RAKEL

TEXTBOOK OF Family Practice

Sixth Edition

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Chapter 59

Nicotine Addiction

ROBERT E. RAKEL and ALAN BLUM

The power of nicotine addiction became clear when I saw malnourished and hungry people trading food rations for cigarettes.

William Foege, M.D. (1989), commenting on refugee camps during the Nigerian Civil War

Tobacco smoking leads to a dependence on nicotine that is indistinguishable from other forms of drug dependence. The fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) of the American Psychiatric Association (1994) classifies tobacco dependence as an addiction. In such a dependency, the drug is needed to maintain an optimal state of well-being. Nicotine, the habituating constituent of tobacco, meets the criteria for addiction because a typical withdrawal syndrome occurs after smoking cessation. Nicotine is more addicting than cocaine because it is easier for addicts to break their addiction to cocaine and heroin than to nicotine (Krasnegor, 1979; Lee & D'Alonzo, 1993).

Cigarette smoking is the chief avoidable cause of death in our society. Each year smoking is responsible for 18% of the total deaths in the United States—seven times more Americans than were killed in the Vietnam War. "Clearly, smoking has killed more Americans during this century than were killed in battle or died of war-related diseases in all wars ever fought by this nation" (Pollin & Ravenholt, 1984).

Approximately 40% of all deaths from cancer and 21% of deaths from cardiovascular disease are caused by smoking. Tobacco contributes to about 400,000 deaths annually in the United States, as compared with 47,000 deaths each year in motor vehicle accidents (McGinnis & Foege, 1993).

More young women than young men smoke cigarettes, and in 1986 lung cancer passed breast cancer as the leading cause of cancer death in women. Smoking kills 10,000 more women than breast cancer does, yet we have a breast cancer awareness month and a great deal of attention focused on breast cancer but no public outcry against the needless deaths from lung cancer (Centers for Disease Control and Prevention [CDC], 1988).

Although cigarette smoking in adults declined from 42% to 27% in the United States between 1964 and 1992 (after publication of the Surgeon General's first report on smoking and health in 1964), 28% of men and 24% of women continue to use tobacco daily. Approximately 1.3 million persons per year stop smoking. However, each day approximately 3000 individuals start smoking, most of whom are young (Pierce et al, 1989). Half of high-school seniors who smoke started by age 14 years. Almost half of all smokers start smoking before 18 years of age, and only 5% start after the age of 20 years. Although 80% of those who smoke say that they would like to stop, only 20% of those who try actually succeed in stopping for good. The likelihood of success in stopping increases with the number of attempts, and those with a college education are twice as likely to break the habit as less educated smokers.

In 1964, only a single life insurance company, State Mutual of Massachusetts, offered a reduced price to nonsmokers. Today, virtually all life insurance companies, even those owned by tobacco conglomerates, now offer significant discounts to persons who do not smoke. Actuarial data leave little doubt that the average life expectancy of a 32-year-old man who smokes cigarettes is 72 years versus 79 years for someone who does not smoke. Smoking-related chronic obstructive pulmonary disease (COPD) is the largest cause of disability payments, and lung cancer is no longer a rarity among men and women in their 40s.

Much is heard about the need to increase tobacco taxes to pay for the increased health care of those who smoke, but the tobacco industry has effectively blunted significant increases. By world standards, cigarette taxes in the United States are very low, ranking 22nd when tax is compared with the total price. U.S. cigarette taxes average 30% of the retail price, whereas the proportion in Denmark is 85%, in Ireland 76%, in India 75%, and in Germany 73% (*American Medical News*, September 5, 1994).

HEALTH RISKS ASSOCIATED WITH SMOKING

Cancer

Forty percent of all cancer deaths are attributable to cigarette smoking. Besides lung cancer, smoking is the major cause of cancer of the larynx, oral cavity, and esophagus (Table 59–1). It is a contributory factor in cancer of the pancreas, bladder, kidney, stomach, and uterine cervix. Recent studies have implicated smoking in leukemia, colon cancer, Graves' disease, depression, and renal disease in persons with diabetes mellitus. A dose-response relationship exists between smoking and all these diseases.

Lung. Lung cancer is 22 times more likely to develop

Table 59–1. Diseases or Conditions Caused Directly or Indirectly by Cigarette Smoking

Cancer	Cardiovascular	Respiratory	Pregnancy	Infants and Children	Other
Lung	Coronary heart disease	COPD (emphysema)	Growth retardation (low birth weight)	Low birth weight	Infertility
Larynx	Stroke	Bronchitis	Preterm labor	Congenital abnormalities	Impotence
Esophagus	Subarachnoid hemorrhage	Pneumonia	Spontaneous abortion	Sudden infant death syndrome	Osteoporosis
Pancreas	Aortic aneurysm	Asthma	Abruptio placentae	Neonatal death	Early menopause
Uterine cervix	Hypertension	Otitis media	Placenta previa		Premature wrinkling
Ovary	Peripheral vascular disease		Bleeding		Peptic ulcer
Colon			Premature rupture of membranes		Alzheimer's disease
Bladder					Graves' disease
Kidney					Insomnia
Breast					Depression
Brain					
Blood (leukemia)					

COPD, chronic obstructive pulmonary disease.

in male smokers and 12 times more likely in female smokers than in those who have never smoked. A clear dose-response relationship exists between lung cancer risk and daily cigarette consumption, and those who smoke more than a pack of cigarettes a day have a risk that is at least 20 times that of nonsmokers.

Unfortunately, early detection does not improve the survival rate for lung cancer. The 5-year survival rate is less than 10% and has not changed since the early 1960s. However, the risk of death from lung cancer is reduced when smoking is discontinued.

The foremost conclusion of the 1964 Surgeon General's report on smoking and health was that cigarettes are the major cause of lung cancer in men (U.S. Department of Health, Education, and Welfare [U.S. DHEW], 1964). Although squamous cell cancer is the most common form in men and adenocarcinoma predominates in women, all four principal histologic types of lung cancer, including small cell and large cell, are associated with smoking. (Damber & Larsson, 1986). A diminished risk for lung cancer is experienced in former smokers after 5 years of cessation; however, the risk remains higher than that of nonsmokers for as long as 25 years (U.S. Department of Health and Human Services [U.S. DHHS], 1990).

From 1950 to 1990, the death rate for lung cancer increased fourfold for men and sevenfold for women. Lung cancer is the principal cause of cancer death for both sexes, and smoking accounts for 87% of lung cancer deaths.

Increasing data regarding the genetic predisposition to lung cancer are emerging. The gastrin-releasing peptide receptor (GRPR) gene, which is activated by nicotine and is located on the X chromosome, may explain the greater risk for smoking-related lung cancer in women. In addition, nicotine addiction and difficulty withdrawing from nicotine appear to be related to the presence of a dopamine receptor gene.

Larynx. The risk for laryngeal cancer is 20 to 30 times greater in smokers. Seventy percent of oral and 85% of laryngeal cancer deaths are directly attributable to smoking.

In several major prospective studies investigating the relationship between smoking and laryngeal cancer, mortality ratios could not be calculated because all the deaths from laryngeal cancer occurred in individuals who had smoked cigarettes. There appears to be a synergistic, multiplicative effect between smoking and drinking such that the risk for development of cancer of the larynx is as much as 75% higher in people who use tobacco and alcohol versus those who are exposed to either substance alone (U.S. DHEW, 1979).

Esophagus. Cigarette smoking is a factor in over half of the cases of esophageal cancer, and the 5-year survival rate is only about 3%. Heavy smokers (more than one pack per day) have 10 times the mortality from esophageal cancer as do nonsmokers.

Pancreas. An equally dismal picture occurs with cancer of the pancreas, for which the 5-year survival rate is only 2%. Because of the nonspecific nature of the initial symptoms and the difficulty in making a diagnosis, the mean survival time after diagnosis is less than 6 months. Smokers have two to three times the risk of pancreatic cancer as nonsmokers do, and the risk is proportional to the amount smoked. Switching from nonfiltered to filtered cigarettes does not decrease the risk. Over one fourth of pancreatic cancer (27%) is attributable to cigarette smoking (Silverman et al, 1994).

Cervix Uteri and Ovary. Women who smoke cigarettes have four times the risk of cervical cancer as nonsmokers. Even women who smoke only 100 cigarettes during their lifetimes more than double their risk of cervical cancer. The risk from smoking is greater in women younger than 30 years than in those older than 30 (Slattery et al, 1989).

Constituents from cigarette smoke are distributed by the blood throughout the body and have been detected in the cervical mucus of smokers at levels 40 to 50 times those in serum.

The risk of ovarian cancer is three times greater in women who smoke cigarettes (Qian et al, 1989).

Bladder and Kidney. Forty percent of bladder cancers are smoking related, and higher rates of kidney cancers are also noted in smokers. Smokers have a three to four times higher risk of bladder cancer than do people who never smoked. The kidneys and bladder are the final common pathway for the concentration of toxic products of tobacco smoke and provide the longest direct exposure to carcinogens and radioactive substances,

such as polonium 210, in tobacco smoke (Winters & Di Franza, 1982).

