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The Impact of nicotine use on wound healing and postoperative complications in plastic and reconstructive surgery, with a particular focus on the chronology and duration of preoperative cessation

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Absract:

Background: In elective plastic surgery, achieving safe outcomes and meeting patient expectations are paramount. The absence of complications remains the primary determinant of satisfaction, making the management of modifiable risk factors essential. Nicotine dependence is a well-established factor impairing tissue healing.

Aim: To determine the impact of nicotine use on plastic surgery clinical outcomes, analyze the pathophysiological mechanisms of impaired wound healing, and assess preoperative cessation strategies and objective laboratory verification methods.

Material and methods: A systematic review (2014–2026) was conducted across PubMed, Google Scholar, and Cochrane Library databases following PRISMA guidelines. Out of 1,200 initially identified articles evaluating the direct impact of tobacco, e-cigarettes, and NRT on surgical complications (necrosis, infection, dehiscence), 52 publications met the strict inclusion criteria.

Results: Both traditional tobacco smoke and electronic cigarette aerosols exhibit comparable toxicity to the microcirculation, inducing tissue hypoxia, vasoconstriction, impaired immune response, and inhibited fibroblast collagen synthesis. This directly elevates the risk of infection and surgical flap necrosis. When complete nicotine abstinence is unachievable, nicotine replacement therapy (NRT) provides a superior tissue safety profile compared to vaping. Furthermore, patient self-reporting is highly unreliable.

Conclusions: Absolute preoperative nicotine cessation for a minimum of 4 weeks should serve as a standardized criterion for plastic surgery qualification. To eliminate patient self-reporting bias, the implementation of routine, preoperative urine cotinine screening is highly justified to directly reduce postoperative morbidity and generate substantial economic savings for the healthcare system.

Key words: plastic surgery, wound healing, smoking cessation,smoking-induced surgical complications, preoperative care, surgical flaps.

1. Introduction

Tobacco smoking remains one of the most critical modifiable risk factors in plastic surgery. Epidemiological data demonstrate that active smokers account for 10% to nearly 16% of all patients qualified for aesthetic surgery procedures.[1] However, a major clinical challenge in this patient population is the deliberate concealment of their addiction. Screening studies based on the objective analysis of urine cotinine—a nicotine metabolite—reveal that a substantial percentage of patients who declare complete abstinence during medical history taking are, in reality, still actively smoking tobacco. [2,3,4] This phenomenon is widely attributed to the fear of disqualification from the scheduled procedure or postponement of the surgery date. Since many plastic surgeons, aware of the significant complication risks associated with nicotine, refuse to perform elective aesthetic procedures on active smokers, patients resort to dishonesty, unknowingly exposing themselves to a drastic increase in postoperative risks.

The scientific basis regarding the toxicity of tobacco smoke is well-defined in the medical literature. Nicotine and tar components exert a proven, destructive impact primarily on the cardiovascular and respiratory systems. Contemporary population-based studies, including the multicenter PATH cohort study, indicate a clear, positive trend toward steadily increasing health awareness among patients.[5]

Nevertheless, in plastic surgery, the negative impact of nicotine - coupled with low patient awareness on the subject - generates an exceptionally high risk of complications. The specificity of this field relies on the dissection of large cutaneo-subcutaneous flaps and the suturing of tissues under precisely defined mechanical tension. The vascularity of such dissected flaps becomes critically dependent solely on peripheral microcirculation and small perforating vessels.[6] Any disruption in tissue perfusion - which nicotine induces through potent vasoconstriction and the promotion of hypoxia - drastically increases the risk of localized tissue hypoxia.[7] Consequently, this leads to complications that directly undermine the goal of plastic surgery: flap margin necrosis, wound dehiscence, and the formation of wide, hypertrophic scars. In operations where the primary objective is an optimal aesthetic result, these complications constitute a therapeutic failure.[6,7] A cohort study involving 40,465 plastic surgery patients demonstrated that active tobacco smoking drastically impairs the mechanical integrity of the wound, increasing the risk of wound dehiscence by as much as 84% (OR=1,84) and raising the risk of infection by 40%.[4]

Given the magnitude of the nicotine dependence problem among plastic surgery patients and their tendency to conceal this fact from physicians, it is crucial to thoroughly systematize knowledge regarding the actual surgical risks associated with this habit.

