Introduction

Skin lesions are a frequent occurrence in the head and neck. Many times this will be the patient’s reason for seeking medical attention, while at other times it will be noticed during the course of a routine physical examination. It is important for the otolaryngologist to be able to distinguish between obviously benign lesions and those that are malignant or pre-cancerous. The greatest chance for cure with minimal cosmetic and functional deficits depends on early diagnosis and treatment. This discussion will focus on basal cell (BCCa) and squamous cell (SCCa) carcinomas of the head and neck including their epidemiology, presentation, diagnosis and treatment. Several benign lesions that are often confused with these cancers will also be discussed, as will lesions that are generally considered pre-malignant. For the purposes of this discussion, skin malignancy will refer only to BCCa and SCCa. Melanoma, Mohs’ surgery and reconstruction of defects after the treatment of cutaneous malignancies are the subjects of separate Grand Rounds presentations and will therefore be omitted.

Epidemiology and Etiology

Skin malignancies are the most common cancers in humans with 600,000 to 800,000 new cases in the United States each year. Basal cell is the most common type, accounting for 90% of all cutaneous neoplasms in the head and neck and almost one quarter of all cancers diagnosed in the U.S. SCCa is the second most common histopathologic type with nearly 100,000 new cases in the U.S. each year. Although highly dependent on sex, geographic location and cell type, the lifetime probability of developing BCCa varies from 11 to 28% and 1.5 to 11% for SCCa. For both cell types, the incidence increases with age. Generally, those over 60 are at highest risk for developing SCCa, while BCCa is relatively common in patients over 40. The incidence is increasing in younger patients, perhaps due to changes in fashion, and tumors of either cell type may be seen in patients in their 20’s. Men are 2 to 3 times more likely than women to develop a skin malignancy. In both sexes, 80% of skin cancers arise in the head and neck.

Numerous different factors seem to interact to cause skin cancer. The most important of these factors seems to be exposure to ultraviolet radiation (UV). It is estimated that 90% of non-melanoma skin cancers are due to UV exposure. Laboratory experiments indicate that UV light in the ultraviolet B (UVB) band (wavelength 280 to 320 nm) has the greatest potential for carcinogenesis. It is radiation in this frequency range that is responsible for the common sunburn. UVB has been shown to induce photochemical damage to DNA and its repair mechanisms, and has been shown to be immunosuppressive. Ultraviolet A (UVA, wavelength 320 to 400 nm) was initially believed to be non-carcinogenic. It is much more prevalent than UVB. Recent studies have indicated that UVA does have some carcinogenic potential in laboratory animals with exposure to high doses for long periods of time. In addition, UVA seems to augment UVB responses and thereby may act as a co-carcinogen. This has raised concerns over the safety of tanning beds that emit UVA light. Ultraviolet radiation C (UVC, wavelength 200 to 290 nm), is a potent carcinogen that
is filtered by the ozone layer of the earth’s atmosphere. The depletion of the ozone layer as a result of the widespread use of chlorofluorocarbons has prompted concerns over the potential for a drastic increase in the incidence of skin malignancy. Mathematical models predict a 2 to 4% increase in the incidence of skin cancer for every 1% reduction of the ozone layer. This could result in an additional 30,000 cases per year in the United States.

Race and ethnic heritage have strong influence in the risk of developing a skin malignancy. Whites of Celtic ancestry (Irish, Scottish and Welsh) have the highest incidence, while blacks have the lowest incidence but the highest mortality. Skin malignancies in blacks seem to be less dependent on sun exposure as they tend to be more common in non-exposed skin and frequently occur at the site of scarring, burns or previous trauma. This difference in the incidence of skin cancers among ethnic groups is attributed to the melanin content of skin and the ability to tan. A pale complexion with the inability to tan, the tendency to freckle and sunburn and red or blonde hair indicate a high risk for the development of skin cancer. Most studies indicate that the ability to tan is the single most important protective factor. Persons of Scandinavian descent who are fair-skinned but tan easily have relatively low rates of skin cancer, while persons of Irish descent who are unable to tan have a much higher incidence. Patients who have a history of skin malignancy have a 12 to 15-fold increased risk for the development of second skin cancer within 5 years of diagnosis of the first.

