

**KAISER PERMANENTE**

The Smoker's Book

# Independence from Smoking

Read the entire booklet all the way through to help you prepare to be smoke-free. It may take a few extra minutes, but it is well worth your time. Then set a specific quit date using the contract on page 17.

When you have successfully quit, you will feel a great deal of personal satisfaction at having accomplished a very difficult task, and the rewards may be more than you imagined.

## Preparing to Quit

Think about what keeps you from quitting.

The benefits of kicking the smoking habit are numerous. They include freedom from the mess, smell, inconvenience, expense, and dependence associated with smoking.

To develop the motivation you need to ensure your success, make a list of all your reasons for smoking and all your reasons for quitting. State the reasons most important to you.



## Pros

Example:

*Smoking helps me control my weight.*

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## Cons

Example:

*Smoking excludes me from many activities.*

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You have smoked a lot, and each cigarette has reinforced the strength of your habit. Now you must be equally strong in reversing the process. Once the reasons not to smoke are greater than your reasons to smoke, quitting will be much easier. In order to remind yourself how much you want to stop, post your list of reasons in a prominent place in both your home and office. Look at this list each time you get the urge to smoke.

## Strategies

You can prepare to quit by changing some habits before you quit. What you do *before* you quit can help you succeed. Do at least three of the following:

- Begin to buy cigarettes by the pack; do not buy cartons.
- Change to lower tar and nicotine brands each time you buy a pack of cigarettes. (Not getting accustomed to a specific brand helps prepare you for quitting.)
- Eliminate some of the places where you allow yourself to smoke.
- Make smoking unpleasant. Don't empty your ashtrays, collect all your cigarette butts in one large glass container as a visual reminder of the amount you smoke; the sight and smell of stale cigarette butts will be very unpleasant.
- Practice the four Ds to prevent automatic smoking:
  1. Deep breathe: inhale through your nose and hold for the count of five. Slowly exhale through your mouth.

2. Drink 8–10 glasses (8 ounces each) of water every day.



3. Delay smoking for five minutes when you feel a craving. You will find the urge to smoke goes away whether you smoke a cigarette or not.
  4. Do something—keep busy.
- Picture your success. Spend at least 5 to 10 minutes a day imagining yourself as a comfortable non-smoker in situations where you usually smoke.
  - Move cigarettes to a hard-to-reach place. Stop carrying matches or a cigarette lighter. Make smoking less automatic.

## How to quit

You must mentally prepare yourself to become a nonsmoker. You need to set a specific date you want to stop—your Quit Day—and write it down. Use the contract in this booklet to take this important step. (You do not have to stop smoking immediately, unless you are prepared to quit.)

Now that you have chosen a day, you are ready to decide which method you will choose to stop smoking: either *cold turkey* or *tapering*. If you choose to quit cold turkey, continue to smoke until Quit Day. A quick final break with cigarettes can give you the confidence of knowing you can survive a period of time without smoking at all.

If you decide to taper off, prepare yourself for Quit Day by either gradually decreasing the number of cigarettes you smoke, or gradually increasing the number of uninter-

hours you go without smoking each day. Gradually cutting down may give you the opportunity to mentally prepare to accept not smoking.

Whether you quit cold turkey or taper off, it will take some time—several weeks for most smokers—before your urge to smoke lessens. The degree of your commitment to quit smoking will play a large role in determining what your quitting experience is like.

### Using nicotine replacement therapy

Nicotine replacement therapy (NRT) slowly lessens the level of nicotine in your body. These products include gum, nicotine patches, and Bruprion. (Consult with your health care professional if you think you need NRT.)



Follow the directions on the packaging completely. These are temporary aids to help reduce the physical symptoms, to allow you to work on the behavioral aspects of your smoking habit. ***Never smoke while using these products. It is extremely harmful.***

### Tips to help you quit

The following are general suggestions that will help you quit smoking.

- Increase awareness of your smoking habit by keeping a diary. The diary helps you become aware of (1) when, where, and why you are smoking; (2) how much or how little you smoke in certain situations. It will also help you see more objectively how much smoking is affecting your life. You might want to set the diary up or copy the one on this page.

### Smoking Diary

Number	Time	Need (based on a 1-5 scale)	Place or Activity	With Whom	Mood/Reason

- Rank your cigarettes from 1 to 5. (1 or 2 = I can do without it; 3 = I would like it; and 4 and 5 = I need it.)
- Reread all the reasons you want to quit, every night before going to bed, then repeat one of those reasons out loud to yourself 10 times.
- Tell a friend the date of your Quit Day. Also tell him or her your method of quitting, possible obstacles, and the reward you plan to give to yourself for staying a nonsmoker. Support from others can be very helpful.

