

Clearing the Smoke: The Scientific Rationale for Tobacco Abstinence with Plastic Surgery

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The use of tobacco is a significant contributor to preventable morbidity and mortality in the United States. A significant proportion of cardiovascular diseases, various oral and pulmonary neoplasms, nonmalignant respiratory diseases, and peripheral vascular disorders can be attributed to the use of cigarettes. Surgical outcomes can also be adversely affected as a result of cigarette smoking with intraoperative and postoperative pulmonary, cardiovascular, and cerebrovascular complications as well as increased wound healing complications. These are found across the entire spectrum of surgical specialties. Tissue ischemia and wound-healing impairment secondary to the influence of tobacco is particularly problematic for the plastic surgeon, especially during elective facial aesthetic procedures, cosmetic and reconstructive breast operations, abdominoplasty, free-tissue transfer, and replantation procedures. By educating and providing guidelines to those patients who smoke and by refusing to operate on individuals who fail to abstain, tobacco-associated surgical morbidity in the plastic and reconstructive surgery patient can be eliminated. (*Plast. Reconstr. Surg.* 108: 1063, 2001.)

Cigarette smoking is the leading preventable cause of death and disability in the United States. Despite achievements and advances in clinical knowledge during the 20th century, approximately 48 million American adults continue to smoke cigarettes, and half of those who smoke will eventually die from a smoking-related illness.¹ At least one out of every five deaths in this country can be attributed to the use of tobacco, with the number approaching 430,000 per year.¹ The annual economic burden of tobacco-related diseases is quite staggering as well—nearly \$100 billion in health-care costs and lost productivity each year.²

The use of tobacco has been implicated in a variety of illnesses and is known to cause chronic obstructive pulmonary disease, coronary heart disease, cerebrovascular disease, pe-

ripheral vascular disease, and cancer. An association between impaired wound healing and tobacco use has also been clinically recognized for centuries and, more recently, supported by experimental data.

The reduced capacity for wound healing is of particular concern when it affects the outcomes of elective surgical procedures, as patients have an increased risk of complications and a decrease in the quality of postoperative results. Emotional, physical, and financial strains on patients and surgeons are inevitable in the wake of surgical morbidity and will certainly add to the stresses of developing and managing a clinical practice.

Plastic and reconstructive surgeons are particularly vulnerable to these problems, as they perform a high number of elective operations including facial and breast aesthetic operations, restoration of facial and breast deformities, body contouring, and reconstruction of the trunk and extremities. Success is largely judged on form rather than function; therefore, any factors that increase the incidence of scarring, asymmetry, technical failure, and need for reoperation must be minimized or removed. To optimize surgical results, plastic surgeons should (1) identify patients who use tobacco products, (2) provide patient counseling and smoking cessation information, and (3) insist on eliminating tobacco use from all preoperative candidates.

This article discusses the effects of tobacco smoke on wound healing, reviews the data on tobacco use and plastic surgery complications, and provides recommendations for managing patients who use tobacco products.

BIOCHEMISTRY AND PATHOPHYSIOLOGY

Tobacco smoke is a complex aerosol of particulate matter, volatile acids, and gases. The estimated number of gaseous and particulate compounds in cigarette smoke exceeds 4000, including many that are pharmacologically active, toxic, mutagenic, and carcinogenic.³ These chemicals include carbon monoxide, ammonia, arsenic, butane, hydrogen cyanide, toluene, dichlorodiphenyltrichloroethane, acetone, cadmium, methyl alcohol, and naphthalene.² Radioactive compounds such as polonium are also present in tobacco smoke.²

When cigarette smoke is inhaled into the pulmonary system, many of its toxic constituents, including nicotine, can directly poison protective cilia or pass through the ciliary barrier.⁴ Other toxins such as carbon monoxide and hydrogen cyanide are inhaled in the gaseous phase. The overall cellular effect of these inhaled or absorbed by-products is to produce an environment of relative tissue hypoxia and delayed wound healing mediated by vasoconstriction, abnormal cellular function, and thrombogenesis.

Vasoconstriction is mediated directly and indirectly by nicotine, a colorless, odorless, and poisonous alkaloid. A reduction in tissue perfusion results from elevated cellular levels of nicotine. The indirect pathways of vasoconstriction include the enhancement of thromboxane A_2 and the stimulation of catecholamine release from the adrenal medulla, sympathetic ganglia and nerve endings, and cardiac chromaffin tissue.^{3,5,6}

Numerous cellular functions, critical to uneventful wound healing, are altered by the presence of tobacco by-products. Injury and detachment of endothelial cells from within the lumen of small vessels has been associated with the toxic constituents of tobacco smoke.⁷ Cigarette smoke has been implicated in superoxide anion-mediated degradation of nitric oxide leading to impaired endothelial-dependent vasorelaxation.⁸ The loss of endothelial protection removes the natural anticoagulation and antispasmodic function normally mediated by endothelium-derived relaxing factor.⁹ Platelets may also attach to the damaged endothelia and release thrombotonin and thromboxane A_2 , further inducing vascular constriction and spasm.⁷ Other by-products, such as hydrogen cyanide, inhibit the enzymatic pathways vital for cellular oxidative me-

tabolism and oxygen transport, effectively diminishing the ability for cellular repair and wound healing. Combined with acrolein, another toxic gaseous component, hydrogen cyanide inhibits leukocyte function, further impairing the inflammatory phase of healing.¹⁰ The proliferation of macrophages and fibroblasts, cells integral to the phases of wound healing, is also diminished by the presence of nicotine.¹¹ Additionally, the presence of nicotine and catecholamines stimulates the production of chaperones, hormones that retard and decrease the rate of wound epithelialization.¹² Collagen deposition is also decreased in smokers.¹³

Nicotine is also associated with thrombogenesis by interfering with prostaglandin I_2 (prostaglandin) activity.¹⁴ Prostaglandin I_2 is a potent vasodilator and inhibitor of platelet aggregation. Platelet adhesiveness is augmented, raising the potential for thrombotic microvascular occlusion and subsequent tissue ischemia.

