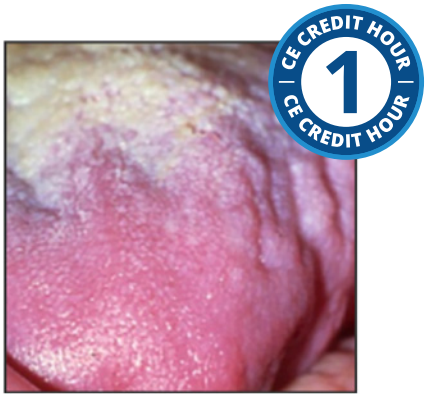
# Biological Effects of Radiation




**Course Author(s):** Hassem Geha, DDS, MDS, Diplomate of the ABOMR
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## Conflict of Interest Disclosure Statement

- The author reports no conflicts of interest associated with this course.

## Introduction – Biological Effects of Radiation

Biological Effects of Radiation discusses the interaction of ionizing radiation with biological systems and how this affects the integrity of cellular proteins, including DNA. In addition, it present the short- and long-term effects of radiotherapy associated with treating head and neck tumors on normal tissues.

## Course Contents



## Overview

This course discusses the interaction of ionizing radiation with biological systems and how this affects the integrity of cellular proteins, including DNA. Short-term and long-term effects of radiotherapy associated with the treatment of head and neck tumors on normal tissues is presented.

## Learning Objectives

**Upon completion of this course, the dental professional should be able to:**

- Explain the difference between nonionizing and ionizing radiation.
- Discuss the mechanisms of free radical generation.
- Discuss potential cell damage related to direct and indirect ionization.
- Discuss, in general terms, the effect of free radicals on cellular proteins as a function of the various phases of the cell cycle.
- Discuss the effect of radiation dose on cell dynamics.
- Discuss the effects of high-dose ionizing radiation on tumor cells.
- Discuss the deterministic effects (short-term and long-term) of high dose radiation normal tissues in the head and neck.
- Discuss stochastic effects and mutations associated with ionizing radiation.

## Introduction

**Radiation biology** is the study of the effects of radiation on biological systems. When radiating energy and living cells interact, energy transfer leads to one of two phenomena: excitation or ionization. Low-energy **nonionizing radiation**, e.g., ultraviolet radiation, microwaves, and extra-low-frequency electromagnetic radiation, causes molecular **excitation**. **Excitation occurs** when an electron changes its energy level without being ejected from the atom's orbit.[1-3]

**Ionizing radiation** is sufficiently energetic to eject an electron from an atom's orbit resulting in an ion pair (the electron and the rest of the atom), i.e., causes **ionization**.[1,4,5] Particulate radiation and electromagnetic radiation can cause ionization. **Particulate radiation** is a stream of atomic or subatomic particles such as α-particles and neutrons produced by nuclear disintegration; high energy **electromagnetic radiation** includes δ-rays and x-rays (photons).[1,5-8]

Radioactive substances emit gamma-rays. X-ray photons are man-made. They are produced by x-ray units when fast-moving electrons interact with tungsten atoms.9 When photons with sufficient energy liberate orbital electrons from atoms and their corresponding molecules, the creation of unpaired electrons results in the formation of highly reactive **free radicals**. Free radicals in a biological system can damage cellular proteins by altering their chemical structure.[1,5-8,10,11]

### Direct versus Indirect Ionization

Direct or indirect ionization can cause cell damage. (Figure 1). **Direct ionization** occurs when charged particles, e.g., electrons, with sufficient kinetic energy, interact with cellular atoms or molecules to create free radicals.[4,6,10,11] The process is known as direct because the interaction occurs directly between a particle and a cellular component without an intermediary step. The charged particle can continue interacting with other molecules until all its kinetic energy is lost.

**Indirect ionization** occurs when non-charged particles, e.g., photons, interact with cellular water. The energy absorbed

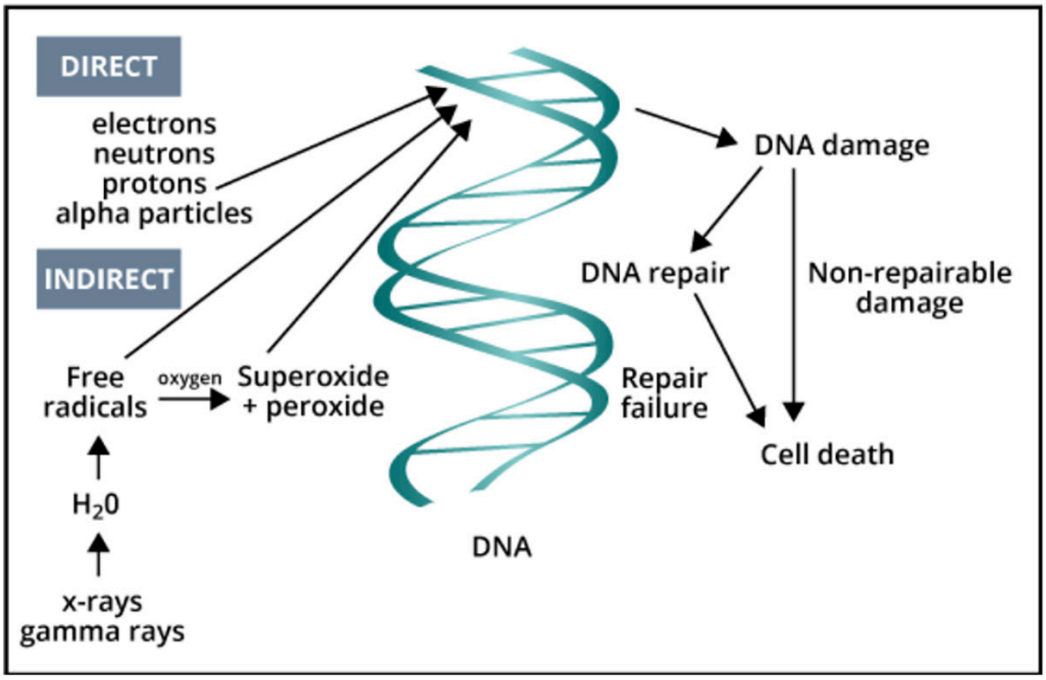


**Figure 1.** Cell damage can be caused by direct or indirect ionization.

by an $H_2O$ molecule will form ion pairs and reactive oxygen metabolites such as hydroxyl radicals.[4,6,11] These free radicals interact with cellular atoms and molecules, damaging cellular proteins, and may form additional free radicals.[2,4,6,10,11] The process is indirect because of the intermediate step of H2O-based free radical formation.