Other factors have been shown to increase the risk of developing a cutaneous malignancy. Ionizing radiation has been shown to be quite carcinogenic. The first radiation-induced cancer was a skin malignancy in an x-ray technician reported in 1902. The incidence of BCCa and SCCa has been shown to be higher than expected in survivors of the Japanese atomic bomb. In addition, many patients treated with radiation for benign skin conditions including ringworm, hirsutism and hemangioma have an increased incidence of skin cancers. Health-care workers with radiation exposure, uranium miners and airline pilots also have an excess of skin cancers. The greatest risk factor seems to be the accumulated dose of radiation. Fortunately, the use of radiation for the treatment of benign conditions has largely been abandoned, and precautions have been put in place to minimize occupational exposure.

Chronic exposure to chemicals including polycyclic aromatic hydrocarbons and arsenic has been shown to cause skin cancer. Arsenic exposure may be either occupational or medicinal. Fowler’s solution was ingested for the treatment of syphilis, asthma and psoriasis. Workers in the refining of ores, pest control, farming, glass blowing and wine growing all experienced arsenic exposure as well. These cancers typically appear years after exposure, are often multiple, may occur on unexposed parts of the skin and are associated with hyperpigmentation and keratoses. Cigarette smoking has not definitively been shown to cause skin malignancies, but animal studies suggest an association.

Several medical conditions may predispose to skin cancers. These include xeroderma pigmentosum, an autosomal recessive disorder that results in the inability to repair DNA damage from exposure to UV radiation with a high incidence of BCCa, SCCa and melanoma. The nevoid basal cell carcinoma syndrome is an autosomal dominant disorder characterized by multiple BCCa, jaw cysts, rib abnormalities, palmar and plantar pits with calcification of the falx cerebri. Other genetic disorders that are at high risk for developing cutaneous malignancies include albinism (autosomal recessive), epidermolytic hyperkeratosis (autosomal recessive), epidermolytic hyperkeratosis (autosomal recessive and dominant) and dyskeratosis congenital (sex-linked recessive). In addition, previous trauma such as burns or ulcers with the resultant scarring is associated with the development of skin malignancies (Marjolin’s ulcer). Immunosuppressed patients, such as those with lymphoma or leukemia, or post-transplant patients also demonstrate an increase in the incidence of cutaneous malignancy and cancers in these patients frequently behave more aggressively.

Normal Skin Histology

The skin is the largest organ of the body and is approximately one sixth of the total body weight. It has four major functions including protection, sensation, thermoregulation and metabolic activity. It varies over regions of the body in thickness, color and presence of appendages such as hairs, glands and nails. Despite these variations, all skin has the same basic structure and organization. The external layer consists
of a keratinized squamous epithelium known as the epidermis. The epidermis may be broken down into five layers representing stages of maturation of epidermal cells. The innermost layer is the stratum basale or germinativum. This is the germinal layer of the epidermis. More superficial is the stratum spinosum. Adjacent to this layer is the stratum granulosum in which cells have granules that contribute to the process of keratinization. Above this lies the stratum lucidum, which may only be appreciated in especially thick skin. The most external layer is the stratum corneum which is made up of flattened, fused cell remnants composed mostly of the fibrous protein, keratin.

Deep to the epidermis is the dermis. This layer is composed of fibro-elastic connective tissue that is highly vascular and serves to nourish and support the epidermis. The hypodermis or subcutaneous layer lies deep to the dermis. This layer contains variable amounts of adipose tissue. Hair follicles, sweat glands, sebaceous glands and nails are termed epidermal appendages as they develop from downward growths of epidermis into the dermis and hypodermis.