Carbon monoxide is another toxic by-product common in tobacco smoke. The oxygen-carrying capacity of blood is reduced by the competitive, inhibitory binding of carbon monoxide to hemoglobin. Carboxyhemoglobin levels rise, and tissue delivery of oxygen is reduced as the oxygen-hemoglobin saturation curve is shifted to the left. The decrease in oxygen available for tissue consumption leads to diminished wound healing.⁴

The resulting hypoxic state stimulates erythropoiesis, red blood cell aggregation, and fibrinogen production, leading to increased blood viscosity, which potentiates an environment already ripe for thrombogenesis.¹⁵ Decreased red blood cell deformability is also noted in smokers but through an unknown mechanism.³

In summary, nicotine, carbon monoxide, and many other toxic tobacco by-products clearly interfere with the dynamics of normal wound repair (Fig. 1). Direct tissue injury occurs within the microvasculature. The cellular populations needed to initiate the inflammatory response and propagate healing are inhibited and, when present, may not function properly because of impaired enzymatic activities. Hormones that actually retard epithelialization and wound repair may also be produced. Vasoconstriction (direct and indirect) reduces blood flow and oxygen delivery to the skin and extremities, and the competitive binding of toxins to hemoglobin further enhances this

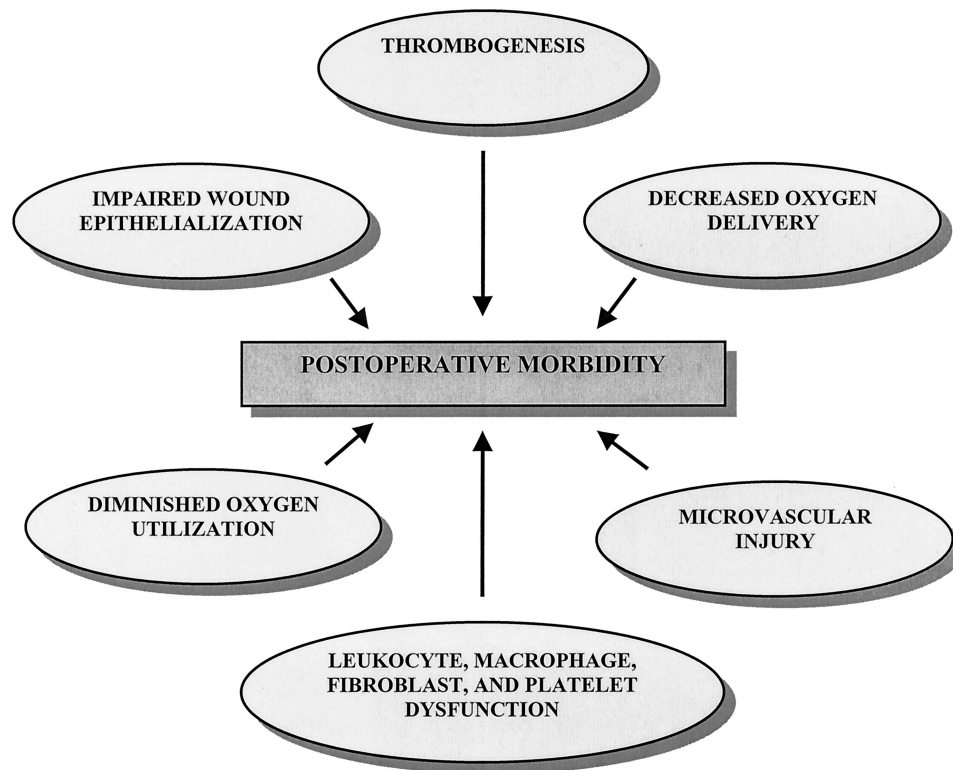


FIG. 1. Etiologies of tobacco-associated wound complications.

hypoxic state. Occlusive microvascular thrombosis occurs more frequently with increases in platelet adhesiveness, plasma fibrinogen concentration, blood viscosity, and altered red blood cell rheology. Delayed healing, wound dehiscence, postoperative infections, and poor scarring are, therefore, at an increased risk of occurring in the patient who smokes tobacco.

LITERATURE REVIEW

Despite centuries of anecdotal experience and decades of objective data, the detrimental effects of tobacco use on wound healing were not reported in the literature until 1977, when diminished healing of a hand wound was observed in an individual with arteriosclerosis.¹⁶ Since then, experimental and clinical studies have demonstrated impairment in random skin flap survival, reduced digital blood flow and replantation success, and increased wound complications associated with trauma, disease, and other surgical procedures.

The regulation of tissue blood flow is quite variable throughout the entire body, and it is dependent on central and local control as well as the degree of vascular resistance within the particular tissue. Cutaneous tissue beds have

the highest sympathetic innervation and are consequently the most sensitive to the influence of tobacco smoke. Logically, the areas of plastic surgery most susceptible to the adverse effects of tobacco are those incorporating large cutaneous flaps (rhytidectomy, breast reconstruction, and abdominoplasty). Digital replantation, muscle flap transposition, intraoral operations, and procedures requiring bone grafting are also affected.

