

Parotid Masses: Face the Facts

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Abstract

Parotid masses present both functional and aesthetic consequences due to the proximity of the facial nerve and the glands' alignment with the contours of the mandible. As the largest salivary glands, the parotids participate in providing moisture to the mouth through the production of saliva. This paper presents an overview of the anatomy and physiology of the parotid glands, signs and symptoms of parotid conditions, relevant diagnostic testing, medical and surgical treatment of parotid masses, postoperative recovery, and the role of the otorhinolaryngology (ORL) nurse in the continuum of care and patient education.

Anatomy and Physiology of the Parotid Glands

The parotid glands are located below the external auditory canals and descend anterior to the angle of the mandible between the skin of the cheek and the masseter muscle (Pick & Howden, 1974). Each parotid gland is divided into superficial and deep lobes by cranial nerve VII, the facial nerve (Bailey et al., 2001). The five branches of the facial nerve, the frontal, zygomatic, buccal, mandibular, and cervical branches, control the mobility and sensation of the face. The anatomical proximity of each gland to the facial nerve, and the obvious change in appearance when in a disordered state, create a reasonable concern for people affected by parotid gland conditions.

The parotid glands are the largest of the three major pairs of salivary glands found in and around the mouth; the parotid, submandibular, and sublingual glands. There are 600 to 1000 minor salivary glands, which are microscopic in size and located beneath the lining of the lips, tongue, hard and soft palates, and inside the mouth, nose, sinuses, and larynx (American Cancer Society, 2004).

Saliva is secreted by these major and minor salivary glands and has five major functions: 1) lubrication and protection, 2) buffering and clearance, 3) maintenance of tooth integrity, 4) antibacterial activity, and, 5) taste and digestion (Cummings, et al., 2005). Saliva coats the structures of the mouth and throat to protect them against harmful enzymes and irritants, as well as providing lubrication for the important functions of speech, mastication, and swallowing. The slight alkalinity of saliva buffers ingested and plaque-produced acids to play a major role in the prevention of dental caries. Saliva also contains minerals such as calcium and fluoride to aid in the formation of tooth enamel. The antibacterial properties of saliva are due to its immunoglobulin components, IgA, and to a lesser extent, IgG and IgM, along with non-immunologic components, such as enzymes which destroy bacteria. The moistening action of saliva facilitates conversion

of solid food into an easily swallowed bolus, promotes the perception of taste on the surface of the tongue, and begins the digestive process with the enzymes it contains. Stensen's ducts, which open into the oral cavity adjacent to the maxillary second molars, are the drainage sites for saliva from the parotid glands (Bailey et al., 2001).

Disorders of the Parotid Glands

Gland Dysfunction

Problems can arise following an alteration in the physiology of the parotid glands and the saliva they produce. In general, this occurs with defective stimulation, obstruction, or changes in salivary composition (Cummings et al., 2005).

Sialadenosis, or sialosis, is bilateral parotid gland swelling that is not inflammatory or due to a neoplasm (Cummings et al., 2005). The main effect of sialadenosis is enlargement of the cells by up to three times their normal size, causing compression of the ducts. The cause of this cellular enlargement is unknown, but classified by the presumed etiologies: endocrine, as in diabetes mellitus, hypothyroidism, pregnancy, and others; dystrophic/metabolic, as in alcoholism, eating disorders, and malnutrition; and, neurogenic, due to the effects of medications altering the autonomic input to the gland. One-half of the cases of sialadenosis will regress with treatment and control of the associated conditions (Cummings et al., 2005).

Obstruction of Stenson's duct due to sialolithiasis, or stone formation, often presents in patients 50 to 80 years of age, and in men more commonly than in women (Cummings et al., 2005). The exact etiology of sialolithiasis is unknown; however, salivary stasis, ductal inflammation and ductal injury are important contributing factors. Salivary stasis alters the mucoid elements of saliva to create a gel, which provides a framework for the deposition of salts. The calculi form when crystalline material precipitates around organic elements, such as epithelial cells, bacteria, or bacterial debris (Cummings et al., 2005). Constriction of the duct

can be congenital, or acquired from trauma, chronic inflammation, tumor compression, scar formation, or previous head and neck radiotherapy. Obstruction typically occurs during meals when saliva starts to flow but cannot exit the gland properly, leading to parotid swelling and pain (AAOHNS, 2004). Ductal dilation and/or surgical stone removal can become necessary if symptoms do not resolve with conservative measures involving hydration, massage, and sialogogues (Harris & Huntoon, 2008).