Colon and Rectum. A strong relationship has been noted between smoking and colorectal cancer, but the induction period is about 35 years. This lengthy induction period would explain why it is just beginning to show up in women and shows that our efforts to prevent smoking among the young should be intensified (Giovannucci et al, 1994).

Leukemia. A greater than 50% increased mortality from leukemia occurs in cigarette smokers (relative risk, 1.53), and the response is dose related. Those smoking more than one pack per day have a twofold increased risk (Kinlen & Rogot, 1988). The risk is greatest for myeloid leukemia and acute nonlymphocytic leukemia. Approximately 14% of all cases of leukemia in the United States may be due to cigarette smoking (Brownson et al, 1993). Overall, smoking cigarettes increases a person's risk for leukemia by 30%.

Chronic Obstructive Pulmonary Disease

Cigarette smoking is the main cause of COPD, which is the leading cause of disability in the United States. Changes in bronchi and the lung parenchyma are proportional to the amount of smoke inhaled. Cigarette smoke inhibits ciliary activity of the bronchial epithelium and phagocytic activity of macrophages in the alveoli. This reduced activity results in decreased clearance of foreign material and bacteria from the lung, which leads to increased infection and tissue destruction.

Even after age 60 years, smokers who quit have better pulmonary function than those who continue smoking. Lung function is inversely related to the number of cigarettes smoked during one's lifetime. Smokers at age 65 or older who quit smoking before age 40 have pulmonary function levels similar to those of people who never smoked (Higgins et al, 1993).

Cardiovascular Disease

CORONARY HEART DISEASE

Nicotine raises systolic blood pressure, the heart rate, and cardiac output and causes vasoconstriction. The relationship between cerebral vasoconstriction and anoxia and the intake of carbon monoxide resulting from cigarette smoking could explain the 50% increase in automobile accidents in smokers. The symptoms associated with carbon monoxide intoxication can be a problem, especially for persons with an already compromised coronary circulation. Carbon monoxide has an affinity for hemoglobin (forming carboxyhemoglobin) that is 245 times stronger than that of oxygen. Thus it reduces oxygen delivery to the myocardium and has a decidedly negative inotropic effect. Carboxyhemoglobin also lowers the threshold for ventricular fibrillation and could help explain the higher incidence of sudden death in those who smoke.

The risk of myocardial infarction is proportional to the number of cigarettes smoked. The trend toward the use of filtered cigarettes does not appear to have reduced the risk of coronary heart disease. Theoretically, filters on cigarettes reduce the amount of tar (the condensate of tobacco smoke that comprises over 3000 compounds, including more than 40 carcinogens), but they may increase the amount of carbon monoxide, thus contributing to the increased mortality from coronary heart disease. Persons who smoke cigarettes containing low amounts of nicotine have the same degree of risk of myocardial infarction as those who smoke cigarettes containing larger amounts. Smokers of these low-dose cigarettes still have three times the risk of myocardial infarction as nonsmokers (Kaufman et al, 1983). The good news is that the risk of sudden death decreases immediately on stopping and, within a few years of stopping, the risk of myocardial infarction decreases to a level similar to that in men who have never smoked, even in heavy smokers who have a positive family history of coronary heart disease (Rosenberg et al, 1985).

Three fourths of myocardial infarctions in women younger than 50 years have been attributed to smoking (Slone et al, 1978). The Chief Medical Examiner of Dade County, Florida, states that a woman between 40 and 50 years of age who dies suddenly is considered to be a cigarette smoker until proved otherwise (J. Davis, personal communication, 1977). The risk of myocardial infarction increases progressively to as much as 20-fold in persons smoking 35 or more cigarettes per day. There is no safe level of smoking. Women who smoke only 1 to 4 cigarettes a day have a 2.5 times greater risk of coronary heart disease. Women who smoke and use oral contraceptives have a risk of heart attack that is 10 times greater than that of women who do neither.

Silent ischemia probably accounts for the majority of all cardiac ischemic events. Patients with coronary heart disease who smoke have three times as many episodes of silent ischemia as nonsmokers, and the duration of each is 12 times longer (Barry et al, 1989). Frequent episodes of myocardial ischemia, even though asymptomatic, must damage the heart. Because smoking also increases platelet adhesiveness and lowers high-density lipoprotein cholesterol, the association with a higher incidence of myocardial infarction is no surprise.

Benefits from stopping smoking can be demonstrated at all ages. No decrease in benefit is seen as one gets older, so it is still worthwhile for someone older than 65 to break the addiction (Hermanson et al, 1988; LaCroix et al, 1991). This benefit can be demonstrated in the cerebral as well as the coronary circulation. Elderly individuals who stop smoking have significantly higher cerebral perfusion levels than do those who continue to smoke. Even those who have smoked for 30 to 40 years have improved cerebral circulation within a relatively short time after stopping smoking (Rogers et al, 1985).

Persons who smoke more than one pack of cigarettes a day are four times more susceptible to Alzheimer's disease than nonsmokers are. As with other smoking-related diseases, this one is also dose dependent; those smoking less than one pack a day are at 1.6 times the risk.

STROKE

Stroke is the third most common cause of death in the United States. Although hypertension is the greatest risk factor for stroke, cigarette smoking is also a significant factor. The incidence of stroke in smokers is 50% higher than in nonsmokers (40% higher in men and 60% higher in women) (Wolf et al, 1988).

The risk of stroke increases in proportion to the amount of smoking; it is twice as great in those who smoke more than 40 cigarettes per day than in those smoking fewer than 10 cigarettes per day.

When compared with women who have never smoked, the risk of stroke increases 2.2-fold in women smoking 1 to 14 cigarettes per day and 3.7-fold in women smoking 25 or more cigarettes daily (Colditz et al, 1988). A clear dose-response relationship has also been noted by Bonita and associates (1986). They found a 3-fold increase in the risk of stroke in smokers in comparison to nonsmokers (Fig. 59–1). The risk is 5.6 times higher in persons smoking more than one pack of cigarettes daily. Cigarette smokers who are also hypertensive have a 20-fold increased risk of stroke.

Sclerosis of the carotid arteries is directly proportional to the amount of smoke exposure. Smoking increases the risk of ischemic heart disease and cerebrovascular disease regardless of the level of serum cholesterol. Jee and colleagues (1999) found that a low cholesterol level did not protect against smoking-related arteriosclerotic cardiovascular disease in patients in South Korea, where the prevalence of smoking is among the highest in the world at 72% of men.

Smoking may increase the likelihood of thrombosis by increasing serum fibrinogen, enhancing platelet aggregation, and increasing blood viscosity.

The risk of stroke declines rapidly after cessation of smoking and, after 5 years, is at the level of nonsmokers, which emphasizes that it is never too late to quit no matter how long one has been smoking.

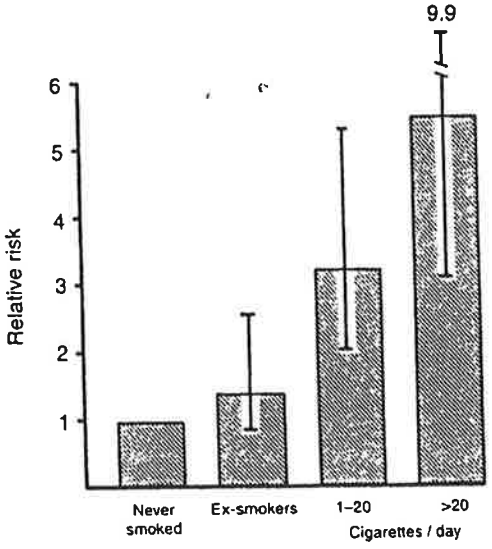

Figure 59–1. Cigarette smoking and risk of stroke, adjusted for age and sex. Bars indicate 95% confidence limits. (From Bonita R, Scragg R, Stewart A: Cigarette smoking and risk of premature stroke in men and women. BMJ 293:6, 1986.)

SUBARACHNOID HEMORRHAGE

Habitual smoking increases the risk of subarachnoid hemorrhage 3.9 times for men and 3.7 times for women. The risk increases to 22 times that of nonsmokers in women who both smoke and use oral contraceptives (Bell & Symon, 1979).

One theory is that structural damage occurs in the wall of cerebral vessels and causes aneurysms that are more likely to rupture. In a meta-analysis of all available data regarding cigarette smoking and stroke, Shinton and Beevers (1989) confirmed the 50% increased risk of stroke associated with cigarette smoking and found that the risk of subarachnoid hemorrhage tripled and was greater in women than men.

Other Diseases and Conditions

Graves' Disease. Smoking appears to be one of the multiple factors causing Graves' disease in genetically predisposed individuals. Family members of patients with Graves' disease may be able to prevent the development of this disease by stopping smoking (Prummel & Wiersinga, 1993).

