

Oral Cancer

Chapter 1 Oral Cancer Facts and Figures

Knowledge About Oral Cancer

For the most part, oral cancers are curable when diagnosed and treated early. Yet the survival rate for these cancers in the United States is one of the lowest for all cancers (52%) and has changed little in nearly four decades.

Results from two national studies—the 1990 and 1992 National Health Interview Surveys (NHIS)—suggest the U.S. adults are ill informed about signs and risk factors for oral cancers. For example, in one survey when asked, "What is one early sign of mouth cancer?" only 25 percent of the respondents correctly identified one early sign (red or white patch or lesion, or sore or lesion in mouth that does not heal) and 44 percent responded that they did not know of one sign of oral cancer. Further, while 83 percent of the respondents knew that heavy alcohol use definitely increases the chance of getting cirrhosis of the liver, few knew that heavy drinking also is a risk factor for throat cancer (18%) and mouth cancer (16%).

Moreover, one of these studies showed that only 15 percent of adults had ever had an oral cancer examination. Further, a recent national pilot survey among 219 U.S. general practice dentists found their level of knowledge about signs and symptoms of oral cancer and their practices to be uneven and less than desirable. Interestingly, although nearly all dentists agreed that patients 40 years of age and older should be given an annual oral cancer examination, 30 percent of them do not provide this exam to this age cohort during a patient's initial visit. Further studies have shown that practitioners are not assessing their patients' use of tobacco and alcohol.

Only 21 percent of responding adults had ever heard of a test or exam for oral or mouth cancer. However, nearly 28 percent of the respondents reported having had an oral cancer examination after it was described to them. Of these, nearly 20 percent had the exam in the past year, whereas over 5 percent reported having had the exam one to three years previously. Adults 40 to 64 years of age were more likely to have had an exam than were those in the younger age group. Whites and others were significantly more likely to have had an exam than those with less education. Adults who had a higher level of knowledge about risk factors for oral cancer were significantly more likely to have had an exam. Nonsmokers and those who smoked less than daily were significantly more likely to have had an exam than were those who smoked every day.

Females had a higher level of knowledge about risk factors than did males. Those with some college education were more likely to have a higher level of knowledge than were those with a high school education or less. Respondents who thought personal behaviors cause more cancers were significantly more likely to be knowledgeable about risk factors for oral cancer than were those who thought factors that they had little control over cause most cancers. Respondents who did not smoke cigarettes and those smoked less than daily were significantly more likely to have a higher level of knowledge than were those who smoked every day.

Source: Horowitz AM, et al. Maryland Adult's Knowledge of Oral Cancer and Having Oral Cancer Examination. *Journal of Public Health Dentistry*, Vol. 58, No. 4, Fall 1998.

What is Oral Cavity and Oropharyngeal Cancer?

Oral Cancer (i.e., cancer of the tongue, floor of the mouth, lip, palate, gingiva and alveolar mucosa, buccal mucosa, or oropharynx) is a relatively unknown, yet deadly disease. Approximately 2/3 occur in the oral cavity, and the remainder occurs in the oropharynx.

The oral cavity starts at the skin edge of the lips. It includes the lips, the *buccal mucosa* (inside lining of the lips and cheeks), the teeth, the gums, the front two-thirds of the tongue, the floor of the mouth below the tongue, the *hard palate* (bony roof of the mouth), and the *retromolar trigone* (area behind the wisdom teeth).

Oropharyngeal cancer develops in the *oropharynx* (the part of the throat just behind the mouth). The oropharynx begins where the oral cavity stops. It includes the *base of tongue* (back third of the tongue), the *soft palate*, the *tonsillar area* (tonsils and tonsillar pillars), and the *posterior pharyngeal wall* (back wall of the throat).

The oral cavity and oropharynx assist with breathing, talking, eating, chewing, and swallowing. *Minor salivary glands* located throughout the oral cavity and oropharynx make saliva that keeps the mouth moist and helps digest food.

The oral cavity and oropharynx contain several types of tissue and each of these tissues contains several types of cells. Different cancers can develop from each kind of cell. The differences are important, because they influence the patient's treatment options and outlook for recovery.

Many types of tumors can develop in the oral cavity and oropharynx. Some of these tumors are *benign*, or noncancerous. They do not invade other tissues and do not spread to other parts of the body. Others are cancerous, which means they can penetrate into surrounding tissues

and spread to other parts of the body. There are also some growths that start off harmless, but sometimes develop into cancer. These are known as *precancerous* conditions.

Benign oral cavity and oropharyngeal tumors

Benign tumors and tumor-like conditions include eosinophilic granuloma, fibroma, granular cell tumor, keratocanthoma, leiomyoma, osteochondroma, lipoma, schwannoma, neurofibroma, papilloma, condyloma acuminatum, verruciform xanthoma, pyogenic granuloma, and rhabdomyoma, as well as odontogenic tumors. The usual treatment for these conditions is surgical removal. Recurrence is very unlikely.

Leukoplakia, erythroplakia, and dysplasia

Leukoplakia and *erythroplakia* are terms that describe an abnormal area in the mouth or throat. Leukoplakia is a white area on the mucosa. Erythroplakia is a slightly raised, red area that bleeds easily, if scraped. The seriousness of leukoplakia or erythroplakia in each person can be accurately determined only by a *biopsy*, a sampling of tissue for examination under the microscope. These white or red areas may be a cancer, *dysplasia* (a precancerous condition) or some relatively harmless condition. There are mild, moderate, and severe forms of dysplasia.

These are based on how abnormal the tissue appears under the microscope and, in turn, help predict how likely the abnormality is to progress to cancer or to go away on its own or after treatment. Often dysplasia will go away if the factor that causes it is eliminated. The most frequent causes of these conditions are smoking or chewing tobacco. Poorly fitting dentures rubbing against the mucosa and irritating it also can cause leukoplakia or erythroplakia. Treatment with *retinoids* (drugs related to vitamin A) can help eliminate some areas of dysplasia or prevent others from forming.

Most of the time, leukoplakia is the result of a benign condition that is very unlikely to develop into cancer. About 5% of leukoplakias, however, are either cancerous when first found or are precancerous changes that progress to cancer within 10 years if not properly treated. Erythroplakia is usually more serious in as much as 51% of these non-specific red lesions are diagnosed as cancer at the time of initial biopsy.

Malignant oral cavity and oropharyngeal tumors

More than 90% of cancers of the oral cavity and oropharynx are *squamous cell carcinoma*, also called squamous cell cancer. Squamous cells are flat, scale-like cells that normally form the lining of the oral cavity and oropharynx. Squamous cell cancer begins as a collection of abnormal squamous cells. The earliest form of squamous cell cancer is called *carcinoma in situ*, meaning that the cancer cells are present only

in the lining layer of cells called the *epithelium*. *Invasive squamous cell cancer* means that the cancer cells have spread beyond this layer into deeper layers of the oral cavity or oropharynx.

Verrucous carcinoma is a type of squamous cell carcinoma that makes up less than 5% of all oral cavity tumors. It is a low-grade cancer that metastasizes rarely but can deeply spread into surrounding tissue. Therefore, surgical removal of the tumor with a wide margin of surrounding tissue is advised.

Minor salivary gland cancers can develop in the minor salivary glands that are found throughout the mucosal lining of the oral cavity and oropharynx. There are several types of minor salivary gland cancers, including adenoid cystic carcinoma, mucoepidermoid carcinoma, and polymorphous low-grade adenocarcinoma.

The tonsils and base of tongue contain *lymphoid* (immune system) tissue that can develop into a cancer. The treatment and *prognosis* (outlook for cure) for minor salivary gland cancers and lymphomas are different from that of squamous cell carcinoma and are not discussed in this document. The information contained in the rest of this document about oral cavity and oropharyngeal cancer refers only to squamous cell carcinoma.

What Are the Key Statistics About Oral Cavity and Oropharyngeal Oral Cancer Facts and Figures Cancer?

- The American Cancer Society estimates about 29,800 new cases (20,000 in men and 9,800 in women) of oral cavity and oropharyngeal cancer will be diagnosed in the United States during 1999.
- An estimated 8,100 people (5,400 men and 2,700 women) will die of oral cavity and oropharynx cancer in 1999. Death rates have been decreasing since the mid-1980s.
- Incidence and mortality rates are almost three times higher for men than women, with the incidence gender ratio having recently narrowed. An increase among younger patients has also been recognized. 95% of oral cancers occur in people over 40, with the average age of diagnosis being 60.
- Blacks have a higher incidence and mortality than whites. In the United States, oral cancer is the seventh most common cancer among males and the fourth most common cancer among African-American males, who in relation to the general population bear a disproportionate burden of this disease. One American dies from this disease every hour, more than from either cervical cancer or melanoma.

- 81% of oral cavity and oropharyngeal cancer patients survive at least one year after diagnosis.
- For all stages combined, the 5-year survival rate is 53% and the 10-year survival rate is 43%.

When patients newly diagnosed with oral and oropharynx cancers are carefully examined, about 15% will have another cancer in nearby areas such as the *larynx* (voice box), *esophagus* (the part of the digestive system between the throat and stomach), or lung. Another 10% to 40% will develop cancer of one of these organs or a second cancer of the oral cavity or oropharynx at a later time. For this reason, it is very important for patients with oral and oropharyngeal cancer to have follow-up examinations for the rest of their lives and avoid risk factors, like smoking and drinking, which increase the risk for these second cancers.

What Are the Risk Factors for Oral Cavity and Oropharyngeal Cancer?

A *risk factor* is anything that increases a person's chance of getting a disease such as cancer. Different cancers have different risk factors. For example, unprotected exposure to strong sunlight is a risk factor for skin cancer, and a diet high in fat and low in fiber is a risk factor for colorectal cancer. Scientists have found certain risk factors that make a person more likely to develop oral cavity and oropharyngeal cancer.

Some people with oral cavity or oropharyngeal cancer do not have any known risk factors, and others with several risk factors never develop the disease. Even if a patient does have one or more risk factors for oral cavity and oropharyngeal cancer, it is impossible to know for sure how much that risk factor contributed to causing the cancer.

Tobacco: 90% of patients with oral cavity and oropharyngeal cancer use tobacco, and the risk of developing these cancers increases with the amount smoked or chewed and duration of the habit.

Smokers are six times more likely than nonsmokers to develop these cancers. About 37% of patients who persist in smoking after apparent cure of their cancer will develop second cancers of oral cavity, oropharynx, or larynx, compared with only 6% of those who stop smoking. Tobacco smoke from cigarettes, cigars, or pipes can cause cancers anywhere in the oral cavity or oropharynx, as well as causing cancers of the larynx, lungs, esophagus, kidneys, bladder, and several other organs. In addition, pipe smoking has a particularly significant risk for cancers in the area of the lips that contact the pipestem.