Experimental evidence obtained from several animal models has supported the fact that tobacco smoke and nicotine exposure contribute to decreased blood flow, impaired wound healing, and diminished skin flap survival.^{12,17-21} Mosley et al.¹² observed that nicotine impaired wound healing in the rabbit ear. Nolan et al.¹⁹ observed a significantly greater percentage of mean flap necrosis in rats exposed to cigarette smoke when compared with control animals. Craig and Rees²⁰ demonstrated that flap survival in hamsters not exposed to smoke (100 percent survival) was superior to that in hamsters exposed to smoke preoperatively (80 percent survival) and in hamsters exposed both before and after surgery (40 percent survival). Impairment of endothelium-

dependent relaxation of systemic arteries has also been observed in rabbits subjected to passive smoking.²¹

Rhytidectomy skin flaps serve as an excellent human model for random pattern circulation and have generated several studies centering on the adverse effects of tobacco. Rees et al.²² retrospectively reported the observation that patients who smoke have an increased risk for skin slough after face lift surgery. They attributed 74 percent of their total number of skin sloughs to the influence of tobacco and calculated that a smoker is 12.5 times more likely to develop skin necrosis than a patient who does not smoke. A subsequent prospective study, performed by Riefkohl et al.,¹⁵ revealed a greater risk of skin slough in smoking patients who had undergone face lifts. Active smokers were shown to have a higher incidence of superficial necrosis (19.5 percent) compared with nonsmokers (5 percent). Webster et al.²³ provided data to the contrary, reporting no skin sloughs in their patient population. They did, however, take into account the available data documenting the ill-effects of nicotine and performed conservative undermining of facial flaps during rhytidectomy.

Large skin flaps are regularly undermined during an abdominoplasty and may be thicker and more resilient than thin facial flaps, but they are still subject to the detrimental effects of tobacco. Anecdotal evidence has suggested an increase risk of abdominoplasty flap necrosis in smokers, but this has not been definitively proven.²⁴

There is more clinical evidence demonstrating adverse outcomes in breast reconstruction regarding donor abdominal and recipient breast flap necrosis. Kroll,²⁵ comparing a series of transverse rectus abdominis muscle (TRAM) flap breast reconstructions, noted a 27.5 percent incidence of abdominoplasty flap necrosis in smokers compared with 5.9 percent in nonsmokers. He also found that umbilical necrosis was more common in smokers (27.5 percent) than in nonsmokers (11.8 percent).²⁵ Bostwick,²⁶ studying TRAM and mastectomy flaps, observed an increase in wound complications affecting those patients exposed to first-hand and second-hand tobacco smoke. Bailey et al.²⁷ evaluated the rate of implant loss in patients receiving immediate breast reconstruction. They found a 33 percent incidence of implant loss in smokers because of wound infection and skin loss, compared with 14 per-

cent in nonsmokers. Hartrampf and Bennett,²⁸ following a group of TRAM breast reconstructions, found that every patient with skin loss in areas other than the flap itself had been exposed to tobacco smoke. Hartrampf and Bennett went on to suggest that, in their experience, smokers experienced at least a two-fold increase in complications of wound healing, skin loss, and flap loss compared with nonsmokers. Chang et al.,²⁹ reviewing 10 years of experience with over 900 free TRAM flaps, concluded that smokers were at a significantly higher risk for mastectomy skin flap necrosis, abdominal flap necrosis, and hernia compared with nonsmokers.

Hand and foot microsurgeons have reported poor microsurgical outcomes when smokers undergo digital replantation surgery. The data indicate that this observation is secondary to vascular spasm and not anastomotic failure. Numerous studies have documented the reduction in digital blood flow in normal fingers as a consequence of nicotine exposure.^{16,30-32} Smoking a single cigarette can result in a reduction in blood flow to the thumb by 24 percent.³² Other reports indicate a reduction of blood flow in the hand (lasting up to an hour) of 42 percent after one cigarette is smoked.³³ Van Adrichem et al.³⁰ performed a controlled prospective clinical study in which they concluded that the microcirculation was significantly reduced in replanted digits of smokers compared with nonsmokers. Chang et al.¹⁴ note that approximately 80 to 90 percent of cigarette smokers will lose their replanted digits if tobacco use occurs within 2 months before surgery. The reason why digital replantations are more adversely affected than free-tissue transfer is unclear, although it is thought to be related to the predominant vasomotor control of digital vascular beds compared with those of skeletal muscle.^{31,34}

The success of free-tissue transfer and microvascular patency rates has also been studied with regard to the use of tobacco products. Endothelial cell damage is thought by some to delay the healing of suture holes at anastomotic sites, allowing for increased platelet aggregation, microvascular thrombosis, and eventual flap failure.⁹ Yaffe et al.³⁵ and Lee,³⁶ in two separate experimental animal models, concluded that although nicotine exposure did result in a reduction of total blood flow, microvascular anastomotic patency rates were unaffected. Reus et al.³⁷ studied the incidence of

free-tissue transfer survival and complications in nonsmokers, active smokers, and patients who discontinued tobacco use before surgical intervention. They found that complications occurred significantly more in active smokers but were limited to the interface between the flap and its bed or an overlying skin graft. This population of patients required more secondary surgical procedures at the recipient site to accomplish ultimate wound closure. There was no statistical difference in anastomotic patency rates or flap survival between the various patient groups. Other studies have documented similar patency success and flap survival rates in smokers.⁷ Despite the devastating effects seen in digital replantation, it seems that free-tissue transfers can be successfully performed in cigarette smokers, though a significant increase in wound complications at the donor and/or recipient site can be expected.

Other types of procedures, common to the practice of plastic surgery, have been shown to be adversely affected by the active smoking status of the patient. Lovich and Arnold³⁸ performed a retrospective review of 300 pedicled muscle flap procedures and determined that active smokers had a significantly higher complication rate than nonsmokers and smokers who had quit. Their observed complications occurred in the immediate postoperative period and included partial muscle necrosis and partial skin graft loss. Jones and Triplett,³⁹ evaluating patients after bone grafting and dental implant placement, discovered that 80 percent of the individuals who had intraoral wound dehiscence and/or infection were active smokers, whereas nonsmokers had only a 10 percent incidence of wound complications. Hollinger et al.,⁴⁰ using an animal wound model, concluded that nicotine administration hindered bone regeneration for up to 4 weeks. Nicotine is also associated with delayed revascularization of cancellous bones grafts, a smaller percentage of total revascularization within the graft, and a higher percentage of bone graft necrosis.⁴¹

PHARMACOLOGIC MANAGEMENT STRATEGIES

Cigarettes are among the most addicting products known, and the vast majority of individuals who stop smoking will relapse within 1 week.⁴² According to the Agency for Health Care Policy and Research (AHCPR), 70 percent of current smokers have expressed a desire to discontinue the habit, but only half have

ever been urged to do so by a physician.⁴³ When factoring in physician advice to quit tobacco use, a 30 percent improvement in smoking cessation rates has been observed in some randomized clinical trials.⁴³ Therefore, the AHCPR Clinical Practice Guideline on Smoking Cessation⁴³ states, "All physicians should strongly advise every patient who smokes to quit." Aside from the powerful influence of communication and patient education, the physician may further aid smokers by recommending nicotine replacement therapy and/or nonnicotine pharmacologic therapy.