Diabetes Mellitus. The risk of diabetes increases with the number of cigarettes smoked. People smoking more than one pack a day have 1.5 times the risk for diabetes as those who smoke 1 to 14 cigarettes. Albuminuria as a sign of early renal damage and retinopathy is greater in patients with insulin-dependent diabetes mellitus who smoke and can be shown to improve significantly if the person stops smoking (Chase et al, 1991).

Depression. Smokers are more likely to experience major depression than nonsmokers are, and the incidence increases steadily with the number of cigarettes smoked. Conversely, it is estimated that one third of smokers are depressed and self-medicate with tobacco. Kendler and associates (1993) suggested that this increased risk could be due to genes that predispose to both conditions.

Insomnia. Smokers are more likely than nonsmokers to have insomnia and, as a consequence, to feel tired in the morning. Smokers will be more restless during sleep and more likely to awaken tired and then smoke during the day for the stimulation. However, smokers also consume more alcohol and caffeine than nonsmokers do, which will contribute to insomnia (Lexcen & Hicks, 1993).

Wrinkles. Every day 3000 children try their first cigarette; 750 of these children will die of a smoking-related disease. We are not very effective in getting the message across to this group—by talking about disease, we may not be speaking their language. The fact that smoking causes wrinkles, bad breath, and yellow teeth may be a more effective message than evidence that smoking kills. Premature wrinkling (crow's feet) increases with the number of cigarettes smoked. Kadunce and associates (1991) found that heavy smokers are five times more likely to have wrinkles than nonsmokers are.

Limb Malformations. Mothers who smoke cigarettes during the first trimester of pregnancy are more

likely to give birth to children with limb reduction malformations, similar to the limb deformities in cattle who feed on the tobacco plant.

Macular Degeneration. Macular degeneration is the leading cause of blindness after age 65, and nothing prevents or delays its progression. Smoking 20 or more cigarettes a day increases the risk of macular degeneration twofold to threefold. As with other smoking-related disorders, macular degeneration also appears to be dose related, with the incidence increasing with the number of pack-years (Christen et al, 1996; Seddon et al, 1996).

OTHER TOBACCO-RELATED HEALTH RISKS

Filtered Cigarettes

Cigarette advertising campaigns have long tried to allay the consumer's concern about smoking. In the 1950s, faced with declining sales after the publication of studies linking smoking to lung cancer, tobacco companies began producing filtertip brands that were claimed to remove certain components of smoke that manufacturers have never publicly acknowledged to be harmful. Incredibly, until the 1980s the American Cancer Society, the National Cancer Institute, and most major health organizations supported the concept of a "less hazardous" cigarette in the belief that most people who smoke would not or could not stop. Today, a mistaken popular belief persists that filtered brands of cigarettes (which now account for more than 97% of those sold in the United States) are safer than nonfiltered cigarettes. Low-tar and low-nicotine filtered cigarettes are now advertised widely. Because the addiction is to nicotine, people who smoke low-nicotine cigarettes undergo "compensatory smoking" in which they inhale more frequently and more deeply to maintain their blood nicotine levels. As a result, tar intake also increases, so the cigarette changes from the low-tar to the high-tar category. Smokers who take 14 puffs per cigarette inhale 58% more tar than do those taking the standard 8.7 puffs per cigarette. Some manufacturers include perforations in the filter to dilute the smoke with air and advertise these cigarettes as ultra-low-tar. Many smokers, however, block the holes with their lips or their fingers to obtain undiluted smoke with a higher concentration of nicotine (Kozlowski et al, 1980).

Cigarettes with reduced yields of nicotine and carbon monoxide are not safer. The fourfold increased risk of myocardial infarction does not vary according to the nicotine content, and the degree of risk is proportional to the number of cigarettes smoked (Palmer et al, 1989). Nicotine blood levels are similar for cigarette smokers, pipe smokers, and users of snuff despite the different methods of absorption.

Only in 1995 did the Federal Trade Commission (charged with monitoring advertised tar and nicotine levels) and the Food and Drug Administration (FDA) recognize the problem of compensatory smoking and challenge the fallaciousness of tar and nicotine ratings. However, should the FDA succeed in mandating a maximum level of nicotine in cigarettes, it may well assist the tobacco industry once again in enabling consumers to rationalize their continued smoking of implicitly less addictive brands. Cigarettes that are very low in nicotine may well facilitate smoking in adolescents.

Cigars

An alarming trend has been the increasing popularity of cigar smoking, perhaps as a result of the mistaken conception that because most cigar smokers do not inhale, cigars are a safer form of smoking. Just the opposite is the case, however. One large cigar carries the nicotine kick of four or five cigarettes and may contain even more carcinogens. Even an occasional cigar such as a few per week can produce nicotine craving (Jacobs et al, 1999).

Cigar smoking increased by nearly 50% between 1993 and 1997 because of promotion by popular "macho" film stars and glamorization in the media. In 1997, 31% of high school boys and 11% of high school girls reported smoking a cigar within the past month. (CDC, 1998)

Iribarren and colleagues (1999) found that cigar smokers were at higher risk for coronary artery disease than nonsmokers were, as well as for cancer of the oropharynx, nose, larynx, esophagus, and lung. As with cigarette smoking, there appears to be a synergistic relationship between cigar smoking and alcohol consumption.

Smokeless Tobacco

Smokeless tobacco comes in two types: snuff, which is dry or moist, and chewing (spitting) tobacco, which comes as loose leaf, plug, or twist. Use of these substances increases the frequency of oral-pharyngeal cancer and gum recession. Long-term users of snuff have a 50-fold increased risk for cancer of the cheek and gum (Koop & Luoto, 1982). Leukoplakia is found in 18% to 64% of users (Connolly et al, 1986).

Smokeless tobacco contains the same carcinogens as cigarette tobacco, but some of them are present in much greater concentration. Nitrosamines, which are powerful chemical carcinogens, are present at levels up to 14,000 times higher than the federal government allows in bacon and beer (Connolly et al, 1986).

A large percentage of the estimated 10 million users of smokeless tobacco in the United States are male adolescents who mistakenly believe it to be a relatively safe alternative to smoking. Most users start at 10 to 12 years of age (Evans, 1988).

Although educational programs have been launched by the National Cancer Institute and Major League Baseball, an upward trend in smokeless tobacco use has occurred in adolescents. College athletes have been found to believe that male peers, coaches, and professional athletes are indifferent to the use of spitting tobacco (Hilton et al, 1994). In one study across geographic lines, 12% of 2000 students in the sixth through

ninth grades reported using smokeless tobacco (Gottlieb et al, 1993). Eighth graders in rural areas are five times more likely to "dip" snuff. Both professional and college rodeos continue to welcome sponsorship by smokeless tobacco companies, as do leading country music singers in concerts held on university campuses, where free samples are distributed. Ominously, in recent years smokeless tobacco manufacturers have promoted candy-flavored snuff products in convenient and less messy tea bag–like pouches (e.g., Skoal Bandits). Internal documents from one company published in the news media in 1995 revealed an apparent strategy to graduate users from sweeter, lower-nicotine products to stronger, higher-nicotine brands.

Involuntary (Passive) Smoking

The effects of tobacco on nonsmokers (passive smoking) can be significant. An estimated 3000 nonsmokers die each year from inhaling secondhand smoke. In addition, 15% of the American public is allergic to cigarette smoke. Two thirds of the smoke from a burning cigarette never reaches a smoker's lungs, but instead goes directly into the air. *Sidestream smoke* is what is emitted into the air from a smoldering cigarette between puffs, whereas *mainstream smoke* is what the smoker inhales directly during puffing. Although diluted by air before being inhaled, sidestream smoke contains greater amounts of toxic substances than mainstream smoke does because of a lower combustion temperature and lack of filtration through the cigarette (Table 59–2).

Over 3000 different chemicals have been identified in cigarette smoke, and at least 40 of them are known carcinogens. The U.S. Environmental Protection Agency has determined that environmental tobacco smoke is a "class A" human carcinogen, in the same class as asbestos, mustard gas, arsenic, and benzene. We have cleared our schools of asbestos, but three fifths of schools have yet to ban smoking. In addition to the 3000 lung cancer deaths a year in nonsmokers, almost 40,000 heart disease deaths each year are linked to secondhand smoke.

A nonsmoker who spends 1 hour in a smoke-filled car on a commuter train inhales the equivalent of nine filtered cigarettes (Aronow, 1979). Similarly, it has been estimated that a nonsmoking musician who plays in a smoke-filled club and lives with a chain-smoking roommate inhales the equivalent of 27 cigarettes a day. Food service workers are also at increased risk. We protect a patron in restaurants who prefers a table in a nonsmoking area, but the workers go unprotected. Siegel (1993) found the level of tobacco smoke in bars to be four to six times higher than that in offices and that in restaurants to be almost twice as high. He believes that this increased level of smoke may result in a 50% higher lung cancer risk in food service workers.