### After you quit

- Discard all smoking paraphernalia—ashtrays, lighters, and extra packs of cigarettes.
- Change your routines, activities, and even the order in which you usually do them. This is especially important for the routines that included smoking in the past. (Take a walk instead of a coffee break, wear gloves while driving, drive a different route to work.) That way, you'll be focusing your thoughts on what you need to do next rather than thinking about missing your usual cigarette. It also may help permanently break the link between certain activities and the urge to light up.
- Fill in the gaps of time that were spent smoking with other activities—start a hobby, visit a friend, begin an exercise program, read a book.



- Substitute other things in your hands or mouth as a replacement for the cigarette (sugarless gum, a hard candy, beverage stirring stick, cinnamon sticks, worry beads).
- Practice deep breathing. Take long deep breaths. This can be relaxing and helps the urge to smoke to pass.
- Constantly imagine yourself as a happy nonsmoker. Focus on the positive benefits to be gained by quitting.
- Plan a major celebration for your six-month anniversary as a nonsmoker!



### Staying smoke-free

The next few weeks may be the most difficult as your body readjusts to being a nonsmoker. You may experience such symptoms of recovery as a change in sleep patterns, lightheadedness, more coughing, etc. Think of these as the positive effects of your body adjusting to a life without tar and nicotine.

You may also find that you still have sudden and strong urges to smoke that seem to appear without warning. The best approach is to *be prepared*—expect these urges to occur and create specific strategies to deal with them and prevent them in the future.

### Positive statements

Here are a few statements that you use to help talk yourself through an urge or craving until it passes (which does whether you smoke or not).

Choose one or two of the following phrases that have the greatest meaning for you and write them down.

1. The urge will pass.
2. I don't smoke anymore.
3. Just one day at a time.
4. There are no excuses.
5. I choose to be in control of my life.
6. Smoking is no longer an option for me.

Read your selected phrases whenever you need to deal with an urge!

### Strategies

Chances are, you tend to smoke at certain times or in certain situations. These situations can "trigger" the urge to smoke, so it is important that you avoid these triggers for awhile and that you also have specific plans for coping with each of these common triggers when they do occur.

**Prevent** situations that trigger the urge to smoke by doing the following:

- Avoid smokers for the first few weeks after you've quit.
- Try not to get too tired or hungry—keep healthy snacks on hand.
- Learn to relax and take some deep breaths throughout the day—before an urge hits.
- Take a walk every day.
- Get plenty of rest and sleep.
- Reward yourself with something everyday.

**Cope** with situations that trigger your urge to smoke by trying some of the strategies that have worked for other smokers trying to quit. The goal is to change your environment, distract yourself, and find something else to do instead of smoking.



# 3

## Three Days to Quit Day!

- Starting today, eliminate one place (car, bathroom, garage, etc.) where you usually smoke. Continue to do this for the next three days.

- Drink more water.

- Continue the following:

1. Delay five minutes before lighting up.
2. Keep your daily diary, and check for a pattern in your smoking.
3. Become used to the idea of being smoke-free.
4. List further tips of your own that will help you quit.

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- Calculate the cost of smoking:

1. \_\_\_\_\_ Average number of packs smoked per day
2. \_\_\_\_\_ Average number of packs smoked per year (multiply figure on line 1 by 365)
3. \_\_\_\_\_ Total number of years smoked
4. \_\_\_\_\_ Total number of packs smoked to date (multiply figure on line 2 by figure on line 3)

5. \_\_\_\_\_ Total cost to date (multiply figure on line 4 by \$2.50, which is the average cost per pack over the last 20 years)



### Cost of future smoking:

6. \_\_\_\_\_ Average number of packs smoked per year (same figure as on line 2)
7. \_\_\_\_\_ Total you would have smoked the rest of your life (first subtract your age from 75 years, then multiply the figure on line 6 by that number)
8. \_\_\_\_\_ Total cost for future smoking (multiply the cost on line 7 by \$5.00, the estimated future average cost per pack)
9. \_\_\_\_\_ Lifetime cost of smoking (add figures on lines 5 and 8)

On a scale of 1-10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_\_

# 2

## In Two Days I Will Be Free of Cigarettes!

- Take the test "Why I Smoke" on page 13.
- Continue the following:
  1. Delay five minutes before lighting up. Think positive thoughts about your ability to resist the urge for the next five minutes.
  2. Keep your daily diary.
  3. Don't smoke in one more customary smoking place.
  4. Practice the deep-breathing exercise.
  5. Visualize yourself as a confident nonsmoker.