Smokeless tobacco, ("snuff" or chewing tobacco) is associated with cancers of the cheek, *gingiva* (gums), and inner surface of the lips. Smokeless tobacco increases the risk of these cancers by about 50

times. Often cancer associated with smokeless tobacco will begin as leukoplakia or erythroplakia.

Alcohol: Alcohol consumption strongly increases the risk of oral cavity and oropharyngeal cancer. Approximately 75% to 80% of all patients with oral cancer frequently consume alcohol. These cancers are about six times more common in drinkers than in nondrinkers. People who smoke and also drink alcohol have a much higher risk of cancer than those using only alcohol or tobacco alone.

Ultraviolet light: More than 30% of patients with cancers of the lip have outdoor occupations associated with prolonged exposure to sunlight.

Irritation: *Chronic* (long-term) irritation to the lining of the mouth caused by poorly fitting dentures has been suggested as a risk factor for oral cancer. But multiple research studies have shown no difference between denture wearers and non-denture wearers in the occurrence of oral cancer. As poorly fitting dentures can tend to trap proven causative agents of oral cancer beneath them, such as alcohol and tobacco particulates, denture wearers should have their dentures evaluated by a dentist at least every 5 years for optimum fit. All denture wearers should remove their dentures at night and clean and rinse them thoroughly every day.

Vitamin deficiency: *Vitamin A deficiency* is associated with an increased risk of developing cancer of the oral cavity and oropharynx.

Plummer-Vinson syndrome: This rare combination of iron deficiency with abnormalities of the tongue, fingernails, esophagus, and red blood cells is associated with an increased risk of oral cancer. However, this syndrome is very rare and is responsible for only a very small number of oral cancers.

Mouthwash: Some studies have suggested that mouthwash with a high alcohol content is associated with an increase in oral and oropharynx cancer risk. More recent research has pointed out that smokers and people who often consume alcoholic drinks are more likely to also use mouthwash than people who neither smoke nor drink.

Human papillomavirus (HPV) infection: Papillomaviruses are a group of about 80 related viruses. Most HPV types cause warts on various parts of the body. A few HPV types are at least partly responsible for about 85% of cancers of the cervix. These HPV types are also associated with some cancers of the vagina and penis. The situation for oral cancers is less clear, however. The types of HPV found in cervical cancer are found in a much lower percentage of oral cancers, but are also found in over 10% of samples of normal oral tissue. The current view is that HPV may be a factor that, together with other influences, contributes to the development of some oral cavity and oropharyngeal cancers.

Immune system suppression: People taking immunosuppressive drugs to treat certain immune system diseases, or to prevent rejection of transplanted organs, may be at increased risk for cancers of the oral cavity and oropharynx.

Being male: Oral and oropharyngeal cancer is twice as common in men as in women. This is because men are more likely to use tobacco and alcohol; however, the rate at which women are developing oral cancer is increasing and the rate at which men are developing oral cancer is decreasing. This is because more women are starting to smoke and more men are quitting the habit.

Can Oral Cavity and Oropharyngeal Cancer Be Prevented?

Most oral cavity and oropharyngeal cancers can be prevented by avoiding risk factors whenever possible.

Tobacco and alcohol are the most important oral cavity and oropharyngeal cancer risk factors. The best way to avoid these cancers is to never start smoking or using smokeless tobacco. Limit your intake of alcoholic beverages, if you drink at all. Quitting tobacco and alcohol significantly lowers your risk of developing these cancers, even after many years of abuse.

Exposure to ultraviolet radiation is an important and very avoidable risk factor for cancer of the lips, as well as for skin cancers. If possible, avoid being outdoors during the middle of the day, when the sun's ultraviolet rays are strongest. Minimize exposure to ultraviolet rays by wearing a wide-brimmed hat and using sunscreen.

Avoiding sources of oral irritation (such as dentures that do not fit properly) may also decrease your risk for oral cancer.

Vitamin deficiencies have been related to oral cavity and oropharyngeal cancers. In general, eating a healthy diet is much better than adding vitamin supplements to an otherwise unhealthy diet. The American Cancer Society recommends choosing most foods from plant sources. Eat at least five servings of fruits and vegetables every day and six servings of other foods from plant sources such as breads, cereals, grain products, rice, pasta, or beans. Eat fewer high-fat foods such as those from animal sources.

Isotretinoin (13-cis-retinoic acid) is a drug chemically related to vitamin A. When used by patients with oral cavity or oropharynx cancer, isotretinoin may reduce the risk of developing a second cancer in the head and neck region; however, it has no effect on the recurrence rate of the first cancer. Because of isotretinoin's side effects (rashes, eye problems, and changes in blood cholesterol and fat levels), it is not recommended for widespread use by the general population. Vitamin A

supplements are not recommended, unless prescribed by a medical doctor for a specific health problem. High doses of vitamin A do not decrease cancer risk. In fact, some studies suggest that too much vitamin A can increase the risk of developing lung or prostate cancer.

Do We Know What Causes Oral Cavity and Oropharyngeal Cancer?

Doctors and scientists can't say for sure what causes each case of oral cavity and oropharyngeal cancer. But we do know what many of the risk factors are and how some of these risk factors cause cells to become cancerous. We know that tobacco and alcohol can damage cells in the lining of the oral cavity and oropharynx, and that cells in this layer must grow more rapidly to repair this damage. Many of the chemicals found in tobacco cause damage to DNA, which contains the cell's instructions for repair and growth. Scientists are not sure whether alcohol directly damages DNA, but they have shown that alcohol increases penetration of many DNA-damaging chemicals into cells. This is one reason why the combination of tobacco and alcohol causes far more damage to DNA than tobacco alone. This damage can cause certain areas of DNA (for example, those in charge of starting or stopping cell growth) to malfunction. Then, abnormal cells can begin to accumulate, forming a tumor. With additional damage, the cells may begin to invade (spread into neighboring tissue) and metastasize (spread to distant organs).

Can Oral Cavity and Oropharyngeal Cancer Be Found Early?

Many cancers of the oral cavity and oropharynx can be found early, during routine screening examinations by a doctor or dentist, or by self-examination. Some early cancers have symptoms that cause patients to seek medical or dental attention. Unfortunately, others may not cause symptoms until after reaching an advanced stage or may cause symptoms that appear to be due to a disease other than cancer, such as a toothache.

Regular dental checkups, that include an examination of the entire mouth, are important in the early detection of oral and oropharyngeal cancers and precancerous conditions. The American Cancer Society also recommends that primary care doctors examine the mouth and throat as part of a routine cancer-related checkup.

Many physicians and dentists recommend that people take an active role in the early detection of oral cavity and oropharyngeal cancer by doing monthly self-examinations. This means looking in a mirror to check for any of the findings included in the following list:

Signs and symptoms:

- a sore in the mouth that does not heal (most common symptom)
- a lump or thickening in the cheek
- a white or red patch on the gums, tongue, tonsil, or lining of the mouth

- ❑ a sore throat or a feeling that something is caught in the throat
- ❑ difficulty chewing or swallowing
- ❑ difficulty moving the jaw or tongue
- ❑ numbness of the tongue or other area of the mouth
- ❑ swelling of the jaw that causes dentures to fit poorly or become uncomfortable
- ❑ loosening of the teeth or pain around the teeth or jaw
- ❑ voice changes
- ❑ a lump or mass in the neck
- ❑ weight loss

Many of these signs and symptoms may be caused by other cancers or by less serious, benign problems. It is important to see a medical doctor or dentist if any of these conditions lasts more than two weeks. Remember, the sooner you receive a correct diagnosis, the sooner you can start treatment and the more effective your treatment will be.

Source: American Cancer Society Jan. 1999. Reprinted with permission.

Chapter 2 Detecting Oral Cancer

A Guide for Health Care Professionals

INCIDENCE AND SURVIVAL

Oral or pharyngeal cancer will be diagnosed in an estimated 30,000 Americans this year, and will cause approximately 8,000 deaths. On average, only half of those with the disease will survive more than five years.

THE IMPORTANCE OF EARLY DETECTION

Early Detection Saves Lives

With early detection and timely treatment, deaths from oral cancer could be dramatically reduced. The five-year survival rate for those with localized disease at diagnosis is 76 percent compared with only 19 percent for those whose cancer has spread to other parts of the body.

Early detection of oral cancer is often possible. Tissue changes in the mouth signal the beginnings of cancer often can be seen and felt easily.

WARNING SIGNS

Lesions That Might Signal Oral Cancer

Two lesions that could be precursors to cancer are leukoplakia (white lesions) and erythroplakis (red lesions). Although less common than leukoplakis, erythroplakis and lesions with erythroplakic components have a much greater potential for becoming cancerous. Any white or red lesion that does not resolve itself in two weeks should be reevaluated and considered for biopsy to obtain a definitive diagnosis.

Other Possible Signs/Symptoms of Oral Cancer

Possible signs/symptoms of oral cancer that your patients may report: a lump or thickening in the oral soft tissues, soreness or a feeling that something is caught in the throat, difficulty chewing or swallowing, ear pain, difficulty moving the jaw or tongue, hoarseness, numbness of the tongue or other areas of the mouth, or swelling of the jaw that causes dentures to fit poorly or become uncomfortable.

If the above problems persist for more than two weeks, a thorough clinical examination and laboratory tests, as necessary, should be performed to obtain a definitive diagnosis. If a diagnosis cannot be obtained, referral to the appropriate specialists is indicated.

RISK FACTORS

Tobacco/Alcohol Use

Tobacco and excessive alcohol use increase the risk of oral cancer. Using both tobacco and alcohol poses a much greater risk than using either substance alone.

Sunlight

Exposure to sunlight is a risk factor for lip cancer.

Age

Oral cancer is typically a disease of older people usually because of their longer exposure to risk factors. Incidence of oral cancer rises steadily with age, reaching a peak in persons aged 65-74. For African Americans, incidence peaks about 10 years earlier.

Gender

Oral cancer strikes men twice as often as it does women.

Race

Oral cancer occurs more frequently in African Americans than in whites.

WHAT YOU CAN DO

A thorough head and neck examination should be a routine part of each patient's dental visit. Clinicians should be particularly vigilant in checking those who use tobacco or excessive amounts of alcohol.

- Examine your patients using the head and neck examination described here.
- Take a History of their alcohol and tobacco use.
- Inform your patients of the association between tobacco use, alcohol use, and oral cancer.
- Follow-up to make sure a definitive diagnosis is obtained on any possible signs/ symptoms of oral cancer.

Note: We offer a slide program, *Detecting Oral Cancer*, consisting of 28 color slides that provide step-by-step instruction in performing an oral cancer examination and illustrate oral lesions that are suspicious for oral cancer. There's an accompanying text that introduces each slide. This program is designed as an educational tool for health care professionals.