## Free Radicals

Free radicals have a high degree of chemical reactivity.[4,10] When they interact with cellular macromolecules, e.g., proteins, and alter their chemical structures, they cause repairable or non-repairable damage with significant downstream effects such as altered cell function or cell death.[5,6,8] The effect of free radicals on deoxyribonucleic acid (DNA), which contains the genetic code, is the most important and varies during the various phases of the cell cycle.[2,4-6,8,11,12]

Normal cells go through five physiological phases: G0, G1, S, G2, and M (Figure 2).[13] The G0 phase is the latent or resting phase. Cells recruited from the G0 phase enter the G1 or the first active phase of the reproductive cycle. In the G1 phase, the cells synthesize ribonucleic acid (RNA), enzymes, and proteins in anticipation of entering subsequent phases of the reproductive cycle. In the $G_1$ phase, the cells synthesize ribonucleic acid (RNA), enzymes, and proteins in anticipation of entering subsequent phases of the reproductive cycle.

The S phase follows the G1 phase. The predominant event in the S phase is the synthesis of DNA. At the end of the S phase, the cells contain twice the original amount of DNA. The G2 phase follows the S phase. During the G2 phase, the mitotic spindle is created, which is essential for cell divisionIn the M or mitotic phase, cell division occurs. Cells are most radiosensitive in the G1, G2, and M phases, respectively. During the S phase, the cells are the most radioresistant. (Figure 3).[8,11]

There is a wide variation in radiosensitivity among different cell types. For example, rapidly dividing cells or cells with a potential for rapid division are more radiosensitive than those that do not divide. In addition, undifferentiated cells are more radiosensitive than highly specialized cells. Finally, within the same cell families, the immature forms rapidly dividing are more radiosensitive than the mature cells that have specialized in function and have ceased to divide.[6,8,10]

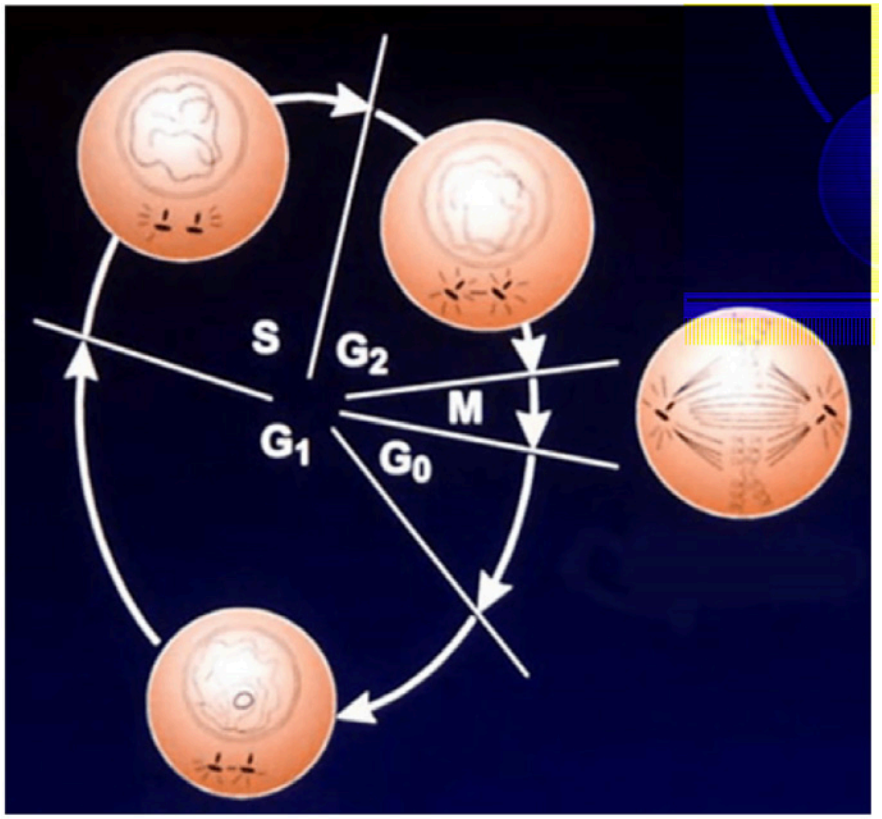


**Figure 2.** Five physiological phases of the cell cycle: $G_0$, $G_1$, S, $G_2$, and M.

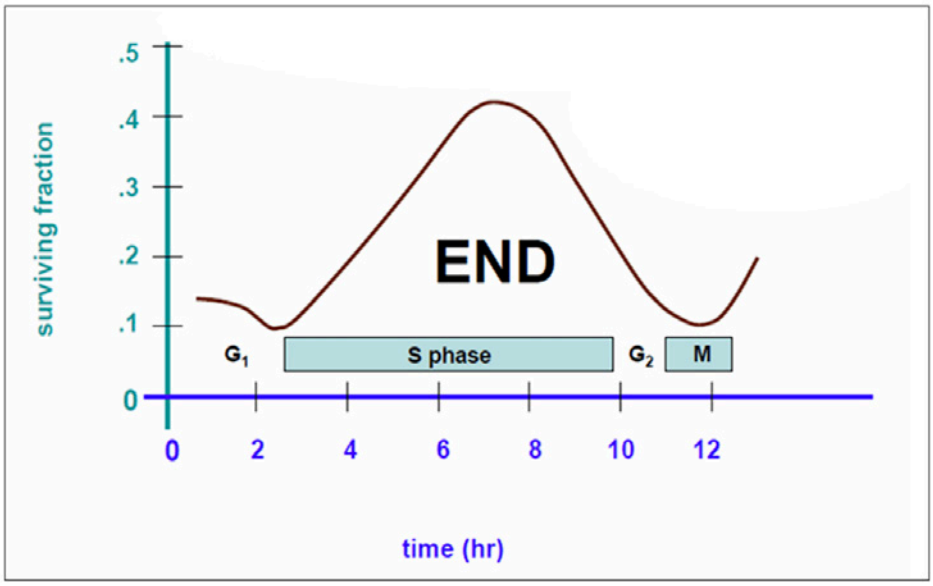


**Figure 3.** Cells are most radiosensitive in the $G_1$, $G_2$, and M phases, respectively; and most radioresistant in the S phase.

In summary, cell radiosensitivity is directly proportional to the rate of cell division and inversely proportional to the degree of cell differentiation, i.e., actively dividing cells or those not fully mature are most at risk from radiation. Highly sensitive cells include germ cells, immature red blood cells, and lymphocytes (an exception to the above). Epithelial cells are moderate to highly radiosensitive. Cells of low radiosensitivity include muscle and nerve.