On a scale of 1-10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_\_

# 1

## One Day Until I Quit Smoking!

Now it is time to clarify your values regarding not smoking.

- Make a list of reasons why you continue to like smoking.
- Review your list of reasons why you want to quit smoking.
- Make a list of substitute activities. Make separate lists for activities involving the hands (knitting, unfolding paper clips); the mouth (chewing gum, cinnamon sticks); relaxation (walking); and deep breathing (suck on plastic straw or beverage stirrer).
- Identify patterns for smoking and nonsmoking times by looking carefully at your smoking diary.
- Practice deep-breathing exercise.

On a scale of 1-10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_\_





## When you're...

around others who smoke:

- Practice saying "No thanks, I've quit."
- Tell your friends you're trying to quit.

feeling bored:

- Find new ways to occupy your time.
- Look through books or magazines.
- Take walks or start a hobby.
- Call a friend.

feeling nervous,  
stressed, or anxious:

- Count to 10.
- Take a walk.
- Learn relaxation techniques.
- Learn how to communicate feelings and emotions.

finished with your meal:

- Don't linger at the table.
- Chew gum or a strong mint.
- Brush your teeth.
- Phone a friend.

watching TV:

- Do something with your hands.
- Sip water.
- Eat popcorn.

talking on the phone:

- Doodle on paper.
- Talk from a different location than your usual spot.

drinking coffee:

- Hold cup in a different hand.
- Drink in a different location.
- Consider switching to tea.

drinking alcohol:

- Avoid drinking and bars while you are trying to quit.

driving in the car:

- Take a different way to work.
- Play relaxing music.
- Have snacks, water, or gum on hand.



## The Five-Day Countdown to Quit Day

# 5

### Five Days Until I Quit Smoking!

- During the next five days, cut your smoking back to less than 20 cigarettes per day of a low-tar and low-nicotine brand. Do **not** go below 10 cigarettes per day. At that level, each cigarette becomes more important, and it may become too difficult to quit.
- Buy cigarettes by the pack instead of by the carton.
- You may switch brands as often as you like, providing they are low-tar and low-nicotine. Try not to smoke two packs of the same brand in a row.
- Keep track of how many cigarettes you smoke by keeping your daily diary.
- Spend plenty of time getting used to the idea of being smoke-free.
- Involve a friend for support.
- Become familiar with this booklet.
- Start a new exercise habit.

On a scale of 1–10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_

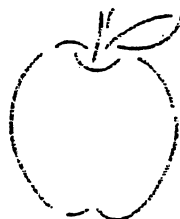
# 4

### Four Days to a Smoke-Free Me!

Make the following changes in your smoking habit in the next four days:

- When you desire a cigarette, *delay five minutes* before lighting it. Try to change your activity by finding distractions, such as starting a conversation, drinking a glass of water, or closing your eyes and imagining yourself as a *confident healthy nonsmoker*.
- Practice deep breathing. (It triggers the same mechanism as inhaling smoke.) Take a deep breath, hold five seconds, exhale slowly through your lips, repeat two to three times.
- Try smoking with the hand you don't normally use.
- List the cigarettes you smoked today that you feel are unnecessary, and try to eliminate them during the next few days.
- Add a healthy snack—fruit, nuts, or popcorn.
- Use positive thinking.

On a scale of 1–10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_





## Zero Day—Quit Day!

- Congratulations! You are on your way to a smoke-free self. Today is a milestone for you. You have already accomplished a lot and your thoughts about quitting smoking have been put into action!
- Throw away all smoking-related items. Clear out all ashtrays, lighters, and cigarettes.
- Keep busy and spend as much time as possible in places where smoking is not allowed.
- Set up an appointment with your dentist to have your teeth cleaned to get rid of tobacco stains.
- Buy yourself a treat or do something special to celebrate.
- Enjoy your new lifestyle as a nonsmoker!

On a scale of 1–10 (1 is not confident, 10 is very confident), how confident are you about becoming a nonsmoker? \_\_\_\_\_

## What to expect when you quit

### Symptoms of Recovery

Within 72 hours after you have your last cigarette, your body begins to heal. As your body repairs itself, instead of feeling better, you may feel worse. These “withdrawal pangs” are symptoms of recovery. Many ex-smokers experience temporary weight gain caused by fluid retention, irregularity,

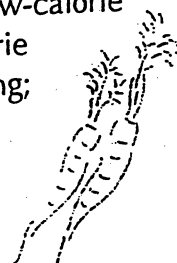
and soreness of the gums or tongue. You may also feel edgy and short-tempered. It is important to understand that these symptoms are only temporary and signals that you are beginning a healthier lifestyle.