# 6491	Slide Program: <i>Detecting Oral Cancer</i>	\$40.00
	S & H	5.50
	Total	\$45.50

Calif. residents add sales tax. Send a check to Homestead Schools, or call (800) 253-0088 for faster service.

THE EXAM

This exam is abstracted from the standardized oral examination method recommended by the World Health Organization. The method is consistent with those followed by the Centers for Disease Control and Prevention and the National Institutes of Health. It requires adequate

lighting, a dental mouth mirror, two 2 x 2 gauze squares, and gloves; it should take no longer than 5 minutes.

EXAM REVIEW

The examination is conducted with the patient seated. Any intraoral prostheses (dentures or partial dentures) are removed before starting the examination. The extraoral and perioral tissues are examined first, followed by the intraoral tissues.

Source:

National Institute of Dental and Craniofacial Research
National Institutes of Health
National Oral Health Information Clearinghouse
1 NOHIC Way
Bethesda, MD 20892-3500

I. THE EXTRAORAL EXAMINATION

- **FACE:** (*Figure 1*) The extraoral assessment includes an inspection of the face, head, and neck. The face, ears, and neck are observed, noting any asymmetry or changes on the skin such as crusts, fissuring, growths, and/or color change. The regional lymph node areas are bilaterally palpated to detect any enlarged nodes and, if detected, their mobility and consistency. A recommended order of examination includes the preauricular, submandibular, anterior cervical, posterior auricular, and posterior cervical regions.

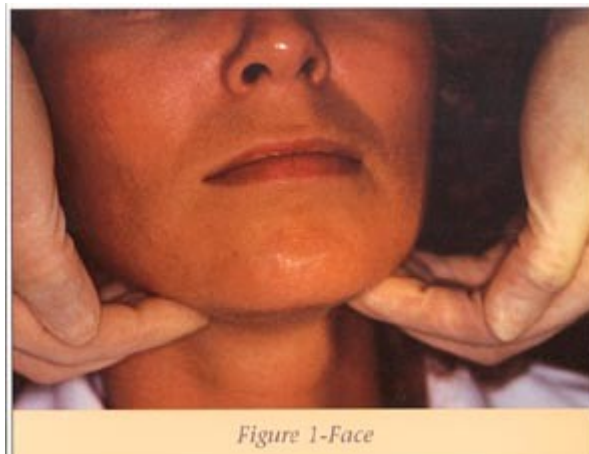
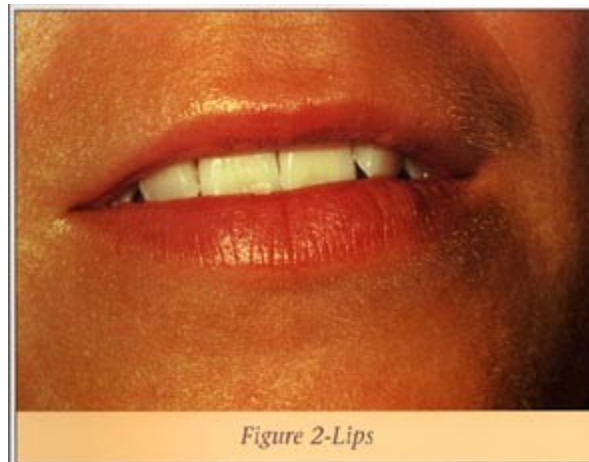


Figure 1-Face

II. PERIORAL AND INTRAORAL SOFT TISSUE EXAMINATION

The perioral and intraoral examination procedure follows a seven-step systematic assessment of the lips; labial mucosa and sulcus; commissures, buccal mucosa, and sulcus; gingiva and alveolar ridge; tongue; floor of the mouth; and hard and soft palate.

- **LIPS:** (*Figure 2*) Begin examination by observing the lips with the patient's mouth both closed and open. Note the color, texture and any surface abnormalities of the upper and lower vermilion borders.



- **LABIAL MUCOSA :** (*Figures 3 and 4*) With the patient's mouth partially open, visually examine the labial mucosa and sulcus of the maxillary vestibule and frenum and the mandibular vestibule. Observe the color, texture, and any swelling or other abnormalities of the vestibular mucosa and gingiva.

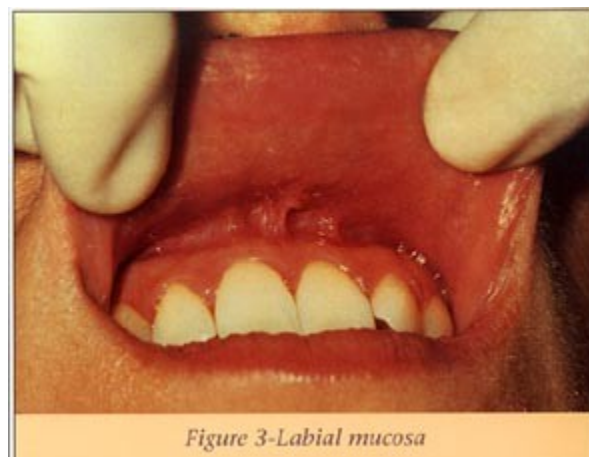




Figure 4-Labial mucosa

- **BUCCAL MUCOSA :** (*Figures 5 and 6*) Retract the buccal mucosa. Examine first the right then the left buccal mucosa extending from the labial commissure and back of the anterior tonsillar pillar. Note any change in pigmentation, color, texture, mobility and other abnormalities of the mucosa, making sure that the commissures are examined carefully and are not covered by the retractors during the retraction of the cheek.

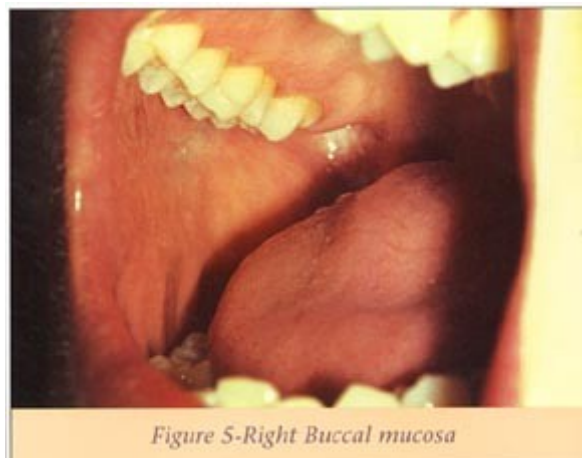


Figure 5-Right Buccal mucosa

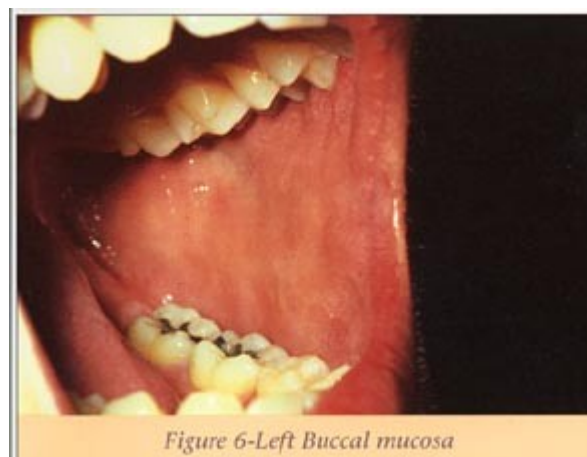
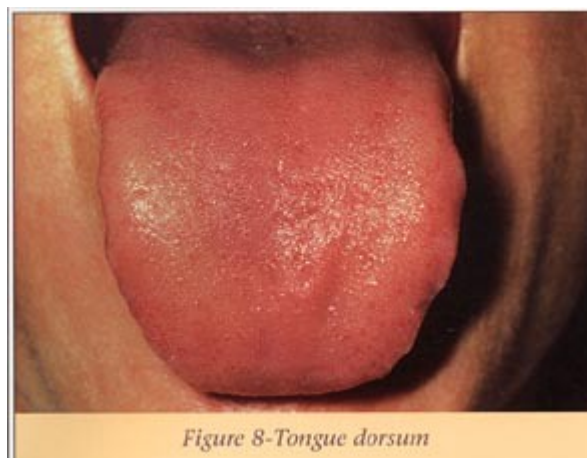


Figure 6-Left Buccal mucosa

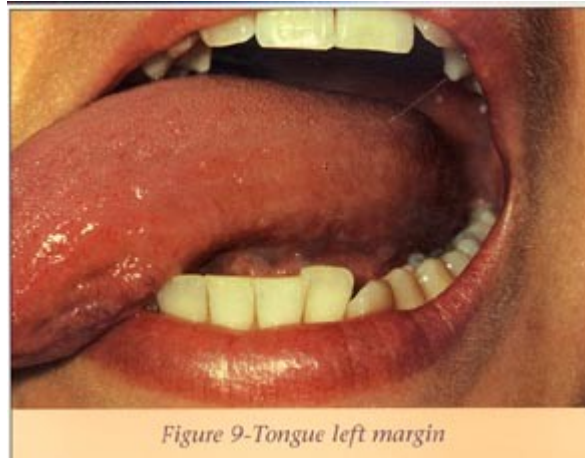
- **GINGIVA :** (*Figure 7*) First, examine the buccal and labial aspects of the gingiva and alveolar ridges (processes) by starting with the right maxillary posterior gingiva and alveolar ridge and then move around the arch to the left posterior area. Drop to the left mandibular posterior gingiva and alveolar ridge and move around the arch to the right posterior area.
- Second, examine the palatal and lingual aspects as had been done on the facial side, from right to left on the palatal (maxilla) and left to right on the lingual (mandible).



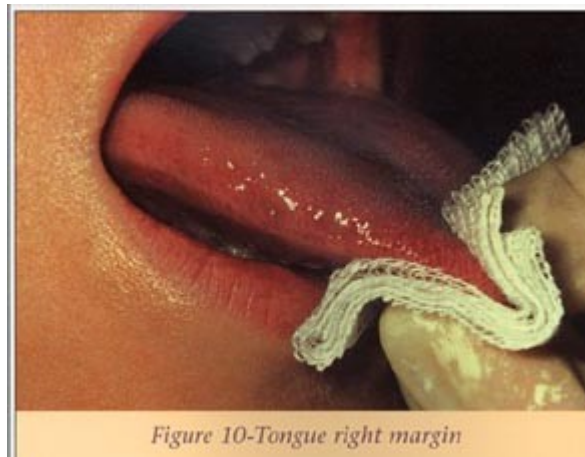
- **TONGUE:** (*Figure 8*) With the patient's tongue at rest, and mouth partially open, inspect the dorsum of the tongue for any swelling, ulceration, coating or variation in size, color, or texture. Also note any change in the pattern of the papillae covering the surface of the tongue and examine the tip of the tongue. The patient should then protrude the tongue, and the examiner should note any abnormality of mobility or positioning.



