

44. Williams MR, Walker KJ, Turkes A, et al. The use of an Lh-Rh agonist (ICI18360, Zoladex) in advanced premenopausal breast cancer. *Br J Cancer* 1986;53:629-636.
45. Dixon AR, Robertson JFR, Jackson L, et al. Goserelin (Zoladex) in premenopausal advanced breast cancer: Duration of response and survival. *Br J Cancer* 1990;62:668-670.
46. Pike MC, Ross RK, Lobo RA, et al. LHRH agonists and the prevention of breast and ovarian cancer. *Br J Cancer* 1989;60:142-148.
47. Gudmundsson JA, Nilius SJ, Bergquist C. Intranasal peptide contraception by inhibition of ovulation with the gonadotrophin-releasing hormone superagonist nafarelin: Six months clinical results. *Fertil Steril* 1986;45:617-623.
48. McLachlan RI, Healy DL, Burger HG. Clinical aspects of LHRH analogues in gynaecology: A review. *Br J Obstet Gynaecol* 1986;45:617-623.
49. Cuzick J, Wang DY, Bulbrook RD. The prevention of breast cancer. *Lancet* 1986;1:83-86.
50. Early Breast Cancer Trialists' Collaborative Group. Effects of adjuvant tamoxifen and of cytotoxic therapy on mortality in early breast cancer. *N Engl J Med* 1988;319:1681-1692.
51. Nayfield SG, Karp JE, Ford LG, Dorr A, Kramer BS. Potential role of tamoxifen in prevention of breast cancer. *JNCI* 1991;83:1450-1459.
52. Nolvadex and Adjuvant Trial Organization (NATO). Controlled trial of tamoxifen as a single adjuvant agent in the management of early breast cancer. *Br J Cancer* 1988;57:608-611.
53. Breast Cancer Trials Committee, Scottish Cancer Trials Office MRC. Adjuvant tamoxifen in the management of operable breast cancer: The Scottish trial. *Lancet* 1987;2:171-175.
54. Rutqvist LE, Cedermark B, Glas U, et al. Contralateral primary tumors in breast cancer patients in a randomized trial of adjuvant tamoxifen therapy. *JNCI* 1991;83:1299-1306.
55. Palshof T, Mouridsen HT, Dahnfeldt JL, et al. Adjuvant endocrine therapy in pre- and postmenopausal women with operable breast cancer. *Rev Endocr Rel Cancer* 1985;17(suppl):43-50.
56. Pritchard KI, Meakin JW, Boyd NF, et al. Adjuvant tamoxifen in postmenopausal women with axillary node positive breast cancer: An update. In: Salmon SE, ed. *Adjuvant therapy of cancer*. Vol V. New York: Grune and Stratton, 1987:391-400.
57. Cummings FJ, Gray R, Davis TE, et al. Tamoxifen versus placebo: Double-blind adjuvant trial in elderly women with stage II breast cancer. In: Proceedings of the NIH Consensus development conference on adjuvant chemotherapy and endocrine therapy for breast cancer. Bethesda, MD: National Institutes of Health, 1996:119-123.
58. Fisher B, Constantino J, Redmond C, et al. A randomized clinical trial evaluating tamoxifen in the treatment of patients with node-negative breast cancer who have estrogen-receptor-positive tumors. *N Engl J Med* 1989;320:479-484.
59. CRC Adjuvant Breast Trial Working Party. Cyclophosphamide and tamoxifen as adjuvant therapies in the management of breast cancer. *Br J Cancer* 1988;57:604-607.
60. Jordan VC. Tamoxifen for the prevention of breast cancer. In: DeVita VT, Hellman S, Rosenberg SA, eds. *Cancer prevention*. Philadelphia: JB Lippincott, 1990:1-16.
61. Kannel WB, Hjortland MC, McNamara PM, et al. Menopause and risk of cardiovascular disease. *Ann Intern Med* 1976;85:447-452.
62. Ross RK, Pike MC, Mack TM, Henderson BE. Oestrogen replacement therapy and cardiovascular disease. In: Drife JO, Studd JWW, eds. *HRT and osteoporosis*. London: Springer-Verlag, 1990:209-222.
63. Barrett-Connor E, Bush TJ. Estrogen and coronary heart disease in women. *JAMA* 1981;265:1861-1867.
64. Paganini-Hill A, Ross RK, Henderson BE. Postmenopausal oestrogen treatment and stroke: A prospective study. *Br Med J* 1988;297:519-522.
65. Love RR, Newcomb PA, Wiebe DA, et al. Effects of tamoxifen therapy on lipid and lipoprotein levels in postmenopausal patients with node-negative breast cancer. *JNCI* 1990;82:1327-1332.
66. McDonald CG, Stewart HJ. Fatal myocardial infarction in the Scottish adjuvant tamoxifen trial. *Br Med J* 1991;303:435-437.
67. Love RR, Mazens RB, Torney DC, et al. Bone mineral density in women with breast cancer treated with adjuvant tamoxifen for at least two years. *Breast Cancer Res Treat* 1988;12:297-301.
68. Turken S, Siria E, Seldin D, et al. Effects of tamoxifen on spinal bone density in women with breast cancer. *JNCI* 1989;81:1086-1088.
69. Yager JD, Yager R. Oral contraceptive steroids as promoters of hepatocarcinogenesis in female Sprague-Dawley rats. *Cancer Res* 1980;40:3680-3685.
70. Fornander T, Rutqvist LE, Cedermark B, et al. Adjuvant tamoxifen in early breast cancer: Occurrence of new primary cancers. *Lancet* 1989;1:117-120.
71. Lipton A, Harvey HA, Hamilton RW. Venous thrombosis as a side effect of tamoxifen treatment. *Cancer Treat Rep* 1984;68:887-889.
72. Falkson HC, Gray R, Wolbert WH, et al. Adjuvant trial of 12 cycles of CMFPT followed by observation or continuous tamoxifen versus four cycles of CMFPT in postmenopausal women with breast cancer: An ECOG Phase III study. *J Clin Oncol* 1990;8:599-607.
73. Jordan VC, Fritz NF, Torney DC. Long-term adjuvant therapy with tamoxifen effects on sex hormone binding globulin and antithrombin III. *Cancer Res* 1987;47:4517-4519.
74. Ross RK. Prostate cancer. In: Schottenfeld D, Fraumeni J, eds. *Cancer epidemiology and prevention*. 2nd ed. Cambridge, England: Oxford University Press, 1992 (in press).
75. Noble RL. The development of prostatic adenocarcinoma in Nb rats following prolonged sex hormone administration. *Cancer Res* 1977;37:1929-1933.
76. Ross RK, Bernstein L, Lobo RA, et al. Evidence for reduced 5-alpha-reductase activity in Japanese compared to U.S. white and black males: Implications for prostate cancer risk. *Lancet* 1992;339:887-889.
77. Lookingbill DP, Dowers LM, Wang C, et al. Clinical and biological parameters of androgen action in normal healthy Caucasian versus Chinese subjects. *J Clin Endocrinol Metab* 1991;72:1242-1248.

SECTION 6

ALAN BLUM

Curtailing the Tobacco Pandemic

By all rights, lung cancer should have been included along with smallpox as one of the diseases that was eradicated in the 20th century. Instead, to the undying shame of the health professions—and due to the untiring energy of the transnational tobacco conglomerates—the production, distribution, marketing, and use of tobacco continue to grow in every corner of the world. Deaths from lung cancer are expected to exceed 3 million a year by the turn of the century.¹

Since U.S. Surgeon General Leroy E. Burney issued a policy statement in 1957 that accepted the cause-effect relation between cigarette smoking and lung cancer,^{1a} each succeeding Surgeon General has been committed to curbing the use of tobacco. In 1964 the Report of the Advisory Committee to the Surgeon General on Smoking and Health reviewed and summarized the devastating scientific case against smoking.² This document and an analysis produced in the United Kingdom in 1962 by the Royal College of Physicians galvanized the medical community and the public alike. The Surgeon

General's report was written by ten eminent biomedical scientists who had been selected by Surgeon General Luther Terry from a list of 150 people (none of whom had taken a public position on the subject of smoking and health) approved by major health organizations and the tobacco industry.

Concerns about smoking had long been raised in the scientific community. In 1928 Lombard and Doering³ reported a higher incidence of smoking among patients with cancer than among controls. Ten years later Pearl⁴ reported that persons who smoked heavily had a shorter life expectancy than those who did not smoke. In 1939 Ochsner and DeBaKey began reporting their observations on the relation between smoking and lung cancer.⁵ They and other outspoken opponents of smoking, such as Dwight Harkin and William Overholt, were met with derision by the medical profession, more than two thirds of whom smoked.