**Pre-Cancerous Lesions**

Several benign lesions are important to recognize as they may precede the development of a skin cancer. The most common of these lesions are actinic keratoses (AK), sometimes called solar keratoses. AK is the most common pre-malignant lesion of the head and neck. They occur almost exclusively on sun-exposed areas of the face and neck and are most common in fair-skinned patients, the same population and sites known to be at high risk for the development of SCCa and BCCa. AK results solely from sunlight induced damage. The risk for progression to BCCa or SCCa is 5% to 20%. The lesions typically have an erythematous base with an adherent scale. They rest superficially on the skin and demonstrate little infiltration. Frequently they develop from a rough scale that may be palpated prior to the lesion becoming clinically apparent. AK occasionally develops marked hyperkeratosis and may present as a cutaneous horn. As the risk for progression is significant, these lesions should be treated and not observed. Depending on the clinical situation, they may be treated with cryotherapy, shave excision, topical 5-fluorocil (5-FU) or trichloroacetic acid (TCA) peel. Full-face dermabrasion may also be useful for severe, diffuse disease.

Keratoacanthoma is often clinically and histopathologically confused with SCCa. Two general types have been described, solitary and multiple. In solitary keratoacanthoma, the lesions usually grow rapidly over several months. They are most common in elderly males. Several phases of development have been recognized: growth, plateau and involution. Typically the tumor appears in the growth phase as a firm, dome-shaped papule that progresses to nodule of 1 to 2.5 cm in greatest diameter over 6-8 weeks. The center often becomes ulcerated and filled with keratinous material. The lesion then enters the plateau phase. The tumor then spontaneously involutes within 6 months, healing with a slightly depressed scar. Ninety five percent of solitary keratoacanthomas occur on sun-exposed areas, and solitary lesions have never been reported on palms, soles or mucosal surfaces. There are reports of keratoacanthoma progressing to SCCa in immunosuppressed patients. It is unclear whether this represents true transformation, or simply initial misdiagnosis. The most common location is the nose, and aggressive lesions with invasion and destruction of the underlying structures have been reported.

Multiple keratoacanthoma has two forms. The first is the multiple, self-healing epithelioma of skin. These lesions usually begin in childhood or adolescence and can be located anywhere on the body including the palms, soles and head and neck. Usually no more than 12 are present at any one time and the course is similar to that of solitary keratoacanthoma with spontaneous involution. The second form is the eruptive keratoacanthoma. These usually do not occur until adulthood when hundreds of characteristic 2-3mm papules appear. The mucosa of the mouth and larynx may be involved.

The treatment of keratoacanthoma can be difficult, as most lesions will spontaneously involute. Surgical excision is the treatment of choice when the lesion cannot be observed. Intralesional 5-FU and methotrexate have induced involution with healing. Mohs’ surgery may be used to treat large lesions and those that are difficult to distinguish from SCCa.

Bowen’s disease is synonymous with carcinoma in situ of the skin and represents pre-invasive SCCa. The histologic appearance is full-thickness dysplasia of the epidermis without a breech of the basement
membrane. Clinically the lesions are well-circumscribed, erythematous plaques with irregular borders. They tend to occur in sun-exposed areas. Lesions may occur in non-exposed skin in patients with a history of arsenic exposure.

**Basal Cell Carcinoma**

The typical presentation of BCCa is that of a raised, nodular lesion with a smooth, pearly border. There is often telangiectasia and prominent vascular on and adjacent to the lesion. The center may ulcerate and crust. Patients may describe pruritis as one of the earliest symptoms, and may describe the lesion early in its development as pimple-like.

Several different clinical types of BCCa have been described. The most common is the nodular or noduloulcerative. These lesions typically are raised, waxy or pink in color with a well-defined border. A capillary network is readily visible. The characteristic presentation and clear demarcation make this type relatively easy to treat. A variant of the nodular type is the cystic BCCa, which has tongues of tumor separated by spaces within the stroma. Some lesions may be confused with melanoma by the production of a brown pigmentation. These lesions differ from nodular BCCa only by the production of the pigment and behave similarly to nodular BCCa.

The superficial multicentric BCCa often presents with one or several erythematous, scaly patches. The lesions have irregular borders and increase in size by spreading peripherally. These lesions are more common on the extremities and trunk and less common in the head and neck. The indistinct borders make treatment more difficult.

Morpheaform BCCa is more clinically subtle and more aggressive in its growth pattern. It has also been known as sclerosing or fibrosing BCCa. The lesion typically is a flat macule with a yellow or whitish plaque. The margins may be quite indistinct and the lesion may resemble a scar. The lesion may go unnoticed for years, but may eventually ulcerate. Histologic evaluation may reveal subclinical, fingerlike extensions into the dermis. This subtle spread makes complete excision difficult.

