Healing is a complex process that is influenced by biological, mechanical and systemic factors. The association between cigarette smoking and delayed wound healing is well recognized in clinical practice. Nicotine is a vasoconstrictor that reduces nutritional blood flow, resulting in ischemia and impaired healing of injured tissue.

The mechanism by which smoking may affect healing is unknown. One possible explanation is that the substances in tobacco and its smoke, particularly nicotine, cotinine, carbon monoxide, and hydrogen cyanide are cytotoxic to those cells that are involved in healing. Hemoglobin, the oxygen carrying molecule in the blood will be unable to carry as much oxygen as normal in smokers, tiny blood vessels become narrow, making it more difficult for both hemoglobin and oxygen to reach where they are needed, resulting in tissue hypoxia. Nicotine increases platelet adhesiveness, raising the risk of microvascular occlusion, and tissue ischemia. It also affect negatively bone healing by diminishing osteoblast function, causing autogenous bone graft morbidity and interferes with the biomechanical properties of the graft.

Smoking is also associated with catecholamine release, resulting in vasoconstriction and decreased tissue perfusion, as well as a significant effect on fracture union, where both current and previous smokers exhibited a significantly higher proportion of delayed union and nonunion of fractures (2 to 10 times higher), reported to be more apparent in those cases requiring bone grafts, as there is an increased chance of de-vascularizing the graft.

Studies had also shown a significant increase in the incidence of localized osteitis (Dry Socket) following tooth removal in cigarette and water-pipe smokers (3-times more prone) compared to other factors like age, sex, usage of oral contraceptives, and increased surgical time, suggesting that the heat from the burning tobacco, and tobacco along with its byproducts could act as a contaminant in the surgical site together with the suction applied to the cigarette that might dislodge the clot from the alveolus interrupting healing of the socket.

Smoking is reported as an important patient-related risk factor responsible for postoperative infection, chemicals in cigarette smoke limit the activity of neutrophils, leading to a higher risk of infection which may require antibiotics or more surgical intervention compared to non-smokers.

Reduction of both bone quantity and quality in traumatic sites were found to be more pronounced in smokers than non-smokers and believed to be due to the increase in bone resorption at the fractured ends resulting from smoking, interfering with the osteoblastic function. Bone formation following distraction osteogenesis was also found to be compromised (lower volume of bone and chondrocytes) in high-dose nicotine exposure cases.

Tobacco, apart from being positively associated with pre-cancerous and cancerous lesions as well as many life-threatening health conditions, is also reported to negatively affect the outcome of almost all routine therapeutic procedures performed in the oral cavity, starting from simple nonsurgical periodontal therapy to orthognathic surgeries.
Both physicians and dental surgeons should aid smoking patients to become tobacco-free, educating the patients by imparting these adverse effects.