The aim of this study was to conduct a detailed analysis of the pathophysiological impact of nicotine on tissue healing processes and to evaluate the incidence and types of postoperative complications in smoking patients undergoing selected plastic and reconstructive surgery procedures. Additionally, the study attempted to assess the efficacy of preoperative smoking cessation strategies and laboratory verification methods, aiming to formulate conclusions that could optimize qualification criteria and enhance patient safety.

2.Research materials and methods

2.1. Search Strategy and Materials

The research material consisted of original and review scientific articles published between 2014 and 2026. A comprehensive literature search was conducted across international medical databases, including PubMed, Google Scholar, Cochrane Library and the JEHS library.

To identify relevant publications, the following keyword combinations were utilized:

2. "nicotine and plastic surgery"
3. "smoking and wound healing"
4. "tobacco and flap necrosis"
5. "abdominoplasty / rhytidectomy and smoking"

2.2. Inclusion and Exclusion Criteria

Studies meeting strictly defined inclusion criteria were qualified for the final analysis:

1. Research articles (clinical, retrospective, and cohort studies) as well as review articles (systematic reviews and meta-analyses).
2. Publications written in English or Polish.
3. Studies analyzing the direct impact of nicotine (cigarette smoking, e-cigarettes, and nicotine replacement therapy [NRT]) on healing processes and complication rates (necrosis, infections, and wound dehiscence) in plastic surgery.

The exclusion criteria were as follows:

- Conference abstracts, letters to the editor, and case reports.
- Articles describing the general health impact of smoking without a direct reference to plastic or reconstructive surgery procedures.
- Duplicate publications and studies published prior to 2014, unless they were deemed essential for conducting a robust and comprehensive meta-analysis.

2.3. Data Selection Process

The literature selection process was carried out in multiple stages in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. In the initial stage, a total of 1,200 articles were identified based on their titles. Following duplicate removal and abstract screening, 1,000 papers were excluded as they did not align with the scope of this meta-analysis. In the final stage, the full texts of the remaining articles were thoroughly evaluated and verified. Ultimately, 51 publications were included in the systematic review.

2.4 AI

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

Discussion:

3. Pathophysiological Mechanism of the Impact of Nicotine on Tissues

3.1. Vasoconstriction

Nicotine is an agonist of nicotinic acetylcholine receptors. Its absorption into the bloodstream triggers rapid stimulation of the sympathetic nervous system and the adrenal medulla, leading to a massive, systemic release of catecholamines—primarily epinephrine and norepinephrine. These hormones, through their affinity for alpha-adrenergic receptors located in the blood vessel walls, induce rapid vasoconstriction. Smooth muscle contraction of the vasculature directly occludes the peripheral microcirculation, drastically limiting tissue perfusion. Additionally, nicotine impairs endothelial nitric oxide synthase (eNOS) synthesis, which is a key, natural vasodilator, introducing a secondary vasoconscriptive mechanism. [8,9] Sørensen et al. investigated blood flow (QBF, SqBF) using laser Doppler flowmetry and ^{133}Xe clearance and tissue oxygen tension (TO_2) was measured using a LICOX O_2 electrode. Their research demonstrated that smoking a single cigarette causes an immediate reduction in cutaneous blood flow by 30% to 40%. This state persists for up to 50–60 minutes after smoking cessation, which, in chronic addiction, results in permanent ischemia of the skin, including the operative site.[10]