Within 20 minutes of your last cigarette, you should notice the temperature of your hands and feet increase to normal. Within eight hours the carbon monoxide level in your blood drops to normal. Within 72 hours the bronchial tubes relax, making breathing easier.

Within a few weeks, you will notice some major changes in your body: your sense of taste returns; your smoker’s cough is gone; your digestive system returns to normal; and your head is clear, with no more headaches from cigarettes. Most important of all, you will feel really alive, full of energy and strength.

Within one to nine months, coughing, sinus congestion, fatigue, and shortness of breath decrease, while your energy level increases.

Although weight gain is common, it can be prevented or kept to a minimum, since it is due in part to an increased appetite and food intake. The weight gain is usually not permanent, since most ex-smokers go back to their former weight in time. If you are concerned about putting on extra weight, you may do the following: drink six to eight glasses of water daily; plan menus to include low-calorie foods and have low-calorie foods on hand for nibbling; chew sugarless gum; and take time each day for exercise.



*Robert B. Baron MD, MS*

## INTRODUCTION

Obesity is one of the most common problems in clinical practice. Defined as a body weight 20% or more above "desirable" weight, over one third of adult Americans are overweight. Perched at the center of chronic disease risk and psychosocial disability for millions of Americans, the prevention and treatment of obesity offers unique patient care and public health opportunities. If all Americans were to achieve a normal body weight, it has been estimated that life expectancy would increase by 3 years, coronary heart disease would decrease by 25%, and congestive heart failure and stroke would decrease by 35%.

Unfortunately, obesity is often one of the most difficult and frustrating problems in primary care for both patients and physicians. Considerable effort is expended by primary care providers and patients with little benefit. Using standard treatments in university settings, only 20% of patients lose 20 pounds at 2-year follow-up, and only 5% of patients lose 40 pounds. This lack of clinical success has created a never-ending demand for new weight-loss treatments. Approximately 45% of women and 25% of men are "dieting" at any one time, spending over 0 billion each year on diet books, diet meals, weight-loss classes, diet drugs, exercise tapes, "fat farms," and other weight-loss aids. The challenge for health care providers is to identify those patients with obesity who are most likely to benefit medically from treatment and most likely to maintain weight loss, and to provide them with sound advice, skills for long-term lifestyle change, and support. For patients not motivated to attempt a weight-loss program, health providers must continue to be respectful and empathic and focus on other health concerns (see Chapter 16). Whenever possible, health providers should emphasize prevention of obesity and further weight gain.

## DEFINITIONS

Obesity represents an excess of body fat; being overweight means having an excess of body weight, including all components of body composition (muscle, bone, water, and fat). In clinical practice, the two are used interchangeably to refer to excess body fat. The two most commonly used terms to quantify obesity are relative weight (RW) and body mass index (BMI). The RW is the actual weight divided by the "desirable weight" (derived from "acceptable weight" tables). The BMI, or Quetelet index, is the actual body weight divided by the height squared (kilograms per square meter). This index more closely corresponds to measurements of body fat and better differentiates being "overweight" due to an increase in muscle mass from that caused by true obesity.

The National Institute of Health defines obesity (somewhat arbitrarily) as a RW of greater than 120% ( $\text{BMI} > 27 \text{ kg/m}^2$ ). "Morbid" obesity is commonly defined as a RW greater than 200% ( $\text{BMI} > 40 \text{ kg/m}^2$ ).

## HEALTH CONSEQUENCES OF OBESITY

The relationship between body weight and mortality is curvilinear, similar to other cardiovascular risk factors. Most studies have demonstrated a {J}-shaped or {U}-shaped relationship, suggesting that the thinnest portion of the population also have an excess mortality. This is primarily due to the higher rate of cigarette smoking in the thinnest group, except in the elderly, in whom malnutrition is predictive of excess mortality independent of cigarette use.

The increase in total mortality related to obesity results predominantly from coronary heart disease (CHD). Although it is not fully established that obesity is an "independent" risk factor for CHD, obesity is clearly an important risk factor for the development

of many other CHD risk factors. Obese individuals age 20-44, for example, have a three- to fourfold greater risk of type II diabetes, five- to sixfold greater risk of hypertension, and twice the risk of hypercholesterolemia. The obese also have an increased risk of some cancers, including those of the colon, rectum, and prostate in men; and those of the uterus, biliary tract, breast, and ovary in women.