Nicotine Replacement Therapy

Nicotine has a chemical structure that enables it to easily produce addictive effects similar to other substances such as cocaine, heroin, and alcohol.⁴⁴ A major barrier to long-term abstinence from tobacco is the nicotine withdrawal syndrome, which includes nicotine craving, irritability, anxiety, difficulty concentrating, restlessness, and increased appetite.⁴⁵ These withdrawal symptoms intensify after only a few hours of tobacco abstinence. The most well-studied and well-documented pharmacologic approach to assist smokers manage nicotine dependence and withdrawal is the therapeutic administration of nicotine replacement medicines. Nicotine medicines enable the tobacco-dependent individual to abstain from the substance by partially replacing the nicotine formerly obtained from tobacco, thus reducing withdrawal symptoms while sustaining desirable mood and attention states.

Nicotine replacement therapy is currently available in several different forms: chewing gum, transdermal patch, nasal spray, and vapor inhaler. Both the nicotine patch and gum are widely used, and both have been shown to be efficacious as an aid to smoking cessation. Nicotine delivered by transdermal and oral routes causes fewer cardiovascular and endocrine effects, but its influence on surgical procedures and wound healing has yet to be fully investigated.

The transdermal patch delivers nicotine throughout the day, although there is a wide variation in the peak plasma levels obtained and the speed of absorption between currently available patch systems. The AHCPR has suggested treatment schedules for individuals using this system of nicotine delivery (Table I). On awakening on the designated quit day, the patch should be applied to a relatively hairless

TABLE I
Agency for Health Care Policy and Research Nicotine Transdermal Patch Treatment Suggestions⁴³

Brand	Duration	Dosage (mg/h)
Nicoderm and Habitrol	4 weeks, then 2 weeks, then 2 weeks	21/24 patch every day, then 14/24 patch every day, then 7/24 patch every day
Prostep	4 weeks, then 4 weeks	22/24 patch every day, then 11/24 patch every day
Nicotrol	4 weeks, then 2 weeks, then 2 weeks	15/16 patch every day, then 10/16 patch every day, then 5/16 patch every day

area of the body between the neck and waist. Rotating patch sites every day for the duration of the treatment period reduces the major side effect, a local skin reaction. Clinicians should be familiar with the package insert regarding formal treatment information and contraindications, and an attempt to individualize treatment strategy should be made on the basis of patient characteristics (previous patch experience, number of cigarettes smoked daily, and degree of addiction).

Compliance with nicotine patch therapy is good, as it is dependent only on whether or not the patient applies the device each day. Nicotine gum therapy is usually of longer duration (up to 3 months), requires the patient to constantly chew and manipulate the gum, and is associated with some dietary restrictions (Table II). Patient preference, previous failure with the nicotine patch, and specific contraindications to the nicotine patch should lead the clinician to consider recommending use of the gum. Again, familiarity with the product package insert and knowledge of all contraindications is imperative.

There has been little research to support the use of one form of nicotine replacement med-

ication over another, and it is acceptable for patient preference and tolerability to the side effects of the therapy to be the main consideration when recommending such intervention. Currently available nicotine replacement products generally do not deliver the drug in doses seen during active cigarette smoking, which can lead to unsuccessful cessation attempts. This has stimulated the initiation of combination therapy (e.g., patch/gum, patch/spray), increased the research and development of new nicotine delivery products (oral lozenges, tobacco-flavored gum, tobacco-free cigarettes), and brought about the quest for nonnicotine replacement therapies.⁴⁴

Nonnicotine Therapy

Nonnicotine medications may be overlapped for up to several weeks in preparation for a quit date, as opposed to nicotine replacement, which requires complete abstinence after cessation of tobacco use. The use of one therapy, however, does not preclude use of another, as nonnicotine agents have been studied in conjunction with nicotine replacement medicines during smoking termination trials. Bupropion (Wellbutrin; GlaxoSmithKline, Research Triangle Park, N.C.; Zyban, GlaxoSmithKline) is the first Food and Drug Administration–approved nonnicotine medicine for use in smoking cessation attempts (Table III). Large, multicenter trials have shown a statistically significant increase in the quit rates of individuals

TABLE II
Agency for Health Care Policy and Research Nicotine Chewing Gum Treatment Suggestions⁴³

Dose (mg/piece)	Guidelines
2	Good for those who smoke ≤ 1 pack per day Good for initial prescription Prescribed for the first months of a "quit" Fixed dosing schedule (1 piece every 1–2 hrs) No more than 30 pieces per day Avoid acidic beverages (coffee, soft drinks, juices) before and during gum use
4	Good for those who smoke ≥ 1 pack per day Good for those who are highly dependent on nicotine, have severe withdrawal, or have failed to quit using 2-mg dosing Fixed dosing schedule (1 piece every 1–2 hrs) No more than 20 pieces per day Avoid acidic beverages (coffee, soft drinks, juices) before and during gum use

TABLE III
Guidelines for Bupropion Therapy⁴⁸

Week 1 (patient still using tobacco)	150 mg PO every day \times 3–5 days, then 150 mg PO twice daily*
Weeks 2–3 (patient prepares for the quit date)	150 mg PO twice daily
Quit date	150 mg PO twice daily
Weeks 3–12†	150 mg PO twice daily

* Some smokers may have success at 150 mg every day.