## Effect of Radiation Dose on Cell Dynamics

Exposure of cells with high mitotic activity to high dose radiation would result in a severe delay in mitotic activity for an extended period before the mitotic rate would return to normal. Conversely, exposure of cells with high mitotic activity to moderate doses of radiation would result in a moderate delay in the mitotic activity for a moderate period, followed by a moderate increase in mitotic activity for a short period before the mitotic rate would return to normal. (Figure 4).[2,6,8]

Exposure of cells with high mitotic activity to low dose radiation would result in an initial mild delay in mitotic activity followed by a short period of increased mitosis before the mitotic rate would return to normal.[2,6,8] In association with dental radiography, neither short-term nor long-term effects of radiation on mitotic activity are discernable.[2,5-7,11] However, to minimize exposure and maximize diagnostic yield, follow the dictum of ALARA - As Low As Reasonably Achievable.

## Effects of High-dose Ionizing Radiation

High-dose ionizing radiation is an effective alternative to surgery or a valuable adjunct to surgery and/or chemotherapy in the locoregional treatment of head and neck malignancies. Oral healthcare providers can expect to be called on to care for head and neck cancer patients undergoing radiotherapy or who may have previously completed radiotherapy. Consequently, it is essential to understand the deterministic biological changes due to high-dose ionizing radiation.[5,6,11]

The therapeutic benefit of ionizing radiation is related to its effects on tumor cell DNA. Radiation can interact and damage DNA directly by causing DNA breaks or indirectly by interacting with water or oxygen molecules, resulting in ion pairs and reactive oxygen metabolites such as H2O2 and hydroxyl

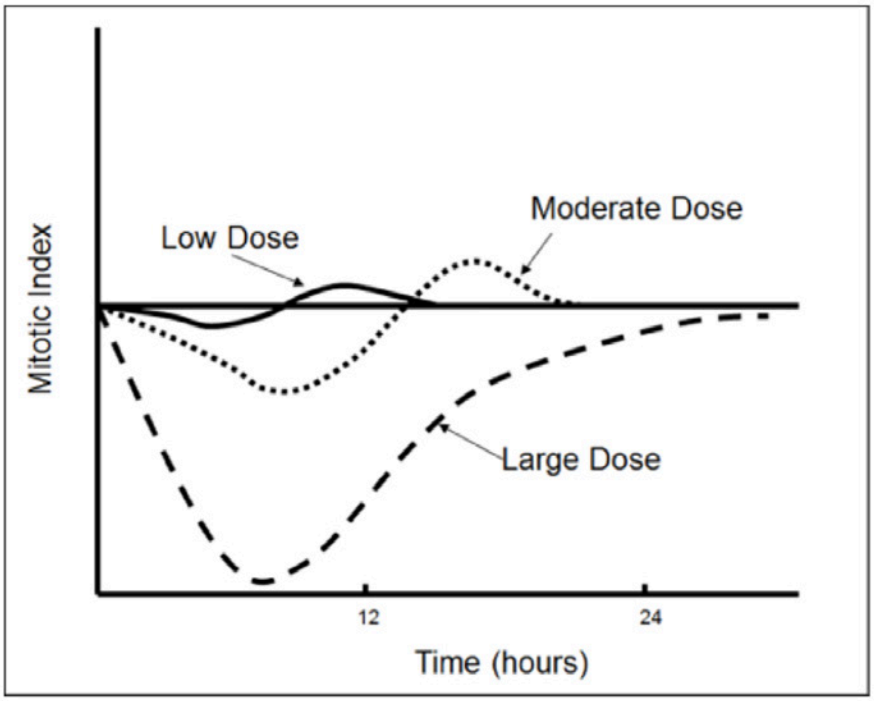


**Figure 4.** Change of mitotic rate as a function of dose and time.

radicals (Figure 5).[13] DNA damage, if not repaired, will likely cause cell death and eliminate malignant activity.[13]

Malignant cells have a decreased capacity to repair radiation damage. They can fall into one of three categories: (1) lethal damage, which occurs when no DNA repair is possible and leads to cell death, (2) sublethal damage, which is repairable as a function of time provided no further radiation damage is incurred prior to the repair, and (3) potentially lethal damage, which is a condition in which cells may survive without repair, depending on post-radiation conditions.[13]

Radiation dose is expressed as the absorbed energy by the irradiated tissue. The unit of the absorbed dose is the Gray (Gy=1 J/Kg). A conventional radiotherapy regimen for head and neck neoplasms consists of delivering 60–70 Gy, fractionated at a rate of 2.0 Gy/day, 5 days/week, for 6 to 7 weeks. However, newer and often more aggressive protocols and techniques may be employed to improve tumor control and/or reduce side effects.[13]

Theoretically, any malignant tumor can be destroyed by ionizing radiation if the dose delivered is sufficient. The limiting factor is the amount of radiation the adjacent normal