Not until the epidemiologic work in the 1950s of Doll and Hill^{6a} in the United Kingdom and Hammond and Horn⁶ in the United States did the medical profession begin to take the problem seriously. Cigarette advertisements continued to appear in the *Journal of the American Medical Association* and other medical journals until the mid-1950s. A Viceroy cigarette advertisement published in medical journals in 1954 thanked the 64,985 doctors who visited Viceroy exhibits at medical conventions that year. Such scientific displays existed at various state medical society meetings until the 1980s. In

1978 the American Medical Association (AMA) issued a report, "Tobacco and Health," which summarized research projects that confirm the findings of the 1964 Surgeon General's report and cemented the association between smoking and heart disease.^{6a} This report was entirely underwritten by the tobacco industry, which in effect had succeeded in muting any official action-oriented stance on the part of the AMA for 14 years.

Nonetheless, since 1985 when it first called for a ban on tobacco advertising, the AMA and its publications have become increasingly outspoken in the effort to curtail the use and promotion of tobacco. The AMA has funded two national conferences on tobacco and has made the subject of smoking and health one of its four top priorities. Pressure by the AMA led the Joint Commission on Accreditation of Healthcare Organizations to institute a policy mandating that accredited health facilities be smoke-free environments as of 1992.

Considering its \$350 million annual income, the American Cancer Society (ACS) has been cautious and conservative in challenging the tobacco industry. Not until 1983 did the organization begin to address the subject of cigarette advertising. On the other hand, the ACS has made several major contributions, including the adoption of the annual stop-smoking day known as the Great American Smokeout, the sponsorship of world conferences on smoking and health (which currently draw 1000 people and are held every 3 years), and the creation of Globalink (a worldwide electronic communication network to aid the sharing of antitobacco strategies). The American Academy of Family Physicians has led medical specialty organizations in confronting tobacco problems by means of training for physicians in smoking cessation and financial support for antitobacco advocacy groups such as Doctors Ought to Care (DOC). Various chapters of the American Lung Association have done substantive lobbying and taken aggressive public stances in accelerating the passage of local clean indoor air legislation.

Governmental agencies, public health organizations, and academic institutions have not exerted much leadership on this issue. A remarkable grassroots antismoking movement that arose in the 1970s with the goal to create smoke-free public places impelled more traditional organizations to action. These groups—Action on Smoking and Health, Group Against Smoking Pollution, and Americans for Nonsmokers' Rights—paved the way for measures such as the federal ban on smoking in aircrafts and local laws that restrict smoking, remove cigarette vending machines, and ban the distribution of free tobacco samples.

Although numerous prospective studies conducted over the past 40 years have documented multifarious disease risks associated with smoking,⁷ cancer has been linked to tobacco use for more than two centuries. In 1761, John Hill,⁸ a London physician, reported an association between the use of snuff and cancer of the nose. The first U.S. Surgeon General's Report on Smoking and Health in 1964 concluded that cigarette smoking was the major cause of lung cancer in men and was causally related to laryngeal cancer and oral cancer in men.² More than 57,000 subsequent studies and 20 additional reports of the Surgeon General have documented the impact of tobacco use on morbidity and mortality in the United States and abroad. It is now understood that approximately 40% of all cancer deaths are attributable to cigarette smoking; smoking

is thus responsible for more than 434,000 deaths per year in the United States, or 18% of all deaths.⁹

Smoking is the major cause of cancers of the lung, larynx, oral cavity, and esophagus and is a contributory factor in cancers of the pancreas, bladder, kidney, stomach, and uterine cervix (Table 20-22). Overall, cigarette smoking has been identified as the chief preventable cause of deaths due to cancer in the United States.⁷

LUNG CANCER

The most prominent conclusion of the 1964 Surgeon General's Report was the determination that cigarette smoking is the major cause of lung cancer in men.^{2,10,11} There is a clear dose-response relation between lung cancer risk and daily cigarette consumption, and those people who smoke more than a pack of cigarettes a day have a risk that is at least 20 times that of nonsmokers.⁷ The four major histologic forms of lung cancer—squamous cell, adenocarcinoma, small cell, and large cell—are all associated with smoking. Squamous cell cancer is the commonest form among men; in women, adenocarcinoma predominates.¹²

The identification of cigarette smoking as the major causative factor in the development of lung cancer led the tobacco industry to respond to such reports with the promotion of "less hazardous" cigarettes, including filtered, low-tar, and low-nicotine cigarettes, creating the illusion that the risk had been eliminated or diminished.^{13-15,16} This recalled the multimillion dollar advertising campaigns developed in the 1940s to allay the public's concerns about cigarette smoking, including R. J. Reynolds' slogan, "More doctors smoke Camels than any other cigarette," American Tobacco Company's boast, "Lucky Strike is less irritating to sensitive or tender throats," and Philip Morris' claim, published in countless magazines, newspapers, and medical journals, "Every case of irritation of the nose and throat due to smoking cleared completely or definitely improved."¹⁷ Lorillard's Kent cigarettes, one of the most widely promoted "health-oriented" brands of the 1950s, contained a filter that was made of asbestos.¹⁷

Over the years, such purported innovations in the design of the product have been met with overwhelming consumer acceptance. For example, between 1976 and 1982 sales of low-tar cigarettes, which offer few if any safety advantages, increased from 17% to 59% of total cigarette sales.¹⁴ Currently, the tobacco industry continues to suggest health benefits to consumers through the use of words such as "light," "ultra-light," "mild," "medium," "slims," and "superslims." Because lung cancer risk is related to years of smoking and to the frequency and depth of inhalation,^{18,19} those people who switch to buying allegedly less hazardous cigarette brands often smoke more and inhale more deeply to attain the satisfied level of nicotine.

Tragically, while smoking rates have declined by an average of 0.5% per year over the past 10 years, and while the incidence of lung cancer among black and white men has leveled off, the incidence of lung cancer continues to rise at a rate of 5% per year among women. Moreover, early detection hardly improves survival; the 5-year survival rate has remained at less than 10% since the 1960s.⁹ Although there is a gradual

TABLE 20-22. Summary of Smoking and Cancer Mortality

Type of Cancer	Gender	Relative Risk Among Smokers		Mortality Attributable to Smoking	
		Current*	Former*	Percentage*	Number†
Lung	Male	22.4	9.4	90	82,800
	Female	11.9	4.7	79	40,300
Larynx	Male	10.5	5.2	81	2400
	Female	17.8	11.9	87	700
Oral cavity	Male	27.5	8.8	92	4900
	Female	5.6	2.9	61	1800
Esophagus	Male	7.6	5.8	78	5700
	Female	10.3	3.2	75	1900
Pancreas	Male	2.1	1.1	29	3500
	Female	2.3	1.8	34	4500
Bladder	Male	2.9	1.9	47	3000
	Female	2.6	1.9	37	1200
Kidney	Male	3.0	2.0	48	3000
	Female	1.4	1.2	12	500
Stomach	Male	1.5	?	17‡	1400
	Female	1.5	?	25	1300
Leukemia	Male	2.0§	?	20§	2000
	Female	2.0	?	20	1600
Cervix		2.1	1.9	31‡	1400
Endometrium		0.7	1.0	—	—

* Except as noted, data from The Health Consequences of Smoking; A Report of the Surgeon General, 1982,⁷ 1989,¹¹ 1990.²⁰

† Data based on Boring et al, 1991.⁴³

‡ Data from Centers for Disease Control, MMWR, 1991.⁷⁹

§ Data from Mills et al, 1990,⁹⁴ and Severson, 1987.⁵⁰

decrease in risk for death from lung cancer after cessation of cigarette smoking, this message is perceived by many of those who smoke to mean that the risk for developing lung cancer will diminish immediately on quitting. This misunderstanding may lead to postponement of cessation in the belief that it does not matter when one stops. Although a diminished risk for lung cancer is experienced among former smokers after 5 years of cessation, the risk among former smokers remains higher than that of nonsmokers for as long as 25 years.²⁰ Any early reduction of health risk after cessation applies only to heart disease,²⁰ whereby a decline in risk for heart problems appears to occur within 1 year of cessation; even then, the remaining decline in excess risk for heart disease is more gradual, approaching those of persons who have never smoked only after many years of smoking abstinence.¹⁶