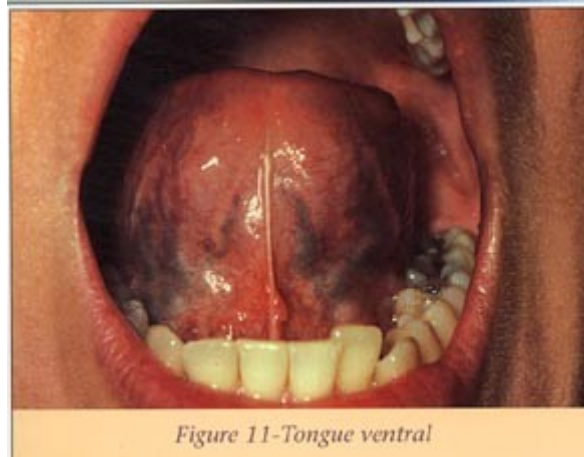
(Figure 9) With the aid of mouth mirrors, inspect the right and left lateral margins of the tongue.



(Figure 10) Grasping the tip of the tongue with a piece of gauze will assist full protrusion and will aid examination of the more posterior aspects of the tongue's lateral borders.



(Figure 11) Then examine the ventral surface. Palpate the tongue to detect growths.



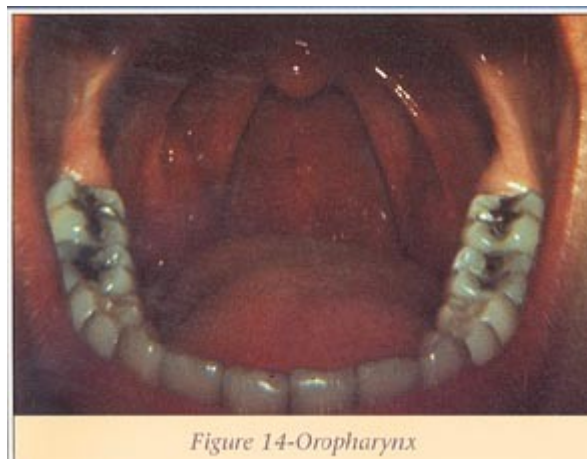
- **FLOOR:** (*Figure 12*) With the tongue still elevated, inspect the floor of the mouth for changes in color, texture, swellings, or other surface abnormalities.



- **PALATE:** (*Figures 13 and 14*) With the mouth wide open and the patient's head tilted back, gently depress the base of the tongue with a mouth mirror. First inspect the hard and then the soft palate.



(Figure 14) Examine all soft palate and oropharyngeal tissues.



(Figure 15) Bimanually palpate the floor of the mouth for any abnormalities. All mucosal or facial tissues that seem to be abnormal should be palpated.

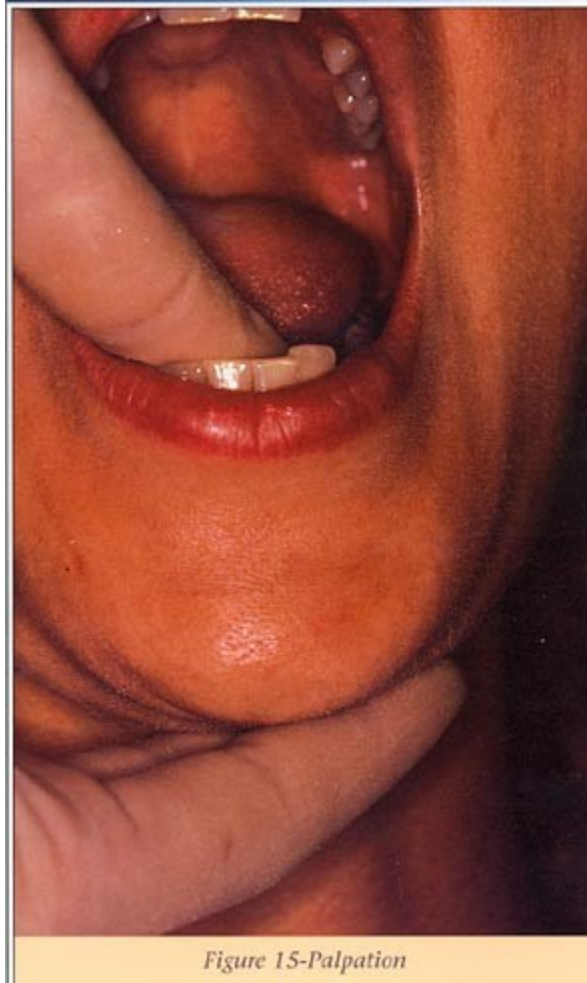
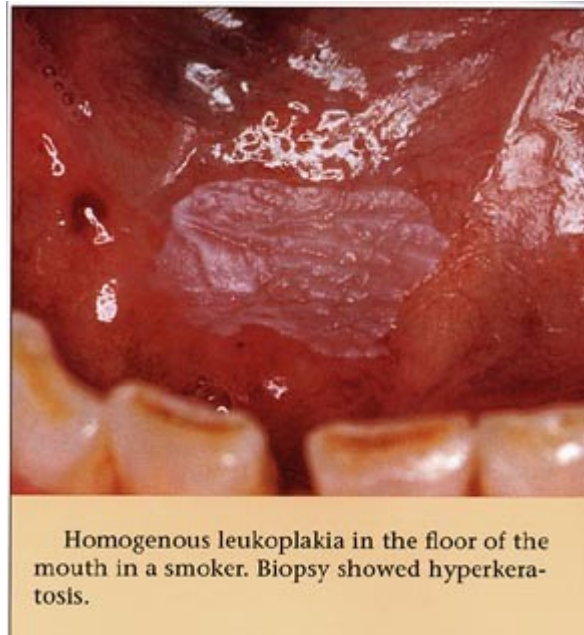


Figure 15-Palpation

ORAL LESIONS

suspicious for Oral Cancer



Homogenous Leukoplakia in the floor of the mouth in a smoker. Biopsy showed hyperkeratosis.

Clinically, a leukoplakia on left buccal mucosa. However, the biopsy showed early squamous cell carcinoma. The lesion is suspicious because of the presence of nodules.



Nodular leukoplakia in left commissure. Biopsy showed sever epithelial dysplasia.



Erythroleukoplakia in left commissure and buccal mucosa. Biopsy showed mild epithelial dysplasia and presence of candida infection. A 2-3 week course of anti-fungal treatment may turn this type of lesion into a homogenous leukoplakia.



Chapter 3 Providing Oral Cancer Examinations For Older Adults

by Janet A. Yellowitz, DMD, MPH

Abstract: Although cancer is not a part of the aging process, malignant neoplasms occur primarily in older adults. As the size of the elderly population increases, there will be many more older adults at risk for oral cancer. Many older adults do not seek dental care because they do not think they need it; and, therefore, they do not receive routine oral

examinations. Dental practitioners need to encourage older patients to seek dental care so they can receive oral cancer examinations.

Each year, close to 30,000 new cases of oral cancer are detected in the United States.¹ As a result of this disease, nearly 9,000 deaths occur, one every hour. Most oral cancers (90 percent) are squamous cell carcinomas, begin as surface lesions, and have a highly variable presentation during their early stages. There is hardly an oral lesion that at one stage or another does not assume the same overt appearance as oral squamous cell carcinoma _ hence the concept of oral cancer as "the great mimicker."² Detecting an oral lesion is primarily dependent upon the clinician having a high level of suspicion and providing a comprehensive oral cancer examination. For the purpose of this article, oral cancer will refer to oral squamous cell carcinoma.

Although cancer is not a part of the aging process, malignant neoplasms occur primarily in older adults. Fiftyfive percent of all cancers and 67 percent of cancer deaths occur in people age 65 and older.³

Older adults not only have a greater risk of developing cancer but are more frequently diagnosed with cancer in an advanced stage. Likewise, most oral cancers are diagnosed in a late stage, after having metastasized to the lymph nodes.

Similarly to other cancers, oral cancer is found disproportionately more often in older adults than in any other age segment. The average age at which oral cancer is diagnosed is 63, with the majority of those lesions found in those 40 years and older. The National Cancer Institutes' Surveillance, Epidemiology and End Results program found that close to half of all oral cancer cases were found in the 65andolder age group.

In another study of close to 1,000 oral cancer cases, 42 percent were in people 65 and older; 60 percent of those cases were in people age 65 to 74 and 40 percent in those 75 and older.⁵ For the total population, the incidence of oral cancer averages about 11 cases per 100,000, peaking at 49 cases per 100,000 people age 70 to 74.⁶ The incidence rate is 30 percent higher for blacks than for whites, peaking at ages 55 to 64. Assuming these rates remain stable, as the size of the elderly population increases, there will be many more older adults at risk for oral cancer.

Today, the average life expectancy is at an alltime high. On average, females born today will live 79 years and males 73 years. Those age 65 today can anticipate an additional 17.6 years of life (19 for females arid 15.8 for males). Between the years 2010 arid 2030, when the babyboom generation reaches 65, the older population will dramatically expand. By 2030, there will be about 70 million older people, more than twice the number in 1997.

Currently, about onethird of oral cancers are diagnosed in an early, localized stage. The fiveyear survival rate for those with regional involvement is 42 percent and, for those with distant rnetastasis, 17 percent .⁴ Despite advances in therapy, little improvement in survival rates for oral cancer has been seen during the past several decades.

Table 1: Population at High Risk for Oral Cancer

60+ years of age

History of tobacco use

History of alcohol use

Low level of education

Occupation of lower socioeconomic category

Retired or not covered by dental insurance

Edentulous or having many nonreplaced missing teeth

Does not use preventive health measures

Risk Factors

The primary risk factors for oral cancer are tobacco use, alcohol use (current and previous), and sunlight exposure (lip cancers). Tobacco and alcohol use have been implicated in close to 75 percent of all oral cancers in the United States. Together, smoking and alcohol have a multiplicative effect on the development of oral lesions.⁷ The time-dose relationship of carcinogens found in tobacco and tobacco smoke is an important factor in causing oral cancer. Cigar and pipe smoking are likely to provide a greater risk than cigarette smoking, and smokeless tobaccos have been implicated in the development of cancer of the gingival and buccal mucosa. In addition, individuals having a prior oral cancer are at highest risk for developing a second lesion.