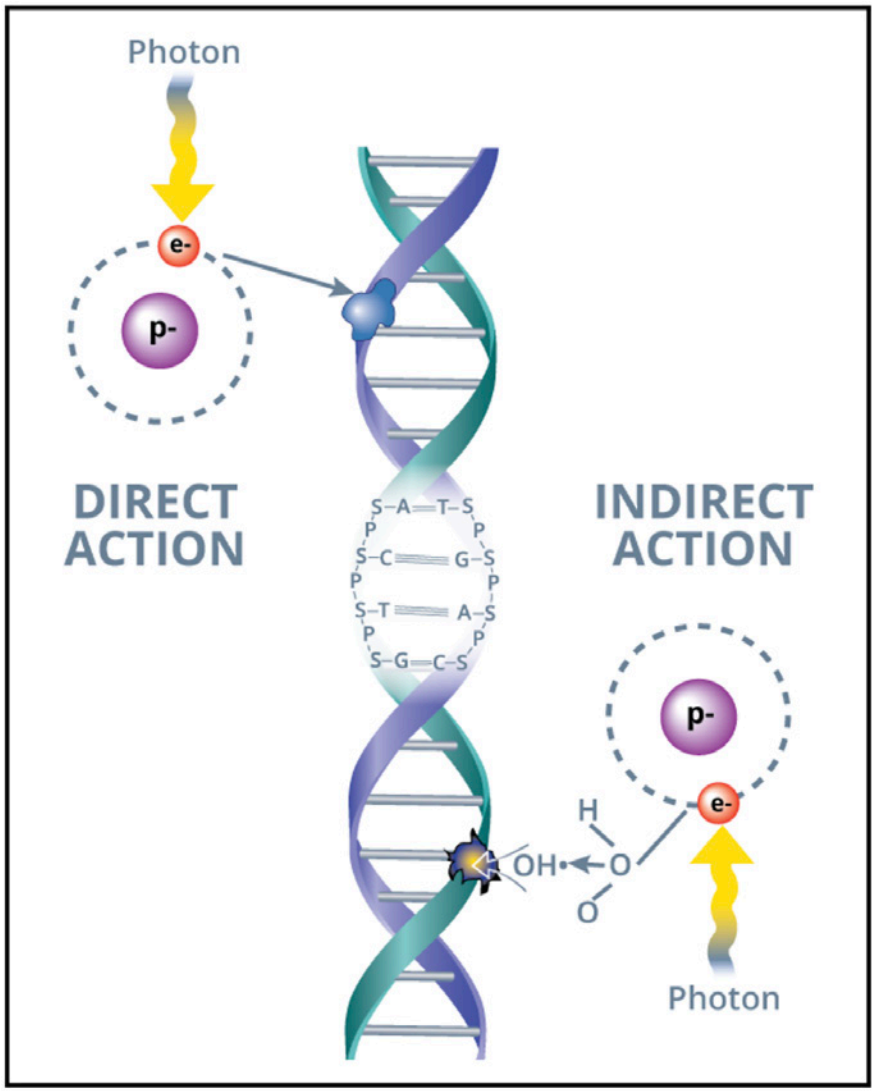


**Figure 5.** It is estimated that two-thirds of radiotherapy-induced damage to DNA is caused by hydroxyl radicals.

tissues will tolerate.[13] These adverse effects can be either direct, i.e., radiation-induced destruction or damage to susceptible cells causing a loss or disruption of tissue function, or indirect, i.e., radiation-induced decrease in vascularity and associated tissue changes.[5,6,8]

**Short-term effects** - Undifferentiated cells of irradiated tissues or organs will be severely damaged. It will lead to acute but transient disruption of the integrity and function of affected tissues or organs. If the doses are relatively low, after some time, the stems cells will be able to differentiate, and healing will take place, followed by at least partial re-establishment of tissue or organ function.[5,6,8]

**Long-term effects** - In radiation therapy, sometimes the radiation doses are high enough to cause damage to the microvasculature of tissues and organs without damaging the differentiated cells. The outcome of this damage will be noticed 6-8 weeks post irradiation. The damage to the microvasculature will lead to a loss of nutrient flow into the organs leading to their degeneration and necrosis. These effects progress for the remainder of the irradiated patient's life.[5,6,8]

### Skin and Mucosa

A biological model proposed to address the changes associated with radiotherapy-induced dermal and mucosal changes suggests an inflammatory, epithelial, ulcerative, and healing phase. During the inflammatory phase, free radicals and cytokines, e.g., interleukin-1β, prostaglandins, and tumor necrosis factor-α (TNF-α), are released in response to irradiation. These chemical mediators of inflammation increase vascular permeability and cause tissue damage.[13]

During the epithelial phase, cellular reproduction reduces, which leads to desquamative pseudomembranous degeneration. The ulcerative phase heralds the period when the ulcerated tissue is most painful and susceptible to infection. In addition, the number of gram-negative bacteria may increase, further stimulating the inflammatory process. The final or healing phase occurs when epithelial regeneration can reestablish tissue integrity.[13]

Radiodermatitis and oral mucositis develop secondary to the depletion of the epithelium's rapidly dividing basal cell layer. Skin reactions to radiotherapy are estimated to affect about 25% of patients and depend on the dose delivered and the tissue volume irradiated. Skin reactions appear within the first three weeks of radiotherapy with conventional doses. These reactions may be erythematous, desquamative, or necrotic.[13]

Radiotherapy-induced mucositis generally appears on non-keratinized tissues such as the labial and buccal mucosa, soft palate, pharynx, the floor of the mouth, and the tongue. It is associated with edema and erythema of the affected tissues, followed by desquamation. Desquamative lesions progress to painful ulcerations and may become colonized by Candida organisms. The progression will result in acute or chronic candidiasis. (Figure 6).[13]

During conventional radiotherapy protocols (i.e., 2 Gy/day, 5 days/week, for 5–7 weeks), cellular repopulation of the epithelium can counteract the destructive effects of radiotherapy dosing of up to 1.8 Gy/day is unlikely. After the first week of therapy, there will be hyperemia and epithelial atrophy followed by edema and erythema due to hyperemia. Painful desquamative pseudomembranous lesions and ulcerations mark the second and third weeks of therapy (Figure 7).[13]

Pharyngeal mucositis may impair the patient's ability to swallow and speak. An estimated 60% of patients undergoing conventional radiotherapy for head and neck cancer develop severe mucositis and require pain control and nutritional supplementation. With more intense radiotherapy, the incidence of mucositis may exceed 90%. However, complete healing in most patients occurs within four weeks after the completion of radiotherapy.[13]

Long-term effects associated with irradiated oral mucosa include tissue atrophy, telangiectasias, and increased risk of chronic ulceration (Figure 8). Cytokines and late-responding endothelial cells in the connective tissue underlie progressive fibrosis and

