Smoking Cessation

Scott P Marlow RRT and James K Stoller MD MSc FAARC

Introduction
Epidemiology of Cigarette Smoking
Nicotine Addiction
Smoking Cessation Interventions: Overview
Behavioral Interventions
Pharmacologic Interventions

Nicotine Replacement Therapy
Smoking Cessation with Chantix
Buy Chantix - https://www.webhealthsearch.com/smoking-cessation/buychantix/
Bupropion
Second-Line Smoking Cessation Drugs

Alternative Smoking Cessation Interventions
Combined Smoking Cessation Interventions
Cost
The Respiratory Therapist's Role in Smoking Cessation Summary

Cigarette smoking is the primary cause of chronic obstructive pulmonary disease, and smoking cessation is the most effective means of stopping the progression of chronic obstructive pulmonary disease. Worldwide, approximately a billion people smoke cigarettes and 80% reside in low-income and middle-income countries. Though in the United States there has been a substantial decline in cigarette smoking since 1964, when the Surgeon General's report first reviewed smoking, smoking remains widespread in the United States today (about 23% of the population in 2001). Nicotine is addictive, but there are now effective drugs and behavioral interventions to assist people to overcome the addiction. Available evidence shows that smoking cessation can be helped with counseling, nicotine replacement, and bupropion. Less-studied interventions, including hypnosis, acupuncture, aversive therapy, exercise, lobeline, anxiolytics, mecamylamine, opioid agonists, and silver acetate, have assisted some people in smoking cessation, but none of those interventions has strong research evidence of efficacy. To promote smoking cessation, physicians should discuss with their smoking patients "relevance, risk, rewards, roadblocks, and repetition," and with patients who are willing to attempt to quit, physicians should use the 5-step system of "ask, advise, assess, assist, and arrange." An ideal smoking cessation program is individualized, accounting for the reasons the person smokes, the environment in which smoking occurs, available resources to quit, and individual preferences about how to quit. The clinician should bear in mind that quitting smoking can be very difficult, so it is important to be patient and persistent in developing, implementing, and adjusting each patient's smoking-cessation program. One of the most effective behavioral interventions is advice from a health care professional; it seems not to matter whether the advice is from a doctor, respiratory therapist, nurse, or other clinician, so smoking cessation should be encouraged by multiple clinicians. However, since respiratory therapists interact with smokers frequently, we believe it is particularly important for respiratory therapists to show leadership in implementing smoking cessation. *Key words: chronic obstructive pulmonary disease, COPD, smoking cessation.*
[Respir Care 2003;48(12):1238–1254. © 2003 Daedalus Enterprises]

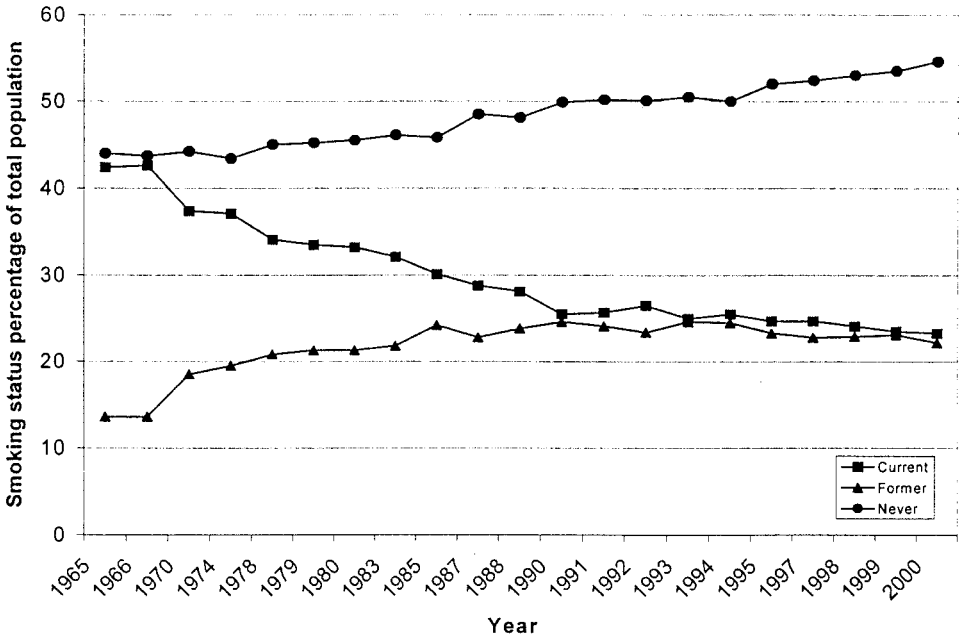

Fig. 1. Percentage of adult current, former, and never smokers. (Adapted from Reference 4.)

Introduction

In reviewing smoking cessation the present report first summarizes the epidemiology of smoking cessation and evidence that smoking causes harm. We next review the physiology of nicotine and smoking addiction and the benefits of smoking cessation. Finally, we present a systematic review of smoking cessation methods, with evidence-based recommendations.

Although primary smoking prevention (eg, education, regulation of advertising) is recognized as an integral part of combating smoking, the reader is referred to other recent publications for comprehensive reviews of this issue, such as the Report of the Surgeon General released in 2000: "Reducing Tobacco Use."[1]

Epidemiology of Cigarette Smoking

Smoking is a modern day epidemic that poses substantial health burden and cost. Worldwide estimates suggest that smoking prevalence has increased to approximately 1.1 billion people (1 in 3 adults), with 80% of these residing in low- and middle-income countries.[2] Smoking remains widespread in the United States, though trends show a substantial decline since 1964. Cigarette smoking was rare in the early 20th century, when the annual per capita United States consumption rate was 54 cigarettes. In 1964 the per capita consumption was 4,345 cigarettes/person/y, and that rate had declined to 2,261 cigarettes/person/y in 1998.[3] As shown in Figure 1, the prevalence of current smokers peaked in 1965 at 42.4% and had declined to 23.4% in 2001.[4,5] This decline has been referred to by the Centers for Disease Control and Prevention as one of the "Ten Great Public Health Achievements in the 20th Century."[6]

Though the decline in smoking certainly represents a favorable trend, smoking is, disturbingly, most common among groups of lower socioeconomic status. Figure 2 shows that the prevalence of smoking is higher among individuals in families with combined incomes $<$ \$9,000 (35% prevalence) than in families with a combined income exceeding \$75,000 (19% prevalence).[7] Figure 3 shows that these trends are also evident by education level, with a higher frequency of smoking among those who have not completed high school (33%) than among those who have completed 4 years of college (14%).[7] For example, in 1965, 51.9% of men and 33.9% of women reported smoking, whereas in 2000 25.7% of men and 21.0% of women reported smoking.[4,8] Smoking rates among high school students continue to exceed the national rate for adults, having increased during the

Scott P Marlow RRT and James K Stoller MD MSc FAARC are affiliated with the Department of Pulmonary and Critical Care Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio.

Scott P Marlow RRT presented a version of this report at the 32nd RESPIRATORY CARE Journal Conference, Chronic Obstructive Pulmonary Disease: Translating New Understanding Into Improved Patient Care, held July 11–13, 2003, in Los Cabos, Mexico.

Correspondence: Scott P Marlow RRT, Department of Pulmonary and Critical Care Medicine, A90, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland OH 44195. E-mail: marlows@ccf.org.

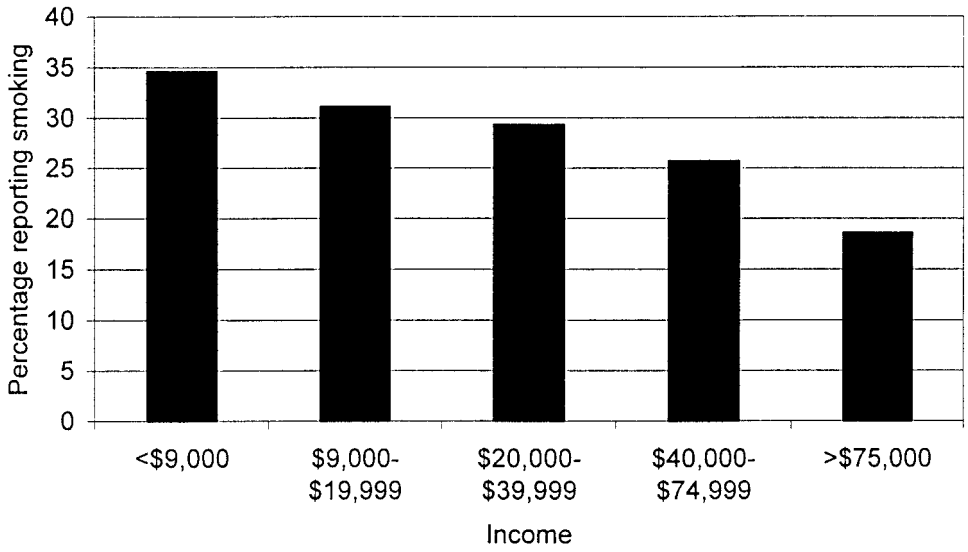

Fig. 2. Percentage of people ≥ 18 years old who reported smoking in the past month, by combined total family income, 1999–2000. (Adapted from Reference 7.)

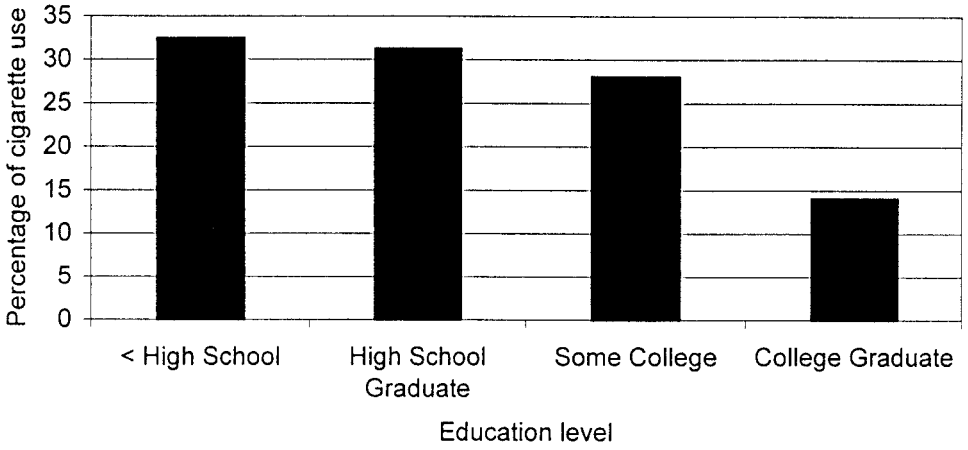

Fig. 3. Percentage of people ≥ 18 years old who reported smoking in the past month, by education level, 1999–2000. (Adapted from Reference 7.)

