40 Tobacco Use

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Definition

Tobacco use may be defined as any habitual use of the tobacco plant leaf and its products. The predominant use of tobacco is by smoke inhalation of cigarettes, pipes, and cigars. *Smokeless tobacco* refers to a variety of tobacco products that are either sniffed, sucked, or chewed.

Technique

Questions regarding tobacco use are traditionally included in the social history with other habits but may fit just as appropriately when inquiring about the patient's pulmonary or cardiovascular status. Most patients are aware that smoking may affect their health and expect to be asked about this habit. Unlike cigarettes, the potential adverse health effects of smokeless tobacco are not well known, and patients are often surprised when questioned. Females and males, young and elderly, should be asked about the use of *all* forms of tobacco.

The technique for inquiring about tobacco use will be divided into two categories: smoked and smokeless tobacco. The direct approach is best, and questions should be asked in a nonaccusatory fashion. Ask: "Do you smoke cigarettes, cigars, or a pipe?" If the patient answers no, then ask: "Have you ever smoked?" and "When did you stop smoking?" Both questions are important and relate to cancer risk and severity of lung disease. If the patient answers affirmatively, then try accurately to quantify the amount. Cigarette smoking is quantified by inquiring about what portion of a pack or how many packs are smoked per day and for how many years. A standard package contains 20 cigarettes. This can be translated into pack years:

Pack years = number of packs per day × years smoked.

Examples: 10 cigarettes per day = ½ pack for 10 years = 5 pack years (½ × 10 = 5).

2 packs per day for 15 years = 30 pack years (2 × 15 = 30).

Cigar smoking can be similarly quantified as the number of cigars per day for how many years (or cigar years). Pipe smoking is more difficult to quantify because many pipe smokers do not actually have their pipe lit all the time. Perhaps the easiest way to quantify pipe smoking is the number of bowlfuls of tobacco used per day, or how long a pouch of tobacco lasts. It is also important to quantify the degree of smoke inhalation, best done by categorizing as no inhalation, mild or moderate inhalation, and deep inhalation. In the history, inquire about the patient's attempts to stop smoking. "Have you ever tried to quit smoking?" "How did you quit?" "How long did you quit?" and "Do you believe you can quit?" will elicit this information. While questioning the patient, attention should be given to the presence of tobacco stains on the teeth and fingers. The odor of tobacco may be obvious and should be noted.

Physicians should inquire specifically about the use of smokeless tobacco because some patients will not volunteer this information. Also, the term *smokeless tobacco* will not be recognized by all patients. The simplest way is to ask, "Do you dip snuff or use chewing tobacco?" It may help to mention specific brand names such as Skoal or Copenhagen. If the patient responds affirmatively, then quantify the amount used and for how many years. It is not unusual to find individuals who started using smokeless tobacco when they were 8 or 10 years old. Smokeless tobacco use can be quantified by finding what portion or how many packages of a particular product are used in a 24-hour period. Snuff is packaged in cans containing 30 to 40 grams of tobacco; chewing tobacco comes in 70- to 80- gram pouches containing square plugs or bricks. Amount used may be described, for example, as a half can of snuff per day for 10 years or one pouch of tobacco per day for 10 years. This could also be recorded as 5 can years or 10 pouch years, similar to the pack years used for cigarette smoking. It is also important to find out the number of hours per day tobacco is kept in the mouth and if the juices are expectorated or swallowed. The longer the tobacco is in the mouth, the greater the local effects; the larger the proportion of juices swallowed, the more nicotine, glucose, and sodium that are systemically absorbed.