There is also growing evidence identifying the human papilloma virus and *Candida albicans* in the development of oral carcinoma. Although denture irritation was once thought to be a cause, it is not a risk factor for oral cancer. From a positive perspective, diets with adequate amounts of iron and vitamins A, C, and E appear to have a protective role.^{8,9}

Although the majority of squamous cell carcinomas are associated with tobacco and/or alcohol use, not all patients with an oral cancer fit this pattern. In a recent five-year review of oral cancer patients treated in a metropolitan hospital, 20 percent reported no history of tobacco use, and 21 percent reported no history of alcohol use.¹⁰

One's risk of being diagnosed with an advanced oral cancer increases as one's utilization of dental services decreases. Oral cancer is often found in those least likely to seek routine oral care (Table 1).

Although most dental practices have policies to recall patients routinely, these policies apply primarily to dentate patients. In general, edentulous patients do not receive routine or preventive dental care. Often, patients wearing a complete set of dentures for many years have not seen a dentist since the dentures were delivered. Many older adults do not seek dental care because they do not think they need it. Hence, many edentulous elders do not receive routine oral examinations.

Early Lesions

Early oral cancers have numerous and variable clinical appearances. Early lesions can appear as subtle, asymptomatic red, red-and-white speckled, or white areas with subtle textural changes. Early lesions can appear as an area of induration or ulceration; can appear as a result of physical, chemical, or thermal trauma; or may resemble lichen planus.

Although oral cancer can occur anywhere in the mouth, most often it is found in cancer-prone sites — the ventral and lateral borders of the tongue, anterior floor of the mouth, and soft palate complex.

Oral Mucosa of Older Adults

The oral mucosa of older adults is often described as atrophic, thin, pale, and friable, with a decrease in capillary blood flow. Although many of these characteristics are found in an older population, these changes are not universal.

Age-related changes of the oral mucosa have not been well-documented or have little scientific data to support their claims. Many of the changes associated with aging were a likely result of systemic disease, poor nutrition, or medications. Aging of the oral mucosa is perhaps best described as a "post-maturational deteriorative change that, with time, leads to all increased vulnerability to challenges."¹¹

The rate of biological aging differs both within an individual and among individuals, presenting great variability in one's tissues, including the oral mucosa. In general, muscle mass is less dense and varicosities are more frequently found in older adults. Differentiating a soft tissue change as being a result of the environment or due to intrinsic aging is often not possible. Without clear criteria, distinguishing between

agerelated changes and potentially malignant changes is more difficult in older adults than in younger ones and requires the clinician to have a higher degree of suspiciousness when completing an oral cancer examination.

Practitioner Challenges

Two conditions increase the difficulty of diagnosing early lesions, the stage at which the patient has the best prognosis. First, the tissue changes common to early lesions are subtle; and, second, patients with early lesions rarely present with symptoms. Once the patient becomes symptomatic, most lesions are easily diagnosed.

Delay in Diagnosis

Oral cancer has been referred to as the "forgotten disease"¹² and has frequently been a low priority of both health care providers and the public.¹³ Delays in diagnosis have been attributed to the attitudes of both clinicians and patients. Many health care professionals underestimate the utility of screening exams for older adults and underestimate their life expectancies. Likewise, older adults tend to be unaware of the risk of oral cancer and their need to have routine oral examinations.

For example, more than onethird of oral cancer patients in a recent study reported not seeking professional advice for more than three months after becoming aware of a lesion.¹⁴ Similarly, Prout found that oral cancer patients averaged 11 visits with medical care providers during the two years prior to their diagnosis.¹⁵ These findings suggest that:

- Patients delay seeking care after being aware of an oral change.
- Patients do not obtain routine oral cancer examinations.
- Patients seek the care of physicians, not dental professionals, for assessment of soft tissue changes.
- Dentists are bestsuited to identify oral changes.

Yet, dentists often do not detect oral lesions in their early stages due to their opinions, practices, and lack of knowledge related to oral cancer.¹⁶⁻

¹⁸ In a recent national survey of general dentists, the vast majority reported their knowledge of oral cancer to be current, yet onethird of the dentists do not perform an oral cancer examination during a patient's initial visit, and 41 percent do not provide this examination to patients during their recall visits.¹⁹ In addition, twothirds of the dentists reported not palpating their patients' lymph nodes, which is one of the key components of an oral cancer examination. Patients treated in dental practices that do not provide comprehensive oral examinations are at an increased risk of not having an oral lesion diagnosed while it is in an early stage.

Comprehensive oral examinations are not routinely provided to all patients. Without definitive criteria to identify those most likely to have an oral carcinoma, annual oral cancer examinations are recommended for all patients. To help ensure that the components of an oral cancer examination are included in one's examination protocol, the oral cancer examination should be delineated as a separate service. Having the oral cancer examination itemized separately may encourage practitioners to provide it.

Table 2: The Components of an Oral Cancer Examination and Their Recommended Sequence

Starting extraorally:

1. Examine the face, head, and neck (include eyes, lips, and ears).
2. Palpate the pre and postauricular lymph nodes.
3. Palpate the occipital lymph nodes (at base of skull).
4. Palpate the superficial cervical lymph nodes (along sternocleidomastoid muscle).
5. Palpate the deep cervical lymph nodes (deep to the sternocleidomastoid muscle).
6. Palpate the supraclavicular lymph nodes.*
7. Palpate the thyroid gland.*
8. Evaluate the function of the temporomandibular joint.

Intraorally:

9. Palpate the lips.
10. Palpate the labial and alveolar mucosa and gingiva.
11. Examine the buccal mucosa.
12. Palpate and milk the parotid gland.
13. Examine the hard and soft palate and alveolar ridges.
14. Examine the oropharynx.
15. Palpate the submental and submandibular glands.
16. Palpate the tongue ** and floor of the mouth.

* Palpation of the supraclavicular lymph nodes and thyroid gland can help to the extent of invasiveness of lesions, however the connection to the oral cavity is less direct than with other nodes and glands.

**To examine the posterior part of the tongue, grasp extended tongue with gauze, distract the tongue to each side to view the opposite, exposed areas. To optimally view the floor of the mouth, gently dry tissues and apply light external pressure.

The Oral Cancer Examination

A comprehensive oral cancer examination includes the following:

- **A review of the patient's medical and dental history.** Well-prepared medical and dental histories provide information pertinent to the etiology of oral changes and aid in the identification of conditions that may increase the risk of disease.²⁰
- **Visual assessment of the head, neck and oral cavity.** Visualization of the mucosal surfaces with good illumination is vital in detecting early changes, which usually have little mass and minimal depth.⁴ Slight drying of mucosal surfaces aids in the recognition of changes.
- **Manual palpation of regional cervical lymph nodes.**²¹ Palpation is particularly significant when a primary lesion is not readily visible. Palpation can occur bimanually or bilaterally. The presence of a metastatic lymph node in the neck can draw attention to a potential primary site.²²

The condition of a patient's cervical lymph nodes provides one of the most important prognostic factors in a patient with oral cancer.²¹ Palpable nodes are the primary sign of current or past lymph node disease and may indicate the presence of an infectious, immune, or neoplastic disease. Normal lymph nodes are not palpable on routine examination; however, small, mobile, discrete, nontender nodes are frequently found in healthy people.²³ In general, tender, soft, enlarged, and freely movable nodes suggest acute infection. When unexplained, enlarged or tender nodes call for a reexamination and assessment. Hard, nontender and fixed nodes suggest a chronic infection or malignancy.

Sequence of Examination

To ensure that no area is overlooked, the clinician needs to establish a systematic routine for the oral examination. The order of the examination is a matter of individual choice to best suit one's work style. Utilizing an orderly, stepbystep protocol helps to increase efficiency and conserve time.

Table 2 identifies the components of an oral cancer examination and a recommended sequence.

Following a review of the patient's medical and dental history, ask the patient if he or she is experiencing discomfort in any areas of the mouth or neck. To reduce patient anxiety and concern about the examination and to inform the patient of the activity, explain the steps and reasons for the examination. At a minimum, patients need to be made aware of the need to bring to their dentists' attention any "lumps" and "bumps"

or painful areas in their mouth, especially any change present for two weeks or longer.

Identification and Initial Management of Findings

Changes in tissue color, symmetry, texture, size, and contour need to be viewed with a higher degree of suspicion and thoroughly evaluated to rule out malignancy. Any change detected must be described in detail, providing exact location, size, color, texture, and other significant characteristics. When possible, photographic documentation is useful for followup comparisons.

When a lesion is detected, probable sources of irritation should be removed; and, when present, the use of alcohol or tobacco should be curtailed. Reevaluation of the area is needed 10 days to two weeks following the initial assessment. Traumatic lesions and areas of chronic irritation usually resolve or markedly improve within that period. Any nonhealing mucosal lesion present for 14 days should be considered suspicious for oral cancer.

When a lesion persists longer than 14 days, a diagnostic workup is required. This workup includes, but is not limited to, the use of diagnostic aids such as toluidine blue staining, cytology brushes, biopsy, and/or referral to an oral surgeon or oncology specialist. In addition, the patient needs to be made aware of the practitioner's concern and the need for immediate care.

Summary

An oral cancer examination needs to be a part of the routine (at a minimum annually) oral evaluation of all patients. As "physicians of the mouth" dentists are trained to detect changes in the oral cavity, including an asymptomatic early carcinoma. The recognition of early oral lesions requires that clinicians maintain a high index of suspiciousness of all soft tissue changes.

Providing a thorough physical examination of the head, neck, and oral cavity is essential for all dentists and any clinician involved in detecting, diagnosing, and treating oral disease. The examination assesses for manifestations of disease and presence or absence of palpable lymph nodes, and provides information critical for the development of appropriate differential diagnoses. Oral cancer must be included in the differential diagnosis for illdefined, variableappearing lesions found in older adults. With prompt action, a clinician can save lives and reduce the morbidity associated with oral cancer.

Providing Oral Cancer Examinations For Older Adults

Currently, the most effective way to manage oral cancer is through early diagnosis followed by adequate treatment. If dental professionals

increase their efforts to identify early lesions and increase patient awareness so that they reduce their risk behaviors, the morbidity of oral cancer will decline. However, it will take many years before real reductions in the number of cancer cases begin to occur. As more people move into the age groups of high risk for oral cancer, it is likely that the occurrence of oral cancer will increase. Thus, for older Americans, oral cancer remains a serious concern requiring constant professional attention.

Author

Janet A. Yellowitz, DMD, MPH, is an associate professor and the director of geriatric dentistry at the University of Maryland Baltimore College of Dental Surgery. Reprinted with permission.

CDA Journal. Vol. 27, No. 9, Sept. 1999.