Hirayama (1981) demonstrated an increased risk of lung cancer in nonsmoking housewives exposed to the secondhand cigarette smoke of their husbands (Fig. 59–2). The risk from passive smoking was one half to one third that of direct smoking. A direct dose-response relationship was observed, with the annual mortality from lung cancer being 8.7 per 100,000 for women whose husbands smoked only occasionally and 18.1 per 100,000 for those whose husbands smoked 20 or more cigarettes daily. The wives of heavy smokers had a twofold greater risk of dying of lung cancer than did wives of nonsmoking men. Their risk was half that of women smokers.

A similar study in Sweden found that women with husbands who smoke have three times the risk of lung

Table 59–2. Toxic and Tumorigenic Agents of Cigarette Smoke; Ratio of Sidestream Smoke to Mainstream Smoke

Agent	Amount per Cigarette	SS/MS Ratio
Gas Phase		
Carbon dioxide	10–80 mg	8.1*
Carbon monoxide	0.5–26 mg	2.5*
Nitrogen oxides (NO_x)	16–600 μg	4.7–5.8
Ammonia	10–130 μg	44–73
Hydrogen cyanide	280–550 μg	0.17–0.37
Hydrazine	32 μg	3
Formaldehyde	20–90 μg	51
Acetone	100–940 μg	2.5–3.2
Acrolein	10–140 μg	12
Acetonitrile	60–160 μg	10
Pyridine	32 μg	10
3-Vinylpyridine	23 μg	28
N-Nitrosodimethylamine	4–180 ng	10–830
N-Nitrosoethylmethylamine	1.0–40 ng	5–12
N-Nitrosodiethylamine	0.1–28 ng	4–25
N-Nitrosopyrrolidine	0–110 ng	3–76
Particulate Phase		
Total particulate matter	0.1–40 mg	1.3–1.9*
Nicotine	0.06–2.3 mg	2.6–3.3*
Toluene	108 μg	5.6
Phenol	20–150 μg	2.6
Catechol	40–280 μg	0.7
Stigmasterol	53 μg	0.8
Total phytosterols	130 μg	0.8
Naphthalene	2.8 μg	16
1-Methylnaphthalene	1.2 μg	26
2-Methylnaphthalene	1.0 μg	29
Phenanthrene	2.0–80 μg	2.1
Benz(*a*)anthracene	10–70 μg	2.7
Pyrene	15–90 ng	1.9–3.6
Benzo(*a*)pyrene	8–40 ng	2.7–3.4
Quinoline	1.7 μg	11
Methylquinoline	6.7 μg	11
Harmane	1.1–3.1 μg	0.7–2.7
Norharmane	3.2–8.1 μg	1.4–4.3
Aniline	100–1200 ng	30
α-Toluidine	32 ng	19
1-Naphthylamine	1.0–22 ng	39
2-Naphthylamine	4.3–27 ng	39
4-Aminobiphenyl	2.4–4.6 ng	31
N'-nitrosonornicotine	0.2–3.7 μg	1–5
NNK	0.12–0.44 μg	1–8
N'-Nitrosoanatabine	0.15–4.6 μg	1–7
N-Nitrosodiethanolamine	0–40 ng	1.2

*In cigarettes with perforated filter tips, the SS/MS ratio rises with increasing air dilution. In the case of smoke dilution with air to 17% the SS/MS ratio for total particulate matter rises to 2.14, that for CO_2 to 36.5, that for CO to 23.5, and that for nicotine to 13.1.

NNK, 4-(methylnitrosamino)-1-(3-pyridyl)-butanone.

From The Health Consequences of Smoking: Cancer. A Report of the Surgeon General. Rockville, MD, US Department of Health and Human Services, Public Health Service, Office on Smoking and Health, DHHS Publication No. (PHS) 82-50179, 1982.

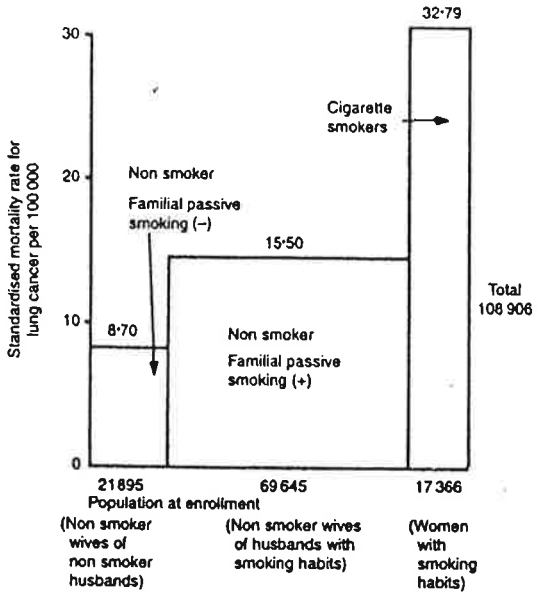

Figure 59–2. Lung cancer mortality in women according to the presence or absence of direct and familial indirect smoking. (From Hirayama T: Nonsmoking wives of heavy smokers have a higher risk of lung cancer: A study from Japan. BMJ 282:183, 1981.)

cancer as wives of nonsmoking husbands (Pershagen et al, 1987). At least 14 studies have shown an association between being married to a smoker and having an increased risk of lung cancer. Overall, about one third of lung cancers occur in nonsmokers living with smokers (Fontham et al, 1994).

Cancer risk appears to be proportional to the total amount of smoke to which an individual is exposed during a lifetime. The risk of development of cancer of any form appears to be dose dependent in that it increases by at least 50% in persons exposed only during childhood or adulthood and more than doubles for those exposed during both periods. The risk of cancer increases significantly with increasing exposure. It is greatest for cancer of the breast and cervix and for leukemia and lymphoma (Garfinkle, 1980; Raeburn, 1989).

Passive smoking increases the risk of cervical cancer. Slattery and associates (1989) found that passive exposure to smoke for 3 hours a day increases the risk of development of cervical cancer 3.43 times. One hour of passive smoking exposes the person to carcinogenic nitrosamines equivalent to smoking one-half pack of filtered cigarettes. Thus the risk of cancer from passive smoking can be as great as that from personal cigarette smoking.

The risks of passive smoking extend far beyond cancer. It is estimated that tobacco smoke in the home and workplace could be responsible for the deaths of 46,000 nonsmokers annually in the United States. Most of these deaths are due to heart disease, and consequently, passive smoking is the third leading preventable cause of death after alcohol and smoking itself. It is estimated that the risk of myocardial infarction is three times higher for a woman whose husband smokes (Wells, 1988).

EFFECTS ON CHILDREN

Parents who smoke are more likely to have children who will take up smoking. Indeed, 75% of those who smoke cigarettes had at least one parent who smoked. The risk of a child taking up smoking doubles with each additional adult family member who smokes. Over 50% of children younger than 5 years live in homes with at least one adult smoker. Children of smoking parents are innocent victims (involuntary smokers) and have been shown to be more likely to suffer more bronchitis and pneumonia during their first year of life and more otitis media when older. Numerous studies have shown that they have an increased incidence of cough, bronchitis, and pneumonia that is proportional to the number of cigarettes smoked by the parents, particularly the mother. In fact, children of parents who smoke at least half a pack a day have nearly twice the risk of hospitalization for a respiratory illness. Asthma is also more prevalent in children whose mothers smoke, and their stature is retarded in proportion to the number of smokers in the home (Charlton, 1994; Rantakallio, 1978). Passive smoking has also been blamed for some instances of sudden infant death syndrome.

Small children are victimized more by passive smoking than adults are. Because of more rapid breathing, they inhale larger amounts of harmful substances. Children exposed to their parents' cigarette smoke have six times the average number of respiratory infections. They also have deficits in growth and in intellectual and emotional development, as well as more behavior disorders, such as hyperactivity.

The risk of cancer is increased by 50% in children of men who smoke. The risk of hematopoietic cancer developing in a child is 4.6 times greater if both parents smoke (Sandler et al, 1985a).

EFFECTS ON PREGNANCY

A dose-response relationship also exists for cigarette smoking during pregnancy. The more a pregnant woman smokes, the lower the infant's birth weight is likely to be. On average, babies born to women who smoke during pregnancy are 200 g lighter than those born to comparable nonsmokers (Fig. 59–3). Heavy smokers have a 130% increased incidence of newborns weighing less than 2500 g. However, a woman who gives up smoking by her fourth month of gestation will have the same risk as a nonsmoker. Mainous and Hueston (1994a) found that women who stopped smoking in the first trimester had 26% fewer preterm deliveries and 18% fewer low-birth-weight infants. Each cigarette smoked per day is associated with a 10-g decrease in infant birth weight, and a direct relationship exists between the degree of smoking and infant weight reduction, with infants born to light, moderate, and heavy smokers weighing 96, 183, and 200 g less, respectively, than those born to nonsmokers (Abell et al, 1991). Pregnant women who do not smoke but whose passive smoke exposure is high are twice as likely as those with low exposure to have a low-birth-weight infant (Mainous & Hueston, 1994b).