1990s to a peak of 36.4% in 1997 and since decreased to 28.5% in 2001 (29.2% among boys versus 27.7% among girls).[9]

Because smoking is common, the economic impact is profound. In 1998 the direct medical cost of smoking was estimated to be $75.5 billion, with productivity losses estimated at $82 billion and smoking-related neonatal costs estimated at $366 million.[10] When all expenses are combined, they represent a total of $3,391 per smoker per year, which amounts to approximately 8% of all annual health care expenditures in the United States.[10]

The morbidity and mortality attributed to smoking are also substantial. For example, between 1995 and 1999 approximately 440,000 deaths annually were attributed to smoking.[10] Smoking also increases the risk of death in many illnesses. Table 1 shows the relative risk of smoking-attributable morbidity and mortality from various conditions, largely including respiratory, cardiovascular, and neoplastic diseases.[11] Table 2 shows estimates of the number of deaths attributed to smoking. Notably, the 35,053 second-hand-smoke-related deaths are not included in those estimates.[10,11]

Smoking confers a risk of serious illness, and smoking cessation offers health benefits, which is evident in Table 1: former smokers have lower relative risk of death in all disease categories.[11] For example, compared to never-smokers, men who are current or former smokers have a higher relative risk of death from cancer of the trachea, lung, or bronchus (23.26 times higher in current smokers

Table 1. Relative Risk of Death: Current Smokers Versus Former Smokers

Cause of Death	Relative Risk of Death			
	Male		Female	
	Current Smoker	Former Smoker	Current Smoker	Former Smoker
Malignant Neoplasms				
Lip, oral cavity, pharynx	10.89	3.40	5.08	2.29
Esophagus	6.76	4.46	7.75	2.79
Pancreas	2.31	1.15	2.25	1.55
Larynx	14.60	6.34	13.02	5.16
Trachea, lung, bronchus	23.26	8.70	12.69	4.53
Cervix uteri	0.00	0.00	1.59	1.14
Urinary bladder	3.27	2.09	2.22	1.89
Kidney and renal pelvis	2.72	1.73	1.29	1.05
Cardiovascular Diseases				
Hypertension	2.11	1.09	1.92	1.02
Ischemic heart disease				
Age 35–64 yr	2.80	1.64	3.08	1.32
Age > 65 yr	1.51	1.21	1.60	1.20
Other heart disease	1.78	1.22	1.49	1.14
Age 35–64 yr	3.27	1.04	4.00	1.30
Age > 65 yr	1.63	1.04	1.49	1.03
Atherosclerosis	2.44	1.33	1.83	1.00
Aortic aneurysm	6.21	3.07	7.07	2.07
Other arterial disease	2.07	1.01	2.17	1.12
Respiratory Diseases				
Pneumonia, influenza	1.75	1.36	2.17	1.10
Bronchitis, emphysema	17.10	15.64	12.04	11.77
Chronic airways obstruction	10.58	6.80	13.08	6.78

(From Reference 11.)

Table 2. Smoking-Attributed Mortality

Cause of Death	Male (*n*)	Female (*n*)	Total (*n*)
Malignant Neoplasms			
Lip, oral cavity, pharynx	3,617	1,191	4,808
Esophagus	6,552	1,622	8,174
Pancreas	3,075	3,526	6,601
Larynx	2,471	602	3,073
Trachea, lung, bronchus	78,459	44,727	123,186
Cervix uteri	0	488	488
Urinary bladder	3,794	1,064	4,858
Kidney and renal pelvis	2,765	227	2,992
Total malignant neoplasms	100,733	53,447	154,180
Cardiovascular Diseases			
Hypertension	2,993	2,693	5,686
Ischemic heart disease	54,945	33,464	88,409
Other heart disease	13,102	8,325	21,427
Cerebrovascular disease	8,200	8,941	17,141
Atherosclerosis	1,422	797	2,219
Aortic aneurysm	6,113	3,080	9,193
Other arterial disease	520	846	1,366
Total cardiovascular diseases	87,295	58,146	145,441
Respiratory Diseases			
Pneumonia, influenza	6,103	4,929	11,032
Bronchitis, emphysema	9,032	7,247	16,279
Chronic airways obstruction	41,046	36,772	77,818
Total respiratory diseases	56,181	48,948	105,129
Totals	244,209	160,541	404,750

(From Reference 11.)

and 8.7 times higher in former smokers).[11] The 1990 report of the United States Surgeon General on the health benefits of smoking cessation concluded that people who quit smoking before the age of 50 have half the risk of dying in the next 15 years, compared to continuing smokers.[12] Smokers have twice the risk of dying of coronary heart disease or stroke, and the risk of coronary heart disease diminishes by half in the first year after cessation.[12] After 5–15 years of abstinence from smoking, the risk of both stroke and heart disease drops to the level of never-smokers.[12]

Another benefit of quitting smoking is a slowing of the accelerated rate of lung function decline that occurs in susceptible smokers. For example, the Lung Health Study randomized and followed 5,887 smokers with early chronic obstructive pulmonary disease in one of 3 arms: usual care; aggressive smoking intervention with ipratropium bromide via metered-dose inhaler; and smoking intervention with placebo inhaler.[13] Long-term follow-up of the Lung Health Study cohort found that 11-year sustained ex-smokers experienced a lower rate of FEV_1 decline (30.2 mL/y among men and 21.5 mL/y among women) than active smokers (66 mL/y among men and 54.3 mL/y among women).[14]

Nicotine Addiction

Concepts of nicotine addiction have evolved over the 40 years since the Surgeon General's report first reviewed smoking. For example, in 1964 the Report of the Advisory Committee to the Surgeon General classified tobacco as "an habituation rather than an addiction" and that preventing the psychogenic drive of the habit was more important than using nicotine substitutes.[15]

Concepts about the physiology of nicotine addiction have since evolved. For example, in 1979 the Report of the Surgeon General cited nicotine as "a powerful addictive drug."[16] And in 1988 the Report of the Surgeon General on The Consequences of Smoking: Nicotine Addiction[17] concluded:

- Cigarettes and other forms of tobacco are addicting.
- Nicotine is the drug in tobacco that causes addiction.
- Pharmacologic and behavioral processes that determine

tobacco addiction are similar to those that determine addiction to drugs such as heroine and cocaine.

Though debated, the physiology of nicotine addiction has recently been characterized as biphasic, in that it stimulates the pleasure response to the brain, and when taken for longer periods, also creates a relaxed state. As with cocaine, amphetamines, and morphine, addiction to nicotine is believed to result from increased release of dopamine in the region of nucleus acumbens.[18–20] Nicotinic acetylcholine receptors are located throughout the central nervous system, but the neurons located in the ventral tegmental area increase activity with nicotine administration and concurrently activate the increased release of dopamine into the nucleus acumbens.[18,21,22] Corrigall et al[23,24] found that self-administered nicotine in vivo is reduced by lesions to these pathways or by a nicotinic antagonist infused into the ventral tegmental area.

As with all addictions, nicotine withdrawal elicits a number of clinical consequences, avoidance of which promotes smoking. Nicotine withdrawal symptoms are time-limited, can last for several weeks, and include physical symptoms of irritability, anxiety, depression, difficulty concentrating, weight gain, restlessness, and impatience.[25] The intensity of these withdrawal symptoms can be related to the level of nicotine dependence. A common measuring tool is the Fagerstrom Test for Nicotine Dependence, which rates addiction on a 0–10 scale. The test places the most emphasis on the length of time after waking before the first cigarette and the number of cigarettes smoked per day.[26] As evidence of the power of nicotine addiction, estimates suggest that 70% of smokers would like to quit, that approximately 41% try to quit each year, but that only 4.7% remain abstinent.[27]

Smoking Cessation Interventions: Overview

The spectrum of available smoking cessation interventions can be classified into behavioral, pharmacologic, and alternative methods (Table 3). Behavioral interventions include physician advice, individual counseling, group counseling, and telephone counseling. Pharmacologic interventions include nicotine replacement therapy, sustained-release bupropion, clonidine, and nortriptyline. Finally, alternative (and less-studied) interventions include hypnosis, acupuncture, aversive therapy, exercise, lobeline, anxiolytics, mecamylamine, opioid agonists, and silver acetate.

Smoking cessation should begin with assessing the smoker's desire to quit. Table 4 describes the 5 "A"s: *ask*, *advise*, *assess*, *assist*, and *arrange*. For smokers unwilling to attempt quitting, the United States Department of Health and Human Services (USDHHS) Clinical Practice Guideline for Treating Tobacco Use and Dependence recommends discussing the 5 "R"s: *relevance*, *risk*, *rewards*, *roadblocks*, and *repetition* (Table 5).[28]

Table 3. Smoking Cessation Interventions

Behavioral Interventions
Physician advice
Individual counseling by nurse or other nonphysician
Group counseling
Telephone counseling
Self help
Drug Interventions
First-Line: Nicotine replacement therapy (transdermal patch, gum, inhaler, nasal spray)
Antidepressant (bupropion)
Second-Line: clonidine, nortriptyline
Other Interventions
Acupuncture
Hypnosis
Aversive therapy
Exercise
Mecamylamine
Lobeline
Anxiolytics

In summarizing the literature and offering recommendations, we first present evidence from the Cochrane Collaboration reviews of smoking cessation interventions, which considered studies up to 2002. We also review data presented in the USDHHS Clinical Practice Guideline for Treating Tobacco Use and Dependence, originally published in 1996 and updated in 2000.[28] To identify the most recent available data, we searched MEDLINE for research conducted in 1999 through June 2003, using the search terms "smoking cessation" and "tobacco." Our ratings of the strength of the available evidence are based on the system adopted by Fiore et al (Table 6).[28] To assess the efficacy of the available interventions, we largely restricted the analysis to studies that compared interventions to no-intervention control groups.

Overall, the available literature supports the efficacy of behavioral counseling, nicotine replacement, and bupropion in smoking cessation (Table 7). Clinicians should know that an ideal smoking cessation program is individualized, accounting for the person's reasons to smoke, the environment in which smoking occurs, available resources to quit, and individual preferences about how to quit.