Less common, but also of a more aggressive nature, is the keratotic BCCa. These tumors are also known as basosquamous or metatypical carcinoma. It was initially thought that this tumor represented a lesion intermediate between BCCa and SCCa. It now appears as though this lesion is a BCCa that keratinizes. The clinical behavior is more aggressive than other histologic types, but the potential for metastasis is limited.

The typical cell in BCCa resembles the cells of the stratum basale of the epidermis but lack intercellular bridges. There is scant cytoplasm and the nuclei are large, oval and elongated with relatively uniform size and configuration. The tumors contain a connective tissue stroma oriented parallel to the cell masses that causes a palisading and stromal retraction. This is known as “peritumoral lacunae.” The mucinous component of the stroma often shrinks with the dehydration of fixation and may retract from the tumor islands. This is known as clefting and may aid in diagnosis.

There are four basic histologic types: solid, keratotic, cystic and adenoid. In the solid type, there is little cellular differentiation, and masses of tumor cells invade the dermis. The peripheral layer may exhibit palisading of the nuclei. Keratotic BCCa show differentiation toward hair structures. They typically have undifferentiated cells with parakeratotic cells and horn cysts. Cystic tumors display spaces within the tumor and are differentiated toward sebaceous glands. Glandular or tubular formation is the hallmark of adenoid tumors. The epithelial cells form strands that form lace-like patterns.

The stroma of the BCCa is essential for its development and growth. Transplantation of tumor cells without stroma is unsuccessful. This dependence on the stroma may account for the low rate of metastasis. Metastatic BCCa is rare with an incidence in the range of 0.0028% to 0.1%. When it occurs, the tumor most commonly spreads to the regional lymph nodes. Hematogenous spread to the lungs and bone may also occur, but is quite rare. Adenoid and keratotic BCCa have a higher propensity for metastasis, but any
of the clinical or pathologic types may do so. Metastases typically occur from primaries in the head and neck that have been present for long periods of time.

While metastatic disease is rare, BCCa may invade locally with destruction of underlying and adjacent structures. The path of growth is along that of least resistance. Invasion of bone, cartilage and muscle is therefore a late occurrence as periosteum, perichondrium and fascia offer a barrier to tumor invasion. The tumor may spread along these structures for some distance however, and recurrence is common in lesions on structures such as the eyelid, nose and scalp where lateral growth occurs along these resistant planes just deep to the skin. In contrast, embryonic fusion planes offer little resistance to deep tumor invasion. The most susceptible areas include the inner canthus, philtrum, mid-lower chin, nasolabial groove, pre-aurocular area and retro-aurocular sulcus. These areas have a very high recurrence rate after what seems to be adequate therapy.

Tumor growth is also highly dependent upon blood supply. BCCa elaborate angiogenic substances that account for the telangiectatic vessels characteristic of their appearance. Large tumors frequently have a necrotic center as a result of growth beyond their vascular supply. Because of insufficient blood supply, BCCa usually does not invade the subcutis. Perineural spread is rare, but is more common with aggressive or recurrent tumors.

### Squamous Cell Carcinoma

While not as common as BCCa, SCCa is a common neoplasm. Chronic sun exposure seems to be even more important for the development of SCCa than for BCCa. The closer one lives to the equator, the smaller the ratio of BCCa to SCCa. In Michigan, the ratio is 8:1, while in Texas, the ratio is 2:1. SCCa usually presents as an erythematous, ulcerated, crusting lesion. The base of the lesion may be quite granular and friable, bleeding with minimal trauma. A marked inflammatory response in the adjacent tissues often causes a surrounding area of induration. The lesion may change slowly over time. When covered with a crust, removal may reveal a rolled margin. Lesions may present as a small chronic ulcer, particularly in an old scar, or may be noted as superficial multifocal change in sun damaged skin. The latter lesions may be difficult to diagnose, requiring multiple biopsies before the diagnosis is made. On occasion, SCCa may be vegetative with a cystic feel. These exophytic lesions frequently do not invade deeply. As with BCCa, SCCa may be pigmented. They are often confused with keratoacanthoma, but the growth of SCCa is slower. Of course when suspicion is high, biopsy is warranted.