3.2. Hypoxia

The pathophysiological process responsible for impaired oxygen delivery to healing skin flaps is driven by the toxic effects of carbon monoxide (CO), a key component of the gas phase of tobacco smoke. Carbon monoxide exhibits an affinity for hemoglobin that is 200–250 times stronger than that of oxygen. Consequently, a stable complex known as carboxyhemoglobin rapidly forms. Its presence in the blood leads to dual biochemical consequences. First, it directly reduces the pool of hemoglobin capable of transporting oxygen. Second, it shifts the oxyhemoglobin dissociation curve to the left, meaning that hemoglobin binds oxygen more tightly and is reluctant to release it in the capillaries.[11]. As a result of the combined effect of vasoconstriction and carboxyhemoglobinemia, deep peripheral hypoxia develops within the operated and undermined tissues. Skin cells and fibroblasts are deprived of the oxygen

required for energy production via the Krebs cycle, which inhibits their replicative and migratory potential, thereby hindering tissue regeneration. Jensen and colleagues reported that smokers who consume a pack of cigarettes per day experience tissue hypoxia for a significant portion of each day.[12]

3.3. Impairment of Collagen Synthesis and Cellular Functions

Profound hypoxia and the direct cytotoxicity of tobacco smoke components impair tissue regeneration. A crucial role is played here by the impairment of fibroblast and macrophage functions. Tobacco smoking drastically inhibits the synthesis of types I and III collagen, which form the structural foundation of a properly healed wound and a pliable scar.[13] This stems from the fact that the hydroxylation of proline and lysine residues is a process strictly dependent on the presence of oxygen and ascorbic acid. Smoking generates massive oxidative stress, leading to systemic destruction of vitamin C - the requirement for this cofactor in smokers increases up to threefold, inducing a profound local deficit.[7,14]

At the immunological level, nicotine impairs the inflammatory phase of wound healing. A phenomenon occurs characterized by impaired neutrophil chemotaxis and reduced phagocytic capacity of macrophages. Smoker's neutrophils lose the capacity to efficiently migrate to the surgical incision site and exhibit diminished oxidative burst activity.[15] Consequently, the wound clearance phase is pathologically prolonged, cytokine signals for neovascularization are lacking, and the risk of bacterial colonization at the surgical site and subsequent infection increases drastically.[16]

4. Clinical Consequences in Smoking Patients

4.1. Flap Necrosis

In plastic surgery, procedures such as abdominoplasty or rhytidectomy characteristically involve the elevation of extensive cutaneo-subcutaneous flaps, which limits their vascularity, making them critically dependent on peripheral microcirculation and perforating vessels. The introduction of nicotine triggers a rapid neurohormonal cascade characterized by the stimulation of the sympathetic nervous system and increased secretion of catecholamines by the adrenal medulla. The state induced in this manner leads to direct stimulation of alpha-adrenergic receptors located in the blood vessel walls, resulting directly in peripheral vasoconstriction.[9] Vasoconstriction of the peripheral vascular bed causes critical perfusion limitation and occlusion of the microcirculation within the surgically elevated cutaneosubcutaneous flaps.[12] Cutting off blood supply to the undermined tissue margins drives the skin at the periphery of the flap into a state of irreversible ischemia, which manifests as cyanosis, blistering, and ultimately flap necrosis and tissue death, thereby undermining the primary aesthetic outcome of the reconstructive procedure. Coon D. et al. demonstrated in their study that active nicotine use in patients undergoing plastic surgery procedures is associated with a drastic increase in necrotic complications; in their study, the odds ratio for tissue necrosis in smokers was OR=4,3, representing a more than fourfold increase in risk compared to non-smokers.[3] Data indicate that to minimize the risk of flap necrosis in smokers, a minimum of one week of preoperative and postoperative abstinence from nicotine is necessary.[17]