Behavioral Interventions

Behavioral interventions differ according to who is performing the intervention: the physician, nurse, nonphysician clinician, telephone counselor, or patient self-help. The present analysis compares cessation rates for each behavioral intervention to a control group with no (or

Table 4. Smoking Cessation Intervention: The 5 "A"s

Ask about tobacco use	Identify and report status
Advise to quit	Be clear, strong, and personalized
Assess willingness to quit	If patient is willing to quit, assess potential intensity of support
	If patient is not willing to quit, see Table 5
Assist in quit attempt	Help with quit plan
	Set a quit date, usually within 2 weeks
	Enlist support and understanding of family and friends
	Anticipate challenges, especially first few weeks
	Remove tobacco products from environment
	Provide practical counseling (eg, problem-solving and skills training)
	Stress abstinence
	Review past quit experience
	Anticipate triggers and challenges
	Review relationship of alcohol to tobacco use
	Point out that having other smokers in the home will increase the difficulty
	Provide treatment and social support
	Provide a supportive clinical environment
	Help obtain extra treatment social support
	Help obtain patient-environment support from family, friends, and coworkers
	Recommend pharmacotherapy
	Provide supplementary materials:
	Sources: organizations that promote smoking cessation, including federal, state, and nonprofit organizations
	Type: Are the materials appropriate for the patient, in relation to culture, race, education, and age?
	Location: Are the materials readily available?
Arrange follow-up	Schedule follow-up
	Timing: Follow up within the first week of the quit date, and follow up again within the first month
	Actions during follow-up:
	Congratulate
	Review
	Stress abstinence
	Remind that lapse is a learning experience
	Identify potential and current problems
	Assess pharmacotherapy
	Consider increased intervention when necessary

(Adapted from Reference 28.)

minimal) intervention. Limitations of available studies are that behavioral interventions are supplied in a variety of environments or with other (confounding) interventions. For example, a study may provide physician and group advice for smoking cessation along with nicotine gum, as in the Lung Health Study,[13] thereby confounding direct comparisons of the nicotine and the advice.

Among the simplest of behavioral interventions, even brief, direct physician advice to quit smoking is effective. For example, in the Cochrane Library review of 16 studies, Silagy et al[29] found that brief physician advice increased the absolute rate of abstinence by 2.5% over usual care (odds ratio [OR] 1.69, 95% confidence interval [CI] 1.45–1.98). Furthermore, the rate of smoking abstinence increased when the intensity of advice was increased and when follow-up visits were included (OR 1.44, 95% CI 1.23–1.68 and OR 2.66, 95% CI 2.06–3.45).[29] In agreement with the Cochrane Collaboration reviews, findings from the USDHHS Clinical Practice Guideline review of available studies showed that brief (ie, 2–5 min) physician advice was associated with a 2–3% higher rate of smoking cessation (OR 1.3, 95% CI 1.1–1.6).[28] Denny et al[30] reported that 70% of smokers who had seen their doctor within the last 12 months received advice to quit smoking. However impressive that 70% figure appears, the 30% of smokers who did not receive quit-smoking advice is approximately 1,915,000 smokers in the 10-state survey area—a tremendous number of missed opportunities to encourage smoking cessation.[29] If only 2.3–2.5% of those individuals had quit after brief advice, an additional 44,000–48,000 smokers may have quit.

With regard to counseling by nurses, Rice and Stead[31] reported small increases in smoking cessation rate follow-

Table 5. Enhancing Motivation to Quit Tobacco: The 5 "R"s

Relevance	Why would quitting be personally relevant? Consider family, children, health concerns, previous experience, work
Risk	Clinician should ask patient to identify negative consequences of smoking:
	Highlight those most relevant to patient
	Emphasize that low-tar, low-nicotine, and other forms of tobacco do not eliminate risk
	Acute risks
	Shortness of breath
	Exacerbation of asthma
	Harm in pregnancy
	Impotence
	Infertility
	Increased serum carbon monoxide
	Long-term risks
	Heart attack
	Stroke
	Cancer: lung, larynx, oral cavity, pharynx, esophagus, pancreas, bladder, cervix
	Chronic obstructive pulmonary disease
	Long-term disability
	Environmental risks
	Increased risk of lung cancer and heart disease in spouse
	Higher rate of smoking among children of tobacco users
	Increased risk of low birth weight, sudden infant death syndrome, asthma, middle ear disease, and respiratory infections in children of smokers
Rewards	Ask patient to identify potential rewards and highlight those most relevant to the patient
	Improved health
	Improved taste for food
	Improved sense of smell
	Save money
	Feel better about yourself
	Home, clothing, and breath will smell better
	Can stop worrying about quitting
	Sets a good example for children
	Healthier babies and children
	Not worry about exposing others to smoke
	Feel better physically
	Reduced wrinkling and aging of skin
Roadblocks	Ask patient to identify barriers to quitting and address elements of treatment that can assist
	Typical barriers
	Withdrawal symptoms
	Fear of failure
	Weight gain
	Lack of support
	Depression
	Enjoyment of tobacco
Repetition	Repeat every time an unmotivated patient visits the clinic setting. Tobacco users who have failed in previous quit attempts should be told that most people make repeated quit attempts before they are successful.

(Adapted from Reference 28.)

ing nurse advice (OR 1.5, CI 1.29–1.73). Pooled results of 16 trials in a Cochrane Collaboration review showed that receipt of nursing advice was associated with a cessation rate of 13.3%, compared with the control group rate of 12.1%.[31] A review of 29 studies by Fiore et al[28] offered 2 conclusions: smoking cessation intervention by nonphysicians increases abstinence, compared to control groups, and should be encouraged (OR 1.7, CI 1.3–2.1), and no specific clinician type demonstrated superiority, so smoking cessation should be encouraged by multiple health care providers. The strength of evidence supporting these recommendations is rated A (see Table 7).

In a Cochrane Library review of 15 studies Lancaster and Stead[32] compared individual smoking intervention by

Table 6. Strength of Evidence Categories for Recommendations

Strength of Evidence Classification	Criteria
Strength of evidence = A	Multiple well designed randomized clinical trials, directly relevant to the recommendation, that yield a consistent pattern of findings
Strength of evidence = B	Some evidence from randomized clinical trials support the recommendation, but the scientific support is not optimal. For instance, few randomized trials exist, the trials that exist are somewhat inconsistent, or the trials are not directly relevant to the recommendation.
Strength of evidence = C	Reserved for important clinical situations where the panel achieved consensus on the recommendation in the absence of relevant randomized controlled trials

(From Reference 28.)

a counselor trained in smoking cessation to no intervention and found that counseling by an individual improved the abstinence rate by 4% (OR 1.62, CI 1.35–1.94). The USDHHS Clinical Practice Guideline used a different approach; Fiore et al[28] assessed the effectiveness of individual counseling by pooling 58 studies involving physicians, nurses, and nonphysicians. They reported an overall 6% absolute increase in the abstinence rate (OR 1.7, CI 1.4–2.0). In summary, available meta-analyses and key individual studies establish the efficacy of individual counseling from a physician, nurse, or nonphysician in increasing smoking cessation rates.[28,32]

Available studies also suggest that group counseling is effective in promoting smoking cessation.[28,33] Examples of group formats include the American Lung Association's "Freedom from Smoking" program and the American Cancer Society's "Fresh Start" program. In a Cochrane Library review of 54 trials of various group intervention formats, Stead and Lancaster[33] found a 10% higher abstinence rate in the 6 trials that compared group intervention to no intervention (OR 2.19, CI 1.42–3.37). In a concordant analysis of pooled studies, the USDHHS study reported an overall 3% absolute rise in the abstinence rate after group counseling (OR 1.3, CI 1.1–1.6).[28] The difference in cessation rates (10% vs 3%) between the 2 reviews may reflect the fact that the USDHHS review did not directly compare group intervention to no intervention, but rather pooled 58 studies and used a variety of comparators.[28] Overall, the evidence supporting the efficacy of group counseling satisfies level A (see Table 7).

Telephone counseling is simple and permits reaching a large number of people at critical cessation moments. Telephone counseling can be provided in lieu of or as an adjunct to face-to-face intervention and can be provided proactively or reactively (eg, telephone help lines). Meta-analyses by Stead et al[34] and Fiore et al[28] found a similar magnitude of effect. Telephone counseling, compared to minimal or no intervention, conferred an approximately 2% absolute rise in the cessation rate (OR 1.56, CI 1.38–1.77 and OR 1.2, CI 1.1–1.4, respectively).

Telephone help lines have been harder to assess. However, in a comparison of smokers who received mailed self-help material to those who received self-help material and notification of a help line, Ossip-Klein et al[35] reported a 2.6% absolute increase in abstinence among those who knew about the help line. Evidence that telephone contact is effective in supporting smoking cessation is rated A (see Table 7).

Self-help information is marginally beneficial for increasing smoking cessation. Examples of self-help materials include booklets, leaflets, brochures, videotapes, compact discs, help lines, and various computer and Internet interventions. For example, Lancaster and Stead[36] confirmed benefit in a review of 12 studies that compared self-help cessation materials to no intervention; self-help materials slightly improved cessation rates (OR 1.24, CI 1.07–1.45). Enhanced or tailored self-help material was associated with better cessation rate than standard self-help material (OR 1.36, 1.13–1.64), but the addition of self-help material to counseling did not increase cessation rate, nor did the use of multiple self-help interventions, such as multiple mailings.[36] Fiore et al[28] reported only a minimally better cessation rate with self-help materials than with no intervention (OR 1.2 1.02–1.3). Overall, though the effectiveness was nominal, self-help also achieved evidence level A (see Table 7).[28,36] Other reasons that self-help, despite its small impact, should be included in smoking programs are increased population awareness, low expense, and the opportunity to customize the message.

