroidal anti-inflammatory drugs (NSAIDs). However, by day 5 following treatment, makeup can be used. By day 7 it should not be obvious that any major treatment has been carried out. In contrast to laser resurfacing, improvement takes approximately 1 year to
become fully apparent because of the slow process of collagen conversion and maturation. The best results are seen when PCI is carried out in conjunction with iontophoresis and sonophoresis because this provides excellent skin rejuvenation.

With all these treatments it is possible to maintain youthful skin, and even though we cannot prevent the action of gravity we can make significant steps towards rejuvenation.

SUMMARY

We need to replace light-sensitive vitamins, such as vitamins A, C, and E; carotenoids; and other antioxidants, in our skin each day to keep our skin healthy and youthful. If the skin has been photodamaged, then we can still rehabilitate skin to a certain degree with simple treatments used at home or the consulting room/esthetician’s salon. For more serious damage, we need to resort to more intensive remedies like iontophoresis, sonophoresis and enhanced penetration by microneedling of the skin. Percutaneous collagen induction is another minimally invasive technique to improve skin quality.

REFERENCES


240  Esthetic surgery of the face