† Treatment is efficacious for 7–12 weeks, but may be extended longer depending on the needs of the patient.

treated with bupropion and in combination with the nicotine patch.^{46,47} Studies and anecdotal observations have revealed that bupropion decreases the desire for cigarettes and relieves common nicotine withdrawal symptoms, including irritability, frustration, anger, anxiety, concentration difficulties, restlessness, and depression.⁴⁸ The package insert lists a profile of the possible side effects (minimal and well-tolerated) and details the necessary prescribing precautions.

In today's health-care environment, economics plays a significant role in determining standard of care with regard to diagnostic modalities and treatment algorithms. Studies have been performed to analyze the cost-effectiveness of various smoking cessation strategies, and it has been determined that bupropion therapy is a more cost-beneficial treatment than the nicotine patch.⁴⁹

There are a variety of other medications not currently approved by the Food and Drug Administration for use in smoking cessation (Table IV). There is no statistical evidence to support using these medicines as first-line therapy, because their efficacy is lacking, side-effect profiles are less favorable, and patient monitoring is required during treatment.

Limiting the detrimental effects of tobacco smoke using pharmacologic therapy has also been attempted. This alternative treatment strategy could provide an additional management tool for those patients suspected of non-compliance (despite reported history) or for those urgent situations in which the recommended period of abstinence cannot be met (e.g., digital replantation, trauma, and oncologic reconstruction).

Calcium channel blockers, nitrates, antiadrenergics, and other miscellaneous drugs have been studied in different animal models providing variable experimental data. Davies et al.,⁵⁰ focusing on phenoxybenzamine, nifedipine, and nitroglycerin, evaluated pharmacologic interactions with the vasoactive proper-

ties of nicotine. In a rat model, the vasodilating drugs were compared for their ability to improve random-pattern skin flap survival after exposure to smoke. This study concluded that nifedipine and nitroglycerin significantly improved postoperative results and may ultimately be clinically useful in salvaging skin flaps of a smoking patient. Pentoxifylline, a drug that acts to increase the deformability of red blood cells, decrease platelet aggregation, and reduce blood viscosity, has shown promise in improving survival of skin flaps.⁵¹ Although its efficacy has yet to be demonstrated in the patient who smokes, it would seem a logical pharmacologic choice. More controlled studies, especially comparing various classes of drugs, are certainly needed before the safe and widespread use of any medicine to counter the clinical effects of tobacco and its by-products.

Alternative Strategies

Drug therapy is the most attractive means of smoking cessation for many patients and physicians; however, it is not a panacea. Behavioral therapy has been studied extensively, and cessation rates have averaged 20 percent for those willing to participate.⁵² Behavioral modification often incorporates aversion techniques (covert sensitization and rapid smoking strategies) to make the action of smoking less appealing or, in fact, repulsive. Counseling is an important adjunct to this type of cessation attempt. The main disadvantages are that intensive programs are difficult to find, costly, and attract only that small percentage of smokers interested in attending classes or individual sessions.

Behavioral therapy can be made more focused, brief, and appealing by individualizing the treatment. Self-help programs exist and can be tailored to the needs of the individual smoker, thereby increasing compliance. Quit rates have been shown to nearly double when such approaches are incorporated.^{53,54}

Hypnosis and psychotherapy have become

TABLE IV
Medications Not Approved by the Food and Drug Administration for Nicotine Dependence⁴⁸

Medication (class)	Proposed Mechanism in Smoking Cessation
Nortriptyline, doxepin, fluoxetine (antidepressant)	Manages underlying depressive disorder in smokers
Diazepam, buspirone, alprazolam (anxiolytic)	Alters anxiety associated with withdrawal symptoms
Mecamylamine (nicotine antagonist)	Reverses effects of nicotine without perpetuating withdrawal
Naltrexone (opioid antagonist)	Reinforces effects of nicotine by endogenous opioids
Clonidine (alpha agonist)	Reduces craving and certain withdrawal symptoms

some of the most advertised methods of smoking cessation. These techniques attempt to alter subconscious beliefs and attitudes that may be impeding an individual's efforts to quit smoking. Their eventual success depends on the smoker's motivation to quit the habit. Although controversial, results with hypnosis alone seem to be modest at best. However, the probability of success can be increased when multiple sessions are performed and the technique is used with other quitting methods.

Electrical stimulation has been used to treat a variety of drug dependencies, but its efficacy in the treatment of smoking has not been supported.⁵⁵ Acupuncture is frequently used for smoking cessation, although its use has been shown to be no more effective than placebo.^{56,57} Nutritional supplementation and herbal medicinal therapies are also increasing in popularity, although they lack clinical and experimental support.

RECOMMENDATIONS

The most effective strategy for reducing or eliminating the detrimental sequelae of tobacco is total abstinence, although realistically, patients are more compliant if avoidance guidelines are provided. With regard to timing of surgical intervention, there is no uniform agreement on when to discontinue tobacco use, and no well-performed studies exist to support a particular interval of time. Recommendations found in the literature vary from 1 day to 4 weeks preoperatively and from 5 days to 4 weeks postoperatively. Gradual normalization of blood and plasma viscosities, hematocrit, blood cell filterability, plasma fibrinogen levels, and total white blood cell count has been observed in smokers who have abstained for at least 8 weeks, with no such change occurring in those who failed to comply.⁵⁸ Chang et al.,²⁹ in their review of a large number of free TRAM results, did note that smoking-related complications were significantly reduced when the patient stopped smoking at least 4 weeks before surgery.