Another lesson from available meta-analyses of behavioral interventions is that adding formats confers incremental effectiveness.[28] As shown in Figure 4, combining up to 3–4 formats (eg, self-help with individual counseling, or individual counseling and telephone counseling) may increase the absolute cessation rate by 12%.[28]

Also, increasing the intensity of interventions enhances smoking cessation rates (Figures 5–7). Fiore et al[28] found a strongly dose-related increase in cessation rate as the number of separate interventions increased. Factors increasing effectiveness include the duration of each individual session, the total time spent in all sessions, and the number of sessions. With minimal (< 3 min) counseling, the cessation rate was 13.4%; with low-intensity counseling (3–10 min), the rate was 16.0%; with high-intensity counseling (> 10 min), the rate was 22.1%.[28] Total contact time ranged from zero to > 300 min, with no en-

Table 7. Summary of Behavioral and Pharmacologic Smoking Cessation Interventions

Intervention	Description	Number of Studies*	Odds Ratio (95% Confidence Interval)	Effect Size (%)†	Conclusion
Brief physician contact	*Advice differs widely*	*16‡*	*1.69 (1.45–1.98)*	*2*	Physician advice is effective and should be routinely offered.
	Brief = 3–5 min	7	1.3 (1.1–1.6)	2.3	Evidence = A
Nursing intervention	*Nursing intervention only*	*16*	*1.5 (1.29–1.73)*	*1*	Marginal but measurable benefit from intervention.
Nonphysician	Any health care worker other than physician	29	1.7 (1.3–21)	5.6	Improvement increased with pooled USDHHS data. Evidence = A
Group counseling	*Two or more meetings scheduled*	*6*	*2.19 (1.42–3.37)*	*10*	Better margin of improvement with the Cochrane Collaboration data because of looking only at group versus no intervention. If group versus self-help were included, effect size would diminish. Group interventions are effective. Evidence = A
	Groups pooled with other interventions	58	1.3 (1.1–1.6)	3.1	
Individual counseling	*Health care specialist trained in smoking cessation*	*14*	*1.62 (1.35–1.94)*	*4*	Health care specialist counseling improves cessation and should be provided. Evidence = A
	Any health care specialist	58	1.7 (1.4–2.0)	6	
Telephone counseling	*Proactive calls for each analysis*	*13*	*1.56 (1.38–1.77)*	*2.4*	Proactive telephone calls are successful for improving abstinence. Evidence = A
		58	1.2 (1.1–1.4)	2.3	
Self-help	*Multiple formats for each analysis*	*12*	*1.24 (1.07–1.45)*	*1*	Marginal effectiveness but an important adjunct to smoking cessation
		58	1.2 (1.02–1.3)	1.5	Evidence = A, but improvement marginal
Nicotine gum (polacrilex)	*2-mg and 4-mg doses for each analysis*	*51*	*1.66 (1.52–1.81)*	*8*	Evidence clear that nicotine gum improves success of smoking cessation. Increase dose to 4 mg for highly dependent smokers (odds ratio 2.18, 95% confidence interval 1.48–3.7) Evidence = A
		13	1.5 (1.3–1.8)	6.6	
Nicotine patch	*Combined review, including: doses of 7 mg, 14 mg, and 21 mg. 16-h and 24-h patches*	*34*	*1.74 (1.57–1.93)*	*6*	Evidence supports nicotine patch for improved smoking abstinence. No difference found between 16-h and 24-h patch. Evidence = A
		27	1.9 (1.7–2.2)	7.7	
Nicotine nasal spray	*Spray provides 0.5 mg per spray. Dose is 1 spray to each nostril (ie, total dose 1 mg)*	*4*	*2.27 (1.61–3.2)*	*12*	Improvement in smoking cessation rate. Limited number of studies compared to other NRT interventions. Evidence = A
		3	2.7 (1.8–4.1)	16.6	
Nicotine inhaler	*Each inhaler cartridge contains 10 mg of nicotine*	*4*	*2.09 (1.49–3.04)*	*8*	Evidence supports inhaler use. Limited number of studies compared to other NRT interventions. Evidence = A
		4	2.5 (1.7–3.6)	12.3	
Bupropion (300 mg/d sustained-release)	*150 mg/d for 3 d, then 300 mg/d*	*7*	*2.54 (1.9–3.41)*	*10*	Evidence is strong that bupropion increases cessation rate. May also prove effective with NRT. Evidence = A
		2	2.1 (1.54–3.0)	13.2	

*When possible, studies compare intervention to no or minimal intervention.
†Absolute increase in smoking cessation rate (ie, intervention vs control)
‡Italicized data is from the Cochrane Collaboration reviews.
USDHHS = United States Department of Health and Human Services
NRT = nicotine replacement therapy
(Adapted from References 28, 29, 31–34, 36, 38, 39.)

hanced effectiveness beyond 90 min of counseling.[28] A review of the impact of the number of counseling sessions found that the greater the number of sessions, the greater the chance for cessation. Programs with 0–1 sessions had a quit rate of 12.4%, whereas those with ≥ 8 sessions had a quit rate of 24.7% (see Fig. 7).[28] In the Cochrane Collaboration review, the intensity of interventions was defined differently. For example, nursing interventions were

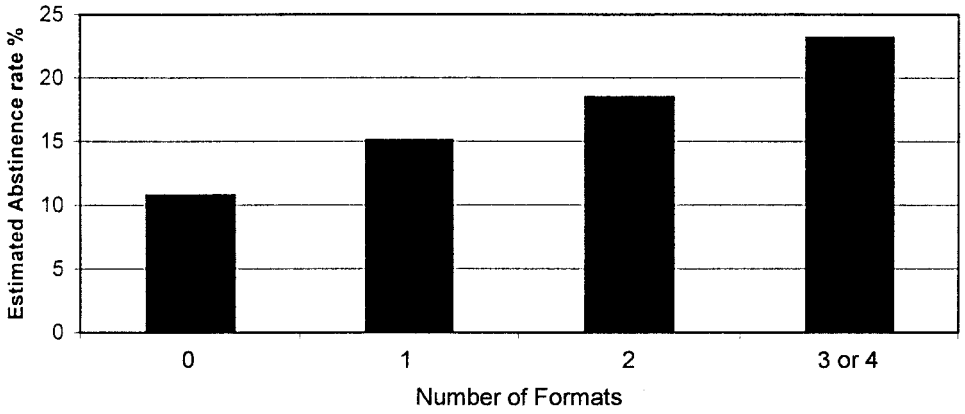

Fig. 4. Meta-analysis of estimated cigarette-smoking abstinence rates relative to number of smoking cessation formats used. The formats included self-help, proactive counseling, group counseling, and individual counseling (n = 54). (Adapted from Reference 28.)

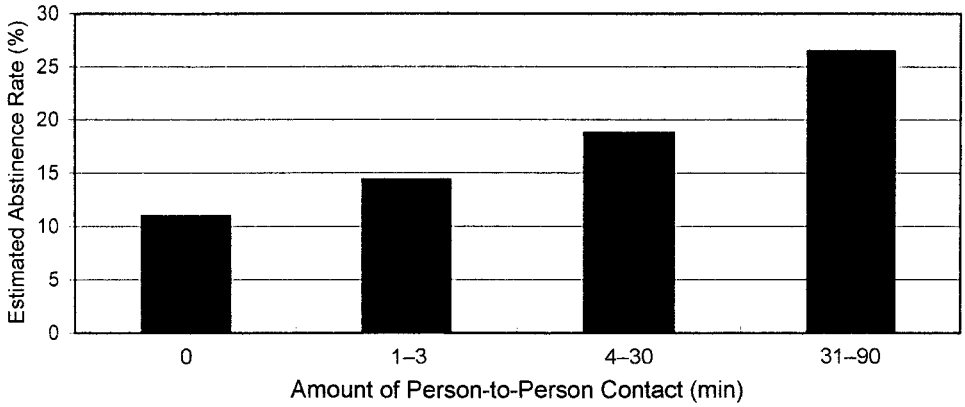

Fig. 5. Estimated cigarette-smoking abstinence rates relative to the duration of the individual counseling session (n = 43 studies). (Adapted from Reference 28.)

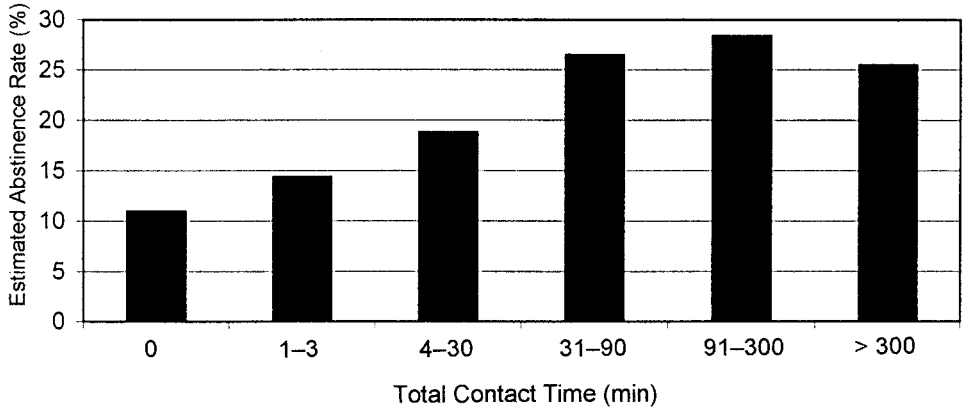

Fig. 6. Meta-analysis of estimated cigarette-smoking abstinence rates relative to the total amount of contact time (n = 35 studies). (Adapted from Reference 28.)

considered low intensity if $\leq$ 10 min and high intensity if $>$ 10 min with a follow-up appointment,[30] whereas physician interventions were defined as minimal intensity if $\leq$ 20 min with one follow-up visit and intensive if $>$ 20 min with one follow-up.[28] Based on these definitions, increased intensity of nursing intervention did not significantly increase cessation rates (low intensity OR 1.67, CI 1.14–2.45, high intensity OR 1.47, CI 1.26–1.72),[30] whereas

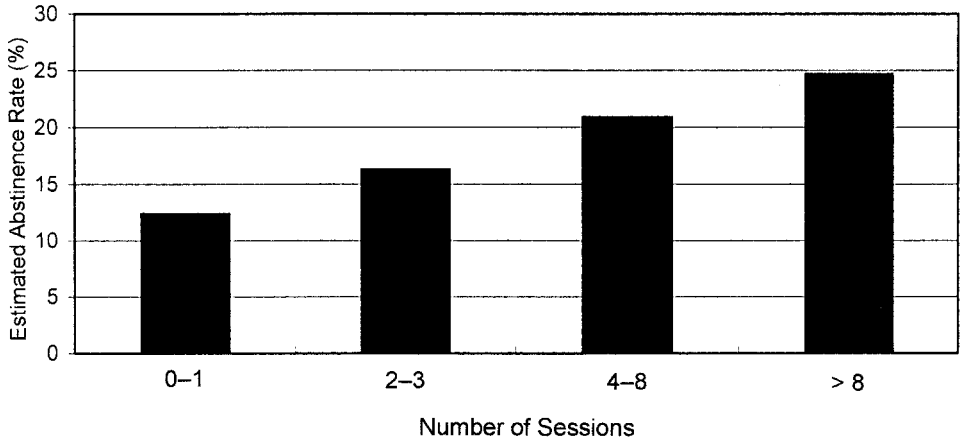

Fig. 7. Estimated cigarette-smoking abstinence rates relative to the number of counseling sessions (n = 43 studies). (Adapted from Reference 28.)

increasing intensity of physician intervention did modestly increase the smoking cessation rate (OR 1.67, CI 1.45–1.98).[28] In contrast, in an analysis of 3 trials involving individual counseling with various intensities, Lancaster and Stead[32] did not find evidence to suggest that increased intensity of individual counseling increased cessation rate (OR 0.98, CI 0.61–1.56).

A more recent study by Simon et al[37] randomized 228 patients to either low- or high-intensity intervention, with all receiving nicotine replacement therapy (NRT) via patch. The 1-year abstinence rate was significantly higher in the higher-intensity counseling group (29% vs 20%, OR 1.6, CI 0.96–2.5).[37]

Overall, evidence suggesting that increased intensity of counseling enhances abstinence achieves evidence level A (see Table 7). Limitations of counseling include the cost of counseling sessions, limited availability to large populations, and the time-intensiveness of the interventions for both professionals and patients.