References

1. American Cancer Society Publication No 5008.95. American Cancer Society, Atlanta, 1995.
2. Barasch A, Eisenberg E, Huang YW, Benign or malignant? A guide to the differential diagnosis of cancerous and noncancerous oral lesions in older adults. *Focus on Adult Oral Health* 1:4, 1994.
3. Yancik R, Ries LG, Cancer in the aged. An epidemiologic perspective on treatment issues. *Cancer* 68 (11 Suppl):250210, 1991.
4. Gloeckler Ries LA, Kosary CL et al, eds, SEER cancer statistics review, 1973-1994. US Department of Health and Human Services, Public Health Service, National Institutes of Health, 1997, Bethesda, MD. NIH pub No 971789.
5. S. Salisbury PL, Diagnosis and patient management of oral cancer. *Dental Clin N Am* 41(4):891914, 1997.
6. Swango PA, Kleinman DV, Oral soft tissue diseases in geriatric populations: an epidemiologic overview. In, Squire CA, Hill MW, eds, *The Effect of Aging in Oral Mucosa and Skin*. CRC Press, 1994, pp 19.
7. Blot WJ, McLaughlin JK, et al, Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Res* 48:32827, 1988.
8. McLaughlin JK, Grindley G, et al, Dietary factors in oral and pharyngeal cancer. *J Natl Cancer Inst* 80:123 743, 1988.
9. LaVecchia C, Lucchini F, et al, Trends in cancer mortality in Europe, 1955-1989 1: Digestive sites. *Eur J Cancer* 28:132235, 1992.

10. Reynolds MW, Waheeb N et al, A typical (atypical) oral cancer. Presented as table clinic at University of Maryland, Baltimore, April 1998.
11. Alvares O, Perspectives and future studies. In, Squier CA, Hill MW, eds, *Effects of aging on oral mucosa and skin*. CRC Press, 1994, pp 1516.
12. Meskin LH, Oral cancer: the forgotten disease. *J Am Dent Assoc* 125(8):10425, 1994.
13. Epstein JB, Scully C, Assessing the patient at risk for oral squamous cell carcinoma. *Spec Care Dent* 17(4):1208, 1997.
14. Dimitroulis G, Reade P, Wiesenfeld D, Referral patterns of patients with oral squamous cell carcinoma, Australia. *Eur J Cancer B Oral Oncol* 28B(1):237, 1992.
15. Prout MN, Barber CE et al, Use of health services before diagnosis of head and neck cancer among Boston residents. *Am J Prev Med* 6(2):7783, 1990.
16. Sclinetler JF, Oral cancer diagnosis and delays in referral. *Br J Oral Maxillofac Surg* 30(4):2103, 1992.
17. Pommerenke FA, Weed DL, Physician compliance: improving skills in preventive medicine practice. *Am Fam Physician* 43(2):5608, 1991.
18. Sadowsky D, Kunzel C, Phelan J, Dentists' knowledge, casefinding behavior, and confirmed diagnosis of oral cancer. *J Cancer Educ* 3(2):12734, 1998.
19. Yellowitz JA, Horowitz AM, et al, Knowledge, opinions, and practices of general dentists regarding oral cancer: a pilot survey. *J Am Dent Assoc* 129(5):57993, 1998.
20. Darby ML, Wash MM, eds, *Dental Hygiene Theory and Practice*. WB Saunders Co, Philadelphia, 1995.
21. Shah JP, Lydlatt W, Treatment of cancer of the head and neck. *CA Cancer J Clin* 45(6):35268, 1995.
22. Alvi A, Oral cancer: how to recognize the danger signs. *Postgrad Med* 99(4):14952, 1996.
23. Bates B, Bickley LS, Hoekelman RA, eds, *A Guide to Physical Examination and History Taking*, 6th ed. JB Lippincott Co, Philadelphia, 1995.

Chapter 4

Reducing the Burden of Oral and Pharyngeal Cancers

by Deborah M. Winn, PhD; Ann L. Sandberg, PhD; Alice M. Horowitz, PhD; Scott R. Diehl, PhD; Silvio Gutkind, PhD; Dushanka V. Kleinman, DDS, MScD

Abstract: In the United States, oral and pharyngeal cancers continue to result in significant morbidity and mortality. Dental professionals play a pivotal role in all facets of controlling the burden of oral and pharyngeal cancer—from efforts to prevent its occurrence, to ensuring that oral cancers are detected at the earliest possible stage, to treating these cancers, and to ensuring maximum quality of life and function for oral and pharyngeal cancer survivors. Individually and by making linkages within the community and beyond, dentists can help patients modify their risk of these cancers and can take steps to screen for them, thereby potentially improving survival and function of those who develop oral cancer. Creative partnerships between community dentists and academic and other research centers will help move knowledge of the biological processes involved in carcinogenesis and innovations in treatment into clinical practice. Partnerships between dental and medical professionals may also help efforts to reduce the morbidity related to oral and pharyngeal cancers. Local, state and national multidis-ciplinary initiatives are emerging that focus more broadly on risk factor control or oral and pharyngeal cancer issues. These many forms of cooperative approaches offer excellent opportunities to make a significant impact on reducing the incidence and in treating debilitating and disfiguring malignancies.

During the past 25 years, remarkable progress has been made in both the elucidation of the molecular bases of cancers and their treatment. Yet monumental challenges remain. Cancers of the oral cavity, lip and pharynx affect more than 30,000 people each year;¹ and, collectively, they remain the sixth most common cancer among U.S. white males and the fourth most common among U.S. black males.² These malignancies are among the most debilitating and disfiguring of all cancers, and annual costs of care are estimated to be about \$2 billion.³ Tobacco and alcohol are major risk factors for these cancers.⁴

It is encouraging that oral and pharyngeal cancer incidence (the number of new cases of oral and pharyngeal cancers per 100,000 people) has declined recently. This decline has been most notable among white males. Only in the past few years has a decline in incidence rates for black males occurred. This, fortunately, is a reversal of rates that increased by 1.6 percent per year during the period 1973-1992. Very recently, the incidence rates for black and white females have also

declined.⁵ However, the U.S. population is increasing, and the baby boomers are aging. Thus, the actual number of individuals with oral and pharyngeal cancers has increased by about 20 percent from 1973 to 1992. Similarly, the number of people with many other forms of cancer is also increasing.⁵

A decline in the overall mortality (deaths per 100,000 people) from oral and pharyngeal cancers has also occurred.⁵ However, a striking exception to this finding is that, among people younger than 40, mortality from cancers of the tongue, the most common cancer site within the oral cavity, has been rising for decades. The mortality from oral and pharyngeal cancers in California is similar to that in the United States. However, California has a greater number of these malignancies than most states because of its large population. Of the newly diagnosed patients with oral and pharyngeal cancers in the United States in 1995, 3,000, or 11 percent, were in California.⁶ Nasopharyngeal cancers may be more common in California than elsewhere in the United States since a disproportionately large number of people of Chinese descent, who appear to be more susceptible to these specific cancers,⁷ reside in California.

The overall survival rate for individuals with oral and pharyngeal cancers is 52 percent at five years after diagnosis. This is lower than that for colon cancer, cancer of the cervix, and breast cancer.² Although survival has improved for many cancers, the five-year survival of individuals with oral and pharyngeal cancers has not increased over the past four decades. The survival of blacks has actually decreased.² Most oral and pharyngeal cancers (64 percent) are not diagnosed at an early and more easily treatable stage; black people with oral cancer are even less likely to have an early stage diagnosis (Figure 1). Yet, it is clear that survival is better when the cancer is found at an early stage (Figure 2). Also, individuals who survive an initial primary oral cancer are at an elevated risk of developing new primary tumors. The rate of second primaries among oral and pharyngeal cancer patients exceeds that for any other type of cancer.^{8,9}

Oral and pharyngeal cancers, like other cancers, result from a multistage accumulation of genetic aberrations. The genetic changes that have been associated with oral and pharyngeal cancers are not localized to any one chromosome but, rather, are found on many human chromosomes. Mutations in certain genes may promote uncontrolled cell growth by overproducing either growth stimulatory factors or their receptors that, following ligand binding, trigger numerous intracellular processes. Mutations in other genes result in a loss of tumor suppressors, proteins that prevent excessive cell growth. Additional genetic alterations favor vascularization of tumors or enable oral tumor cells to invade the surrounding tissues and migrate within lymph nodes to the lymph nodes in the neck. The intricacies of cancers are further increased by genetic aberrations in transcription factors that regulate the expression of other genes.¹⁰

Table 1: Oral and Pharyngeal Cancer Electronic Information Resources

(Following these links will open a new browser window. You will not lose your place in this course.)

- National Institutes of Health: www.nih.gov
- National Institute of Dental Research, one of the National Institutes of Health: www.nidr.nih.gov
- National Cancer Institute, one of the National Institutes of Health: www.nci.nih.gov
- National Oral Cancer Awareness Program, an ongoing program to inform both the public and health care professionals about oral cancer and related topics: www.oralcancer.org
- Centers for Disease Control and Prevention: www.cdc.gov
- American Cancer Society: www.cancer.org

Oral cancers are often preceded by premalignant lesions including leukoplakia (white mucosal changes) and erythroplakia (red mucosal changes) or mixed white and red lesions.¹¹ Biomarkers are cellular, biochemical, or molecular alterations measurable in human tissues and fluids.¹² Alterations in certain genes may occur in premalignant lesions and may, therefore, provide excellent biomarkers for determination of those individuals who require close monitoring or who may benefit from chemoprevention, that is, the use of natural or synthetic chemicals such as vitamin A related compounds to prevent oral cancer.¹³

Major efforts are currently under way to identify genetic biomarkers both for the early detection of oral and pharyngeal cancers and as indicators for prognosis.¹⁴ For example, the normal p53 suppressor gene inhibits cell growth. Mutations of the gene (resulting in failure of the normal inhibitions of growth) are common in oral cancers.¹⁵ In addition, p53 alterations appear in premalignant oral lesions¹⁶ and also predict recurrence and second head and neck primary cancers.¹⁷ Behavioral and molecular factors are both important in oral cancer etiology. Of interest are recent studies suggesting that individuals with a genetic predisposition to rapidly metabolize alcohol and who also consume

large quantities of alcohol are at the highest risk for development of oral and pharyngeal cancers.¹⁸

The primary objective of any therapeutic regimen for treatment of head and neck cancers is cure. However, current modalities also focus on preservation or restoration of function and appearance. Surgery or radiotherapy, either alone or in combination, is generally utilized for early stage tumors. Although surgery is commonly favored, radiotherapy may be essential because of the size or location of the tumor. In latestage disease (tumor greater than 4 cm and/or lymph node involvement), more aggressive treatment, with resultant functional consequences, may be necessary. Chemotherapy is often added to the treatment regimen in advanced tumors or tumors of certain sites in the hope of increasing control;¹⁹ by itself, chemotherapy is only palliative.²⁰ New techniques and approaches in treatment are emerging. For example, for instances when reconstruction of the mandible or soft tissue is required, techniques have now been developed for tumor resection and bone or skin grafting in a single surgical procedure. Investigators also are now exploring the possibilities of applying immunotherapy and gene therapy to the treatment of cancers of the head and neck.