4.2. Dehiscence

Mechanistically, the mechanical dehiscence of postoperative wound margins is driven by the massive oxidative stress induced by tobacco smoke components and the concurrent deep

hypoxia of the wound bed. This condition leads to systemic depletion of ascorbic acid reserves and localized tissue hypoxia, which directly blocks the enzymatic hydroxylation of proline and lysine residues in procollagen chains. The impairment of this process prevents fibroblasts from synthesizing a stable triple helix of types I and III collagen, which forms the structural foundation of a properly developing scar.[7,13] The extracellular matrix formed under these conditions is characterized by biochemical immaturity, a chaotic spatial arrangement, and reduced tensile strength.[14] Consequently, the tissue lacks integrity, contributing to its mechanical tearing. This process is exacerbated by the physiological tension generated by tightened surgical sutures during flap closure.[18] Goltsman et al. conducted an analysis of 40,000 patients, which demonstrated that tobacco smoking is the strongest independent modifiable risk factor for mechanical wound complications. In smokers, an adjusted odds ratio for wound dehiscence was recorded at OR=1,84 (an 84% increase in risk).[4]

4.3. Surgical Site Infections:

The pathophysiological mechanism underlying the development of surgical site infections (SSI) in patients with nicotine dependence is a direct consequence of localized tissue hypoxia and carboxyhemoglobin accumulation. The oxygen deficit resulting from the presence of carbon monoxide paralyzes the cellular mechanisms of innate immunity, leading to a profound impairment of neutrophil and macrophage chemotaxis and migration to the wound bed. Moreover, the deeply hypoxic microenvironment prevents the intracellular respiratory burst from occurring, which abolishes the oxidative bactericidal activity of leukocytes and precludes effective phagocytosis and pathogen eradication. Prolonged impairment of the local immune response, combined with the presence of tissue micro-necrosis, establishes an optimal and highly selective substrate for uncontrolled bacterial colonization of wounds, thereby drastically increasing the incidence of superficial and deep surgical site infections.[18] In study by Sørensen et al., a total of 228 wounds were evaluated. The wound infection rate was 12% in smokers compared to 2% in never-smokers. Furthermore, the study demonstrated that wound infections were significantly less frequent in abstinent smokers than in continuous smokers at 4, 8, and 12 weeks post-randomization. It was shown that 4 weeks of smoking cessation reduces the incidence of wound infections.[19]

4.4. Unsightly Scars

The final aesthetic outcome in plastic surgery heavily depends on scar remodeling. Chronic ischemia and peripheral hypoxia modify the final phase of connective tissue remodeling, inducing deformities and unsightly hypertrophic scars.[20] The pathological prolongation of the inflammatory phase, resulting from micro-necrosis and infection, stimulates non-physiological activation of pro-fibrotic signaling pathways, with a particular emphasis on transforming growth factor-beta (TGF-β).[21] This leads to a disruption of the normal quantitative ratio of type I to type III collagen and a chaotic reorientation of newly synthesized fibers. [22] The lack of an organized spatial structure of the extracellular matrix results in the formation of a dense, disorganized, and incoherent connective tissue, which clinically manifests as wide scars or deformities of the contour of the operated area. Consequently, instead of a fine, pale, and unnoticeable line, the smoker develops wide, spread scars that exhibit a tendency for tissue contraction, completely undermining the visual result anticipated by the patient.

However, the impact of nicotine use on postoperative scar aesthetics is dual in nature and is sometimes described in the literature as a pathophysiological paradox. On one hand, tobacco smoking drastically increases the risk of unsightly, wide wounds resulting from the mechanical dehiscence of incision margins and infection of the wound bed.[19,23] On the other hand, clinical studies on the biomedical characteristics of the scarring process reveal that due to potent vasoconstriction and impairment of the local inflammatory response, smokers paradoxically exhibit a lower incidence of highly vascularized hypertrophic scars and keloids.[24]

5. The Impact of Nicotine on the Outcomes of Specific Procedures

A systematic review and meta-analysis conducted by Pluvy et al. revealed that tobacco-smoking patients are exposed to a significantly increased risk of skin necrosis, particularly in cases involving extensive skin undermining. Within plastic surgery procedures, three operations were identified as carrying the highest nicotine-related risks. A current meta-analysis evaluated the impact of nicotine use on complications following abdominoplasty, cervicofacial rhytidectomy, and breast reconstruction and reduction.[25]