Basic Science

Nicotine, an alkaloid present in all tobacco products, is well absorbed from mucosal surfaces, the respiratory tract, and skin. It acts at the preganglionic–postganglionic synapse, resulting in stimulation of sympathetic and parasympathetic nerve fibers. It exerts a complex action with a transient depressant effect on the autonomic nervous system and a sympathetic effect on the cardiovascular system. The latter can be summarized as increased heart rate, blood pressure, stroke volume, and cardiac output. Combustion products from smoked tobacco also yield tar, nitrous oxides, and carbon monoxide. Tar and related products have been associated with cancer risk. Autopsy studies in cigarette smokers and patients dying of lung cancer show increased morphologic changes such as squamous metaplasia, acanthosis, dyskeratosis, and mitotic figures in bronchial epithelial cells when compared with controls. These changes are generally considered to be precursors of bronchogenic cancer. Inhaled carbon monoxide leads to increased levels of carboxyhemoglobin by as much as 15%, resulting in reduced tissue oxygenation. In experimental animals, smoke inhalation has been shown to cause focal swelling of aortic endothelial cells and the appearance of microvilli-like processes. Subsequently, these create intimal ridges and folds and may serve as initial areas of mural thrombosis and plaque

formation. Cigarette smoking also increases platelet adhesiveness.

In addition to nicotine, smokeless tobacco contains nitrosamines, sodium, glucose, glycyrrhizinic acid, and grit. The sodium content ranges from 207 to 1201 mg per container or pouch. Chewing tobacco has the highest content of glucose—as much as 50 gm/dl. Licorice, which is used as a flavoring, contains glycyrrhizinic acid in concentrations up to 0.15%, sufficient to induce hypokalemia and mimic hyperaldosteronism.

Table 40.1
Comparative Risk of Cancer in Smokers versus Nonsmokers

Site	Increased risk over nonsmokers
Larynx	6–10 ×
Mouth	3–6 ×
Pharynx	3–6 ×
Esophagus	3–6 ×
Bladder	1.5–3 ×
Pancreas	1.5–2 ×

Clinical Significance

Tobacco products are habituating and addicting. Like other addictive drugs, abstinence leads to a syndrome of restlessness, agitation, and depression that is mostly relieved by nicotine administration. Nearly 4000 compounds have been identified in cigarette smoke, but tar, carbon dioxide, nitrous oxides, and nicotine are responsible for the major health hazards of smoking. Tobacco-specific *N*-nitrosamines are produced in smokeless tobacco from nicotine, nornicotine, and anatabine during the curing and processing. These have been shown to be carcinogenic.

Use of tobacco products, particularly cigarette smoking, is the most important preventable public health problem for developed countries. In 1980 approximately one-third of all adults in the United States were cigarette smokers, and over 22 million used various forms of smokeless tobacco. In the United States alone, it is estimated that over 200,000 deaths per year can be attributed to the effects of tobacco. The economic loss, as measured by temporary or permanent disability, outpatient visits, and hospital days, amounts to billions of dollars.

Many of the consequences of smoking and other forms of tobacco use are well established. The increasing popularity of cigarette smoking during the first half of the century resulted in a dramatic rise in lung cancer deaths. At first this was noticeable in men, but recent data show that lung cancer is becoming the leading cause of death from cancer in women and that smoking-related cancers alone explain the recent increase in mortality from cancer in the United States. Beginning in the late 1940s, the effects of smoking were investigated in several retrospective and prospective studies. These studies have been reported, reviewed, and updated in the Surgeon General's reports (U.S. Public Health Service, 1964, 1979, 1982) and other authorities. These studies showed that the excess total mortality in cigarette smokers was 30 to 80% greater than in nonsmokers, and depended on the age of starting smoking and the number of cigarettes smoked per day. For example, for a man who started smoking 20 cigarettes a day at age 25, life expectancy would be reduced by 4.6 years. If the same man smoked 40 cigarettes a day, life expectancy would be reduced by 8.3 years. Life loss is about 5.5 minutes per cigarette smoked.