As a result of these conditions, relative weights of 130% are associated with an excess mortality of 35%. Relative weights of 150% have a greater than twofold excess death rate. Patients with "morbid" obesity (relative weights greater than 200%) have a greater than 10-fold increase in death rates.

Obesity is also associated with a variety of other medical disorders, including degenerative joint disease of both weight-bearing and non-weight-bearing joints, diseases of the digestive tract (gallstones, reflux esophagitis), thromboembolic disorders, heart failure (both systolic and diastolic), respiratory impairment, and skin disorders. Obese patients also have a greater incidence of surgical and obstetric complications, are more prone to accidents and are at increased risk of social discrimination. Obesity is not, however, associated with an increased risk of major psychiatric or psychological disorders.

In addition to the total amount of excess body fat, the location of the excess body fat (regional fat distribution) is a major determinant of the degree of excess morbidity and mortality due to obesity. Increased upper body fat (abdomen and flank) is independently associated with increased cardiovascular and total mortality. Body fat distribution can be assessed by a number of measurement techniques. Measurements of skin folds (subscapular and triceps) reflect subcutaneous fat. Measurement of circumferences (waist and hip) reflect both abdominal and visceral fat. Computed tomography (CT) and magnetic resonance image (MRI) scans measure subcutaneous and visceral fat. Clinically, measurement of the waist and hip circumference is most useful. The waist is measured at the umbilicus and the hips at the greater trochanter. A waist-to-hip ratio of 1.0 and 0.8 are considered normal in men and women, respectively. Ratios above these values reflect abdominal or visceral obesity (or both) and a greater risk of obesity-related disorders.

## ETIOLOGY OF OBESITY

Numerous lines of evidence, including both epidemiologic studies of adoptees and twins and animal studies, suggest strong genetic influences on the development of obesity. In a study of 800 Danish adoptees, for example, there was no relationship between the body weight of adoptees and their adopting parents but a close correlation with the body weights of their biological parents. In a study of approximately 4000 twins, a much closer correlation between

body weights was found in monozygotic than in dizygotic twins. In this study, genetic factors accounted for approximately two thirds of the variation in weights. More recent studies of twins reared apart and the response of twins to overfeeding showed similar results. Studies of regional fat distribution in twins has also shown a significant (but not complete) genetic influence.

Recent molecular genetic studies have confirmed important genetic determinants of some types of obesity. Studies in animals have identified a gene that, when made useless by mutation, causes obesity. The normal gene produces the protein leptin, which controls appetite. When leptin is defective, mice grow profoundly fat; when leptin is supplemented, the mice lose weight. Human studies have recently confirmed the existence of an almost identical human gene. Although mutations in this gene are rare, it is hypothesized that extra doses of leptin may be effective in reducing human obesity. Recently, a high-affinity receptor for leptin has been identified in the mouse hypothalamus and a possible leptin-induced satiety factor, glucagon-like peptide-1 has been identified. Human studies have also identified a mutation in the gene for the beta-3 receptor in adipose tissue, involved in lipolysis and thermogenesis, that markedly increases the risk of obesity. These studies suggest that genetic factors may result in changes in both energy intake and energy expenditure. Genetic influences on control of appetite and eating behavior have long been considered. Animal studies have demonstrated the influence of dozens of factors on eating behavior, and it is likely that similar factors are at work in humans.

Differences in energy expenditure are also likely to be at least partially determined by genetic influences. Differences in the resting metabolic expenditure (RME), for example, could easily result in considerable differences in body weight since RME accounts for approximately 60-75% of total energy expenditure. The RME can vary by as much as 20% between individuals of the same age, sex, and body build; such differences could account for approximately 400 kcal of energy expenditure per day. Recent evidence suggests that the metabolic rate is similar in family members, and as expected, individuals with lower metabolic rates are more likely to gain weight. Differences in the thermic effect of food, the amount of energy expended following a meal, may also contribute to obesity. Although some investigators have shown a decreased thermic effect of food in the obese, others have not.

Environmental factors are also clearly important in the development of obesity. Decreased physical activity and food choices that result in increased energy intake also clearly contribute to the development of obesity. Medical illness and some medications can also result in obesity, but such instances account for less than 1% of cases. Hypothyroidism and

Cushing's syndrome are the most common. Diseases of the hypothalamus can also result in obesity, but these are quite rare. Major depression, which more typically results in weight loss, can sometimes present with weight gain. Consideration of these causes is particularly important when evaluating unexplained, recent weight gain.