5.1. Abdominoplasty

The abdominoplasty procedure necessitates extensive undermining of the cutaneo-subcutaneous flap, extending from the suprapubic incision line up to the costal margins and the xiphoid process of the sternum. During this process, the primary perforating vessels arising from the deep inferior epigastric vascular system are definitively transected. Consequently, the vascular supply to the dissected abdominal wall is acutely reduced, relying solely on peripheral circulation and the subdermal plexus, which is supplied superiorly and laterally by the intercostal, subcostal, and lumbar arteries.[26] The introduction of nicotine into this compromised vascular system induces immediate vasoconstriction of the subdermal plexus. The periumbilical and suprapubic regions represent the so-called watershed zones of vascularization - areas furthest removed from the new peripheral sources of blood supply. Additionally, the umbilicus itself is isolated as a stalk-based tissue island, rendering it highly susceptible to ischemia.[27] Nicotine-induced vasoconstriction and microthrombosis within the capillaries sever the central zone from any remaining perfusion, triggering hypoxia and metabolic ischemic necrosis of the abdominal skin and periumbilical tissues.

In a study by Manassa et al., the authors retrospectively analyzed the medical records of 132 patients (121 females and 11 males) who underwent abdominoplasty over a 5-year period. The incidence of wound complications and dehiscence demonstrated a statistically significant difference between smokers and non-smokers. According to the study results, 47.9% of smokers experienced wound healing complications prior to hospital discharge, compared to 14.8% of non-smokers. [28] Additionally, Bouhadana et al. performed a meta-analysis summarizing 19 independent cohorts of patients who underwent abdominoplasty and panniculectomy exclusively. It demonstrated that tobacco smoking is associated with a statistically significant and rapid increase in the overall rate of local complications.[29]

5.2. Facelift (Cervicofacial Rhytidectomy)

During a facelift, extremely thin and delicate cutaneosubcutaneous flaps are dissected, most commonly in the subcutaneous plane directly overlying the SMAS (superficial muscular aponeurotic system). These structures are devoid of deep muscular vascular supply and rely exclusively on the capillary network of the dermis. The most critical point of the entire

procedure is the retroauricular area; it is here that the flap experiences the highest tension, is at its thinnest, and is furthest removed from the anterior and superior sources of facial vascularization, namely the branches of the facial and superficial temporal arteries.[30] Nicotine-induced vasoconstriction, coupled with the presence of carboxyhemoglobin in the blood, drastically lowers the partial pressure of oxygen within the retroauricular tissues. Due to the minimal thickness of the facelift flap, even brief occlusion of the microcirculation triggers immediate tissue death. In the pathophysiology of facelifts in smokers, skin loss in the retroauricular region exhibits a massive, up to several-fold increase in risk, leading to fascial exposure, extensive ulceration, and distortion of the hairline and ear anatomy.[31]

Rees et al. reported a skin sloughing rate of 7.5% in smokers compared to 2.7% in non-smokers who underwent rhytidectomy.[32] Significantly, studies show that for smokers, a safer alternative that minimizes skin necrosis is the deep-plane facelift. It is suggested that a deep-plane procedure preserves the skin, subcutaneous tissue, and SMAS as a composite flap, which maintains superior blood supply and reduces skin necrosis rates.[33].