The major causes of death from tobacco use include lung cancer, coronary artery disease (CAD), and chronic obstructive lung disease (COLD). Of the estimated 80,000 lung cancer deaths in the United States, most are associated with smoking. Cessation of smoking reduces the risk of lung cancer mortality compared with that of the continuing smoker. The carcinogenic effect of cigarette smoking is synergistic with that of other carcinogenic agents, such as asbestosis. Cancer of the mouth, larynx, and esophagus are also more common among smokers, especially those who smoke cigarettes (Table 40.1).

Smoking is also related to the development of *cardiovascular disease*, especially *coronary artery disease* (CAD). The particular association of smoking with cardiovascular disease was not appreciated well until publication of the Framingham study. Since then, CAD has been recognized as one of the most important consequences of smoking. As in cancer, the risk is increased with the number of cigarettes smoked per day, the age of starting smoking, and the degree of inhalation. The risk of developing untoward events due to CAD is reduced dramatically once cigarette smoking is stopped, so that 10 years after cessation of smoking, the risk of CAD is the same in ex-smokers as it is in nonsmokers. Interestingly, the effect of smoking is present after adjustments for levels of serum cholesterol, blood pressure, degree of physical activity, and presence of obesity. Other studies show a three- to sixfold incidence of death for nonsyphilitic aortic aneurysms in heavy cigarette smokers over nonsmokers. The progression of thromboangiitis obliterans (Buerger's disease) is primarily determined by smoking. The risk of subarachnoid hemorrhage is increased in women who smoke and use oral contraceptives when compared with women who neither smoke nor use oral contraceptives.

Smoking is the most important determinant of *chronic obstructive lung disease*. In cigarette smokers, the risk of developing COLD is 10 times that of nonsmokers, and for pipe and cigar smokers the risk is increased one and one-half to threefold. The risk of lung disease is also influenced by geographic location and climate. In one study, the risk of COLD among British physicians who smoked 25 cigarettes per day was increased twentyfold, while a group of U.S. veterans who smoked 40 cigarettes per day had an eightfold increased risk.

Recent studies have documented an association between maternal smoking during pregnancy and *infant birth weight*. Babies born to women who smoke during pregnancy have lower birth weight than babies from nonsmoking women of the same social class. Some studies have demonstrated a slightly greater neonatal mortality among infants of smokers. Cigarette smoking has also been associated with peptic ulcer disease. Increased frequency of duodenal ulcer, reduced ulcer healing, and recurrent ulceration have been documented.

The clinical implications of *smokeless tobacco* use can be divided into local and systemic effects. Local effects include irritation of the oral mucosa from the alkaline pH and grit, which result in gum recession, exposure of the neck of the tooth, and abrasion of the enamel surface. Chronic inflammation initiates epithelial dysplasia of the buccal mucosa, eventually resulting in leucoplakia that should be considered a premalignant lesion. The rate of malignant transformation for these leucoplakic areas is between 6 and 30%. Nodular and speckled leucoplakic areas tend to have the highest rate of malignant transformation. Other local ef-

fects include a decreased sense of taste from chronic exposure to an alkaline pH and an increased incidence of caries from the high glucose content.

Multiple systemic effects of smokeless tobacco use include the absorption of significant amounts of nicotine. As described earlier, nicotine exerts a sympathomimetic effect on the cardiovascular system. Several studies have demonstrated a significant rise in blood pressure and pulse with a fall in fingertip skin temperature after using smokeless tobacco. These changes were felt to be hemodynamically significant and could affect the care of patients with hypertension, angina, congestive heart failure, Raynaud's phenomenon, or peripheral vascular disease. Chronic exposure from smokeless tobacco has been reported to cause neuromuscular disease manifested as a loss of muscle bulk, strength, and endurance. Tobacco amblyopia, a gradual loss of visual acuity of bright light secondary to nicotine absorption, has been seen in smokeless tobacco users as well as in smokers. The presence of salt, glucose, and flavorings present in smokeless tobacco may affect the therapeutic regimen of certain groups of patients such as hypertensives, patients with congestive heart failure, and diabetics.

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