Some SCCas arise in an area of actinic change, while others arise de novo. Those arising in sun-exposed areas tend to behave less aggressively than those arising de novo. All SCCa tumors have the potential for invasion and metastasis, but those arising de novo do so more often than those arising in areas of actinic change. Clinically, the skin around the tumor can be examined for signs of solar change, while histologically, actinic change can be seen in the skin surrounding the lesion.

Squamous cell carcinoma has much higher rate of metastasis than BCCa. The rate of lymphatic spread is highly variable between investigators. Lesions arising in areas of actinic change have a likelihood of metastasis in the range of 3% to 5%. It has been estimated that at least 8% of lesions arising de novo develop regional metastasis. Lesions arising in areas of scar or chronic inflammation have a metastatic rate between 10% and 30%. The rate of metastasis rises as the tumor invades deeper, with lesions penetrating to Clark’s levels IV and V having a 20% regional metastatic rate. Tumors with a higher histologic grade (i.e. poorly differentiated tumors with marked anaplasia) also demonstrate a greater risk of lymphatic spread. While SCCa arising in the skin is known to have metastatic potential, it is far less than SCCa arising on a mucosal surface. For this reason, SCCa of the lip is left for a separate discussion. SCCa may also spread via perineural invasion. The midface, lips and skin over the mandibular branch of the fifth cranial nerve seem to be especially at risk. Perineural invasion may lead to central nervous system involvement.

The histology of SCCa reveals masses of epidermal cells proliferating downward into the dermis. These masses may be well differentiated, easily recognized cells, or may be atypical or anaplastic. More differentiated tumors usually show evidence of keratin production such as keratin pearls. Broders’
classification uses a grading scale from 1 to 4, with grade 1 being well differentiated and grade 4 poorly differentiated. The diagnosis of poorly differentiated tumors may rely on immunohistochemical stains. Cytokeratin stains are usually positive in SCCa. Vimentin may be present in poorly differentiated tumors, while markers of keratinocyte differentiation such as involucrin and filagrin are present in well-differentiated tumors but less often in poorly differentiated lesions.

Several histopathologic types of SCCa have been described. These include generic, adenoid, bowenoid, verrucous and spindle-pleomorphic types. The generic type arises in an area of actinic change. The adenoid type demonstrates pseudoglandular arrangement with tubular or alveolar formations resulting from dyskeratosis and acantholysis. The lumina are filled with layers of epithelium and desquamated cells. These tumors have the classic nodular, ulcerative appearance, often arising in skin of the periauricular region. In the bowenoid type, invasive SCCa arises in an area with co-existent Bowen’s disease.

Verrucous SCCa is rarely seen in the skin of the head and neck but is more common in the mucosa of the oral cavity and larynx. It presents as a whitish, cauliflower-like lesion. Histologically, the tumor is well differentiated with marked hyperkeratosis, parakeratosis and acanthosis. Invasion is often with blunt, pseudopod-like growth.

The last histologic type is the spindle or pleomorphic type. This type is the least common. Histologically there is little evidence of differentiation. The cells are quite anaplastic and keratinization is usually lacking. The spindle-shaped cells are intermingled with collagen, arranged in whorls and may have pleomorphic giant cells.

**Staging**

The American Joint Committee on Cancer has designed a staging system based on the TNM classification for BCCa and SCCa of the skin similar to staging systems for cancers of other sites. Clinical staging is based upon physical examination and palpation of the lesion and regional lymph nodes. Pathological staging depends on complete resection of the entire site and confirmation of any lymph node involvement. The system excludes lesions of the eyelid, vulva and penis.

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Treatment

There are multiple treatment options for the management of BCCa and SCCa; many of these are used primarily by the dermatologist. An example of such a method is electrodessication and curettage (ED&C). It is most useful in small, less than 2 cm, basal cell cancers. It may be used with SCCa, but the lesions should be carefully selected. The cure rates range from 92% to 98% in experienced hands. The rationale behind its application is that BCCa and SCCa have a soft feel that allows them to be differentiated from the surrounding normal tissue. Various sizes of curettes are used to remove the palpable tumor in its entirety. After normal-feeling tissue is reached on all margins of the excision, electrodessication of the resulting wound is performed. The process may be repeated several times. The wound is then allowed to heal by secondary intention.