Pharmacologic Interventions

The first-line pharmacologic interventions are NRT and bupropion (an antidepressant). The 2 second-line drugs are clonidine (an antihypertensive) and nortriptyline (a tricyclic antidepressant). Available guidelines suggest that NRT, antidepressants, and certain antihypertensives effectively increase smoking cessation rates.[28,38–40] Table 8 summarizes the 5 first-line Food and Drug Administration (FDA) approved smoking cessation medications.

The mechanisms of drugs to aid smoking cessation differ. NRT offsets the craving for nicotine. Although the precise mechanism is unknown, bupropion is thought to blunt the impact of nicotine withdrawal in smoking cessation by diminishing the uptake of dopamine and norepinephrine, thus decreasing cravings.[41] Though these medications assist in smoking cessation, and some are available over-the-counter, caution is advised not to overestimate their efficacy and undermine the power of will in quitting. Prochazka[42] cautioned against considering cessation medicines a "magic bullet," but instead counsels against unrealistic expectations and advises a detailed understanding of the drugs being used.

Nicotine Replacement Therapy

Nicotine replacement therapy is supplied in several forms: patch, gum (polacrilex), nasal spray, inhaler, and lozenges. Favorable features of NRT are that it is readily available, easy to use, relatively inexpensive, and effective.

Nicotine patch is applied transdermally and nicotine is absorbed through the skin. Nicotine patches are available over-the-counter and come in doses of 7 mg, 14 mg, or 21 mg (generic or Nicoderm CQ patch) or 5 mg, 10 mg, or 15 mg (Nicotrol) (see Table 8).

A Cochrane Library review by Silagy et al[38] of 96 trials comparing all forms of NRT to controls found that abstinence rates were 7% better with NRT (OR 1.74, CI 1.64–1.86). Nicotine patch was associated with a 6% better abstinence rate than controls (OR 1.74, CI 1.57–1.93). Similar findings were reported by the USDHHS Clinical Practice Guideline,[28] which showed an increased quit rate of 7.7%, based on review of 26 studies (OR 1.9, CI 1.7–2.2).

Regarding the duration of patch use, the 16-hour and 24-hour patches appear to confer similar benefit.[38] Still, the long-term effectiveness of NRT has decreased since NRT became available over-the-counter in 1996, possibly because advice from health care providers diminished as the need for a prescription to receive the patch vanished.[43]

Table 8. First-Line Medications for Smoking Cessation

Drug	Dose	Instruction	Daily Dose and Duration	Side Effects	Cost*
Nicotine gum (polacrilex)	Nicorette 2 mg Nicorette 4 mg Generic 2 mg and 4 mg Over-the-counter: 108 per box	Mucosal absorption. Chew until spicy or minty flavor begins, then "park" between cheek and gum. When taste disappears, repeat chew-and-park process for approximately 30 min. Maximum levels of nicotine achieved within 20–30 min.	No more than 24 pieces/d. With patients who smoke ≥ 25 cigarettes/d, use 4-mg type, 1 piece/h. For patients who smoke ≤ 25 cigarettes/d, use 2-mg type, 1 piece/h or as needed. Duration: 12 wk	Jaw fatigue, nausea, hiccups	10 pieces/d = $4.00–$4.60/d Total cost: $360–$414
Nicotine patch	Nicoderm CQ or generic: 21 mg, 14 mg, and 7 mg 24-h patches 14 patches per package Nicotrol: 15 mg, 10 mg, and 5 mg 16-h patch 14 patches per package Dose depends on degree of nicotine dependence	Rotate site daily. Hair-free sites offer best absorption. May remove at night for insomnia Continuous delivery. Requires 2–3 d to reach peak.	21 mg or 15 mg during weeks 1–6. 14 mg or 10 mg during weeks 7–8. 7 mg during weeks 9–10. Patients who smoke ≤ 10 cigarettes/d should start on intermediate dose.	Skin reactions among up to 50% of users. Not recommended for patients who have had a recent (within past 2 wk) myocardial infarction.	1 patch/d = $3.11–$3.57/d Total cost: $218–$321
Nicotine nasal spray	Nicotrol NS: 0.5 mg/spray Dose is 1 spray to each nostril 100 doses per bottle	Tilt head slightly back and spray once into each nostril. Do not inhale, whiff, or swallow. Medication absorbs through the nasal mucosa and throat. Peak effect within 5–10 min. Effect decreases within 30 min.	Initially use 1–2 doses/h Minimum: 8 doses/d Maximum: 40 doses/d Duration: 3 mo	94% of users reported nasal irritation in first 2 d. 81% had continued nasal irritation at 3 wk. Not recommended for patients who have had a recent (within past 2 wk) myocardial infarction.	12 doses/d = $5.28/d Total cost: $475
Nicotine inhaler	Nicotrol Inhaler: Each cartridge contains 10 mg of nicotine Delivers 4 mg/puff	Hold inhaler between fingers and "puff". Requires rapid puffing: 3–4 puffs/min. 80 inhalations over 20 min. Absorbs in mouth and throat.	6–16 cartridges/d Duration: 3 mo	Mouth and throat irritation, cough, rhinitis. Use with caution in patients who have reactive airway disease.	10 cartridges/d = $10.80/d Total cost: $972
Bupropion sustained release	Zyban: 150 mg/d for 3 d, then 300 mg/d (150 mg 2 times/d)	Start 1–2 wk before quit date	150 mg for 3 d, then 300 mg/d Duration: 3 mo	Insomnia and dry cough. Contraindicated in those with seizure disorders.	2 pills/d = $4.40/d Total cost: $389

*Cost data are from average price from 3–4 national pharmacies (Adapted from Reference 28 and manufacturers' information.)

Overall, the evidence supporting nicotine patches warrants a rating of A (see Table 7).

Nicotine gum (polacrilex) has also been available over-the-counter since 1996 and is also effective in promoting smoking cessation.[28,38] Nicotine gum is available in 2-mg and 4-mg doses (Nicorette, Nicorette Mint, or generic). Nicotine gum allows absorption of nicotine through the buccal mucosa.

Regarding efficacy, a Cochrane review of 51 studies by Silagy et al[38] found that nicotine gum increased the effectiveness of cessation attempts by 8%, compared to controls (OR 1.66, CI 1.52–1.81). Similarly, in a meta-analysis of 13 studies Fiore et al[28] estimated a 6.6% better cessation rate with nicotine gum (OR 1.5, CI 1.3–1.8). Patients who are highly nicotine dependent (who smoke > 25 cigarettes/d) or those who have failed the 2-mg dose should use the 4-mg dose, but should use no more than 24 pieces per day.[28,38] In a study of 3,094 patients receiving nicotine gum in both treatment arms of the Lung Health Study, Murray et al[44] found no adverse cardiovascular effects from nicotine gum, even among those who smoked and continued to chew gum. Still, approximately 25% of nicotine gum users experienced one or more adverse effects, including mouth irritation, headache, and indigestion.[44] The evidence supporting the efficacy of nicotine gum to increase smoking abstinence is substantial and is rated A (see Table 7).

Nicotine nasal spray (Nicotrol NS) provides the most rapid nicotine administration of all the NRTs, with peak effects within 5–10 min.[28,38] Nicotine nasal spray must be administered correctly for maximum effectiveness. One squirt (0.5 mg) into each nostril delivers a total dose of 1 mg. The dose should not be inhaled or sniffed and should be delivered with the head slightly tilted.[28] Adverse effects are common, with 94% of users reporting some nasal irritation, which persists in 81% of users for up to 3 weeks after initiation.[28] Attractive features of nicotine spray are that it is rapidly absorbed and can reduce nicotine craving,

offers a substitute for the cues of smoking, and can be administered as needed, up to 40 doses per day. Disadvantages are the adverse effects, which include the social stigma of squirting a spray into one's nostrils, and that the drug is contraindicated in patients with reactive airway disease.[28] The Cochrane Library analysis by Silagy et al[38] of 4 studies reported a 12% absolute increase in the rate of smoking cessation (OR 2.28, 1.61–3.20). The USDHSS review of 3 studies found a higher quit rate: 16.6%.[28] The difference in those cessation rate estimates may be due to exclusion in one meta-analysis of the trial by Hjalmarson et al,[45] which found a 12-month cessation rate of 12%. Evidence supporting the efficacy of nicotine nasal spray is rated A.

Nicotine inhaler (Nicotrol inhaler) is the fifth FDA-approved NRT. The inhaler cartridge contains 10 mg of nicotine that can supply 4 mg of nicotine (2 mg are systemically available) over 80 inhalations (suggested to take place over 20 min; see Table 8). Advantages of the nicotine inhaler include that it mimics smoking (albeit with rapid puffing), it delivers nicotine rapidly, and it has minimal side effects. Disadvantages are that the inhaler is the most expensive form of NRT (average wholesale price $1.08 per cartridge) and requires more intense puffing than smoking. The Cochrane Library meta-analysis of 4 studies demonstrated an absolute 8% increase in the cessation rate over placebo inhalers (OR 2.09, CI 1.49–3.04).[38] The meta-analysis by Fiore et al[28] reported a 12.3% increase in cessation rates with nicotine inhaler (OR 2.5, CI 1.7–3.6). Adverse effects reported by Hjalmarson et al[46] were increased cough (28%) and irritation of the mouth or throat (15%). With caution, because of the small number of studies available, the evidence regarding nicotine inhalers also warrants a rating of A.

Nicotine lozenge/tablet (Commit) is not an FDA-approved first-line medication but does show promise. Nicotine lozenges (polacrilex) come in 2-mg and 4-mg doses. They are easy to use, have minimal adverse effects (heartburn, hiccups, and nausea), and provide 25% more nicotine than similar doses of nicotine gum.[47] In a large, randomized trial ($n = 1{,}818$ smokers) concurrently conducted in the United States and England, Shiffman et al[47] found an increased abstinence rate in high-dependence smokers (receiving the 4-mg dose) of 8.7% over placebo and in low-dependence smokers (receiving the 2-mg dose) of 8.2% over placebo. Though there are few studies reviewing nicotine lozenges or tablets, these medications are promising.

Despite the substantial body of supportive evidence, there is continuing uncertainty about some aspects of NRT. For example, does the use of multiple forms of NRT enhance effectiveness? Is there a dose-response beyond the usual recommended doses? Do nicotine lozenges or tablets have efficacy?