Our current recommendations (Fig. 2) to prospective operative candidates are to completely abstain from all tobacco products; however, procedures will be performed on patients who avoid tobacco products and nicotine replacement therapies for an interval of 4 weeks before and 4 weeks after elective cosmetic or reconstructive surgery. During preoperative consultation, these guidelines are clearly stated

and are not subject to debate. Noncompliance will assuredly be met with a cancellation in the procedure. This unyielding approach to the applicable patient population has resulted in a positive anecdotal experience, with reductions in both skin flap necrosis and wound-healing problems. Moreover, it has often been the catalyst for permanent lifestyle changes for the patient, beginning with the complete discontinuance of tobacco products.

Despite appropriate physician counseling and support, patients may still have difficulty complying with abstinence. Self-help programs, community-based support groups, and professional counseling are available and can be beneficial to the individual who desires to quit using tobacco products. The plastic surgeon who possesses a comprehensive knowledge of pharmacologic smoking cessation strategies can either initiate nicotine replacement and/or nonnicotine therapy or at least offer accurate information to the interested patient who can subsequently seek treatment from his or her primary care provider.

CONCLUSIONS

Although generally not as ominous as the cardiovascular, pulmonary, and neoplastic sequelae of tobacco use, the skin and soft-tissue manifestations do exist and are associated with significant morbidity after elective surgical procedures. It is imperative that plastic and reconstructive surgeons completely understand these consequences and are able to effectively communicate them when counseling surgical candidates.

For plastic and reconstructive surgeons, emphasis is always placed on achieving predictable and reproducible results, especially when operating on aesthetically important regions such as the face, breast, and extremities. Factors that provide an element of uncertainty and unpredictability must be identified and eliminated. Knowledge of tobacco use and its associated postoperative complications will enable the surgeon to encourage abstinence among operative candidates, thereby omitting the negative influence of the substance and its by-products.

Considering that the plastic surgery patient population is frequently more concerned with their appearance, a comprehensive explanation of the external manifestations and consequences of smoking may provide a powerful motivation to discontinue the use of tobacco.

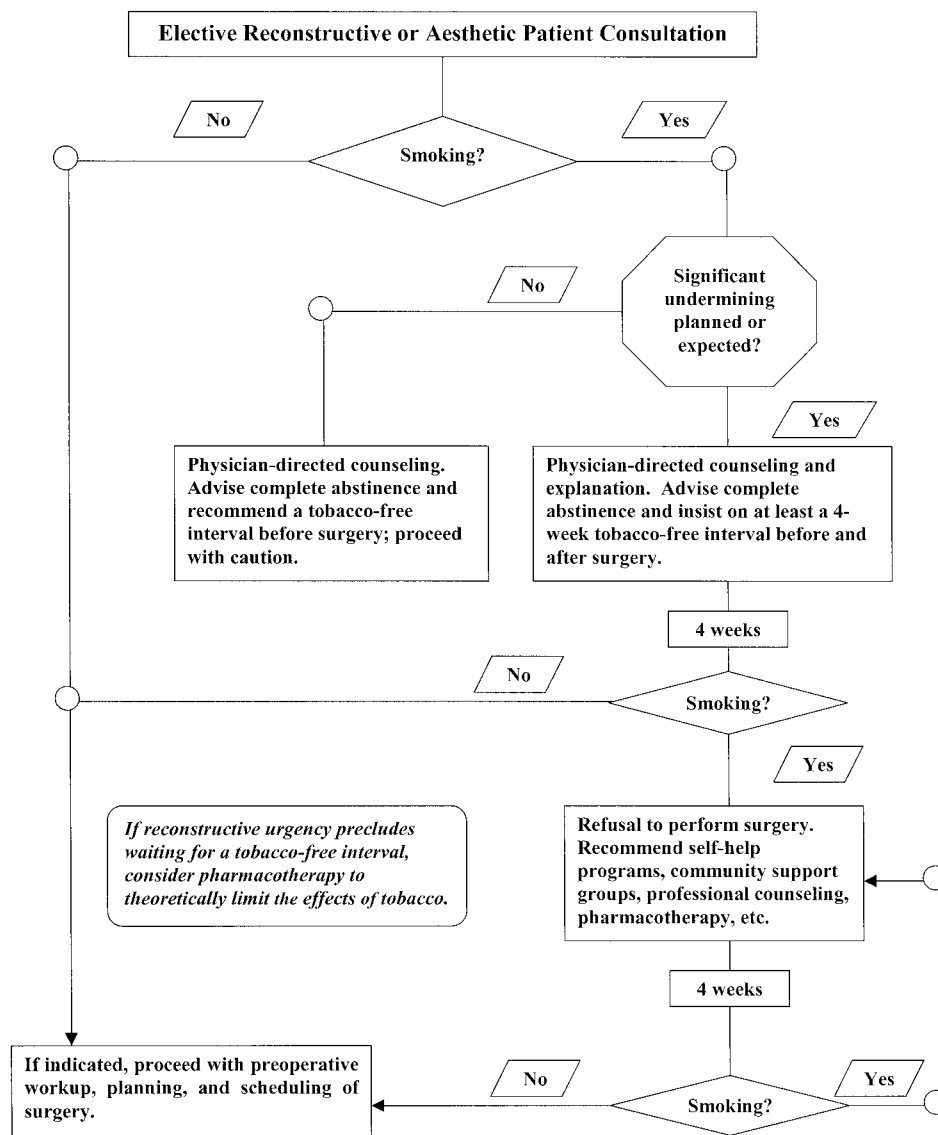


FIG. 2. Treatment algorithm for patients who use tobacco.

Refusing to perform aesthetic or elective reconstructive surgery on individuals who actively use tobacco products may be another incentive and significant motivating factor to abstain. Supplying smoking cessation information and assisting the patient through professional counseling, support groups, pharmacology, and other strategies will provide the necessary tools for an individual to achieve long-term success.