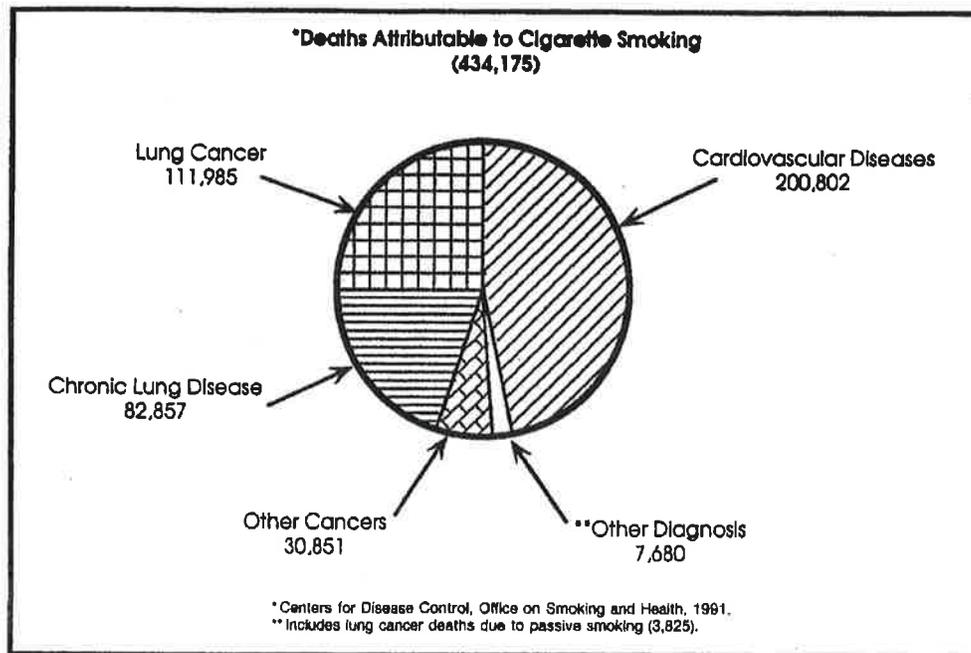
When people who smoke are exposed to other carcinogens at the workplace (e.g., pipefitters and asbestos and uranium miners and radon), their risk for lung cancer is dramatically higher than those who do not smoke; moreover, the combined effects of smoking and occupational exposure to carcinogens is greater than the risk for either alone.^{20a,20b,31}

LARYNGEAL CANCER

Cigarette smoking is the major cause of cancer of the larynx.^{7,21} The 3650 deaths from laryngeal cancer in 1991 in the United States constituted 1% of all deaths from cancer.

Approximately 82% of the 12,500 new cases of laryngeal cancer diagnosed in 1991 were directly attributable to cigarette smoking. In three of the six major prospective studies that have investigated the relation between smoking and cancer of the larynx,^{7,21-26} mortality ratios could not be calculated because all of the deaths from laryngeal cancer occurred in people who had smoked cigarettes.²¹ Overall, deaths from cancer of the larynx were found to have occurred at a rate 6 to 13 times greater among persons who smoked cigarettes compared with nonsmokers. A similar risk for cancer of the larynx has been found among those people who smoke cigars or pipes,²⁷ because 80% of new cases of laryngeal cancer occur in men, it is essential to explode the myth that switching to a pipe or cigar conveys a reduced risk for cancer.

Williams and Horn²⁸ reported a strong dose-response relation between the number of cigarettes smoked per day and the risk for developing cancer of the larynx; other reports have confirmed that people who smoke more than 25 cigarettes a day have cancer mortality ratios 20 to 30 times greater than those who do not smoke.^{7,21} There appears to be a synergistic effect between smoking and drinking, possibly as the result of alcohol acting as a solvent of carcinogens in tobacco smoke or as the result of an alteration in liver metabolism.²⁹ The risk for developing cancer of the larynx is as much as 75% higher in people who use tobacco and alcohol compared with people with exposure to either substance alone.^{21,29} One study describes a typical patient with cancer of the larynx as a 50- to 60-year-old man who smoked cigarettes and was a moderate-to-heavy alcohol drinker.³⁰



ORAL CANCER

There is a dose-response relation between the number of cigarettes smoked per day and cancers of the lip, tongue, salivary gland, floor of the mouth, mesopharynx, and hypopharynx.⁷ The use of pipes, cigars, and spitting tobacco in its various forms (plug tobacco, loose leaf tobacco, twist tobacco, and moist snuff) is also associated with the development of cancers of the oral cavity; the risk of using these forms is of the same magnitude as that of using cigarettes.^{7,21,32} Tobacco use is responsible for more than 90% of tumors of the oral cavity among men and 60% among women.¹¹

There is a 27-fold increase in the rate of oral cancer among men who smoke cigarettes, pipes, or cigars and a sixfold increase among women who smoke.¹¹ Spitting tobacco is a significant cause of leukoplakia,³²⁻³⁵ an abnormal thickening and keratinization of the oral mucosa that is recognized as a precursor of malignancy. The combination of alcohol and tobacco use produces an increase in risk for cancer of the oral cavity on a dose-related basis.³⁶

ESOPHAGEAL CANCER

Prospective and retrospective epidemiologic studies have demonstrated that cigarette smoking is the major cause of cancer of the esophagus in men and women.^{7,21} More than 15,000 Americans die each year from carcinoma of the esophagus (including a disproportionate number of blacks), 80% of which are attributable to smoking.¹¹ Death rates for esophageal cancer are as much as ten times greater among persons who smoke cigarettes, cigars, or pipes compared with those who do not.²⁷ As with laryngeal and oral cancer, alcohol consumption acts synergistically with smoking to increase by 25% to 50% the risk for developing esophageal cancer.^{28,37,38}

In explaining a mechanism for tobacco-induced esophageal

cancer, Newcomb and Carbone note that carcinogens from tobacco smoke have extensive contact with the esophagus because they are swallowed after condensing on the mucous membranes of the mouth and pharynx and as mucus is cleared from the lungs.³⁹

CANCER OF THE UTERINE CERVIX AND OVARY

Recent evidence has strengthened the association between cigarette smoking and cancer of the uterine cervix.^{7,40-42} As many as one third of the 12,000 new cases of cervical cancer in the United States each year are attributable to cigarette smoking.⁴³ Women who smoke cigarettes have four times the risk of nonsmokers for developing cervical cancer.⁴² The finding of nicotine and cotinine in the cervical secretions of cigarette smokers and of the mutagenic activity of these constituents of tobacco smoke in the cervical mucus further supports the epidemiologic findings.^{44,45} It is hypothesized that these carcinogenic metabolites may interact with human papilloma viruses.⁴⁶

OTHER CANCERS

A relation between smoking and bladder cancer was noted in the 1964 Surgeon General's Report.² The 1982 Surgeon General's Report concluded that cigarette smoking is a contributing factor for bladder and kidney cancer. In 1992, researchers at the National Cancer Institute (NCI) reported the results of a large population-based case-control study of cancer of the renal pelvis and ureter that confirms that cigarette smoking is the major cause of these tumors.⁴⁷ Forty percent of bladder cancers (or more than 4000 new cases in the United States each year) and kidney cancers (more than 3600 cases)

currently are believed to be smoking related.¹¹ The kidney and bladder are subject to the longest duration of direct exposure to carcinogens and radioactive substances in tobacco smoke of any organ system.⁴⁸ Occupational exposure by smokers to various dyes, paints, and organic chemicals dramatically increases the risk of bladder cancer. In contrast to the beneficial effects over time of smoking cessation on the incidence of all other tobacco-related cancers, the risk for genitourinary cancer appears to remain elevated among former smokers for more than 15 years.^{48,49}

People who smoke have two to three times the risk for pancreatic cancer that nonsmokers have¹¹; approximately 30% of the 25,000 annual deaths from pancreatic cancer are attributable to cigarette smoking.⁷ This pathogenetic mechanism may relate to exposure to tobacco metabolites in bile acids or blood. Although the 1964 Surgeon General's Report² concluded that there was no relation between smoking and stomach cancer, and although overall mortality has declined, recent evidence has shown a 50% increase in mortality ratios from this disease among those who smoke compared with those who do not.⁷

The fact that cigarette smoke contains at least two known causes of leukemia (benzene and ionizing radiation polonium 210) may explain the epidemiologic association between smoking and lymphoid and myeloid leukemia.⁷ Currently, 20% to 30% of cases of leukemia are attributable to smoking.^{50,50a}

Although there appears to be no relation between smoking and cancers of the colon and rectum, cancers of the liver, anus, penis, and vulva are commoner in persons who smoke than in those who do not.³⁹ An antiestrogenic effect of tobacco smoke is believed to explain the 30% less frequent occurrence of cancer of the uterine endometrium among postmenopausal women who smoke compared with those who do not;⁵¹ in contrast, a 75% increased risk for breast cancer has been found among women who smoke heavily and who began smoking at a young age.⁴⁰

CORONARY HEART DISEASE

Cigarette smoking is a primary risk factor for coronary heart disease (CHD). Overall, persons who smoke have a 70% greater CHD death rate, a twofold to fourfold greater incidence of CHD, and a twofold to fourfold greater risk for sudden death than nonsmokers.⁵² Although women experience lower CHD rates than men, cigarette smoking is a major determinant of CHD in women.⁵³ Cigarette smoking is associated with coronary artery disease and aortic atherosclerosis.⁵² In addition to such chronic conditions, cigarette smoking exerts acute effects, including coronary artery spasm, increased platelet aggregation, and a decreased ventricular fibrillation threshold.^{52,54,55} The risk for myocardial infarction is proportional to the number of cigarettes smoked.⁵²