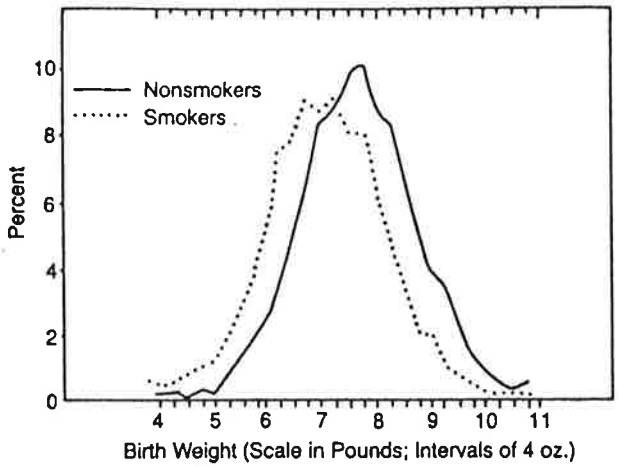

Figure 59–3. Percent distribution by birth weight of infants of mothers who did not smoke during pregnancy and those who smoked one pack or more of cigarettes per day. (From US Department of Health, Education, and Welfare: Smoking and Health. A Report of the Surgeon General, Washington, DC, US Department of Health, Education, and Welfare, Public Health Service, Office on Smoking and Health, DHEW Publication No. [PHS] 79–50066, 1979, pp 8–43.)

Unfortunately, most women smokers do not quit smoking during their pregnancy. In fact, in one study none of the 112 women referred to smoking cessation classes actually attended the classes (Ebrahim et al, 2000; O'Connor et al, 1992).

The term "fetal tobacco syndrome" provides a label for fetal growth retardation when (1) the mother smoked five or more cigarettes a day throughout the pregnancy, (2) the mother had no evidence of hypertension, (3) the newborn has symmetrical growth retardation, and (4) no other cause of intrauterine growth retardation is obvious (Nieburg et al, 1985).

Transplacental exposure to substances absorbed from the mother's smoking during pregnancy may predispose the infant to cancer later in life (Sandler et al, 1985b). Infants born to women who smoke during pregnancy show a significant accumulation of cigarette smoke toxins when tested 1 to 3 days after delivery. Although the levels of such toxins were highest in women who smoked, they were also significantly higher in mothers who were passive smokers than in nonsmokers (Eliopoulos et al, 1994).

The risk of spontaneous abortion in heavy smokers is 1.7 times that in nonsmokers. Smoking during pregnancy increases the incidence of abruptio placentae, placenta previa, bleeding during pregnancy, and premature rupture of membranes. It also increases the incidence of premature births and perinatal deaths (Fig. 59–4). Obviously, pregnancy is an opportune time for the family physician to encourage women to discontinue smoking.

About 25% of women who smoke at the beginning of their pregnancy will stop on their own sometime during the 9 months. Aggressive intervention programs by physicians could influence another 30% to stop. The greatest effort should be directed toward pregnant unmarried white women because they are 40% more likely to smoke than are nonpregnant white women (Williamson et al, 1989).

Strong experimental evidence indicates that maternal smoking causes fetal hypoxia, which could explain the increased incidence of congenital abnormalities noted in babies of smokers (Fig. 59–5). The offspring of mothers who smoke during the 3 months before or after conception are twice as likely to have a cleft palate as the offspring of nonsmokers (Khoury et al, 1989). The increased frequency of placenta previa in women who smoke could be caused by placental hypertrophy occurring as a result of the carbon monoxide hypoxemia (Williams et al, 1991).

Reduced fertility is also a problem in women who smoke cigarettes. Smokers are three to four times more likely to take longer than 1 year to conceive, and heavy smokers have more difficulty than light smokers do. Spermatozoa from smokers also show more morphologic abnormalities and less motility than do spermatozoa from nonsmokers.

Breast-feeding women who smoke cigarettes wean their infants earlier than do women who do not smoke, possibly because of the reduced amount of milk and

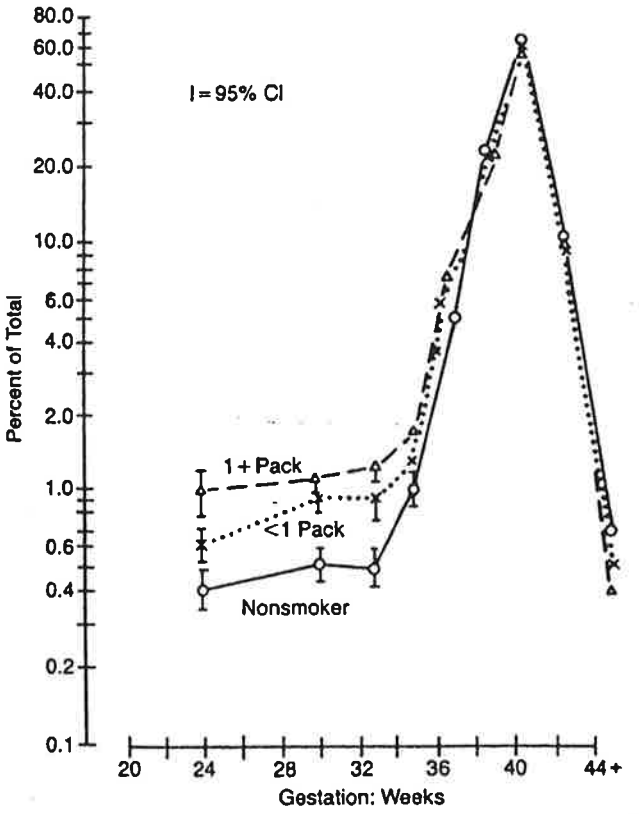

Figure 59–4. Percent distribution by weeks of gestation of births to nonsmokers, smokers of less than one pack per day, and smokers of one pack per day or more. (From US Department of Health, Education, and Welfare: Smoking and Health. A Report of the Surgeon General. Washington, DC, US Department of Health, Education, and Welfare, Public Health Service, Office on Smoking and Health, DHEW Publication No. [PHS] 79–50066, 1979, pp 8–17.)

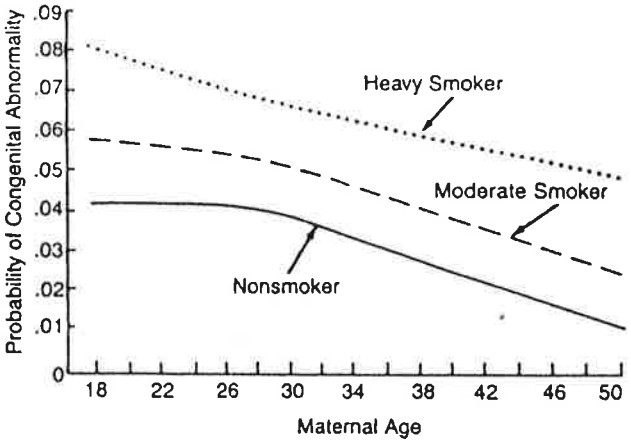

Figure 59–5. Risk of congenital abnormality in an infant according to age and smoking habit of the mother. (From Himmelberger DU, Brown BW Jr, Cohen EN: Cigarette smoking during pregnancy and the occurrence of spontaneous abortion and congenital abnormality: Am J Epidemiol 108:477, 1978. Study supported by Contract No. HSM 99–73–3, National Institute for Occupational Safety and Health.)

lower fat concentration in the milk of these mothers (Hopkinson et al, 1992).

FAMILY PHYSICIAN INVOLVEMENT IN ENDING THE TOBACCO PANDEMIC

A remarkable grassroots antismoking movement that arose in the 1970s has had a major impact on the goal of achieving a smoke-free society and has impelled traditional health organizations such as the American Cancer Society and the American Medical Association to become more outspoken. The first medical organization to develop proven strategies for the clinic, classroom, and community aimed at counteracting tobacco use and promotion was Doctors Ought to Care (DOC), founded in 1977 by a family physician at the University of Miami (Blum, 1980a). Since its inception, DOC has been supported by the American Academy of Family Physicians and the National Conference of Family Practice Residents and Student Affiliates. "Tar Wars," an annual antismoking poster contest for schoolchildren, is a DOC offshoot that has been adopted by numerous state and local family practice organizations.

The five foci of tobacco control, the accepted term for the emerging field of public health, include the following: increases in cigarette excise taxes, bans on tobacco advertising and promotion, restrictions on teenagers' access to tobacco products, pharmacologic and behavioral smoking cessation strategies, and legislation to prohibit smoking in public areas and the workplace.

Other tobacco control efforts include regulatory warning labels on cigarette packages, divestment of tobacco stocks, enforcement of laws against cigarette smuggling, an end to tobacco subsidies, and rejection of donations and research grants from the tobacco industry. The American Cancer Society's most visible antismoking effort is an annual day-long event in November, "The Great American Smoke-Out," during which people who smoke are encouraged to quit and use a nicotine-replacement product instead.