Although we are moving ahead in understanding the etiology and pathogenesis of this disease, there are actions that can be taken now to prevent and control it. Reducing the burden of oral and pharyngeal cancers will require multiple approaches to prevent tobacco use and excessive alcohol consumption, identify precancer lesions and tumors at the earliest possible stage, ensure prompt and coordinated treatment of people with oral cancer, and move promising scientific discoveries rapidly into practice. Dental professionals can contribute to these efforts to reduce the burden of the occurrence of oral cancer and its potentially devastating effects through practicebased efforts to reduce or eliminate patients' risk behaviors and by diagnosing these cancers earlier. Dental professionals can also make a difference through partnerships with the greater community, state and nation.

National and State Programs

Over the past several years, the Centers for Disease Control and Prevention, the National Institute of Dental Research, and the American Dental Association have developed a strategic plan for the prevention and reduction of oral cancer in the United States. It is hoped that this plan will stimulate an effective national campaign for the prevention and control of oral and pharyngeal cancers. Recommendations are made in five broad areas:

- Advocacy, collaboration, and coalitionbuilding;
- Public health policy;
- Public education;
- Professional education and practice; and

- Data collection, evaluation, and research.

Implementation of the plan is under way and involves a wide range of dental, medical, and social service organizations that work with oral cancer patients and those at risk for oral cancer. The national health promotion and disease prevention objectives for the nation have highlighted oral cancer reductions and actions needed for tobacco control.²²

There are several other initiatives at the national level that specifically focus on prevention and control of tobacco use and involve dental professionals. For the past several years, the National Dental TobaccoFree Steering Committee under the sponsorship of the National Cancer Institute has mobilized a consortium to:

- Assess recent developments in tobacco use intervention strategies;
- Define opportunities of dental involvement in tobacco use intervention activities; and
- Promote cooperation among dental and other professional and public interest organizations.

A national program focused on chewing tobacco and snuff, the National Spit Tobacco Education Program, has been under way since 1994. This program was initially funded by Oral Health America, the National Institute of Dental Research, and the National Cancer Institute and is now funded by the Robert Wood Johnson Foundation. Six regional coordinating centers across the country have been established. The National Cancer Institutes' COMMIT program, an acronym for Community Intervention Trial for Smoking Cessation, also included a focus on dental professionals.²³

One example of a stateinitiated program focuses on spit tobacco use. The Spit Tobacco Education and Prevention Plan for the State of Texas is funded by the Texas Cancer Council and administered by the Dental Oncology Education Program in cooperation with the Texas Dental Association. The extremely high use of spit tobacco in Texas stimulated this special initiative. The goal is to diminish and eliminate use of spit tobacco through collaborative integrated research, education, and public policy activities.²⁴

The development, implementation, and evaluation of state models has been suggested as one approach to oral cancer prevention and early detection.^{21,25} A state model is defined as a comprehensive plan that includes implementation and evaluation criteria of appropriate interventions based on the needs of the particular state. The rationale for this approach is that each state has different oral cancer incidence, mortality, and survival rates; racial and ethnic groups; practice acts for health care providers; and laws concerning tobacco use and enforcement practices, as well as differences in both smoking and chewing patterns. Thus, no one model could fit the needs of all states.

Today, no state has a comprehensive state model for oral cancer prevention and early detection, but several states have taken some initial steps to do so. For example, Maryland has begun a partnership for the prevention and early detection of oral cancers. The partnership, which is spearheaded by the state dental director, includes representatives from provider associations, advocacy and consumer agencies, organizations, and other interested groups. It is the intent of the partnership to assist Maryland dental and medical practitioners, policymakers, and residents in receiving the benefits of appropriate and quality oral cancer prevention, education, and training and by advocating oral cancer-related policies that promote and protect health and support healthy behaviors and lifestyles.

Oral Cancer Prevention

Finding innovative means of preventing people from using tobacco and alcohol and developing effective methods to get users to quit will be essential in reducing the occurrence of new cases and the risks of second primary cancers. Based on a very large epidemiologic study in four areas of the United States, it is estimated that about three-fourths of oral and pharyngeal cancers are associated with the use of any form of tobacco and heavy alcohol intake. Tobacco and alcohol independently increase the risk of oral and pharyngeal cancer, and people who use both are at much higher risk than would be expected from the risks among those who only smoke or only drink.²⁶ Quitting smoking reduces the risk of oral and pharyngeal cancer. An advisory group to the surgeon general stated that smokeless tobacco (snuff and chewing tobacco) can cause cancer in humans.²⁷

Although cigarette smoking rates have been declining in adults and have probably contributed to the declines in incidence rates of these cancers, disturbing trends have emerged. Cigarette smoking is increasing among adolescents²⁸. Smokeless tobacco use remains common, based on a survey in 1995 that found that 11.4 percent of high school youth had used smokeless tobacco in the previous month; for white adolescents the figure was 25.1 percent.²⁹ Also of serious concern is the recent popularity of cigar smoking. Compared to nonusers of cigars, cigar smokers experience a four to tenfold higher risk of dying from oral, laryngeal, and esophageal cancer.³⁰

Dentists seldom determine patients' use of tobacco and alcohol products.³¹ Currently, one-third of dental schools do not assess patient risk behavior on their standard patient history forms;³² also, routine risk behavior assessments are not universally used in medical and dental hygiene schools.^{33, 34} Provider knowledge of the patient's risk profile is an essential.

Emerging evidence is demonstrating that interventions in the dental practice setting may be effective in reducing use of tobacco. One recent study compared methods to stimulate smokeless tobacco users to quit.

The intervention compared usual care with a routine oral examination, an explanation of the health risks of smoking, unequivocal advice to quit, and a nineminute video, a selfhelp manual, and a brief counseling session with a dental hygienist. This intervention led to a 50 percent increase in the number of quitters at one year compared to usual care.³⁵ Brief interventions for smokers and other tobacco users suitable for the dental office have also been developed and made available.^{36,37}

New approaches to help tobacco users quit are emerging. One promising strategy is suggested by the recent results of a clinical trial of the antidepressant Bupropion. The results indicated that 19.6 percent of smokers receiving the lowest dosage of the antidepressant were abstinent after one year of followup compared to 12.4 percent among the placebo group, and rates of abstinence for higher dosages of Bupropion were even greater.³⁸ However, because those at highest risk of oral and pharyngeal cancer are smokers who also abuse alcohol, interactions between drugs designed to curb tobacco and alcohol and the challenges of multiple drug dependencies must be considered.

Other mechanisms are also being used to influence tobacco use behaviors. California, for example, has had remarkable success in reducing tobacco consumption through Proposition 99, the tobacco tax initiative. The result of implementation of Proposition 99, which raised taxes on tobacco products and used the funds from the increase to fund tobacco control activities, has been a 27 percent decline in the prevalence of tobacco use in California from 1988 to 1993, a rate of decline three times that of the rest of the United States.³⁹

Early Identification

Identifying cancers at the earliest possible stage is another critical component in mitigating the burden of oral and pharyngeal cancer. Both the patient and the dentist can play a role. White and/or reddish lesions in the oral cavity can progress to malignancies.⁴⁰ Nonhealing sores, pain and swelling are additional signals to a patient to seek a medical or dental examination. However, early detection of oral and pharyngeal cancers is impeded by the public's poor understanding of the risk factors for and the signs and symptoms of oral cancers.^{41,42} For example, only 25 percent could identify one early sign of oral cancer, and 44 percent responded that they did not know any early signs.⁴² Dental professionals can educate patients about their risk for oral and pharyngeal cancer and encourage compliance with visits and examinations to monitor oral and pharyngeal mucosal health. This function in part depends upon undergraduate training and practical experience, as well as continuing education updates.

Oral cancers may be diagnosed and treated earlier if dentists provide oral cancer examinations. Currently, only 14 percent of U.S. adults report that they have ever had an oral cancer examination and only 7 percent had the exam in the past year,⁴³ the frequency recommended by

the American Cancer Society for adults 40 years of age and older. Based on a recent survey in two Maryland counties,⁴⁴ many dentists and other health care providers do not examine all adult patients for oral cancers. In addition, a recent national pilot study showed that dentists' level of knowledge regarding risk factors for and signs and symptoms of oral cancer is inconsistent and less than optimal.⁴⁵ Many dentists have not attended a continuing education course on oral cancer during the past five years.

There are additional barriers to reducing the proportion of oral cancers that are diagnosed at late stage. A significant problem is the lack of reimbursement for oral cancer examinations under the Medicare and Medicaid systems.

Professional Education and Association Opportunities

Dental and dental hygiene schools are uniquely positioned to provide additional training in oral cancer prevention, detection, and care. Also, educational programs have been developed by nonacademic groups including state American Cancer Society groups, the National Oral Cancer Awareness Program, and the National Oral Health Information Clearinghouse, the latter in conjunction with an advisory group that has representatives from patient groups, the Centers for Disease Control and Prevention, the American Dental Association, and the Federation of Special Care.

Associations of dental professionals, working with their medical and other health care personnel colleagues, can also contribute to efforts to reduce oral cancer and encourage activities that improve early detection. State dental and medical boards could require that dental and medical personnel take a special course on oral cancer prevention and early detection prior to licensure and relicensure.⁴¹

Such a requirement has several precedents in California; for example, the newly enacted requirement for all dental personnel to complete continuing education in infection control. Many state and local dental/dental hygiene organizations sponsor cancer education programs at their annual or periodic meetings.

Dental, Medical, and Research Partnerships

Partnerships can focus attention on particular public health problems, potential solutions, and mechanisms for implementing those solutions. Two special types of partnerships will be important: those with the medical community and those with the research community. Other, broader partnerships will be important as well.

Concerted and coordinated effort on the part of dental and medical professionals is essential to make an impact in risk factor management. Responsibility for diagnosis is shared between the dental and medical

professions because of the nature of the presenting symptoms, which may lead patients to seek out dentists, otolaryngologists, or internal medicine specialists, among others.

The patterns of health care utilization by older people suggest that both medical and dental professionals may have opportunities to screen for previously undetected lesions. For example, in one study of head and neck cancer patients residing in Boston, subjects had a median of 10.5 health care visits in the 24-month period just prior to diagnosis, and these visits included a wide range of health care professions and care settings.⁴⁶ Thus, monitoring through both the medical and dental care systems can potentially optimize early detection. Efforts are under way to increase the diagnostic capacity of all health professionals and to introduce preliminary examinations in settings such as the health service centers for homeless shelter residents.⁴⁷

The medical, psychological, and social problems associated with both tobacco use and alcohol abuse are well-known. Thus, it is clear that the individuals at highest risk for oral cancer may have enormous difficulty quitting either of these habits. They may also have medical or emotional problems that can compromise compliance with health care appointments intended to monitor risk factors or oral lesions.