Regarding combined NRT use, the Cochrane Collaboration analysis[38] pooled 5 studies of combined nicotine replacement therapies and observed a small benefit with combination NRT (OR 1.55, CI 1.17–2.05). A recent study by Hand et al[48] found no benefit from combining counseling, nicotine patch, and nicotine inhaler, compared to counseling alone (15% and 14% 1-year cessation rates, respectively). Conversely, Blondal et al[49] found a significantly higher 1-year cessation rate with nicotine patch and nasal spray (28% cessation rate) compared to nicotine patch alone (11% cessation rate). Overall, the evidence is too sparse at present to allow specific recommendations on NRT combinations.

Regarding the dose responsiveness of NRT, the Cochrane Collaboration analysis pooled 6 studies that used higher doses of nicotine patch, but found only marginal evidence of additive benefit (OR 1.2, CI 1.03–1.42).[38]

In summary, all forms of NRT recommended by the FDA as first-line drugs are effective for smoking cessation. At this time there is insufficient evidence to recommend one form of NRT over another. Patients with lesser dependence on nicotine (ie, $\leq$ 10 cigarettes/d) may consider lower-dose or alternative interventions.[28] Given the lack of clear-cut evidence supporting one NRT form over another, patient and physician preference should play a large role in choosing a specific NRT drug.

Bupropion

The antidepressant bupropion is the first non-NRT intervention recommended by the FDA as a first-line drug for smoking cessation.[28,39,50] The Cochrane Collaboration analysis[39] of 7 trials found 10% better cessation among those who received bupropion than among control subjects (OR 2.54, CI 1.9–3.41). The USDHHS Clinical Practice Guideline also found better cessation rate with bupropion: 13.2% higher than controls (OR 2.1, CI 1.5–3.0).[28] With regard to combined bupropion and NRT Jorenby et al[51] found significantly better cessation rates with the combination of NRT and bupropion than with nicotine patch alone (OR 2.07, CI 1.22–3.53 and 2.65 CI 1.58–4.45, respectively).

Another observed benefit of bupropion is its ability to blunt the weight gain that may accompany smoking cessation.[50] For example, Jorenby et al[51] reported that combined bupropion and nicotine patch recipients experienced a lower mean weight gain than did nonrecipients (ie, 2.1 kg vs 1.1 kg at 7 wk).

Overall, the evidence regarding bupropion for smoking cessation merits a rating of A.

Second-Line Smoking Cessation Drugs

Clonidine is an antihypertensive medication that is provided orally or transdermally (Catapres). In a meta-anal-

ysis of 6 studies, Gourlay et al[40] found that clonidine increased smoking cessation rate by 11% (OR 1.89, CI 1.30–2.74). Similarly, Fiore et al[28] analyzed 5 studies and reported a similar enhanced abstinence rate: 11.7% (OR 1.4–3.2). Unfortunately, clonidine can produce important adverse events, such as dry mouth, dizziness, sedation, and postural hypotension, which may discourage its use.[28,40]

Overall, although the evidence supporting clonidine for smoking cessation achieves an A rating, the adverse effect profile relegates it to second-line status. Clonidine has not been approved by the FDA for smoking cessation, but has found use as a salvage regimen with individuals who have failed NRT or bupropion.

Finally, nortriptyline is a tricyclic antidepressant that has been used to assist smoking cessation.[28,39] Results of 3 available studies demonstrate a 12% absolute improvement in cessation rates over controls (OR 1.73, 1.73–4.44).[52] Also, the USDHHS Clinical Practice Guideline[28] review of 2 studies noted 18.4% improvement over control cessation rates (OR 3.2, 1.8–2.7).

Overall, the limited number of trials and the adverse effects of nortriptyline make it a second-line intervention. Evidence supporting the use of nortriptyline in smoking cessation is rated B.

Alternative Smoking Cessation Interventions

Alternative behavioral interventions for smoking cessation include hypnotherapy, aversive therapies, acupuncture, and exercise. Other medications that have been tried for smoking cessation include lobeline, anxiolytics, mecamylamine, opioid agonists, and silver acetate. There is less supportive research for these interventions than for the first-line interventions.

Regarding hypnotherapy, a review of 9 trials by Abbot et al[52] did not find efficacy for smoking cessation. Challenges to validating hypnotherapy include the small size of most of the trials and the confounding issue of separating the impact of time spent with the therapist from the hypnosis itself.[52] The USDHHS Clinical Practice Guideline do not recommend hypnosis.[28]

Aversive therapies were mainly used before current interventions became available; they include rapid smoking, smoke holding, rapid puffing, excessive smoking, and electric shock. The theory underlying aversive therapy is that linking a negative sensation to smoking will encourage cessation. Hajek and Stead[53] reviewed aversive smoking therapies and found rapid smoking to be the most effective. However, they concluded that there was insufficient evidence to support the effectiveness of aversive therapy. In contrast, the USDHHS Clinical Practice Guideline reported that rapid smoking improved abstinence rates by 8% over controls (OR 2.0, CI 1.1–3.5).[28] One important limitation is that, if attempted, rapid smoking should only be done in the presence of a health care professional. Our view is that currently the evidence is insufficient to support aversive therapy.

The 4 available meta-analyses of alternative interventions fail to support efficacy in aiding smoking cessation. For example, 2 available meta-analyses of acupuncture failed to show efficacy (OR 1.08, CI 0.77–1.52 and OR 1.1, CI 0.7–1.6).[28,54] A meta-analysis and a recent study of exercise intervention with 299 smokers showed no increased rate of smoking cessation.[55,56] Similarly, neither a review of available studies nor an unpublished pharmaceutical study of lobeline, a partial nicotine agonist, showed efficacy in aiding smoking cessation.[57]

The Cochrane Collaboration review[58] of trials of anxiolytics (3 trials of buspirone, 1 trial of diazepam, 1 trial of meprobamate, and 1 trial of β blockers) concluded that none of the trials supported efficacy for improving smoking cessation. Adverse effects and the availability of other interventions discourage the use of any of these drugs.

In a review of 2 studies of the nicotine antagonist mecamylamine, Lancaster et al[59] found that mecamylamine combined with nicotine patch produced better cessation rates than nicotine patch alone. However, lack of any long-term studies precludes current endorsement of mecamylamine. Similarly, the USDHHS Clinical Practice Guideline report did not advocate mecamylamine.[28]

Silver acetate is a pharmaceutical aversive therapy that leaves an unpleasant taste in the mouth when combined with cigarettes.[60] The Cochrane Collaboration reported 2 studies comparing silver acetate to placebo and found no measurable improvement in cessation rates (OR 1.05, CI 0.63–1.73). The USDHSS Clinical Practice Guidelines review also found no benefit from silver acetate.[28] In light of current available information, we do not recommend silver acetate.

Combined Smoking Cessation Interventions

Many studies establish the superiority of combined interventions over individual smoking cessation strategies. For example, in the largest available trial with chronic obstructive pulmonary disease patients, the Lung Health Study (n = 5,887 subjects) found that the group that received nicotine gum and counseling (physician counseling and group counseling) had a better smoking cessation rate (22% at 11 years) than the usual-care group (6%).[14]

Regarding the additive effect of bupropion, Tashkin et al[61] compared individual counseling plus proactive telephone calls plus bupropion to individual counseling plus proactive telephone calls plus placebo, and found that the bupropion group had a higher cessation rate than the control group (16% versus 9% at 26 wk, OR 1.74, CI 1.01–3.0). Overall, given the effectiveness of individual strategies and the weight of evidence supporting combined

approaches, current practice often offers both counseling and drug interventions to assist smoking cessation.

Cost

As with all medications, cost is an important issue for smoking cessation therapy. At the same time, the cost of therapy must be offset against the cost associated with buying cigarettes and the personal and societal costs of sequelae of smoking. Currently, the average cost of a pack of cigarettes is $3.15.[62] Thus, the yearly cost of a 1 pack/day habit is $1,149.75. An estimate of the cost of medical care associated with each pack of cigarettes sold is $3.45.[10] Additionally, the estimated cost of lost productivity due to morbidity and mortality from smoking add $3.73 as the societal cost per pack.[10] Thus, the aggregate cost per pack of cigarettes is $7.18. In this context the daily cost of nicotine patches ($3.57) looks quite favorable. Table 8 shows price estimates for the available medications.

The Respiratory Therapist's Role in Smoking Cessation

As both hospital-based and home-care practitioners, respiratory therapists (RTs) have extraordinary opportunities to encourage smoking cessation. The opportunity is advanced by therapists' favorable attitudes toward smoking cessation. For example, Sockrider et al[63] surveyed 354 RTs therapists as to whether they believed that advising smoking cessation was a necessary aspect of their job. The rating system was 5 = strongly agree and 1 = strongly disagree. The mean aggregate score was 4.3. The mean level of agreement that smoking cessation should be included in training was 3.4 and that cessation advice was as important as other aspects of an RT's job was 3.6.[63]

Despite the opportunity and the favorable attitude, the subject of RTs' effectiveness in conducting smoking cessation interventions has received little attention, with only 2 studies identified in our literature search.

In a randomized trial reported by Stevens et al,[64] in which RTs provided counseling, no difference in cessation rates was observed between the group receiving RT counseling and the usual-care group (14.2% and 13.6%, respectively). One possible explanation for RTs' lack of impact in the study was the time constraints that limit RTs' interventions.[64] For example, only 68% of those randomized to RT intervention were seen by the RT (vs 80% contacted by a professional counselor in another study) and only 71% received telephone contact from the RT (vs 99% in another study with professional counselors).[64] Cohn et al[65] reported an impressive 48% 6-week abstinence rate among participants in an RT-led smoking cessation program.

Overall, we conclude that RTs' access to smokers makes them attractive candidates to administer smoking cessation interventions. Also, the paucity of available literature invites further study of RTs' effectiveness in smoking cessation.

Summary

Because smoking remains common and is associated with substantial morbidity, mortality, and costs, aggressive efforts to eradicate smoking are justified. Of the available methods to effect smoking cessation, level A evidence supports the efficacy of various behavioral and pharmacologic interventions: counseling by various health care providers, nicotine replacement therapy, and bupropion. In addition, combination therapy (eg, counseling plus nicotine replacement therapy, nicotine plus bupropion) seems to confer additional benefit. Still, disappointing longer-term abstinence rates for the strongest available studies (ie, $< 25\%$) establish the need for continued investigation of smoking cessation strategies. Finally, we believe that RTs can and should play key roles in smoking cessation programs and that this subject warrants further study.