Lawsuits brought against cigarette manufacturers by individuals made ill by tobacco had been pursued unsuccessfully for 30 years until 1988, when a New Jersey jury awarded $400,000 to the widower of Rose Cippolone, who had died of lung cancer after having smoked for four decades. Although this judgment and others awarded to individual plaintiffs have been overturned by higher courts, they paved the way for larger class-action suits and attempts by state attorneys general and the U.S. Department of Justice to sue for recovery of the Medicaid costs for caring for persons with tobacco-related diseases.

The culmination of litigious activity came in 1998 with the settlement between the tobacco industry and the states attorneys general in the amount of $207 billion to be paid over 25 years. Although the settlement held promise for a vigorous primary prevention effort to reduce demand for smoking in adolescents, little of this funding has been allocated to tobacco control, and various state and bureaucratic agencies charged with this responsibility have lacked creativity and forcefulness. Similarly, although medical societies have unanimously passed resolutions supporting the new war on tobacco, they have not backed up their words with commitments of manpower and money.

SMOKING CESSATION*

Ideally, the validity of the abstinence rate for a method of smoking cessation should rest on the performance of a controlled, double-blind study with follow-up of at least 6 months' duration of all subjects who started out (Schwartz, 1969, 1979, 1987). Few published outcome evaluations meet such criteria. Before the introduction of nicotine replacement products in 1984, smoking cessation techniques in the United States consisted of a hodgepodge of unproven but much-touted chemical remedies, diets, aversive stimuli, hypnotherapy, self-help manuals, special filters, acupuncture, and expensive behavior modification clinics or seminars. Many of these methods are quite costly, but having to pay a high price may well be related to the alleged success of a given method.

When the FDA approved the use of nicotine-containing chewing gum (Nicorette) for smoking cessation, the product gained immediate popularity. However, although the gum was approved for use as an adjunct to a comprehensive program of behavior modification, most physicians offered few instructions and little follow-up. Moreover, some patients became dependent on the gum and perpetuated their smoking by using the gum at times and in places where they were not permitted to smoke. The high success rates reported in clinical trials may be attributed in part to the fact that the research was conducted in clinics that specialize in the treatment of smoking cessation. This difference may further ex-

*Method of Alan Blum.

plain why placebo groups in some studies fared better than the intervention groups of most other methods.

In 1992, all smoking cessation methods began to take a back seat to use of the transdermal nicotine patch. The theory behind the patch is that controlled, continuous release of nicotine provides partial replacement of the nicotine from smoking, thereby reducing the craving and preventing withdrawal. As with users of nicotine gum, relapse is a problem in patients who use the patch. The most significant problem in clinical practice appears to be a combination of the patient's heightened expectations for the patch (based on word-of-mouth testimonials and advertising in the mass media) and the physician's overeager acquiescence in prescribing it. Pharmaceutical company claims notwithstanding, smoking is not simply an addiction to nicotine. Social and psychologic factors also play determining roles. Promotions for various pharmacologic agents for smoking cessation wrongly reinforce the notion that smoking is primarily a medical problem with a simple, prescribable, nonindividualized solution. When a patient requests a drug "that will make me stop smoking," the physician, although not wishing to dash expectations, should emphasize that a drug is an adjunct, not the single solution.

The updated clinical practice guideline *Treating Tobacco Use and Dependence*, published by the U.S. DHHS, has added bupropion sustained release (SR) (Zyban), nicotine inhaler (Nicotrol), and nicotine nasal spray to its list of first-line medications that patients should be encouraged to use (Table 59–3). All three are available exclusively by prescription. Nicotine gum and transdermal nicotine, the only two recommended medi-

Table 59–3. Clinical Guidelines for Nicotine Withdrawal

Nicotine Patch

Patches should be applied as soon as patients awaken on their quit day
At the start of each day, the patient should place a new patch on a relatively hairless location between the neck and waist
No activity restrictions while using the patch
Treatment for 8 wk or less is as effective as longer treatment periods
Dosage
Nicoderm, Habitrol: 21 mg/24 hr for 4 wk, then 14 mg/24 hr for 2 wk, then 7 mg/24 hr for 2 wk
Nicotrol: 15 mg/16 hr for 4 wk, then 10 mg/16 hr for 2 wk, then 5 mg/16 hr for 2 wk
ProStep: 22 mg/24 hr for 4 wk, then 11 mg/24 hr for 4 wk

Nicotine Gum

Gum should be chewed slowly until a "peppery" taste emerges and then "parked" between the cheek and gum to facilitate nicotine absorption through the oral mucosa. The gum should be slowly and intermittently "chewed and parked" for about 30 min
Acidic beverages (e.g., coffee, juices, soft drinks) interfere with the buccal absorption of nicotine, so eating and drinking anything except water should be avoided for 15 min before and during chewing
Instructing patients to chew the gum on a fixed schedule may be more beneficial than ad lib use. Patients often do not use enough gum to get the maximum benefit
Dosage
Nicorette: Available as 2 mg and 4 mg per piece. Smokers of more than 1 pack a day, those who smoke within 30 min of awakening, and those with a history of severe withdrawal symptoms should use 4 mg; light smokers should use 2 mg
Chew 1 piece every 1–2 hr (at least 9 /day) for 6 wk, then 1 piece every 2–4 hr for 3 wk, then 1 piece every 4–8 hr for 3 wk, then discontinue
For the 2-mg dose, do not exceed 30 pieces per day; for the 4-mg dose, 20 pieces per day

Bupropion SR

Contraindicated in patients with a history of a seizure disorder or eating disorder and in those who have used a monoamine oxidase inhibitor in the past 14 days
Side effects are insomnia and dry mouth. If insomnia is present, take the evening dose in the afternoon, but at least 8 hr after the first dose
Dosage
Zyban: 150-mg tablets, 1 every morning for 3 days and then 1 b.i.d. Start 2 wk before the "target quit date" and continue for up to 12 wk

Nicotine Inhaler

Local irritation in the mouth and throat occurs in 40% of patients
Coughing and rhinitis are also common. The severity and frquency of these symptoms decline with continued use
In cold weather the inhaler and cartridges should be kept in an inside pocket or warm area because nicotine delivery declines significantly at temperatures below 40°F
Dosage
Nicotrol Inhaler: 10 mg per cartridge (4 mg delivered and 2 mg absorbed). Each cartridge lasts about 20 min with frequent puffing and is equivalent to about 2 cigarettes. Use 6–16 cartridges per day for the first 12 wk, then reduce gradually over 12 wk

Nicotine Nasal Spray

Moderate nasal irritation for first 3 wk or more. Nasal congestion and transient changes in sense of smell and taste may also occur
Should not be used in patients with severe reactive airway disease
Do not sniff, swallow, or inhale through nose while administering doses
Deliver with head tilted slightly back
Dosage
Nicotrol NS: One spray (0.5 mg) to each nostril (1.0 mg total). Use 1–2 doses per hr and 8–40 doses per day (maximum of 5 doses per hr). Each bottle contains 100 doses. Use for maximum of 12 wk

Some patients may prefer the nasal spray or inhaler because of the more rapid delivery of nicotine simulating smoking. Others may prefer bupropion because it is nonnicotine therapy. Bupropion should be considered especially in those with a history of depression.

Modified from US Department of Health and Human Services: Treating Tobacco Use and Dependence: A Clinical Practice Guideline. Rockville, MD, Agency for Health Care Policy and Research, Public Health Service, 2000.

cations in the original guideline in 1996, remain on the list (U.S. DHHS, 1996). The gum is now available exclusively as an over-the-counter medication in either 2- or 4-mg strengths; the latter is recommended for highly dependent smokers. Clonidine, in doses of 0.1 to 0.75 mg/day delivered either transdermally or orally, is recommended as a second-line agent to treat tobacco dependence. Because of a paucity of data, no other pharmacotherapies are recommended in the guideline. Apart from bupropion SR (which is contraindicated in patients who are at risk for seizures or who have had a previous diagnosis of bulimia or anorexia nervosa), no other antidepressant agent has been documented as effective for smoking cessation or approved by the FDA for this use. Neither benzodiazepines nor β-adrenergic blocking agents have been found to have a beneficial effect in smoking cessation.

Two large multicenter studies have found bupropion SR efficacious in doubling long-term abstinence rates when compared with placebo (Hurt et al, 1997; Hayford et al, 1999). One advantage of this medication is that it can be instituted a week or two before complete cessation is attempted, unlike nicotine replacement products, which are based on providing gradually reduced amounts of nicotine without the other toxic components of cigarette smoke. The nicotine inhaler both resembles a cigarette and mimics the act of smoking, thus permitting perpetuation of a behavioral ritual, but the nicotine is absorbed through the buccal mucosa rather than the lungs. A course of treatment with bupropion SR ranges from 7 to 12 weeks. Treatment with nicotine replacement products ranges from 6 weeks to 6 months. Some studies have found that 15% to 20% of successful abstainers continue to use nicotine gum for a year or longer.

Combination therapy appears to be a promising, albeit doubly expensive approach. A 9-week study combining bupropion SR with transdermal nicotine found much greater efficacy than with either medication alone (Jorenby et al, 1999). Overall, the guideline found insufficient evidence to recommend combination therapy as a general treatment strategy.