The advantages of ED&C are that it is quick and relatively easy to perform as an in-office, outpatient procedure. It allows for the maximal sparing of normal tissue with high cure rates. The disadvantages include the care of an open wound that may heal with depressed or hypertrophic scarring. Delayed bleeding is also a concern. As stated previously, proper technique with carefully selected lesions can produce high cure rates. Contraindications for this treatment modality include deeply invasive lesions, morphea-like and sclerotic BCCa and recurrent tumors.

Cryotherapy is another method of treatment used primarily in BCCa. Again the treating physician should be experienced in the procedure for maximal efficacy. The most common cryogen is liquid nitrogen administered either by spray or the use of cryoprobes. Temperatures of -30°F to -50°F are considered to be lethal to cutaneous malignant tissue. The tumor and an area of surrounding tissue are frozen to assure adequate ablation. Temperature probes inserted at the margin of the lesion are sometimes used to insure that sufficiently low temperatures are attained. The tissue is allowed to thaw and the freeze-thaw cycle is repeated.

The indications, advantages and success of cryosurgery are similar to those of ED&C. The technique is quite advantageous for lesions overlying cartilage, as it is resistant to necrosis. The disadvantages include a prolonged healing time of 3 to 10 weeks during which time the wound may become markedly edematous. Hypopigmentation of the resultant scar may also occur. Its use is generally limited to well-circumscribed lesions. The technique is contraindicated in morphea-like or recurrent tumors, as there is no method for precise control of the margins.

Most head and neck surgeons have the majority of their experience with surgical excision of cutaneous malignancies. The treatment modality may be successfully applied to both BCCa and SCCa. The success rate is in the range of 93% to 95%. A key advantage is the ability to attain tissue for histological evaluation. Frozen section evaluation of the margins can help to insure complete excision while preserving maximal normal tissue. Defects may be reconstructed by primary closure for amenable wounds, skin grafting, healing by secondary intention and locoregional flap reconstruction. Cosmesis is generally excellent, particularly for defects that can be closed primarily. The disadvantages include the fact that it may be more time-consuming and expensive than alternative techniques. Many surgeons feel that these minor disadvantages are outweighed by the ability to confirm the diagnosis histologically and assure the adequacy of the margins. Morphea-like lesions or those with ill-defined borders are perhaps better treated with Mohs’ surgery as the margins of the tumor may be difficult to discern without microscopic examination, and multiple excisions may be required. It is generally felt that with small, well-
circumscribed tumors, margins of 3 to 5 mm are adequate while large tumors, those with poorly defined borders or recurrent tumors should have a wider excision.

The carbon dioxide laser has been used with some success in the excision of cutaneous tumors from patients with cardiac disease or other medical illnesses that place them at high risk for the use of epinephrine. Laser excision allows bloodless excision without the need for vasoconstrictive agents. Laser vaporization is also useful in the case of multiple small lesions that do not require reconstruction. Lesions up to 7-8 mm may be resected and left with a physiologic dressing to heal with excellent cosmetic results. Laser excision has also been applied for the palliation of multiple lesions in elderly debilitated patients or those with more threatening medical conditions where cure is either unrealistic or impossible.

Mohs’ surgery was the subject of a recent Grand Rounds presentation and will be mentioned here only for completeness. The reader is referred to that presentation for thorough discussion of the history and technique. The concept was introduced in 1930 with initial published results in 1941. In recent years it has come to be the treatment method of choice for the cure of high risk BCCa and SCCa of the skin. Using a rapid fixation or fresh tissue technique, the excision specimen is mapped so that re-excision can be directed only to the precise area where tumor remains. The cure rates have proven to be high, 96% to 99%. The advantages of the technique lie in its ability to precisely locate any residual tumor and remove additional tissue only where necessary. This allows for the greatest assurance of complete tumor removal while preserving all uninvolved tissue. Using the fresh tissue technique, resulting defects may be immediately reconstructed. The most significant advantage is the high cure rate that can be attained in the management of advanced, high-risk or recurrent lesions. This includes larger lesions (0.6 cm to 1 cm) and those with a morpheaform growth pattern with ill-defined borders. The disadvantages include the special expertise required to perform the procedure, time and expense.