Ultimately, the surgeon has the final word in determining the scheduling of cases and whether or not to proceed with a questionable patient. We have all successfully operated on and observed uncomplicated wound healing in a smoker; therefore, the temptation to ignore a “little smoking” is great. Last-minute cancella-

tions are both frustrating and costly, and every effort is given to avoid these circumstances. Some may choose to perform a “lesser procedure” to reduce the overall risk in these higher risk patients. Unfortunately, this subjects the patient not only to a lesser procedure but, more importantly, a lesser result.⁵⁹ Plainly stated, operating on a known, active smoker jeopardizes the physician-patient relationship and the outcome of any elective procedure.

By taking a proactive stance and intervening in a tobacco cycle characterized by prevalence, neglect, and morbidity, we have the power to improve the quality of surgical results, reduce the incidence of postoperative complications, and watch our patients enjoy longer, healthier lives.

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REFERENCES

- Centers for Disease Control and Prevention. Tobacco use: United States, 1900-1999. *M.M.W.R. Morbid. Mortal. Wkly. Rep.* 48: 986, 1999.
- Ruppert, R. A. The last smoke: Your patients can quit smoking for life. *Am. J. Nurs.* 99: 26, 1999.
- Krupski, W. C. The peripheral consequences of smoking. *Ann. Vasc. Surg.* 5: 291, 1991.
- Silverstein, P. Smoking and wound healing. *Am. J. Med.* 93: 22s, 1992.
- Goodman, L. S., and Gillman, A. *Pharmacological Basis of Therapeutics*, 5th Ed. New York: Macmillan, 1975. P. 567.
- Lelcuk, S., Threlfall, L., Valeri, C. R., et al. Nicotine stimulates pulmonary parenchymal thromboxane synthesis. *Surgery* 100: 836, 1986.
- Davis, J. W., Shelton, L., Eigenberg, D. A., and Hignite, C. E. Lack of effect of aspirin on cigarette smoke-induced increase in circulating endothelial cells. *Hemostasis* 17: 66, 1987.
- Dattilo, J. B., and Makhoul, R. G. The role of nitric oxide in vascular biology and pathobiology. *Ann. Vasc. Surg.* 11: 307, 1997.
- Gu, Y. D., Zhang, G. M., Zhang, L. Y., Li, F. G., and Jiang, J. F. Clinical and experimental studies of cigarette smoking in microvascular tissue transfers. *Microsurgery* 14: 391, 1993.
- Eichel, B., and Shahrik, H. A. Tobacco smoke toxicity: Loss of human oral leukocyte function and fluid-cell metabolism. *Science* 166: 1424, 1969.
- Sherwin, M. A., and Gastwirth, C. M. Detrimental effects of cigarette smoking on lower extremity wound healing. *J. Foot Surg.* 29: 84, 1990.
- Mosley, L. H., Finseth, F., and Goody, M. Nicotine and its effect on wound healing. *Plast. Reconstr. Surg.* 61: 570, 1978.
- Hunt, T. K., and Pai, M. P. Effect of varying ambient oxygen tensions on wound metabolism and collagen synthesis. *Surg. Gynecol. Obstet.* 135: 561, 1972.
- Chang, L. D., Buncke, G., Slezak, S., and Buncke, H. J. Cigarette smoking, plastic surgery, and microsurgery. *J. Reconstr. Microsurg.* 12: 467, 1996.
- Riefkohl, R., Wolfe, J. A., Cox, E. B., and McCarty, K. S., Jr. Association between cutaneous occlusive vascular disease, cigarette smoking, and skin slough after rhynchotomy. *Plast. Reconstr. Surg.* 77: 592, 1986.
- Mosley, L. H., and Finseth, F. Cigarette smoking: Impairment of digital blood flow and wound healing in the hand. *Hand* 9: 97, 1977.
- Lawrence, W. T., Murphy, R. C., Robson, M. C., and Heggors, J. P. The detrimental effect of cigarette smoking on flap survival: An experimental study in the rat. *Br. J. Plast. Surg.* 37: 216, 1984.
- Kaufman, T., Eichenlaub, E. H., Levin, M., Hurwitz, D. J., and Klain, M. Tobacco smoking: Impairment of experimental flap survival. *Ann. Plast. Surg.* 13: 468, 1984.
- Nolan, J., Jenkins, R. A., Kurihara, K., and Schultz, R. C. The acute effects of cigarette smoke exposure on experimental skin flaps. *Plast. Reconstr. Surg.* 75: 544, 1985.
- Craig, S., and Rees, T. D. The effects of smoking on experimental skin flaps in hamsters. *Plast. Reconstr. Surg.* 75: 842, 1985.
- Torok, J., Gvozdzakova, A., Kucharska, J., et al. Passive smoking impairs endothelium-dependent relaxation of isolated rabbit arteries. *Physiol. Res.* 49: 135, 2000.
- Rees, T. D., Liverett, D. M., and Guy, C. L. The effect of cigarette smoking on skin flap survival in the face lift patient. *Plast. Reconstr. Surg.* 73: 911, 1984.
- Webster, R. C., Kazda, G., Hamdan, U. S., Fuleihan, N. S., and Smith, R. C. Cigarette smoking and face lift: Conservative versus wide undermining. *Plast. Reconstr. Surg.* 77: 596, 1986.
- Grazer, F. M., and Goldwyn, R. M. Abdominoplasty assessed by survey, with emphasis on complications. *Plast. Reconstr. Surg.* 59: 513, 1977.
- Kroll, S. S. Necrosis of abdominoplasty and other secondary flaps after TRAM flap breast reconstruction. *Plast. Reconstr. Surg.* 94: 637, 1994.
- Bostwick, J. Smoking and atherosclerosis (Editor's Perspective). *Perspect. Plast. Surg.* 3: 167, 1989.
- Bailey, M. H., Smith, J. W., Casas, L., et al. Immediate breast reconstruction: Reducing the risks. *Plast. Reconstr. Surg.* 83: 845, 1989.
- Hartrampf, C. R., Jr., and Bennett, G. K. Autogenous tissue reconstruction in the mastectomy patient. *Ann. Surg.* 205: 508, 1987.
- Chang, D. W., Reece, G. P., Wang, B., et al. Effect of smoking on complications in patients undergoing free TRAM flap breast reconstruction. *Plast. Reconstr. Surg.* 105: 2374, 2000.
- van Adrichem, L. N., Hovius, S. E. R., van Strik, R., and van der Meulen, J. C. The acute effect of cigarette smoking on the microcirculation of a replanted digit. *J. Hand Surg. (Am.)* 17: 230, 1992.
- Harris, G. D., Finseth, F., and Buncke, H. J. The hazard of cigarette smoking following digital replantation. *Microsurgery* 1: 403, 1980.
- van Adrichem, L. N., Hovius, S. E. R., van Strik, R., and van der Meulen, J. C. Acute effects of cigarette smoking on microcirculation of the thumb. *Br. J. Plast. Surg.* 45: 9, 1992.
- Sarin, C. L., Austin, J. C., and Nickel, W. O. Effects of smoking on digital blood-flow velocity. *J.A.M.A.* 229: 1327, 1974.
- Smith, J. J., and Kampine, J. P. *Circulatory Physiology: The Essentials*. Baltimore: Williams & Wilkins, 1990. P. 140.
- Yaffe, B., Cushin, B. J., and Strauch, B. Effect of cigarette smoking on experimental microvascular anastomoses. *Microsurgery* 5: 70, 1984.
- Lee, M. S. Effect of nicotine on blood flow and patency of experimental microvascular anastomosis. *Plast. Reconstr. Surg.* 80: 763, 1987.
- Reus, W. F., III, Colen, L. B., and Straker, D. J. Tobacco smoking and complications in elective microsurgery. *Plast. Reconstr. Surg.* 89: 490, 1992.
- Lovich, S. F., and Arnold, P. G. The effect of smoking on muscle transposition. *Plast. Reconstr. Surg.* 93: 825, 1994.
- Jones, J. K., and Triplett, R. G. The relationship of cigarette smoking to impaired intraoral wound healing: A review of evidence and implications for patient care. *J. Oral Maxillofac. Surg.* 50: 237, 1992.