The introduction of bupropion SR and newer forms of nicotine replacement products, backed by intensive advertising campaigns in both medical journals and the mass media, will doubtless stimulate physicians to take a more informed and personal role in smoking cessation. Such active involvement can be extremely crucial in and of itself. In the 1970s, at a time when efforts by physicians to discourage smoking were much less widespread and accepted, Russell and colleagues (1979) found that just 1 to 2 minutes of simple but unequivocal advice to the patient to stop smoking resulted in a cessation rate of over 5% measured at 1 year as opposed to only 0.3% in the control group. Moreover, when strong advice is given at the time of recovery from a heart attack or other smoking-related disease (combined with a brochure and a promise of follow-up), over 60% stop smoking and stay off cigarettes (measured at 3 years)—more than twice the rate of those who receive less definitive advice (Burt et al, 1974). Although most family physicians routinely ask their patients about smoking and advise them to stop smoking, relatively few provide more than advice and actually counsel patients with state-of-the-art techniques (Lindsay et al, 1994).

Even though many people say that they stopped smoking on their own, such individuals may not consciously attribute part of their success to increasing social pressures that reinforced their decision. Indeed, efforts to curtail tobacco use have become a cornerstone of local and national health promotion efforts. The release in 1993 of a report by the Environmental Protection Agency (National Institutes of Health Publication No. 93–3605, August 1993) implicating environmental tobacco smoke as a significant cause of lung cancer and other diseases in persons who do not smoke provided important evidence for individuals working to implement clean indoor air policies at the workplace. Such policies are now the norm.

Obstacles to Change

Unfortunately, the tobacco pandemic cannot be addressed as though it were a static issue whereby sufficient public health education results in a significant change in societal behavior. Rather, smoking is a dynamic issue, with cigarette advertisers—whose livelihoods depend on maintaining more than 50 million users of tobacco, including 1.25 million teenagers who take up smoking each year—constantly adapting to the challenges brought by the antismoking movement.

Thus, smoking cessation programs for individual patients cannot truly succeed in the long run in the absence of both workplace smoking bans and multimedia counteradvertising strategies that weaken the influence of the tobacco industry and reinforce the physician's office-based efforts (Blum, 1980a).

Although cigarette smoking becomes an addiction, it is first an entirely learned behavior. The "peer pressure" so often cited by tobacco companies as the reason for adolescent smoking is as much a manufactured product as the cigarettes themselves. The purpose of advertising is not just to sell cigarettes, but also to promote and reinforce the social acceptability of smoking and to encourage complacency toward the enormous social and health toll taken by smoking-caused diseases and fires. Today, cigarette manufacturers spend more money annually to promote smoking than is spent to advertise almost any other consumer product.

A variety of factors may inhibit physician involvement in smoking cessation, such as a perceived or real lack of time, lack of reimbursement by third-party payers for such counseling, and lack of "peer group" reinforcement in a technologically oriented, tertiary care–centered, highly intellectualized health care system. Nonetheless, physicians might well find that their increased involvement in efforts to promote smoking cessation among patients, regardless of the minimal enhancement in revenue, becomes a practice-building factor as word spreads about the doctors who care.

Office-Based Strategies

Physicians can do a great deal to become better teachers about smoking, in lieu of relegating this role to

ancillary personnel, a smoking cessation clinic, or a pamphlet off the shelf. The physician can develop an innovative strategy beginning outside the office or building. A bus bench, billboard, or sign in the parking lot with a straightforward or humorous health promotion message helps establish a thought-provoking and favorable image. In the waiting area, removal of ashtrays and placement of signs noting that "In the interest of comfort, safety, and health, this is a smoke-free environment" further reinforce the message.

Magazines with cigarette advertisements ought not to appear in the physician's office in the absence of prominent stickers or rubber-stamped messages calling patients' attention to the deceptive, absurd nature of such ads. Alternatively, felt-tipped pens could be made available for patients to contribute their own antismoking comments or artwork. A commitment on the part of American physicians to not let their offices become vehicles for selling cigarettes would make a substantial contribution to health promotion. Although responsibility for the office-based smoking cessation strategy should rest with the physician, it is invaluable to include all office staff as positive reinforcers for patients. Labeling each chart with a small "No Smoking" sticker to indicate the need for such reinforcement may be helpful, although care must be taken to avoid stigmatizing the patient as a "smoker." One would do well to reconsider using potentially alienating words such as "smoker" or even "quitter."

The key to successful smoking cessation efforts is a positive approach. A discussion about the diseases caused by smoking and the harmful constituents of tobacco smoke is essential—indeed, the physician must not shrink from imparting, through graphic posters, pamphlets, slides, and other audiovisual aids, the gruesome consequences of smoking—but the benefits of not smoking must be emphasized at least as strongly. Moreover, solely educating patients about the facts of smoking in a single office visit is unlikely to result in behavioral change.

In contrast, the physician can, through the use of creative analogies related to the patient's occupation, hobbies, or romantic interest, succeed in changing the patient's entire attitude toward smoking. For example, naming a partial list of the poisons and irritants in tobacco smoke, such as hydrocyanic acid (cyanide), ammonia, formaldehyde, and carbon monoxide (see Table 59–2), may mean little at first. (One pregnant patient proudly stated that she never buys a brand of cigarettes with the warning that mentions harm to the fetus, only those brands that say they contain carbon monoxide.) By noting that cyanide is the substance used in the gas chamber in executions, that formaldehyde is used to preserve cadavers, or that ammonia is the predominant smell in urine, however, the physician is likely to cause the patient to think about smoking a bit differently. No one wishes to have "urine breath." Similarly, it does little good to talk about carcinogens in tobacco in an age when the public believes that "everything causes cancer." Sadly, the concept of relative risk is poorly developed in our society because all too many people who smoke choose to think their millions-to-one odds of winning the state lottery are better than their one-in-seven chance of actually getting lung cancer.

Metaphors that Motivate

A revocabularization on the part of the physician is essential for making progress in office-based smoking cessation. Instead of "pack-year history," a more relevant measure is the "inhalation count." A pack-a-day smoker will breathe in upward of 1 million doses of cyanide, ammonia, carcinogens, and carbon monoxide in less than 15 years, not including the inhalation of other people's smoke (calculated at 10 inhalations per cigarette, 20 cigarettes per pack). Another way to emphasize the enormous amount smoked is to state the financial cost: a pack-a-day cigarette buyer will spend in excess of $1000 a year (calculated at $3 a pack)—or well over $10,000 in a decade if that money were put into a savings account or bond. One can remark about the joyful feeling of finding a $50 bill every 2 weeks—which is what one would indeed find if the money had not been spent on cigarettes. One patient who began smoking in the Marines at age 18 and who still smoked three packs a day at age 33 remarked ruefully that he had "smoked a Porsche."

Thus, whereas patient education in general and smoking cessation in particular rest on knowledge on the part of both the physician and patient of the deleterious aspects of adverse health behavior, the cognitive component alone is insufficient. Both the physician and the patient must be motivated to succeed. Three keys to office-based smoking cessation are to personalize, individualize, and demythologize.

The physician can learn to *personalize* approaches to smoking cessation by carefully screening the pamphlets and other audiovisual aids available in the office. (Ideally, family physicians should consider producing their own.) It is essential to scrutinize all such material as one would with a new drug or medical device. Personally handing a brochure to the patient while pointing out and underlining certain passages or illustrations will provide an important reinforcing message. The pamphlets, posters, and signs should be changed or otherwise updated every few weeks or months.

Individualizing the message to the patient is the cornerstone of success in patient education. The same cigarette counseling method cannot be used for a high-school girl, a construction worker, and an executive already showing signs or symptoms of heart disease. In the case of a high-school girl, the physician should not focus on such abstract concepts as emphysema and lung cancer, but rather emphasize the cosmetic unattractiveness of yellow teeth, bad breath, loss of athletic ability, and the financial drain that results from buying cigarettes. As for the construction worker, the physician might suggest the likelihood of fewer lost paydays, greater physical strength, and even a lengthier sex life were he to stop buying cigarettes.