Radiation therapy (XRT) has been used in the past to treat cutaneous malignancy with some success. It has fallen out of favor for several reasons. It requires a prolonged course of treatment and radiodermatitis with the risk of carcinogenesis occur in the treated area. Serious complications including radiation chondritis and osteoradionecrosis also helped to limit its usefulness. XRT still finds application in the treatment of elderly patients or those with co-morbid conditions that make them poor surgical risks. Due to the potential for carcinogenesis, it is relatively contraindicated in young patients. Tissue for histological examination and control of the margins are unavailable, which makes it impossible to ensure the adequacy of treatment. In addition, recurrent tumors after XRT tend to be aggressive and difficult to manage. The recurrence rate after XRT ranges from 4.4% to 9.5% dependent upon the size of the primary lesion. Given the other options currently available for the treatment of BCCa and SCCa, XRT has a limited role in their management.

Photodynamic therapy (PDT) utilizes a photosensitizing drug that is selectively concentrated in tumors and that, after being activated by exposure to light, induces preferential tumor necrosis. Porphyrin has been the drug used most often in the head and neck. The argon ion dye pump laser has been the most commonly used light source. There has been a wide variation in the drug and light source used however, making the evaluation of the results difficult. It therefore remains an experimental treatment that may gain further acceptance in the future. Research is needed to determine the best treatment regimens and proper indications for its application.

Another investigational treatment modality is Interferon-α (IFN-α). Initial investigation has revealed excellent results in the treatment of nodular and superficial BCCa. IFN-α is administered intranasally at low doses three times a week. The exact mechanism of action is not clear, but it is believed to be due to the anti-proliferative and immunomodulating effects of IFN-α. It is known to cause an increased response to neoplastic tissues by stimulating macrophages and natural killer cells. Side effects include local erythema and pain. Some patients have a flu-like illness that is relatively mild and responsive to anti-pyretics. Leukopenia and thrombocytopenia may also complicate therapy.

Retinoids have had limited use in the treatment of cutaneous malignancy, most often in patients with basal cell nevus syndrome. Partial regression of BCCa has been noted using isotretinoin and etretinate.
Isotretinoin in high-doses has shown a preventative effect, but compliance is often compromised due to unacceptable side effects.

Chemotherapy has some usefulness for locally aggressive or metastatic disease. It may be combined with radiation therapy for palliation of extensive metastatic disease. Cis-platin, bleomycin, cyclophosphamide, 5-fluorouracil and vinblastine have all been studied. Cis-platin has been the most widely used and associated with the longest remissions. Cis-platin, doxorubicin and XRT can achieve palliation in widely disseminated or inoperable disease.

Treatment of the regional lymphatics is indicated in certain cases of SCCa and BCCa of the head and neck. This is more often the case with advanced tumors that invade deep tissues such muscle, bone, cartilage or nerve. Elective parotidectomy is indicated for the complete removal of deeply invasive tumors of the periauricular region or when any lymphadenectomy is to be performed for tumors arising superior to the mandible. Larger SCCa (>2 cm), recurrent tumors and those arising in scarred areas (Marjolin’s ulcer) all behave more aggressively and are more likely to require regional lymphadenectomy.

When dissection of the lymphatics is required, the nodal groups that must be removed are dependent on the location of the primary tumor. For tumors arising in the periauricular region, the preauricular, parotid and postauricular nodes are all at risk. The preauricular nodes receive drainage from the anterior parietal scalp, forehead, temple, ear, eyelid skin overlying the zygoma, and in some cases the nasal ala. The parotid nodes drain a similar area and may be located superficial or deep to the facial nerve. If both are involved, a total parotidectomy may be required to removal all nodes. The facial nerve is spared unless involved with tumor. The postauricular nodes are rarely involved, but must be remembered in dealing with tumors in the posterior parietal scalp, ear and mastoid.