Infectious Disorders

Acute parotitis presents as parotid pain and swelling with fever and purulent drainage from Stenson's duct. Parotitis is a consequence of obstructed flow from any cause, including a calculus, thickened secretions, inflammation, infection, or neoplasm (Cummings et al., 2005). Acute infection is caused by retrograde oral bacterial contamination of the duct. Bacteria can usually be flushed from the duct by high salivary flow rates, so a reduction in salivary flow as in dehydration enhances the risk for infection (Cummings et al., 2005). Treatment is aimed at relieving the obstruction and eradicating the causative microbes, using hydration, massage, sialogogues, such as sugarless sour candies, and antibiotics (Harris & Huntoon 2008). Systemic antibiotics usually treat acute bacterial parotitis effectively, so if symptoms do not improve after 48 to 72 hours of the treatment regimen, an abscess is likely to have formed (Cummings et al., 2005). The infection that forms an abscess requires needle aspiration, or possibly incision and drainage. Incision and drainage should open the infected gland parallel to the facial nerve branches so as to avoid damage. Following incision and drainage, drains are placed to wick the purulence, and antibiotic therapy is continued (Cummings et al., 2005).

The parotid glands are the salivary glands most frequently affected by chronic infection, and ductal obstruction due to sialolithiasis is the most common cause (Cummings et al., 2005). The elderly, the immunosuppressed, and patients with poor oral hygiene are at the highest risk. Recurrent and/or frequent episodes of infection often preclude surgical excision of the gland.

Mumps is the most common non-suppurative salivary gland infection, with 85% of cases occurring in children less than 15 years of age (Cummings et al., 2005). Mumps is a contagious viral infection of the parotid and other salivary glands, with an incubation period of 14 to 21 days, and lasting 10 days (Harris & Huntoon 2008). The illness begins with a gradual onset of general malaise, chills, and headache, then glandular swelling, fevers to 101 to 102 degrees Fahrenheit, and jaw pain with palpation or movement. The best treatment for mumps is prevention through immunization. Active infections are managed by hydration, analgesia, rest, and a soft diet. The most common complication of mumps is orchitis, or testicular inflammation in postpubertal males. Rare complications of mumps infection include sterility following bilateral orchitis, permanent hearing loss, meningitis, and encephalitis.

Inflammatory Disorders

Inflammation of the parotid gland can occur in autoimmune and granulomatous diseases. In autoimmune diseases, the body's own immune system attacks the salivary glands because it fails to recognize the moisture-producing glands as "self." This results in dry mouth and eyes, as in Sjögren's syndrome, which can be classified as primary or secondary (Bailey et al., 2001). Sjögren's syndrome is a slowly progressive and chronic condition which predominantly affects middle-aged women (Cummings et al., 2005). Care for primary Sjögren's syndrome is symptomatic, with frequent use of artificial saliva and tears, vigilant dental hygiene, elimination of fungal overgrowth, and enhancement of saliva production (Cummings et al., 2005). Stimulation of any residual functioning salivary gland can be achieved with sialogogues, such as sugarless chewing gum or candies. The most commonly used systemic sialogogue is the muscarinic-cholinergic agonist, pilocarpine (Cummings et al., 2005; Hodson et al., 2004). Treatment of secondary Sjögren's syndrome is aimed at the underlying autoimmune disease, such as rheumatoid arthritis, systemic lupus erythematosus, or thyroiditis.

Human immunodeficiency virus (HIV) is associated with several salivary conditions, most commonly affecting the parotid glands (Cummings et al., 2005). HIV-associated salivary gland disease (HIV-SGD) involves diffuse enlargement of the salivary glands and can occur as the initial manifestation or at any stage of the disease. The gland enlargement is due to uniform follicular hyperplasia, which correlates with the generalized lymphadenopathy usually associated with HIV. This hyperplasia can lead to obstruction of the salivary duct, reducing the output of the gland, and creating xerostomia that mimics Sjögren's syndrome. HIV-SGD is distinguished from Sjögren's syndrome by the absence of Sjögren's antibodies and the absence of the symptoms of Sjögren's syndrome in other organ systems, such as the lung, gastrointestinal tract, or kidneys. Treatment of HIV-SGD involves anti-viral medications along with good oral hygiene and use of sialogogues. Surgical excision of the parotids is controversial for these patients and is usually avoided by the diagnosis of HIV-SGD using radiological studies or fine needle aspiration (Cummings et al., 2005).