In talking with concerned executives, it is especially important to *demythologize* certain beliefs about smoking, such as that the ultra-low-tar cigarettes they are

Table 59–4. A Consumerist Approach to Smoking Cessation: Helpful One-Liners

" 'Low tar' just means 'low poison.' Would you buy a brand of bread that was advertised as having only 2 oz of poison in every loaf?"	Cigarette smoke contains more than 4000 separate chemicals, over 40 of which are known carcinogens. "Tar" is the concentrate of these poisons, and there is no safe level of it.
"The filter is a fraud. You think filters are safer? Safer than what—fresh air?"	No health benefits accrue from smoking filtered cigarettes, which were widely introduced by tobacco advertisers in the 1950s to allay public fears about smoking. Some early cigarette filters were made of asbestos. A person smoking a low-tar filtered cigarette will often compensate by inhaling more deeply and smoking twice as many, thus increasing exposure to poisons.
"Menthol is an anesthetic."	It is a colorless chemical (not green like the ads imply) that is used to deaden the throat and mask the irritating sensation of the hot smoke.
" 'Light' and 'ultra-light' simply mean more sweeteners."	"When you add 2 tsp of sugar to a cup of black coffee, is there any less coffee?" Cigarettes taste different because different candy flavorings are added. Ultra-lights are easier for teenagers to become habituated to.
"Buying a pack of cigarettes for $3 is like spending $30 for a sandwich or $300,000 for a used car."	Cigarettes cost less than 15 cents a pack to manufacture. They are the highest-profit consumer item in America. Most of the increase in price is set by the tobacco manufacturers, not government taxes.
"Ammonia is what makes cigarette smoke smell like urine."	Another rancid aroma in cigarette smoke is formaldehyde. Other gases include carbon monoxide and cyanide.
"Cigarettes are dead leaves."	"Would you go up to a pile of burning leaves and start inhaling?" Cigarettes are dead leaves laden wtih chemicals. They're designed to keep burning no matter what so that you have to buy more and more.

smoking are safer. On the contrary, the use of so-called low-tar brands, which should be referred to as "low-poison" by the physician (Table 59–4), may in fact result in compensatory deeper inhalation of greater concentrations of chemical additives and noxious gases that increase the risk of a heart attack. One way to highlight the absurdity of the belief that low-tar cigarettes are safer is to ask rhetorically, "Safer than what? Fresh air?" or to wonder aloud whether it is safer to jump from the 50th story of the Empire State Building instead of the top. Another analogy is to point out that one would never think of buying a loaf of bread—or any other consumer product—that was advertised as containing "only 2 mg of cancer causers."

In any event, such dialogue must be practiced over and over again like any medical procedure and individualized to the patient. (Remember that no two construction workers, teenagers, or executives are alike.) The counseling should be designed to call attention not only to the inevitable risks of smoking cigarettes but also to the chemically adulterated tobacco product itself, its inflated price, and the ubiquitous and ludicrous way in which the person's brand is promoted (Blum, 1980b). In effect, the family physician can shift the focus away from a resistant or guilt-ridden smoker and onto the product.

Common Myths

The most important myth surrounding smoking is that it relieves stress. This myth can be debunked by pointing out that the stress that is relieved is what resulted from being dependent on cigarettes—the essence of addiction. At the same time, it is also important to point out that deep breathing in and of itself has a relaxing effect (Woods, 1988).

The second saddest myth, reinforced in advertisements for Virginia Slims and a host of new long, thin cigarettes intended for women and girls, is that smoking keeps weight off. Aside from pointing to all the obese women who smoke and attempting to correct the misapprehension that being overweight is a greater health risk than smoking is, one can point out that by damaging the taste buds and other digestive tract cells, smoking does inhibit appetite, but it also results in more sedentary behavior through loss of lung capacity and cardiovascular fitness. One need not gain weight on stopping smoking if one will relearn to enjoy walking and running as much as one relearns the taste of food. By no means will all persons who stop smoking gain weight. Even among those who do, the average weight gain is 6 lb for men and 8 lb for women (Williamson et al, 1991). Although smokers may weigh slightly less than nonsmokers, when they stop smoking they simply return to the average weight of nonsmokers. Moreover, the slightly lower weight in many who continue to smoke is associated with a higher-risk body fat distribution (Bonithon-Kopp et al, 1989; Shimokata et al, 1989). Because more than 75% of black patients who smoke buy menthol brands, it is important to debunk the myth that this substance in some way "cools" the smoke (U.S. DHHS, 1989). In fact, menthol is an anesthetic that deadens the throat to create the illusion of less irritating smoke (see Table 59–2).

From the physician's standpoint, perhaps the biggest myth that has been encouraged in the medical literature is that the patient must be "ready to quit." Although common sense dictates that those who express a greater interest in stopping smoking will have a greater success rate, patients who do not express an interest in stopping smoking symbolize the overall challenge we face in curbing this pandemic. One of the reasons for lack of motivation of patients may be their sense of inevitability

of failure. It is conceivable that by not educating a nonmotivated smoking patient, the physician is in effect reinforcing the notion that it may be too difficult to stop smoking.

Setting a "quit date," the sine qua non of the smoking cessation literature, may rationalize the continuation of an adverse health practice and may strengthen denial. In other words, it is helpful to remind patients that they can stop now. If they do not stop, it does not mean that you will not treat them the next time, but it is important to give encouragement and not reinforce excuses. Most authors do believe that a quit date targeted only 1 week or a few weeks into the future is useful for a motivated patient, for whom denial is less of a problem. Its purpose is to let the individual build up resolve or to permit a gradual reduction in daily cigarette consumption. Giving patients a few written reminders is very helpful (such as lists of the advantages and disadvantages of smoking, the rewards for not smoking and the penalties for lighting up, the situations and environmental influences that encourage one to smoke, and the myths of smoking and smoking cessation) (Woods, 1988). A prescription with a no-smoking symbol signed by the physician and included with the other prescriptions is a thoughtful gesture.* The physician should not advise "cutting down," switching to a low-tar cigarette, or changing to a pipe or cigar.

Consumer Advocacy Role

Traditional office-based approaches begin by asking, "Do you smoke?," "How much do you smoke?," and "When did you start smoking?" Although this information may provide the physician with relevant data for charting purposes, this approach is all to often a signal for the patient to become defensive and resistant to further discussion, especially if the patient had no intention to stop smoking. However, there are alternative ways of obtaining information and at the same time piquing the patient's interest in the subject. By using and identifying with the vocabulary used by the consumer of cigarettes, the physician can adopt (and be perceived in) the role of consumer advocate, as opposed to medical finger wagger. The most important—and nonthreatening—questions to ask are, "What brand do you buy?" and "How much do you spend on cigarettes?" The patient is likely to be surprised and intrigued by these questions, which can be asked at any time in the course of the interview because they appear to be nonjudgmental. They serve to suggest that the physician is not solely a know-it-all and a preacher on the dangers of the evil weed. In effect, a question about the cost of cigarettes shows concern for the patient's financial well-being. Inquiring as specifically as possible about the brand name—for example, Marlboro Menthol Lights 100s, box—will lead to greater understanding on the part of the physician of the same vocabulary used by the person who buys cigarettes and will narrow the communication gap. The patient may even begin to laugh aloud at the foolishness of such a vocabulary, especially when encouraged to show the physician the package and to appreciate how little information about the product appears beyond the attractive design.

More than 15 different versions of Marlboros are available, which illustrates the way cigarette manufacturers create the illusion of choice, individuality, and degree of safety. A patient who states, "Since my heart attack, I've switched from Marlboro Reds to Marlboro Ultra Lights," has been miseducated to believe that some cigarettes can be less harmful than others. Moreover, the product itself is extremely cheap to manufacture (less than 15 cents a pack), but extremely profitable to tobacco companies at $3 a pack.

Promotions for various pharmacologic agents, mail-order gadgets, and clinics in smoking cessation reinforce the notion that cigarette smoking is primarily a medical problem with a simple, prescribable, nonindividualized solution (Blum, 1984). When a patient requests a "drug that will help me stop smoking," the physician must confront the dilemma of not wishing to dash the patient's expectations while emphasizing that a drug or device is at best an adjunct and not a means of smoking cessation. It is an unfortunate fact of life that many patients will not stop smoking until they have gotten their money's worth at a special smoking cessation clinic; moreover, it seems that regardless of the method used, the more expensive, the better.

Approach to Adolescents

Children and teenagers who smoke cigarettes pose a special challenge because they represent the market most carefully nurtured by tobacco advertisers. If an adolescent turns 18 years without starting to smoke, the chance of ever smoking is only 10%. Regardless of all our educational efforts, however, more than 3000 teenagers in the United States start smoking every day. Almost three fourths of adolescents who smoke buy Marlboros.

Adolescents have a desire for independence and feel invulnerable. We should capitalize on their fierce determination to be autonomous and stress the fact that nicotine creates a potentially insurmountable dependence that persists throughout life.

It is essential to avoid emphasizing the adult and dangerous nature of smoking. Rather, smoking should be referred to as the childish, dumb, and silly-looking practice that it is. The single most important statement that the physician can make to an adolescent is "Come on, you're too old to smoke. That's for the little kids who want to look grown up." Another strategy is for the physician to ask a teenager who smokes to help think of ideas for talking to junior high school and primary school students about ridiculing tobacco company executives and making fun of cigarette brand names.

As a general rule in approaching the subject of smoking cessation with a patient, Schwartz (1987) and others recommend thinking in terms of a strategy that includes

*These symbols are available, along with a wide variety of stickers, posters, and newsletters, from DOC, Department of Family Medicine, Baylor College of Medicine, 5510 Greenbriar, Houston, TX 77005 (telephone: 713–798–7729; fax: 713–798–7775).

interventions designed to enhance motivation and those that will help reduce dependence. Time and commitment on the part of the physician will result in greater success. The biggest obstacle to smoking cessation is complacency on the part of the physician.

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