CEREBROVASCULAR DISEASE

Stroke is the third leading cause of death in the United States.¹¹ The risk for stroke increases with the number of cigarettes smoked and declines after cessation of smoking; in 5 years

former smokers have the same risk for stroke as persons who have never smoked.^{20,52,56} Women who smoke cigarettes experience an increased risk for subarachnoid hemorrhage; the concurrent use of cigarettes and oral contraceptives magnifies this risk.⁵²

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Cigarette smoking is the main cause of chronic obstructive pulmonary disease (COPD), the leading cause of disability in the United States. In the 1960s, the most widely advanced hypothesis on the cause of COPD linked progressive decline in lung function to recurrent respiratory infection and atmospheric pollution.⁵⁷ However, this theory could not explain the increasing number of people with COPD living in the Great Plains of the United States where pollution was a minimal risk. Epidemiologic investigations have since confirmed the predominant role of cigarette smoking in causing COPD.^{11,58} Cigarette smoke inhibits ciliary activity of the bronchial epithelium and the phagocytic activity of the macrophages in the alveoli.⁵⁷ This results in the decreased clearance of foreign material and bacteria from the lung, which leads to increased infection, tissue destruction, and decreased lung function.

WOMEN AND SMOKING

In 1964, at the time of the first Surgeon General's Report discussing the smoking epidemic, lung cancer was the leading cause of death due to cancer in men and the fifth leading cause of cancer mortality among women.² This difference in lung cancer mortality rates can be explained by the fact that until the 1920s, it was socially unacceptable—and in some states illegal—for women to smoke.^{56a} Men had taken up cigarette smoking in large numbers toward the end of the 19th century—in part because antispitting ordinances to curtail the spread of tuberculosis had led the tobacco companies to switch from the promotion of chewing tobacco and cigars to the inhalation of tobacco smoke by means of the cigarette. Smoking did not take hold among women until the 1920s when the American Tobacco Company began a mass media advertising campaign with the slogan, "To keep a slender figure, reach for a Lucky Strike instead of a sweet." At that time women did not smoke as many cigarettes or take as many puffs per cigarette as men.^{56b} The appearance by motion picture heroines, athletes, and socialites in cigarette advertisements in the 1930s led to an increase in smoking among women so that by World War II a third of American women were smoking.

In 1968 cigarette maker Philip Morris began to associate smoking with the women's liberation movement by launching its Virginia Slims brand on a massive scale in the broadcast and print media with the slogan, "You've come a long way, baby." The brand name also underscored the constant pressure on women to be thin. When overt cigarette advertising was no longer permitted on television in 1971, the company created the Virginia Slims Tennis Circuit, telecasts of which circumvent the tobacco advertising ban by featuring players

as young as 14 amid dozens of court-side billboards for Virginia Slims.

In 1981, in an article in an advertising journal headlined, "Women top cigarette target," the chief executive officer of R. J. Reynolds described the women's market as "probably the largest opportunity" for the tobacco company.⁵⁹ Currently, women continue to be a primary target for cigarette advertisers.

Smoking rates among less educated young women are increasing, as is the amount they smoke.¹¹ In 1990, the marketing plan for a new brand of R. J. Reynolds cigarettes, Dakota, identified a specific target: "virile females" ages 18 to 20 who have no education beyond high school and who aspire "to have fun with [their] boyfriends and partying."⁶⁰ Other "female" brands include Eve (Liggett), Style (Loews), Satin (Loews), Capri (BAT), More (R. J. Reynolds), and Misty (American Tobacco). The manufacturers sponsor a host of activities, including fashion shows, art exhibitions, and family reunions and offer T-shirts, diaries, and fashion accessories free of charge or in exchange for proof of purchase.

Such promotional efforts have undermined all efforts to educate young women about the adverse effects of cigarette smoking. The emphasis of public health campaigns on the dangers of smoking has failed to address the ubiquitous, sophisticated, and carefree appeal of tobacco advertising. Currently, lung cancer has surpassed breast cancer as the leading cause of cancer deaths among women,¹¹ a fact that is virtually unreported in women's magazines, of which only a handful do not accept tobacco advertising. The issue receives scant coverage on television, probably due to the advertising clout of the subsidiaries of tobacco conglomerates.

Cigarette smoking results in other problems for women, especially during pregnancy. There is a confirmed association between maternal smoking and low-birth-weight infants, and there is an increased incidence of premature birth, spontaneous abortion, stillbirth, and neonatal death.⁶¹

Although there has been a dramatic decline in smoking among physicians, medical students, and most other health professionals during the past several decades, smoking among nurses has not declined. Jacobson attributes this to anger by nurses at their subordination within a health service dependent on women but controlled by men.⁶²

ETHNIC MINORITIES

Black and Hispanic Americans have the highest rates of lung cancer and cardiovascular disease in the United States.⁶³ The disproportionately high rates of smoking-related diseases among ethnic minorities can be attributed to the successful marketing of tobacco products to minority communities.⁶⁴ Billboards advertising cigarettes appear four to five times more often in inner city neighborhoods than in middle class suburbs.⁶⁵ Tobacco and alcohol constitute as much as 80% to 90% of the products advertised on billboards in inner city areas. Cigarette advertising in black and Hispanic magazines and newspapers represents a major source of revenue for these publications.^{64,66,67} In more than 40 years of publication, the leading black-oriented magazine, *Ebony*, has carried few articles on smoking; not surprisingly, cigarette companies are a leading source of revenue. Major black and Hispanic civic

organizations, such as the National Association for the Advancement of Colored People, the Urban League, the United Negro College Fund, and La Raza, receive funding from tobacco companies; an exception is the National Coalition of Hispanic Health and Human Services Organizations.

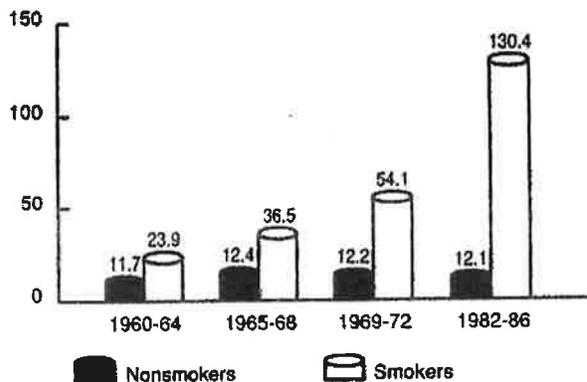
The result of such successful marketing targeted to ethnic minorities is a higher rate of smoking among blacks⁶⁸ and an increase in smoking among Hispanic women.^{69,70} Recent data from the 1987 National Health Interview Survey reveals that 32.9% of blacks smoke compared with 25% among the white middle class population.⁶⁹ Little if any change can be expected in smoking-related mortality among blacks and Hispanics, given the paucity of mass media efforts to counter tobacco use and promotion.

"LESS HAZARDOUS" CIGARETTES

In the 1950s, confronted with declining cigarette sales after the publication of studies linking smoking to lung cancer, tobacco companies began producing filter-tipped brands that were claimed to remove certain components of smoke, which manufacturers have never acknowledged to be harmful.¹⁵ Brown & Williamson purchased advertising space in the Medicine section of *Time* to claim that Viceroy cigarettes offered "double-barrel health protection" and advertisements for Liggett & Myers' filter L & Ms claimed they were "Just what the doctor ordered." Until the 1960s tobacco companies promoted cigarettes at meetings of the AMA and other health organizations by means of scientific exhibits that sought to demonstrate the alleged benefits of one brand over another. Consumer demand soared. Currently, 97% of those who smoke buy filtered brands.

In the 1960s, to allay public anxiety about cancer after the publication of the first Surgeon General's Report on Smoking and Health, tobacco companies began marketing brands with purportedly lower levels of "tar" and nicotine. Throughout the 1970s the ACS, the NCI, and most major health organizations promoted the concept of a safer cigarette in the belief that most people who smoke cannot stop.¹⁵ Persons who switch to allegedly low-tar cigarettes have been found to employ compensatory smoking, whereby they inhale more frequently

Age-standardized death rates per 100,000 women



Source: Cancer Prevention Studies I and II, American Cancer Society

and more deeply to maintain a satisfied level of nicotine.^{14,15,71,72} More simply, "low tar" can be translated as "low poison."⁷³ Tar is a composite of more than 4000 separate solid poisons, including at least 43 known carcinogens.^{11,71} Cigarettes with reduced yields of tar, nicotine, and carbon monoxide are not safer. A recommendation to switch to such brands is misguided.