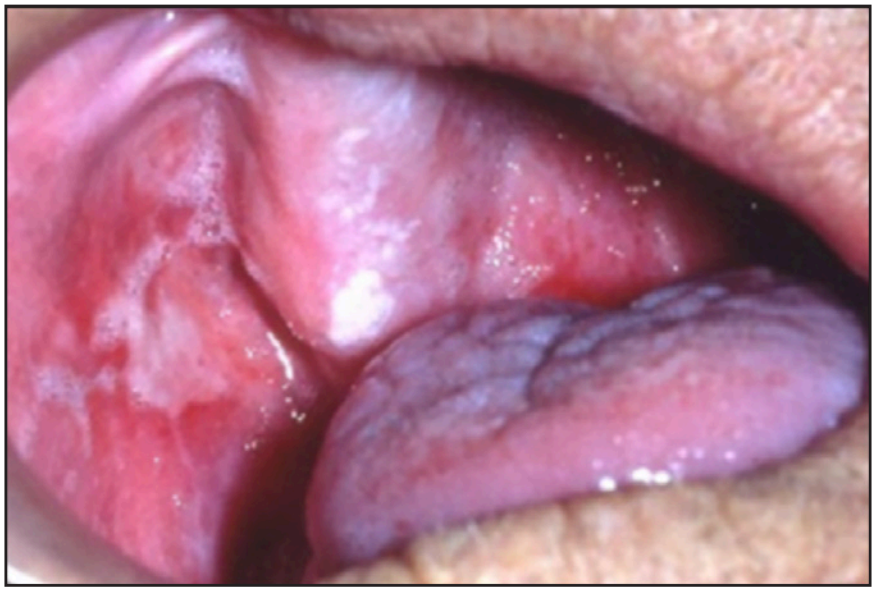

**Figure 6.** Acute radiotherapy-induced mucositis and candidal infection.

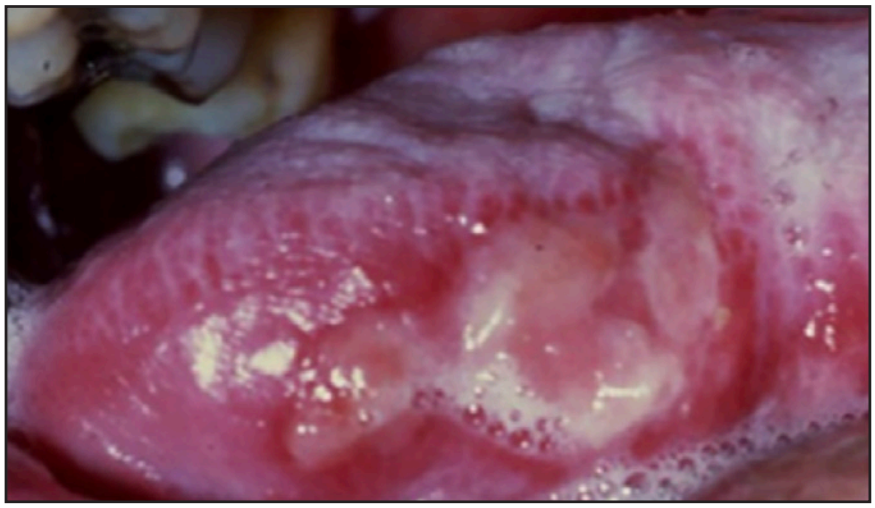

**Figure 7.** Acute radiotherapy-induced ulceration during the 2nd and 3rd week of therapy, also note the thick, viscous and frothy character of saliva.

thrombosis of small vessels in the dermis and submucosa. Radiodermatitis may result in hyperpigmentation, permanent loss of hair, and increased risk of skin cancers, typically basal cell carcinomas.[13]

## Salivary Glands

The healthy patient produces up to 1.5L of saliva per day. The major salivary glands produce 80-90% of the total volume.
The salivary glands secrete serous saliva (parotid glands), mixed seromucous saliva (submandibular glands), or mucous saliva (sublingual and minor salivary glands). Minor salivary glands produce 70% of the mucins. The parotid glands produce the most stimulated saliva, and the submandibular glands produce the most unstimulated saliva.[13]

The well-differentiated and slow replicating salivary gland cells are highly radiosensitive for reasons not clearly understood.1 Radiotherapy causes acute degeneration and necrosis of acinar cells. There will be a reduction of the salivary output by 80% within the first two weeks of conventional radiotherapy. As a result, the saliva will be scant, thick, viscous, and ropy or frothy. Acinar recovery, if any, may occur about 12 to 18

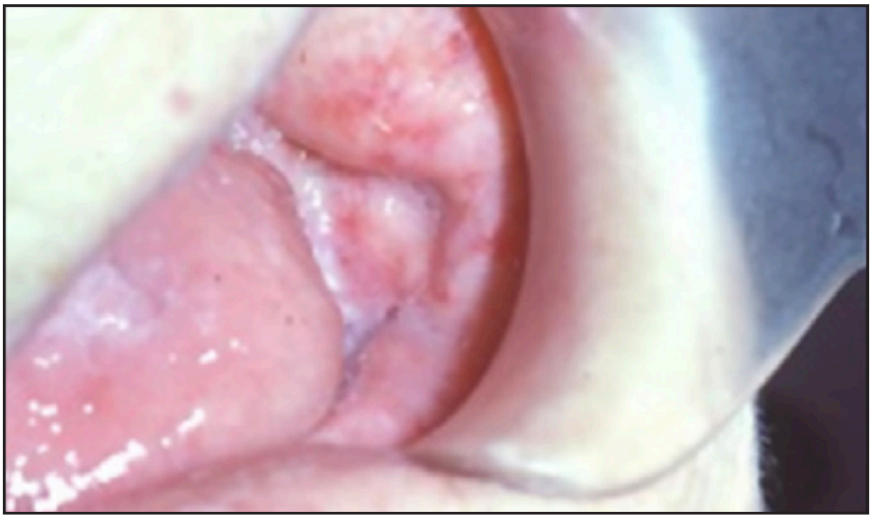

**Figure 8.** Long-term effects associated with irradiated oral mucosa include tissues atrophy and telangiectasias.

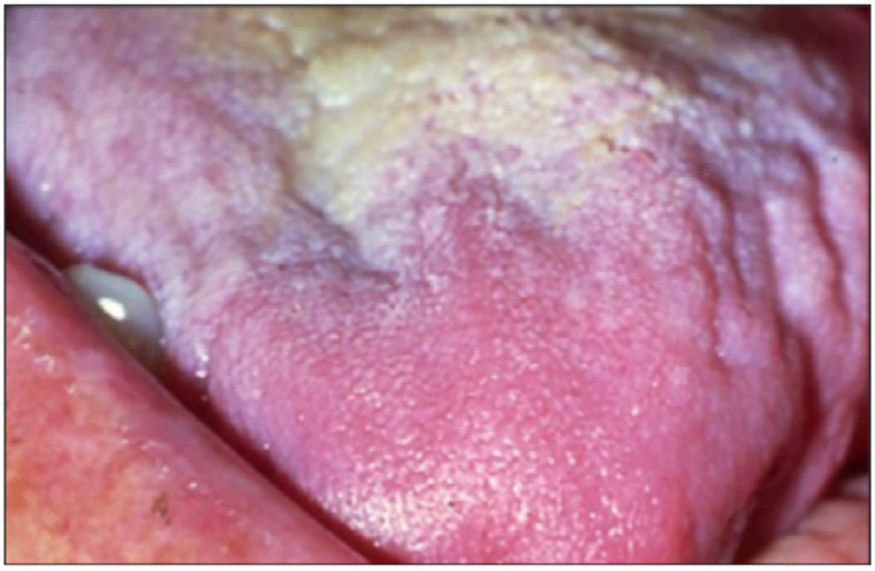

**Figure 9.** Xerostomia and chronic candidal infection are the most frequently reported long-term side effect of head and neck radiotherapy.