## PATIENT SELECTION FOR WEIGHT LOSS

Weight loss is indicated to assist in the management of obesity-related conditions, particularly hypertension, diabetes mellitus (type II), and hyperlipidemia, in any patient who is obese ( $RW > 120\%$ ,  $BMI > 27 \text{ kg/m}^2$ ). Many patients with relative weights of 100–120% ( $BMI 25\text{--}27 \text{ kg/m}^2$ ) who have one of these conditions (particularly hypertension, diabetes, lipid disorders, or significant psychosocial disability) also often dramatically benefit from weight reduction.

Weight loss to prevent complications of obesity in patients without current medical, metabolic, or behavioral consequences of obesity is more controversial. In young and middle-aged individuals, particularly those with a family history of obesity-related disorders, treatment should be based on the degree of obesity ( $RW > 120\%$ ) and body fat distribution. Such individuals with upper body obesity (waist-hip ratios  $> 1.0$  in men and  $> 0.8$  in women) should be considered for treatment; those individuals with lower body obesity and no significant consequences of obesity, can be reassured and followed for development of additional upper body obesity and its metabolic consequences. Many such patients, however, desire weight loss for psychological, social, and cosmetic reasons. A careful discussion of the risks and benefits of weight loss in such instances helps patients make informed decisions about various weight-loss strategies.

A medical or psychosocial indication for weight loss is necessary but not sufficient, however, to begin treatment. Only motivated patients should be treated. Considerable effort should be made to assess the patient's motivation for significant diet and exercise changes. Questions should focus on how the current attempt compares with previous attempts, a realistic assessment of the patient's goals for the amount and rate of weight loss; the extent to which outside stresses, mood disorders, or substance abuse might impair the attempt; and the degree to which others can provide support. Patient motivation can be further assessed by requiring the patient to complete specific pretreatment assignments. For example, patients can be asked to complete a 3-day diet record and to submit an exercise plan that includes both the type of aerobic exercise the patient plans to begin and how the patient plans to fit it into his or her schedule. When obesity coexists with other significant psychiatric disorders, particularly depression and substance abuse,

treatment should initially be directed at the concurrent disorder. (See Chapter 16 for further discussion of assessment of patients' readiness to change.)

## DIET THERAPY

Standard dietary treatment of obesity should use the same nutritional principles as diet recommendations for healthy people. Total fat intake should be limited to 30% or less of total calories, protein to 15%, and carbohydrate (primarily complex carbohydrates) to 55% or greater. A total energy intake should be recommended to result in a daily energy deficit of 500–1000 kcal. Since 1 pound of fat equals approximately 3500 kcal, these deficits should result in a 1–2 pound weight loss per week. No dietary manipulation of macronutrients or other nutritional components can change these basic thermodynamic concepts, yet, virtually all popular diets are based on attempts to circumvent thermodynamics. Diets can easily be designed in medical settings with the assistance of clinical dietitians or other health professionals, or patients can be referred to well-established community resources that follow these principles.

A major development in the dietary treatment of obesity is the use of safe and effective very-low-calorie diets (VLCDs). Also known as protein-sparing modified fasts and protein-formula liquid diets, these diets restrict calorie intake to 400–800 kcal/day. Patients ingest only preformulated, usually liquid, food that provides adequate protein, vitamins, and minerals. Additional intake is limited to 2–3 quarts of calorie-free beverages per day. The major advantage of these diets is the "complete removal of patients from the food environment" to facilitate compliance. In addition, the significant energy deficit results in rapid weight loss, usually 2–4 pounds per week, encouraging the patient to continue. Ongoing concerns about these diets include their cost, side effects and complications, and long-term results. Recent studies suggest that the use of 800-kcal VLCDs can lower cost and prevent most of the significant side effects associated with the lower-calorie (400–600) VLCDs, including gallstones and fluid and electrolyte disorders, with equal long-term efficacy.

As with standard diet therapy of obesity, VLCDs require compliance during the diet, and long-term nutritional and behavioral changes to maintain weight loss. Well-planned programs that combine VLCDs with behavior modification, exercise, and social support report improved long-term results. For example, Nunn and colleagues report an average weight loss of 55 pounds with 75% and 52% of the loss maintained at 1- and 2½-year follow-up, respectively, and Hartman and associates reported maintenance of an average of 24 pounds after 2- to 3-year follow-up. Although these results are significantly better than results from published reviews of standard weight-loss

programs (in which approximately only 5% of patients lose and maintain 40 pounds at 1-year follow-up), the even-longer-term efficacy and cost-effectiveness of VLCDs programs remains controversial.