Not until 1980 did the NCI drop its research effort to develop a less hazardous cigarette, choosing instead to concentrate on efforts to educate heavy smokers to stop.⁷⁴

SPITTING TOBACCO

Snuff-dipping, the practice of placing a pinch of powdered flavored tobacco in the cavity between gum and cheek and sucking on the "quid," has increased dramatically among adolescents in the past 20 years. The consumption of chewing tobacco, the use of which involves a "chaw" that is held in the inner cheek area, has also increased.⁷⁵ Both forms of tobacco require continual expectoration, hence the term *spitting tobacco*. The manufacturers of these products prefer the term *smokeless tobacco*, implying that it is a safe alternative to smoking. After the publication in 1964 of the Surgeon General's Report on Smoking and Health, sales of spitting tobacco began to increase.³ Between 1960 and 1970 sales of snuff and chewing tobacco increased 25% and between 1970 and 1980 sales doubled again. Connolly estimates that there are 16 million users of these products in the United States alone, of whom 3 million are younger than the age of 16.⁷⁶

Snuff can appreciably accelerate a litany of destructive changes, including gingival recession, tooth abrasion, and periodontal bone destruction. Leukoplakia (also called *snuff-dipper's keratosis*), a nonspecific white patch involving the epithelium of the oral mucosa, is most often attributed to the use of tobacco and is found in 18% to 64% of users.⁷⁶ About 1 in 20 cases of leukoplakia will undergo malignant transformation into an epidermoid carcinoma. *N*-nitrosornicotine, one of four tobacco-specific nitrosamines that have been isolated from snuff, has been shown to be tumorigenic in experimental animals.⁷⁵ Snuff has been found to contain other potent carcinogens, including polycyclic aromatic hydrocarbons and radiation-emitting polonium.

In India, where there is widespread chewing of betel nut and tobacco in combination, Jayant and colleagues found a sixfold higher risk for cancer of the oral cavity relative to the nonchewer, nonsmoker.⁷⁷

Until recently, snuff dipping in the United States was a practice confined largely to black women in the rural Southeast, in whom the chance of contracting oral cancer has been found for long-term users to be 50 times that of nonusers of snuff.⁷⁸ Similarly, for most of the 20th century, tobacco chewing was largely a custom among rural men. In 1980 Christen and associates called attention to widespread snuff-dipping and tobacco-chewing habits among baseball and football players in colleges, high schools, and elementary schools.⁷⁹

Such a phenomenon came at the heels of national television and print media advertising by the United States Tobacco Company (UST) for its Skoal and Copenhagen snuff products that featured testimonials of well-known professional athletes and country music performers. A pioneer in the practice of

offering free samples of snuff by mail and at concerts and sporting events, UST boasted in a tobacco trade journal in 1984 that its advertisements in such publications as *The National Enquirer*, *Playboy*, *Sports Illustrated*, and *The New York Times Magazine* generated 400,000 written requests for samples in just 3 months.⁸⁰ Although television advertising for spitting-tobacco products was prohibited by the Comprehensive Smokeless Tobacco and Education Act of 1986, the promotion of these products on television has continued virtually unabated in the form of sponsored sporting events. In 1991, the Federal Trade Commission acted to limit the violations of the law by the Pinkerton Tobacco Company, sponsors of the televised "Red Man Chew Tractor Pulling Series," but it remains to be seen if other companies' brand names, such as UST's Skoal, equally visible in televised auto racing and rodeo, will disappear from the airwaves. (The Justice Department, which is entrusted with enforcement of the law that since 1971 has prohibited cigarette advertising on television, has never challenged the ubiquitous presence of tobacco promotion in sports on television; in contrast, the Federal government in Australia, following the lead of the states of Victoria and New South Wales, banned tobacco sponsorship of sports in 1992.)

Efforts of Connolly⁷⁶ and others have led to a ban on spitting tobacco in New Zealand (1987), Ireland (1988), Hong Kong (1988), and Australia (1990). In 1991, the European Bureau for Action on Smoking Prevention successfully campaigned for a ban on these products in the European Economic Community.

INVOLUNTARY (PASSIVE) SMOKING

Two thirds of the smoke from a burning cigarette never reaches the smoker's lungs, but instead goes directly into the air.⁸¹ The 1986 Report of the Surgeon General, dedicated to a discussion of involuntary or passive smoking, defined environmental tobacco smoke (ETS)—also called *secondhand smoke*—as the combination of sidestream smoke that is emitted into the air from a burning cigarette between puffs and the fraction of mainstream smoke that is exhaled by one who smokes.⁸¹

An increasing number of studies has explored the health risks of the nonsmoker who is exposed to ETS.^{11,81,82} The toxic and carcinogenic effects of ETS are similar to those of tobacco smoke inhaled by active smokers. The National Research Council has estimated that ETS is responsible for as many as 6000 lung cancer deaths among nonsmokers per year.⁸²

At least 14 studies have demonstrated a risk of lung cancer in nonsmoking wives exposed to the secondhand smoke of their husbands.⁹ Passive smoking has been found to increase the risk of leukemia, lymphoma, and cancer of the breast and uterine cervix.^{8,42}

The risks of passive smoking extend 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.^{31,82a,82b} Most of these (32,000) are due to heart disease, making passive smoking the third leading preventable cause of death after smoking and the consumption of alcohol. Additionally, children of parents who

smoke have an increased incidence of cough, bronchitis, otitis media, and pneumonia.¹¹ Children exposed to their parents' cigarette smoke have six times the average number of respiratory infections.⁹

EFFORTS TO CURTAIL TOBACCO USE

Although there is hardly a child or adult who has not heard that smoking is dangerous to health, the prevalence of smoking has declined by only 0.5% per year in the United States during the past 10 years.¹¹ By repeatedly citing seemingly improving prevalence figures and mentioning the 40 million Americans who have stopped smoking since 1964, health agencies underemphasize the fact that the number of current smokers has remained virtually constant at more than 55 million. Women, blue-collar workers, and minority groups in general are not appreciably reducing their cigarette consumption, and smoking rates among adolescents appear to be approaching the rates found in adolescents in the mid-1970s.⁸³ Although physicians and other health professionals should be working to end the tobacco pandemic, comparatively few are taking concerted action.^{9,18,84,85} One obstacle is complacency stemming from the belief by some health professionals and some of the public that the war on smoking has been won.

The remaining discussion in this chapter concerns the challenge to health care professionals to reexamine their approaches, their attitudes, and their vocabulary and to begin looking at the tobacco problem as much in terms of promoting a consumerist message of not *buying* cigarettes as in terms of promulgating a health behavior of not smoking. Such a view may lead to a better understanding of why tobacco advertising has been more successful than health education and why the tobacco companies could be considered among the leading health educators.

INITIAL EFFORTS: PUBLIC INFORMATION AND SMOKING CESSATION

In the late 19th century and early 20th century, the crusading efforts of people such as Lucy Page Gaston led to the enactment of numerous laws prohibiting smoking in public places. Much of this success was undone by efforts on college campuses to portray smoking as a symbol of women's emancipation and by medical societies that raised money to send cartons of cigarettes to the soldiers during World War I. Although the impact of publicity that surrounded the release of the Surgeon General's Report in 1964 was demonstrated by an increased awareness of smoking-related health risks, this short-term dissemination of information did little to solve the problem.¹⁶ Although programs emerged to help adults in their efforts to stop smoking, comparatively few resources have been devoted to primary prevention. The longstanding focus of tobacco control activities on cessation assumes that the major determinants of smoking behavior are within the individual person; the propaganda that promotes the initiation of tobacco use and helps perpetuate it has been ignored largely by government health agencies and researchers.

Approximately 300 cessation methods have been reported in the literature.⁸⁶ Popular techniques in the 1960s and 1970s included 5-day plans, group therapy, hypnosis, conditioning-

based approaches such as rapid smoking and satiation, self-help manuals, special filters, and over-the-counter pharmaceutical products containing either nicotine analogs or aversive chemicals. Approaches that were popularized in the 1980s included acupuncture, nicotine chewing gum, and physician counseling. In 1992, the introduction of transdermal nicotine patches through extensive promotional efforts aimed at pharmacists, physicians, and the lay public has created intense interest in smoking cessation. As with previous pharmacologic aids, the great expectations for the patch are unlikely to be fulfilled.

"Quit clinics" have been developed in the past 10 years by the ACS (FreshStart Program) and the American Lung Association (Freedom From Smoking) designed to be implemented in small-group sessions to help participants understand why people smoke, to handle withdrawal symptoms, and to manage stress. Such methods focus on cognitive and behavioral approaches, mostly neglecting attitudinal objectives.

In 1982, the NCI initiated its Smoking, Tobacco, and Cancer Program (STCP) as part of a restructuring of its cancer control activities. Out of the STCP, the NCI developed the Community Intervention Trial for Smoking Cessation (COMMIT), the largest smoking intervention trial in the world. The project, which includes 11 pairs of matched communities (one community in each pair serves as the intervention site and one as the control site), focuses on interventions primarily among heavy smokers. Changes in community smoking prevalence rates are being monitored throughout the trial.