With granulomatous disease, granular tumors of the lymphoid and epithelioid cells within the salivary glands develop due to tuberculosis, sarcoidosis, cat scratch fever, or HIV (Harris & Huntoon 2008). There is an infiltration of inflammatory cells and necrotizing inflammation of glandular parenchyma into the blood vessels to create nodular masses (Cummings et al., 2005). Spontaneous drainage and fistula formation can occur. Cultures, skin tests, or bloods tests are performed to identify the underlying disorder, so the appropriate antibiotics can be selected for treatment.

Benign Neoplasms

Perhaps the most anxiety-producing condition of the parotid gland is the presence of a tumor. Fortunately, approximately 80% of parotid tumors are benign pleomorphic adenomas (Bailey et al., 2001). Their

presentation is that of a painless, mobile, and slow-growing mass, with 90% of the tumors located in the accessible superficial lobe. Also designated as benign mixed tumors, pleomorphic adenomas have a mixture of various types of tissue elements which can be poorly encapsulated. These features account for tumor recurrence when an adequate margin of normal gland is not taken with the tumor during surgical excision (Bailey et al., 2001).

Warthins tumors represent the second most prevalent (6 to 10%) benign parotid tumor (Bailey et al., 2001). Warthins tumors usually present as a slow growing mass in the lower portion of the parotid and are most likely to affect caucasian men 40 to 70 years of age. Smoking is a risk factor for Warthins tumors (Dutra, Battaglia, Kern, & Kies, 2005). Approximately 10% of Warthins tumors present bilaterally.

Malignant Neoplasms

Malignant parotid tumors have been linked with several lifestyle and environmental factors, such as sunlight exposure, alcohol consumption, and hair dye chemicals (*Pain, swelling or facial paralysis, 2004*). There is also an increased risk with HIV, herpes, breast cancer (Harris & Huntoon 2008), and prior head and neck radiation therapy (Cummings et al., 2005). In general, signs of a malignancy in the parotid gland include a rapid growth rate, pain, facial nerve involvement, and cervical adenopathy (Dutra, Buttaglia, Kern, & Kies, 2005). Only 6 to 29% of parotid gland malignancies present with pain, and 6 to 13% present with facial paralysis (Cummings et al., 2005), so malignant and benign salivary tumors can be indistinguishable.

The most common primary parotid gland malignancy, accounting for 29 to 43% of all parotid gland malignancies, is mucoepidermoid carcinoma (Cummings et al., 2005). Mucoepidermoid carcinoma is the most common malignant tumor in children and adolescents younger than 20 years of age (American Cancer Society, 2004). A histologic grade of low-, intermediate-, or high-grade is applied to mucoepidermoid carcinoma (Cummings et al., 2005). Low-grade tumors have a higher percentage of mucinous cells and a better prognosis, while high-grade tumors have more epithelial cells and a worse prognosis. The five-year survival rates for low-, intermediate-, and high-grade tumors are 95%, 72%, and 0%, respectively (Cummings et al., 2005).

In most cases of mucoepidermoid tumors, the histologic type defines the grade and helps to predict the biological behavior. Low-grade mucoepidermoid tumors are usually small, partially encapsulated, and resemble benign neoplasms. High-grade tumors are relentless and metastasize easily, often resembling squamous cell carcinoma. The usual course of therapy for high-grade mucoepidermoid tumors is aggressive treatment, including excision of the tumor with local lymph nodes and sacrifice of the facial nerve if involved, followed by radiation therapy (Bailey et al., 2001).

Adenoid cystic carcinoma is the second most common parotid malignancy (Cummings et al., 2005).

Adenoid cystic carcinoma has a slow and relentless course with frequent perineural invasion and potential distant extension along the perineural lymphatic vessels. Regional and/or distant metastasis, usually to the lungs, develops in 40% of those affected (American Cancer Society, 2004). Treatment includes excision, along with possible lymph node dissection and radiation therapy. Adenoid cystic carcinoma has three distinct histologic patterns, cribriform, tubular, and solid, which correlate with prognosis and behavior. Although these types can coexist in the tumor, the predominance of a cribriform pattern has the best prognosis, while a solid pattern has a poor prognosis. The tubular histology has an intermediate prognosis (Cummings et al., 2005).