40. Hollinger, J. O., Schmitt, J. M., Hwang, K., Soleymani, P., and Buck, D. Impact of nicotine on bone healing. *J. Biomed. Mater. Res.* 45: 294, 1999.
41. Daftari, T. K., Whitesides, T. E., Jr., Heller, J. G., Goodrich, A. C., McCarey, B. E., and Hutton, W. C. Nicotine on the revascularization of bone graft: An experimental study in rabbits. *Spine* 19: 904, 1994.
42. Henningfield, J. E. Nicotine medications for smoking cessation. *N. Engl. J. Med.* 333: 1196, 1995.
43. United States Department of Health and Human Services. *Clinical Practice Guideline No. 18: Smoking Cessation*. Washington, D.C.: United States Government Printing Office, 1996. AHCPR Publication No. 96-0692.
44. Fant, R. V., Owen, L. L., and Henningfield, J. E. Nicotine replacement therapy. *Clin. Primary Care* 26: 633, 1999.
45. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th Ed. Washington, D.C.: American Psychiatric Association, 1994.
46. Hurt, R. D., Sachs, D. P. L., Glover, E. D., et al. A comparison of sustained-release bupropion and placebo. *N. Engl. J. Med.* 337: 1195, 1997.
47. Jorenby, D. E., Leishchow, S. J., Nides, M. A., et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *N. Engl. J. Med.* 340: 685, 1999.
48. Ferry, L. H. Non-nicotine pharmacotherapy for smoking cessation. *Clin. Primary Care* 26: 653, 1999.
49. Nielsen, K., and Fiore, M. C. Cost-benefit analysis of sustained-release bupropion, nicotine patch, or both for smoking cessation. *Prev. Med.* 30: 209, 2000.
50. Davies, B. W., Lewis, R. D., and Pennington, G. The impact of vasodilators on random-pattern skin flap survival in the rat following mainstream smoke exposure. *Ann. Plast. Surg.* 40: 630, 1998.
51. Takayanagi, S., and Ogawa, Y. Effects of pentoxifylline on flap survival. *Plast. Reconstr. Surg.* 65: 763, 1980.
52. Prochazka, A. V. New developments in smoking cessation. *Chest* 117: 169S, 2000.
53. Stretcher, V. J., Kreuter, M., Den Boer, D. J., Kobrin, S., Hospers, H. J., and Skinner, C. S. The effects of computer tailored smoking cessation messages in family practice settings. *J. Fam. Pract.* 39: 262, 1994.
54. Shiffman, S., Paty, J. A., Rohay, J. M., Di Marino, M. E., and Gitchell, J. The efficacy of computer-tailored smoking cessation material as a supplement to nicotine polacrilex gum therapy. *Arch. Intern. Med.* 160: 1675, 2000.
55. Georgiou, A. J., Spencer, C. P., Davies, G. K., and Stamp, J. Electrical stimulation therapy in the treatment of cigarette smoking. *J. Subst. Abuse* 10: 265, 1998.
56. White, A. R., Resch, K. L., and Ernst, E. Randomized trial of acupuncture for nicotine withdrawal symptoms. *Arch. Intern. Med.* 158: 2251, 1998.
57. White, A. R., Resch, K. L., and Ernst, E. A meta-analysis of acupuncture techniques for smoking cessation. *Tob. Control* 8: 393, 1999.
58. Ernst, E., and Matrai, A. Abstinence from chronic cigarette smoking normalizes blood rheology. *Atherosclerosis* 64: 75, 1987.
59. Rohrich, R. J. Cosmetic surgery and patients who smoke: Should we operate? *Plast. Reconstr. Surg.* 106: 137, 2000.