More recently, the NCI (with logistic support from the ACS) has embarked on a major tobacco control project called the *American Stop Smoking Intervention Study for Cancer Prevention* (ASSIST). The project, which provides funds to the health departments in 17 states, began in 1991 and concludes in 1998. Each of the 17 funded states has assembled a coalition to disseminate materials through specific channels of intervention, including health care agencies, worksites, schools, media, and community networks. The ambitious goal of this \$120 million project is to assist the NCI in achieving its goal of reducing cancer mortality rates by 50% by the end of the century. Because the tobacco industry will spend more than \$28 billion on advertising and promotion during the years of ASSIST, critics decry this goal as overly optimistic.

Although 1.5 million Americans stop smoking each year, a similar number of adolescents begin smoking. At the same time, tobacco companies have maintained and increased efforts to promote smoking. Their appeals to freedom, wealth, glamour, manliness, athletic prowess, and sexual attractiveness undermine public health efforts.

Smoking cessation programs for the individual person cannot truly succeed in the absence of both workplace smoking bans and multimedia counter-advertising strategies that weaken the influence of the tobacco industry and reinforce the physician's office-based efforts.

Although cigarette smoking becomes an addiction, it is first a learned behavior. The "peer pressure" cited by tobacco companies as the reason for adolescent smoking is as much a manufactured product as the cigarette. The purpose of advertising is to sell cigarettes, 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. Cigarette manufacturers spend more money annually to promote smoking than is spent to advertise any other consumer product, including automobiles and food. More money is spent in 1 day in the United States to advertise cigarettes—\$10 million—than the entire annual budget of the Office on Smoking and Health.

A CONSUMERIST APPROACH TO SMOKING CESSATION

Ideally, the validity of the success rate of a smoking cessation method should rest on the results of a controlled, double-blind study for which there is a follow-up of at least 6 months' duration of all participating subjects.^{86,87} Few published outcome evaluations meet such criteria. Despite insufficient evidence to back up advertised claims, expensive commercial aids and clinics for smoking cessation proliferate. Many methods are costly, but having to pay a high fee for an alleged smoking cure may be the most motivating aspect of the method's success.

The physician's active involvement in smoking cessation, akin to his or her role in the prevention of smoking among adolescents and children, can be extremely crucial. More than 10 years ago, at a time when efforts to discourage smoking were much less widespread and accepted, Russell and colleagues found that 1 or 2 minutes of simple but unequivocal advice to stop smoking on the part of the physician resulted in a cessation rate of more than 5% measured at 1 year compared with 0.3% in the control group.⁸⁸

Although many people say they have stopped on their own, such persons may not consciously attribute their success to the increasing social pressures that reinforced their decision. Not only has organized medicine become united in the past few years on the need for more assertive office-based and community-wide strategies to end smoking, but other forces in society, including large corporations and governmental agencies, have implemented smoke-free policies.

OFFICE-BASED STRATEGIES

Many factors may inhibit physician involvement in smoking cessation, such as time constraints, the lack of reimbursement by third-party payers for such counseling, and the absence of peer group reinforcement in a technologically oriented, tertiary care-centered health care system.

There is much the physician can do to become a better teacher about smoking in lieu of relegating this role to ancillary personnel, a smoking cessation clinic, or a pamphlet. 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.

Magazines with cigarette advertisements should not appear in the physician's office in the absence of prominent stickers or rubber-stamped messages calling patients' attention to the deceptive, often absurd nature of such ads. 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."

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—the physician would do well to impart, through graphic posters, pamphlets, slides, and other audiovisual aids, the gruesome consequences of smoking—but the benefits of not smoking must be emphasized as strongly. Educating patients about the facts of smoking in a single office visit is unlikely to result in behavioral change.

Through the use of creative analogies related to the patient's occupation, hobbies, or romantic interest, the physician can succeed in changing the patient's 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, may mean little at first. By noting that cyanide is the substance used in the gas chamber in executions, that formaldehyde is used to preserve cadavers, and that ammonia is the predominant smell in urine, the physician is likely to make the patient think differently about cigarettes.

METAPHORS THAT MOTIVATE

A change in vocabulary on the part of the physician is essential for making progress in office-based smoking cessation. Instead of *pack-year history*, a more relevant term is the *inhalation count*. A pack-a-day smoking patient will breathe as many as 1 million doses of cyanide, ammonia, carcinogens, and carbon monoxide in less than 15 years, not including the inhalation of other peoples' smoke. Another way to emphasize the enormous amount smoked is to state the amount smoked in financial terms: a pack-a-day cigarette buyer will spend in excess of \$800 a year (calculated at \$2.25 a pack)—or in excess of \$10,000 in 10 years if that money were put into a savings account or bond.

Although patient education and smoking cessation rest on the knowledge 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 existing pamphlets and other audiovisual aids or by producing one's own handout. 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 provides 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 student, a construction worker, and an executive already showing signs or symptoms of heart disease. In the case of a high-school student, the physician should focus not only on such topics as emphysema and lung cancer, but also should emphasize the cosmetic unattractiveness of yellow teeth, bad breath, the loss of athletic ability, and the financial drain that results from buying ciga-

rettes. To the construction worker, the physician might suggest the likelihood of fewer lost paydays, greater physical strength, and a greater ability to work if he or she should stop smoking.

In talking with the concerned executive, one should demythologize certain beliefs about smoking, such as that the ultra-low-tar cigarettes being smoked are safe. To the contrary, use of so-called low-tar brands may result in compensatory deeper inhalation of greater concentrations of chemical additives and noxious gases that increase the risk for heart attack.

DEBUNKING COMMON MYTHS

An important myth surrounding smoking is that it relieves stress. This idea can be debunked by pointing out that the stress that is relieved is that which resulted from being dependent on nicotine—this is the essence of addiction. At the same time, deep breathing has a relaxing effect. The physician can suggest that the patient try to postpone for 5 minutes every time he or she intends to light up, then inhale deeply for 5 minutes, then reconsider whether the cigarette is important.

Another myth reinforced in advertisements for Virginia Slims and other cigarettes aimed at women and girls is that smoking keeps weight off. 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 less than 5 lb.⁸⁹

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 smoking cessation will have a greater success rate, those patients who do not express an interest in smoking cessation symbolize the overall challenge to be faced in curing the pandemic. One of the reasons for the lack of motivation of patients may be their sense of inevitability of failure. It is conceivable that by not educating the nonmotivated smoking patient, the physician is reinforcing the notion that it may be too difficult to stop smoking.

Setting a quit date, the essential element of the smoking cessation literature, may rationalize the continuation of an adverse health practice and may strengthen denial. It is helpful to remind patients that they can stop now. If they do not stop, this does not mean the physician will not treat them the next time, but it is important to give encouragement and not reinforce excuses. It is helpful to give patients a few written reminders such as lists of the advantages and disadvantages of smoking, a set of rewards for not smoking and penalties for lighting up, the situations and environmental influences that encourage one to smoke, and the myths of smoking and smoking cessation. 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 may provide the physician with

relevant data for charting purposes, this approach is too often a signal for the patient to become defensive and resistant to further discussion, especially if the patient had no intention to stop smoking. 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 a know-it-all and a polemicist. A question about the cost of cigarettes shows concern for the patient's financial well-being.

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, easy to prescribe for, nonindividualized solution. When a patient requests a "drug that will help me stop smoking," the physician must confront the dilemma of not wanting to dash the patient's expectation while emphasizing that a drug or device is, at best, an adjunct and not a means of smoking cessation.

It is an unfortunate fact that many patients will not stop smoking until they have gone to a smoking cessation clinic.

APPROACH TO ADOLESCENTS

Children and adolescents who smoke cigarettes pose a special challenge, because they represent the market most carefully nurtured by tobacco advertisers. It is essential to avoid emphasizing the adult and dangerous nature of smoking. Smoking should be referred to as the self-deceiving and short-sighted practice that it is. The single most important statement the physician can make to an adolescent is, "Come on, you're too old to smoke. That's for 11- and 12-year-old children who are trying to look grown up." Another strategy is for the physician to ask the adolescent who smokes to help think of ideas for talking to junior high school and primary school students who are just taking up smoking.

As a general rule in approaching the subject of smoking cessation with a patient, 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.

ENDING THE TOBACCO PANDEMIC

In 1977, a physician-based organization, DOC,* was founded to educate the public, especially young people, about the major preventable causes of poor health and high medical costs. Its primary goal is to tap the highest possible level of commitment from every physician, resident, and medical student in ending the tobacco pandemic.

* For more information about DOC and its programs, write to DOC, c/o Department of Family Medicine, Baylor College of Medicine, 5510 Greenbriar, Houston, TX 77005.