months following completion of therapy.[13]

Qualitative and quantitative salivary changes lead to reduced lubrication; reduced lavage and cleansing of oral tissues; decreased immunoglobulin levels, i.e., reduced antibacterial, antiviral, and antifungal activity; and further loss of mucosal integrity. As a result, patients may experience difficulty swallowing, chewing, speaking, and wearing prostheses, which along with hypogeusia or ageusia, may lead to impaired nutrition and weight loss.[13]

If the radiation dose is sufficient, regeneration of acinar cells fails, leading to fibrosis and atrophy of glandular tissue. While a mean dose of 60 Gy is the accepted threshold for producing irreversible damage, in some cases, mean doses of as little as 26 Gy have been implicated. As a general rule, following head and neck radiotherapy, patients will experience a significant reduction in salivary flow, i.e., xerostomia, for the remainder of their life. (Figure 9).[13]

The loss of saliva's buffering capacity and the

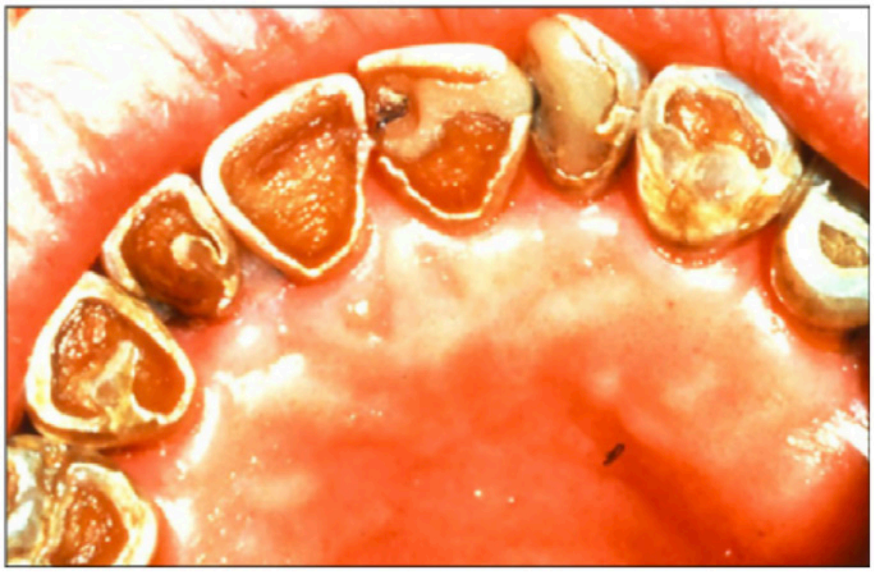

**Figure 10.** A distinctive form of rampant caries, termed "radiation caries", is frequently observed following head and neck radiotherapy.

decrease in the salivary immunoglobulin levels will increase cariogenic oral microflora, leading to "radiation caries". Radiation caries begin on plaque-forming surfaces and areas of exposed dentin resulting in circumferential lesions at the cementoenamel junction and smooth surface caries on cusp tips and incisal edges. (Figure 10).[13]

## Taste Buds

Taste buds are located on the circumvallate and fungiform papillae of the tongue and to a lesser extent on the tonsillar pillars, base of the tongue, soft palate, pharynx, and larynx. Hypogeusia - reduced ability or ageusia - loss of ability to taste sweet, sour, bitter, or salty substances is an early and frequent complaint associated with head and neck radiotherapy. Histologically, taste buds show signs of atrophy at 10 Gy. Higher doses will obliterate the taste buds.[13]

Taste acuity may decrease by a factor of 1,000 to 10,000 due to direct radiation damage to the taste buds and/or secondary to impaired salivary function and mucositis. Perception of acid and bitter is affected early, whereas sweet and salt acuity is affected as treatment continues. Restoration of taste sensation usually occurs within 2-4 months following radiotherapy; however, some patients may experience a life-long alteration in or loss of taste perception.[13]

## Teeth

Radiotherapy to the head and neck area may significantly affect the developing dentition due to a tooth's developmental stage and the absorbed dose. A dose of 10 Gy can permanently damage maturing ameloblasts. Ameloblastic activity ceases after exposure to 30 Gy. Radiation-induced defects may include partial or complete anodontia, tooth dwarfism, incomplete root development, premature closure of apices, and localized enamel defects.[13]

## Bone

Radiation doses above 60 Gy kill endothelial cells. The loss of endothelial cells will cause obliterative endarteritis and periarteritis. Also, it will overwhelm the reparative capacity of bone. As a result, osteoblasts and osteocytes undergo necrosis, and the periosteum and marrow spaces undergo fibrosis. Consequently, the irradiated bone becomes hypocellular, hypovascular, and hypoxic. The potential clinical outcome of these effects is osteoradionecrosis. (Figure 11).[13]

The effect of radiotherapy on the developing bone of a child depends on the child's age during treatment, the quality and quantity of the radiation dose delivered, and the location and extent of the bone in the treatment field. Doses over 20 Gy may significantly impair bone

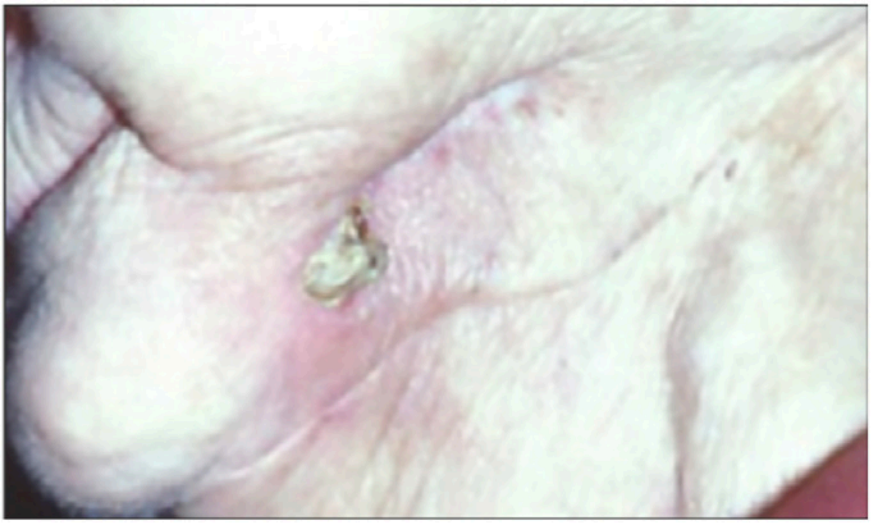

**Figure 11.** Chronic radiodermatitis and ulceration and osteoradionecrosis of the mandible.

growth and development, resulting in maxillary and mandibular hypoplasia. In addition, the use of asymmetrical radiation portals may lead to hemifacial hypoplasia.[13]