REFERENCES

1. US Department of Health and Human Services. Reducing tobacco use: a report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2000 Surgeon General's Report. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
2. Curbing the epidemic: governments and the economics of tobacco control. Developments in practice. The World Bank Group. Washington DC; 1999.
3. Centers for Disease Control and Prevention. Achievements in public health, 1900–1999: tobacco use—United States, 1900–1999. MMWR: Nov 05. 1999;48(43):986–993. Available at http://www.cdc.gov/mmwr (accessed 10/2/03).
4. Center for Disease Control and Prevention. Percentage of adults who were current, former, or never smokers, overall and by sex, race, Hispanic origin, age and education. National Health Interview Surveys, selected years- United States, 1965–2000. Tobacco Information and Prevention Source (TIPS). Apr 11, 2003. Available at http://www.cdc.gov/tobacco/research_data/adults_prev/adstat1.htm (accessed 10/2/03).
5. Center for Disease Control and Prevention. Prevalence of current cigarette smoking among adults and changes in prevalence of current and some day smoking—United States, 1996–2001, MMWR. Apr 11, 2003;52(14):303–307. Available at http://www.cdc.gov/mmwr (accessed 10/2/03).
6. Center for Disease Control and Prevention. Ten great public health achievements in the 20th century. 1900–1999. Office of Communication. Media Relations. Sep 13, 2001. Available at http://www.cdc.gov/od/oc/media/tengpha.htm (accessed 10/2/03).
7. U.S. Department of Health and Human Services. Office of Applied Studies, Substance, Abuse and Mental Health Services Administration (SAMHSA). Tobacco use, income, and educational level. The NHSDA Report. May 3, 2002. Available at http://www.samhsa.gov/oas/2k2/Tob/tob.htm (accessed 10/11/03).

8. Center for Disease Control and Prevention. Women and smoking, a report of the Surgeon General-2001. Pattern of tobacco use among women and girls-fact sheet. Tobacco Information and Prevention Source (TIPS). Mar 27, 2001. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
9. Center for Disease Control and Prevention. Trends in cigarette smoking among high school students—United States 1991–2001. MMWR 2002;51(19):409–412. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5119a1.htm (accessed 10/2/03).
10. Center for Disease Control and Prevention. Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. MMWR. Apr 12, 2002;51(14):300–303. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5114a2.htm (accessed 10/2/03).
11. Centers for Disease Control and Prevention. Smoking attributable mortality, morbidity, and economic costs (SAMMEC): Adult SAMMEC and Maternal and Child Health (MCH) SAMMEC software, 2002c. Available at: http://www.cdc.gov/tobacco/sammec (accessed 10/2/03).
12. US Department of Health and Human Services. The health benefits of smoking cessation: a report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, DHHS Publication No. (CDC) 90–8416.1990. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
13. Buist AS, Connett JE, Miller RD, Kanner RE, Ownes GR, Voelker HT. Chronic Obstructive Pulmonary Disease Early Intervention Trial (Lung Health Study). Baseline characteristics of randomized participants. Chest 1993;103(6):1863–1872.
14. Anthonisen NR, Connett JE, Murray RP. Smoking and lung function of Lung Health Study participants after 11 years. Am J Respir Crit Care Med 2002;166(5); 675–679.
15. US Department of Health and Human Services. Reducing the health consequences of smoking: a report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1964 Surgeon Generals Report: pg 34. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
16. US Department of Health and Human Services. Smoking and health: a report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1979 Surgeon Generals Report. Pg xv. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
17. US Department of Health and Human Services. Health consequences of smoking: nicotine addiction: a report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1988 Surgeon Generals Report. Pg ii. Available at http://www.cdc.gov/tobacco/sgr/index.htm (accessed 10/2/03).
18. Pidoplichko VI, DeBiasi M, Williams JT, Dani JA. Nicotine activates and desensitizes midbrain dopamine neurons. Nature 1997; 390(6658):401–404.
19. Leshner AI, Koob GF. Drugs of abuse and the brain. Proc Assoc Am Physicians 1999;111(2):99–108.
20. Clark PB. Mesolimbic dopamine activation—the key to nicotine reinforcement? Ciba Found Symp 1990;152:153–162: discussion 162–168.
21. Dani JA, De Biasi M. Cellular mechanisms of nicotine addiction. Pharmocol Biochem Behav 2001;70(4) 439–446.
22. Dani JA, Ji D, Zhou FM. Synaptic plasticity and nicotine addiction. Neuron 2001;31(3):349–352.
23. Corrigall WA, Coen KM. Selective dopamine antagonist reduce nicotine self-administration. Psychopharmacology 1991;104:171–176.
24. Corrigal WA, Coen KM & Adamson KL. Self–administered nicotine activates the mesolimbic dopamine system through the ventral tegmental area. Brain Res 1994;653:278–284.
25. Hughes JR, Higgins ST, Bickel WK. Nicotine withdrawal versus other drug withdrawal syndromes: similarities and dissimilarities. Addiction. 1994;(Nov):89(11):1461–1470.
26. Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom test for nicotine dependence: a revision of the Fagerstrom tolerance questionnaire. Br J Addict 1991;86(9):1119–1127.
27. Center for Disease Control and Prevention. Cigarette smoking among adults–United States, 2000. MMWR Highlights. Jul 26, 2002;51;29: 642–645. Available at http://www.cdc.gov/mmwr/ (accessed 10/7/03).
28. Fiore, MC, Bailey WC, Cohen SJ, et al. Treating tobacco use and dependence. Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service. Jun 2000. Available at http://www.surgeongeneral.gov/tobacco/tobaqrg.htm (accessed 10/11/03).
29. Silagy C, Stead LF. Physician advice for smoking cessation. Cochrane Database Syst Rev 2001;(2):CD000165.
30. Denny CH, Serdula MK, Holtzman D, Nelson DE. Physician advice about smoking and drinking: are U.S. adults being informed? Am J Prev Med. 2003;24(1):71–74.
31. Rice VH, Stead LF. Nursing interventions for smoking cessation. Cochrane Database Syst Rev 2001;(3):CD001188.
32. Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. Cochrane Database Syst Rev 2002;(3):CD001292.
33. Stead LF, Lancaster T. Group behaviour therapy programmes for smoking cessation. Cochrane Database Syst Rev 2002;(3):CD001007.
34. Stead LF, Lancaster T, Perara R. Telephone counseling for smoking cessation. Cochrane Database Syst Rev 2003;(1):CD002850.
35. Ossip-Klein DJ, Giovino GA, Megahed N, Black PM, Emont SL, Stiggins J, et al. Effects of a smoker's hotline: results of a 10-county self-help trial. J Consult Clin Psychol 1991;59(2):325–332.
36. Lancaster T, Stead LF. Self-help interventions for smoking cessation. Cochrane Database Syst Rev 2002;(3):CD001118.
37. Simon JA, Carmody TP, Hudes ES, Snyder E, Murray J. Intensive smoking cessation counseling versus minimal counseling among hospitalized smokers treated with transdermal nicotine replacement: a randomized trial. Am J Med 2003;114(7):555–562.
38. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation. Cochrane Database Syst Rev 2002;(4):CD000146.
39. Hughes JR, Stead LF, Lancaster T. Antidepressants for smoking cessation. Cochrane Database Syst Rev 2003;(2):CD000031.
40. Gourlay SG, Stead LF, Benowitz NL. Clonidine for smoking cessation. Cochrane Database Syst Rev 2000;(2):CD000058.
41. Johnston AJ, Ascher J, Leadbetter R, Schmith VD, Patel DK, Durcan M, Bentley B. Pharmacokinetic optimization of sustained-release bupropion for smoking cessation. Drugs 2002;62 Suppl 2:11–24.
42. Prochazka AV. New developments in smoking cessation. Chest 2000; 117(4 Suppl):169S–175S.
43. Pierce JP, Gilpin EA. Impact of over-the-counter sales on effectiveness of pharmaceutical aids for smoking cessation. JAMA 2002; 288(10):1260–1264.
44. Murray RP, Bailey WC, Daniels K, Bjornson WM, Kurnow K, Connett JE, et al. Safety of nicotine polacrilex gum used by 3,094 participants in the Lung Health Study. Lung Health Study Research Group. Chest 1996;109(2):438–445.

45. Hjalmarson A, Franzon M, Westin A, Wiklund O. Effect of nicotine nasal spray on smoking cessation: a randomized, placebo-controlled, double-blind study. Arch Int Med 1994;154(22):2567–2572.
46. Hjalmarson A, Nilsson F, Sjostrom L, Wiklund O. The nicotine inhaler in smoking cessation. Arch Int Med 1997;157(15).1721–1728.
47. Shiffman S, Dresler CM, Hajek P, Gilburt SJ, Targett DA, Stahs KR. Efficacy of a nicotine lozenge for smoking cessation. Arch Int Med 2002;162(11):1267–1276.
48. Hand S, Edwards S, Campbell IA, Cannings R. Controlled trial of three weeks nicotine replacement treatment in hospital patients also given advice and support. Thorax; 2002;57(8):715–718.
49. Blondal T, Gudmundsson LJ, Olafsdottir I, Gustavsson G, Westin A. Nicotine nasal spray with nicotine patch for smoking cessation: randomised trial with six year follow up. BMJ 1999;318(7179):285–288. *Erratum in BMJ 1999;318(7186):764*
50. Jorenby D. Clinical efficacy of bupropion in management of smoking cessation. Drugs 2002;62 Suppl 2:25–35.
51. Jorenby DE, Leischow SJ, Nides MA, Rennard SI, Johnston JA, Hughes AR, et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. N Engl J Med 1999;340(9):685–691.
52. Abbot NC, Stead LF, White AR, Barnes J, Ernst E. Hypnotherapy forsmoking cessation. Cochrane Database Syst Rev 2000;(2): CD001008.
53. Hajek P, Stead LF. Aversive smoking for smoking cessation. Cochrane Database Syst Rev 2000;(2):CD000546.
54. White AR, Rampes H, Ernst E. Acupuncture for smoking cessation. Cochrane Database Syst Rev 2002;(2):CD000009.
55. Ussher M, West R, McEwen A, Taylor A, Steptoe A. Efficacy of exercise counseling as an aid for smoking cessation: a randomized controlled trial. Addiction 2003;98(4):523–532.
56. Ussher MH, West R, Taylor AH, McEwen A. Exercise interventions for smoking cessation. Cochrane Database Syst Rev 2000;(3):CD002295.
57. Stead LF, Hughes JR. Lobeline for smoking cessation. Cochrane Database Syst Rev 2000;(2):CD000124.
58. Hughes JR, Stead LF, Lancaster T. Anxiolytics for smoking cessation. Cochrane Database Syst Rev 2000;(4):CD002849.
59. Lancaster T, Stead LF. Mecamylamine (a nicotine antagonist) for smoking cessation. Cochrane Database Syst Rev 2000;(2):CD001009.
60. Lancaster T, Stead LF. Silver acetate for smoking cessation. Cochrane Database Syst Rev 2000;(2):CD000191.
61. Tashkin D, Kanner R, Bailey W, Buist S, Anderson P, Nides M, et al. Smoking cessation in patients with chronic obstructive pulmonary disease: a double-blind, placebo-controlled, randomized trial. Lancet 2001;357(9268):1571–1575.
62. Center for Disease Control and Prevention. Office of Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion. State Tobacco Activities Tracking and Evaluation (STATE). 2000. Available at http://www2.cdc.gov/nccdphp/osh/state (accessed 10/11/03).
63. Sockrider MM, Maguire GP, Haponik E, Davis A, Boehlecke B. Attitudes of respiratory care practitioners and students regarding pulmonary prevention. Chest 1998;114(4):1193–1198.
64. Stevens VJ, Glasgow RE, Hollis JF, Mount K. Implementation and effectiveness of a brief smoking-cessation intervention for hospital patients. Med Care 2000;38(5):451–459.
65. Cohn RC, Dodson D, French A, Ervin B, Ciarlariello S, Wilson TN. A pilot smoking cessation program run by pediatric respiratory care practitioners for parents. Clin Pediatr (Phila) 2000;39(2):121–124.