5.3. Breast Reconstruction, Mastopexy, and Reduction Mammaplasty

In breast reduction and mastopexy surgeries, the nipple-areola complex (NAC) is transposed on a specially prepared glanduloadipose pedicle, which becomes its sole source of vascular supply.[34] In reduction mammaplasty, vasoconstriction within the pedicle cuts off blood supply to the nipple-areola complex, leading to cyanotic discoloration, hypoxia, and ultimately partial or total necrosis.[35]. In a meta-analysis of 40 studies involving 5,908 patients, Liu et al. demonstrated that in smokers undergoing breast reduction surgery, the risk of infection was 2.03 times higher (95% CI 1,24–3,31; $p < 0,01$),and the risk of tissue dehiscence was 2.34 times higher (95% CI 1,38–3,98; $p < 0,01$).[36]

Conversely, in breast reconstruction utilizing implants or tissue expanders, the entire glandular parenchyma is removed, leaving behind a very thin skin envelope known as the mastectomy flap, beneath which the implant is placed. A study by Jeon et al. demonstrated that following gland removal, the flap becomes drastically thin, and the necrosis rate following immediate implant placement reaches up to 30% in certain centers.[37] In implant-based reconstructions, nicotine-induced occlusion of the microcirculation within the mastectomy flaps triggers marginal necrosis. This establishes a direct pathway of communication between the external environment and the implant pocket. Bacteria easily colonize the hypoxic, defenseless connective tissue of the wound bed, culminating in an acute surgical site infection (SSI). An infection surrounding a foreign body cannot be eradicated with antibiotic therapy alone and inevitably necessitates surgical removal (explantation) of the implant, representing a catastrophic failure of the breast reconstruction.[38]

6. Preoperative Strategy – Smoking Cessation

6.1. Optimal Duration of Smoking Cessation

Establishing a precise timeframe for smoking cessation is a fundamental component of preoperative patient optimization in plastic and reconstructive surgery. As demonstrated by Kolar et al. in a prospective study of 128 patients, smoking cessation at least 4 weeks prior to a scheduled procedure significantly reduces the risk of wound complications during the 30-day postoperative follow-up period. Conversely, continuing the habit up to less than 2 weeks

before surgery is associated with a significantly higher rate of surgical failure and prolonged healing times, which mandates that structured smoking cessation programs be treated as a priority element of surgical planning.[40]

The timeframe for smoking cessation is directly derived from cellular dynamics and the period required to reverse the pathophysiological effects of tobacco toxins on the body. The impact of tobacco smoke on the biomechanics of wound healing is multidirectional and sequential. As indicated by Sørensen in his systematic review, tobacco smoking transiently reduces oxygen perfusion and aerobic metabolism within the wound bed, a dysfunction that impairs both the inflammatory and proliferative phases.[14] It has been shown that smoking cessation immediately restores normal tissue oxygenation; however, cellular repair mechanisms require a longer duration for regeneration. The review revealed that after 4 weeks of tobacco cessation, only a partial normalization of the inflammatory response is observed, while proliferative processes within this timeframe still exhibit signs of profound inhibition.[14]

In a study by Sørensen et al., it was proven that implementing effective tobacco abstinence for a minimum of 4 weeks preoperatively allows for a radical, measurable reduction in the rate of postoperative wound infections and superficial incision margin necrosis.[20]
This relationship is confirmed from both macroeconomic and population-based perspectives by comprehensive systematic analyses. In a meta-analysis by Mills et al. evaluating the impact of smoking cessation duration on surgical complications, it was shown that interventions lasting a minimum of 4 weeks yield the most spectacular and statistically significant decrease in postoperative complications. The meta-analysis demonstrates a linear correlation between the length of the smoke-free period and the clinical benefit to the patient, proving that each additional week of preoperative abstinence increases the therapeutic effect by an additional 19%.[39]

The contemporary medical consensus and clinical standards mandate absolute cessation of tobacco smoking and nicotine intake in any form for a minimum of 4 weeks before a scheduled procedure. Preoperative nicotine abstinence prepares the tissues for surgical trauma and facilitates a proper postoperative healing course. It is worth noting that the specificity of certain plastic surgery procedures, which relies on the dissection of extensive, delicate cutaneosubcutaneous flaps, renders the healing process of these regions extremely sensitive to fluctuations in tissue perfusion. Therefore, maintaining complete abstinence postoperatively dictates the ultimate aesthetic success. During the aforementioned meta-analysis, no definitive timeframes for postoperative smoking cessation were found; thus, a greater body of research is required in this area.