### Temporomandibular Joint (TMJ) and Muscles of Mastication

The Radiation portals that include the temporomandibular joint and/or muscles of mastication may affect the capsule or muscular integrity. The radiotherapy-induced cellular damage and eventual tissue fibrosis can lead to muscle spasm, trismus, and limited function of radiation dose to the area. These effects manifest 3 to 6 months post-radiotherapy with threshold doses between 15 Gy and 50 Gy.[13]

## Dental Radiography Post-Radiotherapy

A common question gets asked about the consequences of exposing a patient to a dental x-ray after radiation therapy. The radiation doses from intraoral, panoramic radiographs, Cone beam, CT, or even medical CT are shallow compared to the doses of radiotherapy. The only concern would be obtaining intraoral radiographs within six months of radiotherapy, i.e., before mucosal healing, and further traumatizing the mucosa in the process.[6]

## Late Somatic Effects of Radiation

Regardless of the radiation dose, there is always the risk of stochastic effects. In addition, radiation can cause cancer by modifying DNA. Most of these cancers appear ten years after exposure. It is important to note that radiation will not cause the development of new cancers. Instead, it will increase the risk of developing additional cancers in the population. It is of note that the risk of late somatic effects in children is twice that noted in adults.[2,5,6,8,11]

## Hereditary Effects of Radiation

Radiation that damages the DNA of germ cells in the gonads can cause gene mutations. These mutations increase with dose. Similar to the late somatic effects, there will not be new mutations but an increased frequency of spontaneous mutations. Mutations in the male germ cells are higher than in the female germ cells. However, there is a reduction in the rate of mutations if the time between exposure and conception increases.[5,6,8,11]

## Conclusion

Ionizing radiation generates free radicals. Free radicals interact with biological tissues, most importantly DNA. The severity of radiation-induced damage depends on the cell's sensitivity and the type and dose of radiation. There may be a DNA damage repair, but there are deterministic effects visible over a short-term period and a long-term period. The late somatic effects may manifest as malignancy and other genetic mutations.

## Course Test Preview

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**1. Which of the following statements related to radiating energy is correct?**

A. When radiating energy and living cells interact, energy transfer leads to one of two phenomena: radiation or ionization.
B. Ultraviolet radiation, microwaves, and extra-low-frequency electromagnetic radiation are high-energy ionizing forms of radiation.
C. Ionizing radiation is sufficiently energetic to eject an electron from an atom's orbit resulting in an ion pair, i.e., the electron and the rest of the atom.
D. Photons with sufficient energy can liberate orbital electrons from atoms resulting in the formation of x-ray radiation.

**2. Which of the following statements related to direct and indirect ionization is correct?**

A. Direct ionization occurs when charged particles, e.g., electrons, with sufficient kinetic energy interact with a water molecule to create free radicals.
B. Indirect ionization occurs when non-charged particles, e.g., photons, interact with the DNA.
C. Energy absorbed by an $H_2O$ molecule can results in the formation of ion pairs and reactive oxygen metabolites such as hydroxyl radicals.
D. Direct ionization and indirect ionization are only the result of x-ray interacting with water molecules.

**3. Which of the following statements related to free radicals and the cell cycle is correct?**

A. Free radicals have a high degree of chemical reactivity.
B. When free radicals interact with cellular macromolecules they cause only non-repairable damage.
C. In general, cells are most radiosensitive in the S phase of the cell cycle and most radioresistant in the G1, G2, and M phases, respectively.
D. Cell radiosensitivity is inversely proportional to the rate of cell division and inversely proportional to the degree of cell differentiation.

**4. With dental radiography neither short-term nor long-term effects of radiation on mitotic activity are discernable. What dictum one should follow to minimize exposure and maximize diagnostic yield?**

A. ALARA – As legal as radiographically attainable.
B. ALARA – At lower additional risk achievable.
C. ALARA – At lowest alternative radiation attainable.
D. ALARA – As low as reasonably achievable.

**5. Which of the following statements related to the effects of high-dose radiation on malignant cells is correct?**

A. The therapeutic benefit of ionizing radiation on tumor cells is related to its effects of tumor cell cytomplasm.
B. Radiation can interact with and damage DNA directly by causing DNA breaks.
C. Radiation cannot cause indirect damage to the DNA.
D. In comparison to normal cells, malignant cells have an increased capacity to repair radiation damage.

**6. Which of the following statements related to radiotherapy is correct?**

A. Radiation dose is expressed as the absorbed energy by the irradiated tissue and the unit of the absorbed dose is the REM (REM=1 Kg/J).
B. A conventional radiotherapy regimen for head and neck neoplasms consists of delivering 6–7 Gy, fractionated at a rate of 0.2 Gy/day, 5 days/week, for 6 to 7 weeks.
C. Theoretically, any malignant tumor can be destroyed by ionizing radiation if the dose delivered is sufficient, the limiting factor is the amount of radiation the adjacent normal tissues will tolerate.
D. Short-term effects of ionizing radiation, which are related to damage to the microvasculature will be noticed within 6-8 weeks post irradiation.

**7. Which of the following statements related to the biological effects of radiation on skin and mucosa is correct?**

A. Radiodermatitis and oral mucositis develop secondary to the depletion of the rapidly dividing basal cell layer of the epithelium.
B. Radiotherapy-induced mucositis generally appears on keratinized oral tissues, e.g., the gingivae.
C. Edema and erythema due to hyperemia and epithelial atrophy of the oral mucosa appear after 8 weeks of radiotherapy.
D. Short-term effects associated with irradiated oral mucosa include tissues atrophy, telangiectasias, and increased risk of chronic ulceration.

**8. Which of the following statements related to the biological effects of radiation on salivary glands is correct?**

A. The well-differentiated and slow replicating salivary gland cells are highly radio-resistant.
B. Salivary output may be increased by as much as 80% within the first 2 weeks of conventional radiotherapy .
C. As a general rule, following head and neck radiotherapy, patients will experience no significant permanent reduction in salivary flow.
D. Radiation caries begin on plaque forming surfaces and areas of exposed dentin at the cementoenamel junction and on cusp tips and incisal edges.