## HEALTH CONSEQUENCES OF DIETING & WEIGHT LOSS

Surprisingly few studies have examined the effects of weight loss on morbidity and mortality. Studies examining the effect of weight loss on cardiovascular risk factors generally show beneficial changes with weight loss as predicted. Descriptive studies on mortality, however, show inconsistent results. Such descriptive studies are unable to clarify if changes in mortality are caused by the weight change, if disease or other factors that contribute to disease, such as cigarette smoking, cause weight loss, or if both are related to a third factor. No randomized trials of long-term effects of voluntary weight loss on mortality have been published.

Since so many Americans are dieting at any one time, and having so little long-term success, considerable interest has been focused on the potential adverse effects of weight cycling ("yo-yo" dieting). Numerous adverse effects of weight cycling have been hypothesized, primarily from animal studies. These include making further weight loss more difficult, increasing total body fat and central obesity, increasing subsequent calorie intake, increasing food efficiency, decreasing energy expenditure, increasing levels of adipose-tissue lipolytic enzymes and liver lipogenic enzymes, increasing insulin resistance, increasing blood pressure, and increasing blood cholesterol and triglyceride levels. Most experts currently feel that these phenomena occur inconsistently, if at all. Descriptive studies that have addressed this question by looking at the impact of weight fluctuations on CHD incidence, CHD mortality, and total mortality have shown mixed results.

There is also debate over whether weight loss diets cause eating disorders or binge eating. Although a history of dieting often precedes the development of eating disorders, there is no evidence proving a causal link. In addition, approximately half of individuals with binge eating disorder report that bingeing preceded dieting. There is also some evidence to suggest that successful weight loss may reduce binge eating in the obese (see Chapter 20).

Thus, there is only indirect evidence suggesting that dieting has negative health effects. This remains an important question, however, and reinforces the idea that casual attempts at quick weight loss should be avoided. At the present time, however, committed attempts at long-term weight loss should not be discouraged because of adverse health effects or the potential of regaining weight.

Dieting also has a significant effect on energy bal-

ance both during and after weight loss. As every successful dieter has observed, the rate of weight loss slows during the course of dieting. Because this can be quite discouraging to the unwary patient (or uninformed health care provider), it is important to inform the patient prior to initiating a weight-loss diet that this is likely to occur. Weight loss is most rapid during the initial days of hypocaloric feeding due to changes in sodium and water balance. This is due to early loss of glycogen and protein (both contain water) and, depending on the degree of calorie deficit and type of diet, to sodium losses associated with ketonuria. Following this initial phase, weight loss depends on the extent of energy deficit. With time, however, the rate of weight loss slows again as the body's metabolic rate decreases and the energy deficit becomes smaller. This change in metabolic rate can be two to three times greater than that predicted from changes in body weight. The lower the energy content of the diet, the lower the metabolic rate. Although it was initially suggested that exercise occurring during a period of hypocaloric feeding could prevent this decrease in metabolic rate, recent studies have suggested that it has no direct effect during hypocaloric feeding (but exercise does increase the postdiet metabolic rate by preserving lean body mass).

Following the period of hypocaloric feeding (resumption of normal energy intakes), the resting metabolic rate increases, but to a level below that observed before beginning the diet. This reduction is in part a reflection of the loss of lean body mass and in part due to additional, poorly understood effects on energy metabolism. Overall energy expenditure is further reduced due to a decrease in the thermic effect of food (the individual eats less) and in differences in physical activity (it takes less energy to perform the same amount of activity for a smaller person). Thus, in order to maintain weight loss, individuals need to consume less energy than before dieting (and increase energy expenditure by increasing the amount of physical activity).

## EXERCISE

Exercise offers a number of significant advantages to patients attempting to achieve long-term weight loss. First and foremost, exercise increases energy expenditure, helping to create the energy deficit necessary for weight loss. Unfortunately, the amount of energy expended during most aerobic exercises (walking, jogging, swimming, etc) for the typical periods performed (20-30 minutes four to five times per week) is modest, approximately 500-1000 kcal per week. Thus, exercise can be predicted to have little effect on short-term weight loss. Clinical trials reflect this modest effect: some studies demonstrate weight loss with exercise alone or extra weight loss when exercise plus diet is compared with diet alone, but other studies do not show such an effect.

The importance of exercise for successful maintenance of weight loss is more clearly established. In addition to the cumulative effect of increased energy expenditure ( $500\text{--}1000\text{ kcal/week} \times 52\text{ weeks} = 7\text{--}15$  pounds per year), exercise affects the composition of the body substance lost during weight loss. When exercise is directly compared with diet, or when exercise plus diet is compared with diet alone, exercise results in greater preservation of lean body mass. That is, for each pound of weight lost, less fat and more muscle is lost during weight-loss programs without exercise. This is particularly important since the body's resting metabolic expenditure (a major portion of the total daily energy expenditure) is closely correlated with lean body mass.