However, postoperative cessation appears to be more sustainable than preoperative cessation. Van Slyke et al., in a study evaluating long-term postoperative outcomes (mean follow-up period: 63.3 months), demonstrated that among 42 patients declaring daily tobacco smoking prior to surgery, 10 individuals (23.8%) permanently quit smoking, maintaining total abstinence from the moment of operation. Additionally, 17 patients (40.5%) discontinued daily cigarette smoking over the long-term follow-up period.[41] Thus, there is a documented positive association between undergoing plastic surgery procedures and long-term cessation of tobacco addiction. The implementation of a mandatory requirement for preoperative nicotine abstinence serves as an effective behavioral intervention, promoting permanent smoking cessation among patients in the long-term follow-up.[41]

6.2. Timeline of Tissue Regeneration

The reversal of pathophysiological and biochemical disruptions induced by nicotine proceeds according to a strictly defined chronology. Understanding these cellular-level changes at specific time points allows for the precise determination of timeframes for pre- and postoperative smoking cessation.

Table 1 outlines this chronological progression, corroborating the broad timeframes mentioned above and providing a rigorous substantive justification for recommending a minimum of 4 weeks of tobacco abstinence prior to surgery.

Table 1. Effect of smoking cessation duration on pathophysiological mechanisms and clinical wound healing parameters.

Time Since Cessation	Systemic objective	Pathophysiological Mechanisms	Clinical significance for the wound and tissues	References
12 Hours	Elimination of carbon monoxude (CO) and restoration of oxygen transport.	Carboxyhemoglobin (COHb) occludes the oxygen-binding sites on hemoglobin and shifts the oxyhemoglobin dissociation curve to the left. After about 12 hours, COHb levels return to baseline values below 1–2%, rendering hemoglobin molecules available for oxygen binding.	Acute tissue hypoxia diminishes, allowing for faster and enhanced tissue oxygenation, particularly within skin flaps and the marginal zones of the wound bed.	(Kambam et al., 1986) [42]

2 Weeks	Enhanced microcirculation and normalization of hemorhelogy.	Tobacco stimulates prothrombotic factors (increased fibrinogen, blood viscosity, and platelet aggregation) and decreases vasodilatory nitric oxide (NO) production.	Adequate circulating blood volume is restored within the wound; vascular microemboli are eliminated, and reduced plasma viscosity facilitates the delivery of nutrients.	(Morita et al., 2005)[43] (Sørensen et al., 2004) [44]
4 Weeks	Restitution of the baseline pool of circulating endothelial progenitor cells, responsible for vascular repair.	Tobacco impairs macrophage migration, suppresses their bactericidal properties, and blocks fibroblast-mediated collagen synthesis. Following cessation, fibroblast restitution is achieved and the stimulation of collagen synthesis is restored.	A decrease in the incidence of surgical site infections (SSIs) and mitigation of pathological damage and vascular endothelial dysfunction, which promotes microcirculatory stabilization and enhances the structural integrity of the healing wound.	(Kondo et al., 2004)[45]

6.3. E-cigarettes and Nicotine Replacement Therapy (NRT)

It is erroneously suggested that electronic cigarettes are healthier than traditional cigarettes; consequently, patients may fail to report their use or perceive them as a safer alternative.[46] As indicated by numerous studies, nicotine itself—including that delivered via alternative methods such as e-cigarettes, heated tobacco products, or transdermal nicotine replacement therapy (NRT) systems—is associated with an elevated risk of postoperative wound complications. [47]

In a cohort study by Troiano et al. involving 45 rats, it was demonstrated that exposure to e-cigarette vapor induces necrosis in random-pattern skin flaps at a frequency comparable to

traditional tobacco smoke.. In both cohorts, necrotic complications occurred significantly more frequently than in unexposed subjects. This suggests that aerosol inhalation exerts an equally destructive effect on microcirculation and tissue viability, and vaping should not be treated as a more favorable alternative to cigarette smoking in a surgical context.[48]