**9. All of the following statements related to biological effect of radiation on taste buds are correct EXCEPT which one?**

A. Hypogeusia or ageusia is an early and frequent complaint associated with head and neck radiotherapy.
B. Histologically, taste buds show signs of atrophy at 1 Gy.
C. Perception of sweet and salt acuity are affected early, whereas acid and bitter is affected as treatment continues.
D. Restoration of taste sensation usually occurs within 2-4 months following radiotherapy.

**10. Which of the following statements related to biological effect of radiation on teeth is correct?**

A. Radiotherapy to the head and neck area will not affect the developing dentition.
B. Maturing ameloblasts may be permanently damaged with as little as 1 Gy.
C. Radiation-induced defects may include partial or complete anodontia, tooth dwarfism, incomplete root development, premature closure of apices, and localized enamel defects.
D. A single intra-oral x-ray can cause permanent damage to a developing tooth.

**11. Which of the following statements related to biological effect of radiation on bone is correct?**

A. Radiation doses in excess of 60 Gy kill endothelial cells, causing obliterative endarteritis and periarteritis, and overwhelm the reparative capacity of bone.
B. Osteoblasts and osteocytes undergo necrosis and the periosteum and marrow spaces undergo regeneration.
C. Irradiated bone becomes hypocellular, hypovascular, and hypoxic; the potential clinical outcome of these effects is osteoradionecrosis.
D. After a dose of 60 Gy, the repair and regeneration of endothelial cells will cause obliterative endarteritis and periarteritis.

**12. Which of the following statements related to biological effect of radiation on developing bone is correct?**

A. The effect of radiation on developing bone depends on the child's age, the radiation dose delivered, and the extent of the bone in the treatment field.
B. Doses in excess of 20 Gy may significantly impair bone growth and development, resulting in maxillary and mandibular hypoplasia.
C. The use of asymmetrical radiation portals may lead to hemifacial hypoplasia.
D. The dose level is not a contributing factor to the damage to the bone in a child.

**13. Radiation portals that include the temporomandibular joint and/or muscles of mastication may affect the capsule or muscular integrity; cellular damage and tissue fibrosis can lead to muscle spasm, trismus, and limited function as a function of radiation dose to the area.**

A. True
B. False

**14. All of the following statements related to late somatic effects of radiation is correct EXCEPT which one?**

A. Regardless of the radiation dose to which a patient is exposed, there is always the risk of stochastic, i.e., random, effects.
B. Radiation can cause cancer by modifying DNA.
C. Most of radiation-related cancers are unique, i.e., new cancers not normally seen in the population.
D. The risk of late somatic effects in children is twice that noted in adults.

**15. All of the following statements related to biological effect of radiation on mutations is correct EXCEPT which one?**

A. Radiation that damages the DNA of germ cells in the gonads can cause gene mutations.
B. Gene mutations increase with dose and represent an increase in the rate of unique mutations that are different than those attributed to spontaneous mutations.
C. Mutations in the male germ cells are higher than in the female germ cells.
D. The rate of radiation-induced mutations is reduced as the time between exposure and conception increases.

## References


1. Curry TS, Dowdey JE, Murry RC, et al. Christensen's physics of diagnostic radiology, 4th ed. Philadelphia, PA. Lippincott Williams & Wilkins. 1990.
2. Sprawls P. Physical principles of medical imaging, 2nd ed. Madison, WI. Medical Physics Pub. 1995.
3. Bushberg JT, Seibert JA, Leidholdt EM, et al. The essential physics of medical imaging, 3rd ed. Philadelphia, PA. Lippincott Williams & Wilkins. 2012.
4. Juhl JH, Crummy AB, Kuhlman JE. Essentials of Radiologic Imaging, 7th ed. Philadelphia, PA. Lippincott-Raven Publishers. 1998.
5. Frommer HH, Stabulas-Savage JJ. Radiology for the Dental Professional, 9th ed. London, UK. Elsevier Health Sciences. 2014.
6. White SC, Pharoah MJ. Oral radiology: principles and interpretation, 7th ed. St. Louis, MO. Mosby. 2013.
7. Iannucci JM, Howerton LJ. Dental radiography: principles and techniques, 5th ed. St. Louis, MO. Saunders. 2017.
8. Bushong SC. Radiologic science for technologists: physics, biology, and protection, 11th ed. St. Louis, MO. Elsevier. 2017.
9. Geha H. Basic Radiation Physics. dentalcare.com. Accessed March 18, 2019.
10. Miles DA, et al. Radiographic imaging for the dental team, 4th ed. St. Louis, MO. Saunders. 2009.
11. Hall EJ, Giaccia AJ. Radiobiology for the radiologist, 7th ed. Philadelphia, PA. Lippincott Williams & Wilkins. 2012.
12. Langland OE, Langlais RP, Preece JW. Principles of dental imaging, 2nd ed. Baltimore, MD. Lippincott Williams & Wilkins. 2002.
13. Huber MA, Terezhalmy GT. The head and neck radiation oncology patient. Quintessence Int. 2003 Oct;34(9):693-717.


### Additional Resources

- No Additional Resources Available

## About the Author


### Hassem Geha, DDS, MDS, Diplomate of the ABOMR


Hassem Geha is a Professor in the Department of Comprehensive Dentistry, and the director of the Advance Oral and Maxillofacial Radiology program at the University of Texas Health Sciences Center. He received his dental degree from Saint Joseph University, School of Dental Medicine – Beirut in 1997, and two specialty degrees in oral biology and Maxillofacial radiology from the Lebanese University, School of Dentistry in Beirut in 2001. In 2002, he relocated to the United States. He became a Diplomate of the American Board of Oral and Maxillofacial Radiology in 2004. He received a Master of Dental Sciences (MDS) degree from the University of Connecticut Graduate School in 2005. Dr. Geha was appointed Assistant Professor at New York University College of Dentistry. In 2010 he joined UTHSCSA where he is Oral Radiology course director for the DS3 and he is heavily involved in the post-graduate program at the dental school. He also is a Clinical Associate in Otolaryngology and Head and Neck Surgery at the American University of Beirut Medical Center. Dr. Geha was the recipient of Albert G. Richards Award in 2003 and the Radiology Centennial Scholarship Award in 2004 given by the American Academy of Oral and Maxillofacial Radiology. He has given many presentations and continuing education courses at national and international meetings and authored many scientific manuscripts and abstracts in national and international journals. Hashem's main research focuses on enhancing digital imaging based on mathematical models. He chaired and served in several academic committees including many MS theses supervising committees in Oral and Maxillofacial Radiology.


Email: geha@uthscsa.edu