The suboccipital nodes lie along the occipital artery and may be involved with tumors arising on the scalp posterior to an imaginary line drawn through the external auditory canal. The external jugular node lies superficial to the sternocleidomastoid adjacent to the greater auricular nerve and drains the lateral face, ear and upper neck. The facial artery nodal group lies along the nasolabial portion of the facial artery from the nose to the mandible and drains the lateral nose, upper lip, medial face and nasolabial fold. The submental nodes lie between the anterior bellies of the digastric and drain the lips, nasal vestibule and chin. The lymphatics of the posterior triangle of the neck (level V) may be involved with cancers of the neck, ear and scalp. The submandibular nodes (level I) drain the cheek, chin, nose and nasolabial regions. The anterior jugular nodes (levels II-IV) receive drainage from throughout the head and neck and are potentially involved in cancers arising in any location. The supraclavicular nodes located along the transverse cervical artery may be involved in cancers of the lower neck or upper chest.

The same principles of neck dissection for cancers of the upper aerodigestive tract apply in dissection for skin malignancy. This includes the sparing of uninvolved structures, but sacrificing those vessels or nerves that are involved with tumor. Post-operative XRT is indicated for multiple positive nodes, extracapsular spread and lymphovascular invasion.

**Prognosis and Recurrence**

The recurrence rate of primary BCCa varies from 1% to 39%. These rates vary depending on the histologic type. Nodular BCCa has a low recurrence rate in the range of 1% to 6%, while morpheaform tumors have much higher rate of 12% to 30%. Larger tumors arising in the midface, morpheaform or keratotic types and deep invasion all have higher recurrence rates. Some evidence suggests that women and younger patients have a higher rate of recurrence suggesting a reluctance to obtain wide margins in favor of cosmesis rather than adhere to sound oncologic principle. The majority of tumors will recur within 3 years of initial treatment, and the risk of subsequent recurrences increases after initial treatment failure.

The risk of recurrence for SCCa varies widely dependent on investigator. Seventy percent of patients who develop recurrences do so within 2 years of initial treatment and 95% do so within 5 years. Tumors located on the midface, size over 2 cm and perineural invasion all carry a higher rate of recurrence. Perineural invasion carries a much higher risk of recurrence (47.2%) and metastasis (34.8%). Treatment of
such neurotrophic SCCa with Mohs’ micrographic surgery and XRT dropped the rate of recurrence to 0% with a metastatic rate of 5.9% in one study.

Weber reports a higher risk of recurrence with aggressive skin cancers that are greater than 2 cm in greatest diameter, invade muscle, cartilage, bone, or nerves, or have regional lymphatic metastasis. Of the 134 patients in his study (45 with SCCa and 89 with BCCa), 70% with any one of these factors recurred, while only 20% of those patients without one of these features had a recurrence. Complete resection with frozen section confirmation of adequate margins successfully controlled 88 of 89 patients with BCCa. Of the 45 patients with SCCa, 14 had a recurrence.

The exact mortality from non-melanoma cutaneous malignancies is difficult to accurately determine. This is because they are not often reported to cancer registries within large centers in the U.S. One study revealed a death rate of 0.44 per 100,000 persons per year with the most deaths resulting from widespread SCCa arising in the periauricular region. Approximately 2,000 to 3,000 deaths per year in the United States can be attributed to BCCa and SCCa of the skin. Patients from 65 to 70 years of age seem to be particularly at risk. While mortality is not high when compared to other tumors, the impact on health care is significant given the prevalence of the disease and non-negligible recurrence rate.

**Conclusion**

Cutaneous malignancies are common disorders that are encountered by physicians in all medical specialties. There are multiple treatment options that offer a high success rate. Lesions that are advanced are more difficult to treat however. Thus early recognition, diagnosis and treatment offer the best chance for cure. Even more importantly, prevention through patient education regarding the harmful effects of ultraviolet radiation can prevent significant morbidity with its resultant socioeconomic and psychosocial impact. Perhaps further investigation will reveal mechanisms to reverse the damage inflicted in skin by radiation. As the head and neck is the most common location for these neoplasms, the otolaryngologist must be ever vigilant so that these lesions may be treated expeditiously with the greatest chance for cure.
References