TABLE 20-23. Thirteen Steps to End the Cigarette Pandemic

1. Paid mass media counteradvertising
2. Dedicated excise tax to purchase counteradvertising
3. Clean indoor air legislation
4. Removal of tax exemptions from tobacco advertisers
5. Advertising and promotion bans
6. School-based campaigns to engender ridicule toward tobacco companies and cigarette advertising
7. Lawsuits against tobacco advertisers by relatives of dead and dying smokers
8. Enforcement of existing financial penalties for violating 1969 Public Health Smoking Act ban on TV cigarette advertising: \$10,000 per violation; enforcement of criminal conspiracy laws
9. Divestment of tobacco stocks by universities, hospitals, health groups, insurance companies, and teacher pension funds
10. Legislation to reduce adolescent access to tobacco through bans on vending machines, free samples
11. Worldwide coordination of efforts to curtail U.S. and U.K. cigarette exports and promotion
12. Agricultural changes to end tobacco subsidies and World Bank support of tobacco growing
13. Smoking cessation programs

DOC's unique, multilayered approach involves the creation of strategies for the clinic, the classroom, and the community (Table 20-23). Although there have been significant strides made by the NCI and the AMA during the 1980s to encourage greater involvement of physicians with tobacco control, most programs have underused physicians, physicians-in-training, and other health professionals.

To begin to realize a smoke-free society, physicians and other health care professionals must expand their vision beyond the stream of individual patients passing through their examining rooms to a concern for proactively and systematically dealing with the health needs of the larger community.

REFERENCES

1. Peto R. Report on smoking-attributable mortality. World Conference on Smoking or Health. Buenos Aires, Argentina, April 1982.
- 1a. Burney LE. Policy over politics: The first statement on smoking and health by the Surgeon General of the United States Public Health Service. *NY State J Med* 1983;83:1252-1253.
2. US Department of Health, Education, and Welfare. Smoking and health: Report of the Advisory Committee to the Surgeon General. Atlanta, GA: Centers for Disease Control (PHS) 1103, 1984.
3. Lombard HL, Doering CR. Cancer studies in Massachusetts: Habits, characteristics, and environment of individuals with and without cancer. *N Engl J Med* 1928;198:481-487.
4. Pearl R. Tobacco smoking and longevity. *Science* 1938;87:216-217.
- 4a. Doll R, Hill AB. Lung cancer and other causes of death in relation to smoking: Second report on mortality of British doctors. *Br Med J* 1966;2:1071-1081.
5. Ochaner A, DeBakey ME. Primary pulmonary malignancy: Treatment by total pneumonectomy: Analysis of 79 collected cases and presentation of 7 personal cases. *Surg Gynecol Obstet* 1939;68:435.
6. Hammond EL, Horn D. Smoking and death rates—Report on forty-four months of follow-up of 187,783 men. *JAMA* 1956;166:1294-1308.
- 6a. American Medical Association Committee for Research on Tobacco and Health. Tobacco and health. Chicago: American Medical Association Education and Research Foundation (AMA-REF), 1978.
7. US Department of Health and Human Services. The Health Consequences of Smoking: Cancer: A Report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Office on Smoking and Health. DHHS (PHS) 82-50179, 1982.
8. Redmond DE. Tobacco and cancer: The first clinical report, 1761. *N Engl J Med* 1970;282:18-23.
9. Rakel RE, Blum A. Nicotine addiction. In: Rakel RE, ed. *Textbook of family practice*. 4th ed. Philadelphia: WB Saunders, 1980:1612-1623.
10. Royal College of Physicians. Smoking and Health: Summary and Report of the Royal College of Physicians of London on smoking in relation to cancer of the lung and other diseases. New York: Pitman, 1962.
11. US Department of Health and Human Services. Reducing the health consequences of smoking—25 Years of Progress: A report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Office on Smoking and Health. DHHS (CDC) 89-8411, 1986.
12. Damber LA, Larsson LG. Smoking and lung cancer with special regard to type of smoking and type of cancer. *Br J Cancer* 1986;53:673.
13. Kaufman DW. Constituents of cigarette smoke and cardiovascular disease. *NY State J Med* 1983;83:1267-1268.
14. Rickett WS. "Less hazardous" cigarettes: Fact or fiction? *NY State J Med* 1983;83:1269-1272.
15. Miller GH. The "less hazardous" cigarette: A deadly delusion. *NY State J Med* 1985;85:313-317.
16. US Department of Health and Human Services. Strategies to control tobacco use in the United States: A blueprint for public health action in the 1990s. Washington, DC: US Department of Health and Human Services, National Cancer Institute. DHHS (NIH) 92-3316, 1991.
17. Blum A. When "more doctors smoked Camels": Cigarette advertising in the journal. *NY State J Med* 1983;83:1347-1352.
18. Lubin JH, Blot WJ, Berrino F, et al. Modifying risk of developing lung cancer by changing habits of cigarette smoking. *Br Med J* 1984;288:1953-1956.
19. Lubin JH, Blot WJ, Berrino F, et al. Patterns of lung cancer risk according to type of cigarette smoked. *Int J Cancer* 1984;3:569-576.
20. US Department of Health and Human Services. The health benefits of smoking cessation: A report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Office on Smoking and Health. DHHS (CDC) 90-8416, 1990.
- 20a. Berry G, Newhouse ML, Antonis P. Combined effect of asbestos and smoking on mortality from lung cancer and mesothelioma in factory workers. *Br J Med* 1985;42:12-18.
- 20b. Selikoff I, Seidman H, Hammond EC. Mortality effects of cigarette smoking among amosite asbestos factory workers. *JNCI* 1980;65:507-513.
21. US Department of Health, Education, and Welfare. Smoking and health: A report of the Surgeon General. Washington, DC: Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW (PHS) 79-50066, 1979.
22. Doll R, Peto R. Mortality in relation to smoking: 20 years observation on male British doctors. *Br Med J* 1976;2:1525-1536.
23. Kahn HA. The Dorn study of smoking and mortality among U.S. veterans: Report on eight and one-half years of observation. In: Haenszel W, ed. *Epidemiological approaches to the study of cancer and other chronic diseases*. Washington, DC: National Cancer Institute Monograph No. 19, US Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, 1966:1-125.
24. Hammond EC. Smoking in relation to the death rates of one million men and women. In: Haenszel W, ed. *Epidemiological approaches to the study of cancer and other chronic diseases*. Washington, DC: National Cancer Institute Monograph No. 19, US Department of Health, Education, and Welfare, Public Health Service, National Cancer Institute, 1966:127-204.
25. Weir JM, Dunn JE. Smoking and mortality: A prospective study. *Cancer* 1970;25:105-112.
26. Hirayama T. Smoking in relation to the death rates of 265, 118 men and women in Japan: A report on five years of follow-up. Clearwater Beach, FL, American Cancer Society's Fourteenth Science Writers' Seminar, March 24-29, 1972:1-4.
27. Cullen JW. Principles of cancer prevention: Tobacco. In: DeVita VT, Hellman S, Rosenberg S, ed. *Cancer: Principles and practice of oncology*. 3rd ed. Philadelphia: JB Lippincott 1989:181-195.
28. Williams RR, Horn JW. Association of cancer sites with tobacco and alcohol consumption and socioeconomic status of patients: Interview study from the Third National Cancer Survey. *JNCI* 1977;58:525-547.
29. Flanders WD, Rochman KJ. Interaction of alcohol and tobacco in laryngeal cancer. *Am J Epidemiol* 1982;115:371-379.
30. Marks RD, Putney FJ, Scragg HJ, et al. Management of cancer of the larynx. *J SC Med Assoc* 1975;71:333-336.
31. US Department of Health and Human Services. The health consequences of smoking: Cancer and chronic lung disease in the workplace: A report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Office on Smoking and Health. DHHS (PHS) 85-50207, 1985.
32. US Department of Health and Human Services. The health consequences of using smokeless tobacco: A report of the Advisory Committee to the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service. NIH Publication No. 86-2874, 1986.
33. Banoczy J, Sugar L. Progressive and regressive changes in Hungarian oral leukoplakias in the course of longitudinal studies. *Commun Dentist Oral Epidemiol* 1975;3:194-197.
34. Roed-Petersen B, Banoczy J, Pindborg JJ. Smoking habits and histological characteristics of oral leukoplakias in Denmark and Hungary. *Br J Cancer* 1973;28:576-579.
35. Sugar L, Banoczy J. Follow-up studies in oral leukoplakia. *Bull WHO* 1969;41:289-293.