Adenocarcinoma, usually of a high-grade, accounts for 15 to 20% of all salivary gland carcinomas (Cummings et al., 2005). The parotid gland is the second most common site for adenocarcinoma after the minor salivary glands (Bailey et al., 2001). Parotid adenocarcinoma tumors have a wide range of biologic behaviors, but the aggressive type, often referred to as "not otherwise specified," is estimated to account for 9% of salivary malignancies (Cummings et al., 2005). As in all high-grade tumors, the treatment involves excision of the tumor and any involved lymph nodes, followed by radiation therapy to guard against metastasis.

Acinic cell carcinoma is a rare salivary tumor (6 to 8%), usually found in the parotid gland (Cummings et al., 2005). Women in their fifties are the most commonly affected (Bailey et al., 2001). Treatment includes wide surgical excision of the parotid gland along with any involved lymph nodes. Survival rates for acinic cell carcinoma are the best of any salivary malignancy. Occasionally, the mass grows quickly, involves the nerve, or produces pain. Patients with this subtype of acinic cell have a poor prognosis and should be treated with postoperative radiation (Cummings et al., 2005).

Squamous cell carcinoma is rarely a primary tumor of the parotid gland, but is the most common type of parotid gland cancer due to metastasis from squamous carcinomas of the skin, usually originating from the forehead, temple, or ear (Cummings et al., 2005). Surgical resection, along with regional lymphadenectomy and subsequent radiation therapy, are the recommended treatments for primary and secondary squamous cell carcinoma (Cummings et al., 2005). Primary squamous cell carcinoma of the parotid has a five-year survival rate of 24 to 50%.

Lymphomas can occur in salivary glands as a primary disease, and more commonly occur in the parotid gland than in the submandibular gland (Bailey et al., 2001). Only 1 to 2% of all salivary neoplasms are lymphomas; however, patients with Sjögren's syndrome have a forty-times greater risk of developing primary lymphoma than does the normal population (Cummings et al., 2005). Treatment involves chemotherapy and/or radiation. The prognosis for a primary salivary lymphoma is better than that of a systemic lymphoma, with a five-year

survival of 90% for the Hodgkin's type versus 43% for non-Hodgkin's lymphomas (Cummings et al., 2005).

The American Joint Committee on Cancer staging system, with its T (primary tumor), N (regional lymph nodes), and M (distant metastasis) tumor descriptors, is commonly used for major salivary gland cancers (Bailey et al., 2001; Cummings et al., 2005). The prognosis for parotid gland cancer is based on the clinical stage at presentation, so smaller and localized lesions have a better prognosis. Data collected from the Surveillance, Epidemiology, and End Results (SEER) database of the National Cancer Institute from 1981 to 1995 explain the differences in survival rates for major salivary gland cancers (Dutra et al., 2005). Survival at five years was 75% for stage I, 59% for stage II, 57% for stage III, and 28% for stage IV.

Tumor Transformation and Mixed Tumors

Pleomorphic adenomas, and benign mixed neoplasms, can transform into carcinomas. These transformed tumors represent 2 to 5% of salivary malignancies (Bailey et al., 2005). The probability of such a transformation is low, with only 1.5% transforming in less than 5 years, and 9.5% transforming after 15 years (Cummings et al., 2005). Even less common than the occurrence of tumor transformation is the diagnosis of malignant mixed tumors. Although less likely to occur than other forms of cancer, both transformed and malignant mixed tumors are aggressive and have high rates of nodal and distant metastasis. The prognosis is poor despite aggressive surgical resection and adjuvant radiation therapy.

Diagnostic Procedures for Parotid Neoplasms

Preoperative evaluation can be useful in identifying the tumor characteristics through the use of ultrasound, magnetic resonance imaging (MRI), computed tomography (CT) scan, or fine needle aspiration (FNA). Modern ultrasonography has proven to be useful and reliable in the differential diagnosis of tumors in the preauricular area, submandibular area, and cheek (Bialek, Jakubowski, & Karpinska, 2003). Ultrasound has been especially helpful in guiding FNA (Cummings et al., 2005).

There are conflicting preferences for imaging to evaluate parotid neoplasms (Cummings et al., 2005). MRI provides excellent soft tissue definition and does not require contrast for sufficient detail, but does not clearly delineate the borders of bone and soft tissue. Intravenous contrast-enhanced CT provides definition of tumor volume and its relationship to lymphatic, vascular, and bony structures. The usefulness of positron emission tomography (PET) has yet to be clearly defined for salivary gland malignancies (Cummings et al., 2005). Currently, the main role for PET scanning is in tumor staging, evaluation for distant and regional metastases, and follow-up assessment for patients who have recurrence.