This is further corroborated by a study by Sifil et al., in which among 15,172 patients, 1,184 (7.8%) reported nicotine use.[2] Complications occurred in 15.2% of nicotine users compared to 3.8% of non-users; notably, complications developed in nicotine-using individuals regardless of the delivery method, including e-cigarettes and nicotine replacement therapy.[2]

In light of the available data, the most justified clinical approach is to recommend complete nicotine abstinence during both the preoperative and postoperative phases of plastic surgery procedures. In cases where complete nicotine cessation is unfeasible for the patient, the implementation of nicotine replacement therapy (NRT) is acceptable. NRT exhibits a superior tissue safety profile and should be strictly preferred over the inhalation of electronic cigarette aerosols.[49]

6.4. Biochemical Verification of Patient Nicotine Status

One report demonstrated that in patients with elevated cotinine levels, the risk of postoperative wound complications doubles.[50] Conversely, another study showed that introducing objective preoperative biochemical verification drastically reduces the number of complications; up to 21.3% of patients undergoing preoperative urinalysis tested positive, proving that despite medical recommendations, a substantial proportion of patients conceal or deny tobacco use during the perioperative period.[51]

Within the screened cohort, a more than twofold decrease in the incidence of delayed wound healing was observed, dropping from 41.5% (in the untested group) to 20.3%.Routine testing enabled a reduction in the overall postoperative complication rate from 42.3% to 18.1%.[51]

Implementing routine measurement of cotinine levels in clinical practice effectively eliminates self-reporting bias resulting from patient concealment of their addiction. Consequently, as an objective predictor of surgical risk, this biomarker facilitates the precise identification of patients who would benefit most from aggressive, preoperative smoking cessation support programs. Additionally, the implementation of mandatory preoperative urine nicotine tests ensures an objective verification of the patient's smoking status. This strategy directly reduces postoperative morbidity and generates substantial economic savings for the healthcare system. [52]

7. **Conclusions**

In conclusion, the clinical and pathophysiological evidence reviewed in this study underscores that nicotine represents one of the primary modifiable risk factors for surgical failure in plastic surgery. Tobacco smoking induces tissue hypoxia and vasoconstriction, impairs the local immune response within the wound, and inhibits fibroblast-mediated collagen synthesis—directly exacerbating the risk of surgical site infections and surgical flap necrosis. Based on the pathophysiology and chronological timeline of tissue regeneration, absolute smoking cessation for a minimum of 4 weeks prior to the procedure should serve as a standardized criterion for surgical qualification. Concurrently, further investigation is warranted to define the optimal timeframes for postoperative cessation. Crucially, enforcing a

mandatory preoperative abstinence requirement acts as a powerful behavioral catalyst, fostering long-term permanent smoking cessation.

Furthermore, electronic cigarette aerosols exhibit microcirculatory toxicity comparable to traditional tobacco smoke. In scenarios where complete nicotine discontinuation is unachievable, nicotine replacement therapy (NRT) offers a significantly superior tissue safety profile over vaping alternatives. Finally, given the unreliability of self-reported patient history, the integration of routine preoperative urine cotinine screening is essential to directly reduce postoperative morbidity and generate substantial cost savings for the healthcare system.

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Author Contribution

Conceptualization, Aneta Synakiewicz; methodology, Julia Kępska; writing - original draft

preparation, Aneta Synakiewicz; literature search, Kamil Wójcik, Nikola Murawska, Weronika Olech, Wiktor Kowalczyk; data curation, Julia Kępska, Weronika Olech;

visualization (tables), Wiktor Kowalczyk, Arina Drobashko; investigation, Kamil Wójcik, Natalia Lewińska, Nikola Murawska, Aleksandra Pociej, writing - review and editing, all authors; supervision, Aneta Synakiewicz. All authors have read and agreed to the published version of the manuscript.

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