Discussion

MacIntyre: For the last 6 months in North Carolina I've been working with the Lung Association to try to raise the cigarette excise tax. As you might imagine in a tobacco state like North Carolina, that is a daunting challenge. Do you have any data that indicate that as you raise cigarette taxes, cigarette use goes down? That might be helpful to me. How sensitive is cigarette smoking to price pressure?

Marlow: What is the relationship of price change to smoking? I don't have data regarding that. There are data out there. We chose to limit this report just to smoking cessation interventions.

Mannino: I can address that. There are ample data.[1,2] Basically, for every 10% increase in the cost of tobacco, you see about a 4% decrease in tobacco use. I think it's called price elastance. So if cigarettes are selling in North Carolina for $3.00 a pack and you get a 30¢ tax increase, you can expect to see about a 4% decrease. That is currently what's happening in Georgia, where we got a cigarette-tax increase because of a state budget crisis. We got a 25¢ per pack tax increase, from 12¢ to 37¢. And there's been a flurry of people calling their physicians or the tobacco quit line. One would think that 25¢ wouldn't make that much of a difference, but to some people it really does; since they buy their cigarettes by the carton, they're suddenly paying $30, as opposed to $27, which apparently is enough to make them want to quit.

REFERENCES

1. Ross H, Chaloupka FJ. The effect of cigarette prices on youth smoking. Health Econ 2003;12(3):217–230.
2. Lewitt EM, Coate D. The potential for using excise taxes to reduce smoking. J Health Econ 1982;1(2):121–145.

MacIntyre: If I understand you correctly, down at the 5–10% range nationwide, a higher tax could raise a ton of money and double that rate.

Fahy: The problem in Phoenix is that our patients go out to the Indian reservation and buy cigarettes dirt cheap. It's very frustrating.

MacIntyre: As I understand it, a major lobby in North Carolina blocking the tax increase is the smugglers. They make a lot of money because we have a very low tax and they haul cigarettes off to New York and sell them directly to retailers.

Fahy: An intervention that you didn't discuss, and that I saw at the

American Thoracic Society conference, is the Commit nicotine lozenge produced by GlaxoSmithKline. Patients really like them because they can self-dose and it doesn't stick to their dentures.

Marlow: Yes, the Commit lozenge, which contains polacrilex, comes in 2-mg and 4-mg doses. It can cause a bit of abdominal discomfort, but it seems to be pretty effective.

Hill: I feel a little uncomfortable raising this sore topic, but you mentioned the role of RTs in smoking cessation. At the hospitals where I've worked, the hospital worker category with the highest proportion of smokers seems to be RTs. Do you have any data on smoking among RTs and what's being done to help them quit? It makes it very difficult to convince patients to quit smoking when they can smell the smoke on the RT's breath.

Marlow: The study of RTs by Sockrider et al[1] did address that issue. I think we should practice what we preach.

REFERENCE

1. Sockrider MM, Maguire GP, Haponik E, Davis A, Boehlecke B. Attitudes of respiratory care practitioners and students regarding pulmonary prevention. Chest 1998; 114(4):1193–1198.

Enright: You conducted a very thorough study. The results, though—from the perspective of the percentage improvement in quit rate—looked dismal. I believe the number you showed is *added* to the baseline cessation rate. So perhaps you should say that the baseline rate is 5%, and a 5% increment doubles the smoking cessation rate.

A major problem physicians have is their perception that they have to spend time with 20 patients before one patient will successfully quit smoking, and so they give up on smoking cessation efforts. In the Lung Health Study a 20% smoking cessation rate at 11 years was considered dramatic, and that was just using Nicorette gum, because that's all we had back then.

Stoller: A point of clarification. As Paul Enright pointed out, those are absolute reduction rates; they're not percentage increments in the rate. The Cochrane Collaboration report reported absolute reduction rates.[1]

REFERENCE

1. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation. Cochrane Database Syst Rev 2002;(4):CD000146.

Heffner: Could you repeat those numbers?

Stoller: At the top, 5%, and those were the tables that reflected 5% absolute reduction, not a 5% change.

Heffner: I see. It wasn't clear what they represented, which makes them look much worse than they actually are.

Marlow: For example, in that study, if the control group had a 10% cessation rate, the addition of the intervention increased cessation rates 2%, meaning that it pushed the 10% up to 12%.

Mannino: Some people, such as Mike Fiore in Wisconsin, have put forth the idea of smoking as a vital sign, and there are little stamps and things you can put on patient charts that remind you to check the patient's smoking status in addition to the usual measurement of weight, blood pressure, and temperature. All patients are asked, "Are you a current, former, or never smoker?" Current smokers receive counseling from both the intake nurse and the physician. Former smokers receive an "attaboy"-type compliment and encouragement to continue to be a former smoker.

Heffner: I hadn't thought of this point before Paul Enright's comment, but it might be interesting to translate the absolute risk reduction into a number-needed-to-treat, which is a metric that physicians more commonly use to assess whether the effort for a given intervention is worthwhile. If my math is right, the number-needed-to-treat might be about 20, which is a value that might justify the intervention.

My second point is in regard to RTs' role in smoking cessation. Dave Pierson wrote a wonderful review for RESPIRATORY CARE 2 years ago on the future of respiratory care.[1] He commented on the role of RTs to help with smoking cessation. We took his observation and employed our RTs at my home institution to assist with the Center for Medicaid/Medicare Services' smoking cessation indicators. So far the program is working well. Respiratory therapists are going by the bedside to identify patients who have smoking histories and initiating our smoking cessation program. I believe the Center for Medicaid/Medicare Services core indicators may become an engine that will drive respiratory therapists to become more involved with smoking cessation.

REFERENCE

1. Pierson DJ. The future of respiratory care. Respir Care 2001;46(7):705–718.

Hill: The issue of smoking cessation becomes moot, of course, if people don't smoke in the first place. It's well known that most smokers start smoking in their teen years. There was a disconcerting increase in the number of teen smokers during the 1990s, but more recently there's been a decline, almost back to where it was before that 1990s' increase. Did you come across anything that explains why teen smoking has been going down and how we can keep it going down?

Marlow: I did not find anything that described why the decline. We men-

tioned prices; typically, increased taxes tend to decrease smoking among kids. I know there have been very active marketing campaigns by community organizations to decrease smoking throughout the country. Even the media have been trying to make a difference. Possibly, some of those external factors are affecting cessation rates.

Mannino: I can comment on that. In the 20-year history of active tobacco control (in the United States at least) there are 3 things that have worked, both for decreasing smoking and keeping kids from starting. One is to increase the price of tobacco, usually through graduated tax increases. Second is limiting where people can smoke. I strongly encourage everyone to get involved in their communities and support ordinances to limit where people can smoke. In DeKalb county, Georgia, we just got 100% smoke-free restaurants. Lexington, Kentucky, in the heart of tobacco country, is 100% smoke free in all indoor businesses, including bars, which was a major step forward. The third, which might be related to the settlement between the tobacco industry and some of the states, is hard-hitting anti-smoking advertising: California, Massachusetts, and Florida have been pretty good models for that. What we know *doesn't* work are those tobacco-industry-sponsored youth-access things that prompt store owners not to sell cigarettes to kids. That just makes cigarettes a little more challenging to obtain. They don't really work.

Shrake:* Your data suggest that there's a willingness among RTs to teach the importance of smoking cessation, but in hospitals they don't do a very good job at it. I would like to suggest a reason for that. You alluded to it. It is that it's not high on the priority list in the hospital.

I would like to make my pitch to this august group that you should assist your hospitals and RTs in implementing protocols so that we can reduce the amount of unnecessary respiratory therapy delivered in hospitals, so we can shift the focus of our trained respiratory practitioners to important tasks such as disease management and smoking cessation. Dr Stoller's research at The Cleveland Clinic Foundation supports the value of respiratory care protocols.

Enright: In the 15 years I've been involved in smoking cessation, my biggest disappointment has been that there are no predictors—demographic, cigarettes-per-day, questionnaire answers, or instruments—of which smoking cessation method will work for an individual patient. So that's a wide open field, to develop such an instrument. Right now it's just totally empirical. You mentioned you should tailor a smoking cessation program to the patient's characteristics, but there are no patient characteristics that predict who will respond to what intervention. You've just got to try one method after the other, and it's usually a matter of economics or availability that dictate the choice.

Pierson: I would just point out that we have been cheering the wonderful results of 22% smoking cessation at 11 years in, if not the only, certainly by far the *major* intervention shown to have any impact on the natural history of the disease this conference is about. Twenty-two percent success in getting people to stop smoking at 11 years! I think that's a call for whatever needs to be done in any way to push ahead with smoking cessation. Not to denigrate all the other treatment modalities we're talking about, and not to make light of the enormous burden that getting people to stop smoking represents, but here's where we really ought to be targeting every resource available in our health care system. It's clear from what Neil MacIntrye and others have said that there are powerful forces, at least in the United States, working against that specific goal.

Hill: I think—for nonsmokers—one of the truly welcome changes we've seen in the last 15 years is the virtual elimination of cigarette smoking from public places. Yet in Europe it's still the way it used to be 15 or 20 years ago. In Europe we're just assaulted with cigarette smoke in public. When I was in Brussels recently, someone lit up in an elevator and I almost had a stroke, but then remembered where I was. Wisia, can you comment? Can we some day expect smoke-free public places in Europe?

Wedzicha: I think the problem is that we in the United Kingdom, for instance, do not have a ban yet on smoking in public places. I think the United Kingdom is more advanced in their smoking cessation efforts than other countries in Europe, but, particularly in Eastern Europe, the situation is pretty bad. I think until we have a full smoking ban in public places, we will not see benefits.

---

* Kevin L Shrake MSc RRT FAARC, Chief Operating Officer, American Association for Respiratory Care, Dallas, Texas.