Tumor sampling is the mainstay in diagnosis and treatment planning for parotid tumors (Cummings et al., 2005). Fine needle aspiration is the procedure used to harvest cells from the mass for microscopic analysis. A fine-gauge needle attached to a syringe directly punctures the mass to withdraw cells, which are placed onto a microscope slide to be evaluated by a cytopathologist. The advantages of FNA over more invasive surgical procedures for diagnosis include minimal pain and infrequent complications such as bleeding, bruising, and infection (*Fine needle aspiration*, 2006). The accuracy of FNA ranges from 54 to 98% (Cummings et al., 2005). An estimated 90% of patients who have parotid tumors will consent to the next course of action, surgical excision with frozen tissue samples (Fee & Tran, 2003).

Treatment of Parotid Tumors

Bailey and colleagues (2001) discuss the treatment for parotid tumors as a function of the extent of the mass. Superficial parotidectomy is the treatment of choice for both benign and malignant tumors in the superficial lobe. A total parotidectomy with preservation of the facial nerve is recommended for benign and malignant tumors involving the deep lobe or both lobes, providing the tumor does not involve the facial nerve. Sacrifice of the facial nerve is necessary if there is malignancy directly infiltrating the nerve. Repair of a separated nerve or grafting of the resected nerve ends can be performed microscopically with fine nylon suture. A mandibulotomy affords access to the deep lobe extending into the parapharyngeal space if necessary (Dutra et al., 2005).

Inspection of regional lymph nodes is recommended, with biopsy of any suspicious nodes, and subsequent modified neck dissection for histologically positive nodes. Levels II and III lymph nodes are most commonly involved (Gold & Annino, 2005). Level II lymph nodes are related to the upper one-third of the jugular vein, extending from the base of the skull to the inferior border of the hyoid bone. Level III lymph nodes include the middle region of the jugular vein located between the hyoid superiorly and a horizontal plane defined by the inferior border of the cricoid cartilage (Lalwani, 2004).

Radiation therapy is indicated for malignancies that: 1) are high-grade; 2) have positive or close surgical margins; 3) involve the deep lobe; 4) are recurrent; 5) invade the skin, bone, nerve, extra-parotid tissue, or regional lymph nodes; and 6) are not completely resectable (Dutra et al., 2005). Adjuvant radiation therapy is usually administered four to six weeks after surgery to allow time for adequate healing and to decrease the risk of wound breakdown. There is no consistent data to support the use of adjuvant chemotherapy (Dutra et al., 2005).

Intraoperative Nursing Care

ORL nurses must understand the relevant anatomy and physiology, as well as the potential surgical risks, in

order to be most effective in preparing the patient and the environment for efficient and effective care. Patients who undergo superficial or total parotidectomy, with or without neck dissection, are placed in the supine position, with the operative side of the face upward (Rothrock, Smith, & McEwen, 2003). Following application of an antiseptic skin preparation to the face and neck, the incision for parotidectomy is made in the preauricular area, extending inferiorly to the attachment of the pinna lobule, then extending posteriorly to the mastoid tip, and sweeping anteroinferiorly in the neck (Bailey et al., 2001). Using a shoulder roll can maximize exposure of the surgical site through hyperextension of the neck.

Specialized equipment and supplies for the procedure (Table 1) are often surgeon-dependent. Because the length of surgery is from one to three hours, depending on the procedure(s) performed, a comfortable body alignment with protection of bony prominences and neurologically sensitive spots is routine, along with the use of sequential compression stockings to prevent deep vein thrombosis by returning blood from the legs to the heart (Rothrock, 1999). A urinary catheter can assist in fluid management if requested by the surgeon and anesthesia provider (Vaiden, Rothrock, & Fox, 2000).

Table 1: Specialized equipment and supplies needed for parotidectomy
Antiseptic skin prep
Shoulder roll
Facial nerve monitor
15 or 19 French fluted drains with reservoir
Urinary catheter
Nerve dissector
Dressing material
Antibiotic ointment

Postoperative Nursing and Medical Care following Parotidectomy

Surgical recovery can present many challenges for the patient. As with any outpatient procedure, care after the day of surgery becomes the duty of the patient and family unit. The ORL nurse can provide support and education to optimize the recovery. The information regarding care of the wound and drain helps to prevent infection and promote healing. Patients should be instructed about the signs and symptoms of infection, keeping the wound dry and clean for at least the initial 24 hours, and applying antibiotic ointment. Care of the drain, including emptying, reactivation, and monitoring the quality and quantity of drainage, should be reviewed. Pain management extends beyond the use of oral analgesics to include nursing interventions, such as head elevation and bracing.