Smoking and alcohol abuse and their consequences may complicate oral cancer treatment. Finally, once diagnosed and treated for oral cancer, many patients face functional and other problems that impact oral and systemic health and must be closely monitored for recurrences and new primaries. Ongoing dentalmedical communication and coordination should help maximize function, quality of life, and early detection of new problems in these patients.

The research community and dental practitioners can form new relationships in the future to take advantage of scientific innovations. The Internet provides the practicing dentist with excellent opportunities to follow these advances: The National Institute of Dental Research and the National Cancer Institute Web pages are constantly updated with new research findings. The research community needs to establish links with dentists in communities as a source of patients for studies and as a possible setting for studies of the effectiveness of procedures for risk factor control, early detection, and followup after diagnosis and initial therapy.

Conclusion

Dental professionals play a pivotal role in all facets of oral and pharyngeal cancers. They must be involved in attempts to prevent occurrence of these malignancies through promotion of tobacco and alcohol control and be well-versed in examination procedures to detect lesions at the earliest possible stages. They must also be instrumental in reducing the morbidity and mortality of oral pharyngeal cancers by

promptly referring patients for appropriate treatment and, subsequently, monitoring them for recurrence or the development of second primary tumors.

Close teamwork among dentists, hygienists, physicians, maxillofacial or head and neck surgeons, radiation and medical oncologists, prosthodontists, psychologists or psychiatrists, and rehabilitation specialists would optimize diagnosis, therapy, and maintenance or restoration of function and quality of life. Consortia that focus on the control of oral and pharyngeal cancers in the United States have been established. Their multi-disciplinary cooperative approaches, in addition to escalated progress in research and more rapid translation of research findings to a clinical setting, would make a significant impact on reducing the incidence of and treating these debilitating and disfiguring malignancies.

Authors

Deborah M. Winn, PhD, is a senior investigator in the Division of Intramural Research, National Institute of Dental Research, National Institutes of Health.

Ann L. Sandberg, PhD, is director of the Neoplastic Diseases Program and the Comprehensive Oral Health Research Centers of Discovery Program in the Division of Extramural Research, National Institute of Dental Research, National Institutes of Health.

Alice M. Horowitz, PhD, is a senior scientist in the Office of Science Policy and Analysis, National Institute of Dental Research, National Institute of Health.

Scott R. Diehl, PhD, is chief of the Molecular Genetic Epidemiology Section in the Division of Intramural Research at the National Institute of Dental Research, National Institutes of Health.

Silvio Gutkind, PhD, is chief of the Oral and Pharyngeal Cancer Branch, Division of Intramural Research, National Institute of Dental Research, National Institutes of Health.

Dushanka V. Kleinman, DDs, MScD, is deputy director, National Institute of Dental Research, National Institutes of Health.

Reprinted with permission.

CDA Journal. Vol. 26, No. 6, June 1998.

References

1. Landis SH, Murray T, et al, Cancer Statistics, 1998. *CA A CA J for Clin* 48:629, 1998.
2. HARRAS A, EDWARDS BK, et al, *Cancer Rates and Risks*. NIH Pub No 96691, Bethesda, MD, 1996, pp 2830, 32.
3. Reisine S and Locker D, Social, psychological, and economic impacts of oral conditions and treatments. In, Cohen LK and Gift HC, *Disease Prevention and Oral Health Promotion*. Munksgaard, Copenhagen, 1995.
4. Blot WJ, Devesa SS, et al, Oral and pharyngeal cancer. *Cancer Surv* 19:2342, 1994.
5. Kosary CL, Ries LA, et al, *SEER Cancer Statistics Review, 1973-1992*. Tables and Graphs, National Cancer Institute, NIH Pub No 962789. Bethesda, MD, 1995, pp 17, 34, 52, 355, 361.
6. American Cancer Society, *Cancer Facts and Figures _ 1995*. American Cancer Society, Atlanta, 1995.
7. Miller BA, Kolonel LN, et al, eds, *Racial/Ethnic Patterns of Cancer in the United States 1988-1992*, National Cancer Institute. NIH Pub No 964104. National Institutes of Health, Bethesda, MD, 1996,
8. Boice JD, Curtis RE, et al, *Multiple Primary Cancers in Connecticut and Denmark*. U.S. Government Printing Office, Washington, DC, 1985.
9. Winn DM and Blot WJ, Second cancer following cancer of the buccal cavity and pharynx in Connecticut, 1935-82. In Boice JD, Curtis RE, et al, *Multiple Primary Cancers in Connecticut and Denmark*. U.S. Government Printing Office, Washington, DC, 1985.
10. Weinberg RA, How cancer arises. *Scientific American* 275:6271, 1996.
11. Silverman S Jr, Diagnosis and management of leukoplakia and premalignant lesions. *Oral Maxillofac Surg Clinics N AM* 10: 1323, 1998.
12. Hulka BS, Overview of biological markers. In, Hulka BS, Wilcosky TC, and Griffith JD, *Biological Markers in Epidemiology*. Oxford University Press, New York, 1990, pp 315.
13. Lippman SM, Benner SE, and Hong WK, Chemoprevention. *Cancer* 1993:98490.
14. Sidransky D, Molecular genetics of head and neck cancer. *Curr Opinion Oncol* 7:22933, 1995.

15. Patterson IC, Eveson JW, and Prime SS, Molecular changes in oral cancer may reflect aetiology and ethnic origin. *Eur J Cancer B Oral Onco* 32B:1503, 1996.
16. Sciubba JJ, Oral leukoplakia. *Crit Rev Oral Biol Med* 6:14 760, 1995.
17. Shin DM, Lee JS, et al, P53 expression: Predicting recurrence and second primary tumors in head and neck squamous cell carcinoma. *J Natl Cancer Inst* 88:51929, 1996.
18. Harty LC, Caporaso NE, et al. Alcohol dehydrogenase 3 genotype and risk of oral cavity and pharyngeal cancer. *J Natl Cancer Inst* 89(22):16981705, Nov 1997.
19. Vokes EE, Athanasiadis, I. Chemotherapy for squamous cell carcinoma of head and neck: The future is now. *Am Oncol* 7:1529, 1996.
20. Shah FP, Treatment of cancer of the head and neck. *CA Cancer J Clin* 45:35268, 1995.
21. Proceedings: National Strategic Planning Conference for the Prevention and Control of Oral and Pharyngeal Cancer, 1997.
22. Department of Health and Human Services. *Healthy People 2000*. US Public Health Service, Washington, DC, 1991. DHHS pub no (PHS) 9150212.
23. National Cancer Institute, *Community based Interventions for Smokers: The COMMIT Field Experience*. Smoking and Tobacco Control Monograph 6, NIH Publ No 954028. National Institutes of Health, Bethesda, Md, 1995.
24. Texas Cancer Council, *Texas Spit Tobacco Education and Prevention Plan: A Guide for Action*. Texas Cancer Council, 1997.
25. Horowitz AM, Goodman HS, et al, The need for health promotion in oral cancer prevention and early detection. *J Public Health Dent* 56:31930, 1996.
26. Blot WJ, McLaughlin JK, et al, Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Res* 48:32827, 1988.
27. Advisory Committee to the Surgeon General, *The Health Consequences of Using Smokeless Tobacco*. NIH Pub. No. 862874. National Institutes of Health, Bethesda, Md, 1986, p xxiii.
28. Office on Smoking and Health and Division of Adolescent and School Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention,

Surveillance for selected tobacco behaviors _ United States, 1900-1994. *Morb Mort Wkly Rep* 43(SS3):143, 1994.

29. Office on Smoking and Health and Division of Adolescent and School Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Tobacco use and usual source of cigarettes among high school students _ United States, 1995. *Morb Mort Wkly Rep* 45(20):4138, 1996

30. Office on Smoking and Health Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control, US Department of Health and Human Services, *Health Consequences of Cigar Smoking*, 1998.

31. Dolan T, McGorray SP, et al, Tobacco control activities in US dental practices. *JADA* 128:166979, 1997.

32. Yellowitz JA, Goodman HS, et al, Assessment of alcohol and tobacco use in dental schools' health history. *J Dent Ed* 59:10916, 1995.

33. Ahluwalia KP, Yellowitz JA, et al, An assessment of oral cancer prevention curricula in US medical schools. *J Cancer Educ*, in press.

34. Gurelian JR, McFall DB, et al, Documenting oral cancer risk factors on health history forms (abstract), *J Dent Res* 75:306, 1996.

35. Stephens VJ, Severson H, et al, Making the most of a teachable moment a smokeless tobacco cessation intervention in the dental office, *Am J Public Health* 85(2):2315, 1995.

36. Lichtenstein E, Hollis JF, et al, Tobacco cessation interventions in health care settings: rationale, model, outcomes. *Addict Behav* 21:70920, 1996.

37. Mecklenburg RE., Christen AG, et al, *How to Help Your Patients Stop Using Tobacco: a National Cancer Institute Manual for the Oral Health Team*. NIH Publ. No. 913191. National Institutes of Health, Bethesda, Md, 1991.

38. Hurt RD, Sachs DP, et al, A comparison of sustained-release Bupropion and placebo for smoking cessation. *New Engl J Med* 337:1195202, 1997.

39. Traynor MP, and Glantz SA, California's tobacco tax initiative: the development and passage of Proposition 99. *J Health Politics Policy Law* 21:54385, 1996.

40. Silverman S Jr, Leukoplakia and erythroplasia. In: Silverman S Jr, ed, *Oral Cancer*, 4th ed. BC Decker, 1996, pp 2540.

41. Horowitz AM, and Nourjah PA, Patterns of screening for oral cancer among U.S. adults. *J Public Health Dent* S6:3315, 1996.
42. Horowitz AM, Nourjah P, et al, U.S. adult knowledge of risk factors for and signs of oral cancers: 1990. *J Am Dental Assoc* 126:3945, 1995.
43. Centers for Disease Control and Prevention, Examinations for oral cancer United States, 1992. *Morbidity Mortality Weekly Report* 43:198200, 1994.
44. Yellowitz JA, and Goodman HS, Physicians and dentists' oral cancer knowledge, opinions and practices. *J Am Dent Assoc* 126:5360, 1995.
45. Yellowitz JA, Horowitz AM, et al, Dentists' knowledge, opinions and practices regarding oral cancer: A pilot survey. *J Am Dent Assoc*, in press.
46. Prout MN, Heeren TC, et al, Use of health services before diagnosis of head and neck cancer among Boston residents. *Am J Prev Med* 6:7783, 1990.
47. Prout MN, Morris SJ et al, A multidisciplinary educational program to promote head and neck cancer screening. *J Cancer Educ* 7:13946, 1992.