36. Blot WJ, McLaughlin JK, Winn DM, et al. Smoking and drinking in relation to oral pharyngeal cancer. *Cancer Res* 1988;48:3282-3287.
37. Schottenfeld D, Ganitt RC, Wynde EL. The role of alcohol and tobacco in multiple primary cancers of the upper digestive system, larynx, and lung: A prospective study. *Prev Med* 1974;3:277-293.
38. Schottenfeld D. Epidemiology of cancer of the esophagus. *Semin Oncol* 1984;11:92-100.
39. Newcomb PA, Carbone PP. The health consequences of smoking. *Med Clin North Am* 1992;76:305-331.
40. Baron JA, Byers T, Greenberg, ER, et al. Cigarette smoking in women with cancers of the breast and reproductive organs. *JNCI* 1986;77:677-680.
41. Brinton LA, Schainer C, Haenszel W, et al. Cigarette smoking and invasive cervical cancer. *JAMA* 1986;255:3265-3269.
42. Slatery ML, Robison LM, Schuman K, et al. Cigarette smoking and exposure to passive smoke are risk factors for cervical cancer. *JAMA* 1989;261:1593-1598.
43. Boring CC, Squires TS, Tong T. Cancer statistics 1991. *CA* 1991;41:19.
44. Sasson IM, Haley M, Hoffman D, et al. Cigarette smoking and neoplasia of the uterine cervix: Smoke constituents in cervical mucus. *N Engl J Med* 1985;312:315-316.
45. Holly EA, Petrakis NL, Friend NF, et al. Mutagenic mucus in the cervix of smokers. *JNCI* 1986;76:983-986.
46. Koutsky LA, Galloway D, Holmes KK. The epidemiology of genital papilloma virus infection. *Epidemiol Rev* 1988;10:120.
47. McLaughlin. Silverman DT, Hsing AW, Ross RK. Cigarette smoking and cancers of the renal pelvis and ureter. *Cancer Res* 1992;52:254-257.
48. Hartge P, Silverman D, Hoover R, et al. Changing cigarette habits and bladder cancer risk: A case-control study. *JNCI* 1987;78:1119.
49. Burch JD, Rohan TE, Howe GR, et al. Risk of bladder cancer by source and type of tobacco exposure: A case-control study. *Int J Cancer* 1989;141:622.
50. Severson RK. Cigarette smoking and leukemia. *Cancer* 1987;60:141.
- 50a. Mills PK, Newell GR, Beeson WL, et al. History of cigarette smoking and risk of leukemia and myeloma: Results from the Adventist health study. *JNCI* 1990;82:1832-1836.
51. Baron JA. Smoking and estrogen related disease. *Am J Epidemiol* 1984;119:9.
52. US Department of Health and Human Services. The Health consequences of smoking: Cardiovascular disease: A report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Office on Smoking and Health. DHHS (PHS) 84-50204, 1983.
53. Willett WC, Green A, Stampfer MJ, et al. Relative and absolute risks of coronary heart disease among women who smoke cigarettes. *N Engl J Med* 1987;317:1303-1309.
54. Martin JL, Wilson JR, Ferraro N, et al. Acute coronary vasoconstrictiveness effects of cigarette smoking in coronary heart disease. *Am J Cardiol* 1984;54:56-60.
55. Fitzgerald GA, Oates, JA, Nowak J. Cigarette smoking and hemostatic function. *Am Heart J* 1988;115:267-271.
56. Wolf PA, D'Agostino RB, Kannel WB, et al. Cigarette smoking as a risk factor for stroke: The Framingham study. *JAMA* 1988;259:1025-1029.
57. Stuart-Harris CH. The epidemiology and evolution of chronic bronchitis. *Br J Tuberculosis Dis Chest* 1954;48:169-178.
58. Stuart-Harris CH. Chronic bronchitis: I. *Abstr World Med* 1968;42:649-669.
- 58a. Sobel R. They satisfy: The cigarette in American Life. New York: Anchor Press/Doubleday, 1978.
- 58b. Einster V. Mixed messages for women: A social history of cigarette smoking and advertising. *NY State J Med* 1985;85:335-340.
59. O'Conner JJ. Women top cigaret target. *Advertising Age* 1981;52:9, 93.
60. Trone Advertising Inc. V. F. Year: I. Promotion recommendations. North Carolina: Trone Advertising, 1989.
61. US Public Health Service. The Health Consequences of Smoking: 1969 Supplement to the 1967 Public Health Service Review. Washington, DC: US Department of Health, Education and Welfare, Public Health Service. PHS Publication No. 1696-2 (Supplement), 1969.
62. Jacobson B. The lady killers. London: Pluto Press 1981:53.
63. US Department of Health and Human Services. Report of the task force on black and minority health. Washington DC: US Department of Health and Human Services, 1985.
64. Blum A. The targeting of minority groups by the tobacco industry. In: Jones LA, ed. Minorities and cancer. New York: Springer-Verlag, 1989:153-162.
65. Citizens action handbook on tobacco and alcohol billboard advertising. Washington, DC, Scenic America, 1990.
66. Cooper R, Simmons B. Cigarette smoking and ill health among black Americans. *NY State J Med* 1985;85:344-347.
67. Ramirez A. A cigarette campaign under fire. *The New York Times*, January 12, 1990: D1, D4.
68. Romano PS, Bloom J, Syme SL. Smoking, social support, and hassles in an urban African-American community. *Am J Public Health* 1991;81:1415-1422.
69. US Department of Health and Human Services. Smoking tobacco, and cancer programs: 1985-1989 status report. Washington, DC: Public Health Service, National Institutes of Health, National Cancer Institute. NIH Publication No. 90-3107, 1990.
70. McGraw S, Smith K, Schensal J, Carrillo E. Sociocultural factors associated with smoking behavior by Puerto Rican adolescents in Boston. *Soc Science Med* 1991;33:1355-1364.
71. US Department of Health and Human Services. The health consequences of smoking: The changing cigarette: A report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Office on Smoking and Health. DHHS (PHS) 81-50156, 1981.
72. Benowitz NL, Hall SM, Herring RI, et al. Smokers of low-yield cigarettes do not consume less nicotine. *N Engl J Med* 1983;309:139-142.
73. Blum A. Cigarettes are so Kool—teenagers and the smoking epidemic. In: *Encyclopaedia Britannica, Medical Annual*, 1982.
74. Blum A. "Safe" cigarettes and other cons. *Phys Assist Health Pract* 1980;4:48.
75. Blum A. Smokeless tobacco. *JAMA* 1980;244:192.
76. Connolly G. personal communication, 1992.
77. Jayant K, Balakrishnan V, Sanghvi LD, et al. Quantification of the role of smoking and chewing tobacco in oral, pharyngeal, and esophageal cancer. *Br J Cancer* 1977;35:232-235.
78. Blum A. Using athletes to push tobacco to children: Snuff-dippin' cancer-lipped men. *NY State J Med* 1983;83:1365-1367.
79. Christen AG, McDaniel RK, Doran JE. Snuff dipping and tobacco chewing in a group of Texas college athletes. *TX Dent J* 1979;97:6-10.
80. Abrams R. Attorney General Abrams speaks out against smokeless tobacco. *NY State J Med* 1985;85:471.
81. US Department of Health and Human Services. The health consequences of involuntary smoking: A report of the Surgeon General. Washington, DC: Public Health Service, Centers for Disease Control, Office on Smoking and Health, Department of Health and Human Services, (CDC) 87-8398, 1986.
82. National Research Council, National Academy of Sciences. Environmental tobacco smoke: Measuring exposures and assessing health effects. Washington, DC: National Academy Press, 1986.
- 82a. Steenland K. Passive smoking and the risk of heart disease. *JAMA* 1992;267:94-99.
- 82b. Repace JL, Lowrey AH. Risk assessment methodologies for passive smoking-induced lung cancer. *Risk Anal* 1990;10:27-37.
83. Centers for Disease Control. Tobacco use among high school students—United States, 1990. *MMWR* 1991;40:617-619.
84. Every physician as a (potential) prevention specialist. In: The health care system and drug abuse prevention. Washington, DC: US Department of Health and Human Services, National Institute on Drug Abuse, 1981.
85. Blum A. Medicine vs. Madison Avenue: Fighting smoke with fire. *JAMA* 1980;243:739-740.
86. Schwartz JL. Review and evaluation of smoking cessation methods: The United States and Canada, 1978-1985. Washington, DC: US Department of Health and Human Services, Public Health Service, National Institutes of Health, NCI. NIH Publication No. 87-2940, 1987.
87. Schwartz JL. A critical review and evaluation of smoking control methods. *Public Health Rep* 1969;84:483-506.
88. Russell MA, Wilson C, Taylor C. The effects of general practitioners' advice against smoking. *Br Med J* 1979;2:231-235.
89. US Department of Health, Education, and Welfare. The health consequences of smoking: 1977-1978. Washington, DC: Office of the Assistant Secretary for Health, Office on Smoking and Health. DHEW Publication No. (PHS) 79-50065, 1979.