Both superficial and total parotidectomy surgeries bear the risk of temporary or permanent facial nerve

damage, altering both motor and sensory function on the involved side. There is a greater risk for facial nerve damage during resection of parotid gland malignancies, with resulting dysfunction being especially notable in the initial weeks following surgery. A temporary paresis, evaluated within two weeks postoperatively, occurs in 17 to 65% of patients, but only 1 to 5% of patients have permanent paralysis persisting two years after surgery (Ellingson, Cohen, & Anderson, 2003).

After every parotid gland surgery, the mobility of the facial nerve should be evaluated. The most important branch of the facial nerve is the zygomatic branch, which controls the movement of the eyelids. Corneal injury can result from the dryness that occurs with an inability to close the eyelid and blink. If eyelid weakness is noted, administer moisture with artificial tears throughout the day and ointment followed by taping the eyelid shut during sleep (Harris & Huntoon 2008). During the acute postoperative phase, during which recovery has begun, patients with facial weakness can benefit from facial retraining through biofeedback, working in front of a mirror, and prescribed exercises (Brach & VanSwearingen, 1999). Long-term therapies for facial weakness include muscle transfers and gold weight placement into the upper eyelid to facilitate closure. The decreased sensory perception requires the use of protection for the face and ear. Recommendations to the patient should include the use of an electric razor for men, and avoidance or protection from skin exposure to extreme temperatures from the environment or from therapeutic application of hot or cold packs (*Parotidectomy*, n.d.).

A potential consequence of the surgery is gustatory sweating, or "Frey's syndrome," which should be explained to the patient preoperatively during the surgical consent process. Frey's syndrome involves perspiration, flushing, and a sensation of warmth over the preauricular and temporal areas occurring in response to eating and/or aromas (Bailey et al., 2001). The syndrome occurs as transected parasympathetic secretory fibers regenerate and follow the sympathetic nerve sheaths of denervated facial sweat glands. The sweat glands are then activated during mastication through cholinergic neurotransmission. Frey's syndrome is reported by 30 to 60% of patients following parotidectomy; however, only 10% of patients are bothered with the symptoms and request therapy (Bailey et al., 2001). Commonly used interventions include topical administration of glycopyrrolate or 20% aluminum chloride (deodorant) to inhibit the facial perspiration, botulinum toxin injection, or simply dabbing the area with a napkin (Cummings et al., 2005). A surgical technique to prevent gustatory sweating involves placing barrier tissue at the time of the parotidectomy to impede nerve regeneration. A surgical technique, transcanal neurectomy of the tympanic plexus, can provide relief from gustatory sweating that has had an unsatisfactory response to medical therapy (Cummings et al., 2005).

Sialocele or salivary fistula is a relatively common complication following parotidectomy (Bailey et al.,

2001). Fluid can drain through the wound or collect beneath the flap when a residual edge of the gland leaks saliva. Although this problem is usually self-limiting, use of a pressure dressing to overcome the secretory pressure can be used, with or without needle aspiration of the fluid.

Several factors can contribute to an alteration of self-image for patients with parotid masses and following their resection. Identity is correlated with one's face, so distortions of the face due to a tumor, the indentation created by its removal, asymmetry of facial movement, or presence of sweat on the skin can impact the self-concept. Education and social support are strategies for coping with these issues. Knowledge of the parotid gland condition and its management, strategies to control the gustatory sweating and facial paresis, and self-care therapies for facial paralysis can empower patients to improve their body image. An important resource for those diagnosed with cancer of the parotid gland can be found through their peers in the Support for People with Oral and Head and Neck Cancer organization (www.spohnc.org). "Let's Face It" is an international support network for people with facial disfigurement, their families, and friends (www.faceit.org). Both organizations value education and offer a newsletter.

Conclusions

The parotid gland and surrounding structures have important functions. Moisture, mastication, taste, facial sensation, and facial movement are all related to the parotid gland. Education about the anatomical and physiological features of the parotid gland can facilitate understanding and management of parotid gland conditions. ORL nurses have the responsibility and privilege to empower their patients through their care, compassion, and teaching.

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