The observation that much of the long-term effect of exercise is through preservation of lean body mass has resulted in an increased interest in the potential role of resistance training (weight lifting, circuit training, etc). Preliminary results suggest that resistance training during dieting does result in maintenance of lean body mass compared with the result from dieting alone. Thus, highly motivated patients can be instructed to add resistance training to their aerobic exercise program.

Regular aerobic exercise results in a number of other benefits to the obese patient, including improved cardiovascular training effect (increased exercise tolerance), decreased appetite (per calorie expended), a general sense of well-being, decreased blood pressure (in hypertensives), improved glucose metabolism and insulin action (in diabetics), improved blood lipids (in lipid disorders), and, in the long-term, decreased mortality from cardiovascular disease and all other causes.

Young patients with mild-to-moderate obesity can be started directly on a regular aerobic exercise program. Patients are commonly instructed to select two exercises and to perform either one of them four to five times per week for 30 minutes per day. Patients are taught to take their pulse and to generate a sustained tachycardia at 70–80% of their maximum predicted heart rate. Sedentary patients, older patients, and patients with severe obesity are instructed to begin walking programs without initial concern about meeting target heart rates. As weight loss proceeds, and patients become used to exercising regularly, they can be advanced to formal aerobic programs.

## BEHAVIOR MODIFICATION & SOCIAL SUPPORT

Sustained weight loss requires long-term changes in eating behavior. Patients must learn specific skills to facilitate decreased calorie intake and increased energy expenditure. Although formal behavior modification programs are available, most patients can be taught basic behavioral strategies in the office.

The single most useful behavioral skill is planning and record keeping. Patients can be taught to plan both menus and exercise programs in advance. Patients are instructed to record actual food intake and exercise behaviors. The act of record keeping itself aids in behavioral change, and the availability of records also helps the health care provider assess progress and make specific suggestions for additional problem solving. Specific reward systems are also useful for many patients. Refundable financial contracts have been shown to be effective in a number of small studies. Additional behavioral strategies include breaking behaviors into identifiable parts (antecedents, consequences, etc) and specific techniques for stimulus control. Methods for slowing eating may also be effective. Efforts to identify and understand lapses and to prevent relapses are particularly important for long-term weight maintenance.

Social support is an additional essential component for any successful weight-loss program. Most successful programs use peer group support. Diet partnerships are effective for some patients. Involvement of family members is also important. A comprehensive review of published results of weight-loss programs strongly suggests that close provider-patient contact is a better predictor of success than the particular weight-loss intervention.

## MEDICATIONS FOR TREATMENT OF OBESITY

Medications for the treatment of obesity are widely available, both over the counter and with prescription. Amphetamines (DEA schedule II) have extremely high abuse potential and are almost never recommended for weight loss. The most commonly prescribed appetite suppressants are fenfluramine (Pondimin), phentermine (Adipex-P, Fastin, Ionamin), diethylpropion (Tenuate, Tepanil), mazindol (Sanorex, Mazanor), and the antidepressants fluoxetine (Prozac) and sertraline (Zoloft). Dexfenfluramine (Redux), an isomer of fenfluramine, was approved by the FDA in the spring of 1996. Phenylpropanolamine is available without prescription.

Considerable controversy exists as to the efficacy of these agents and specific indications for their use. Numerous barriers to the use of medications for obesity have been described recently by advocates for their use. These include public perception of obesity as a disease of lack of willpower, expectation that medications should "cure" obesity, hindrance by state licensing agencies, limited research on long-term efficacy, and the abuse potential of some of the medications. Numerous short-term studies of appetite-suppressant drugs (ASDs) have been conducted. A recent meta-analysis of 36 studies using mazindol and fenfluramine showed that after a median duration of 12 weeks these drugs resulted in a mean weight loss 3 kg



greater than placebo. In those studies with follow-up, discontinuation of the drugs most commonly resulted in weight gain. Advocates of drug therapy point out, however, that this is true for medications for most chronic diseases, such as hypertension, cholesterol, diabetes, peptic ulcer disease; stopping a successful medication results in a recurrence of the condition being treated. In these cases as well, medications do not cure the illness. A recent long-term study with phentermine (15 mg) and fenfluramine (60 mg) showed 9.4-kg weight loss after 3 years (Of the 121 who started the study, 51 completed 190 weeks; these mean results do not include those who dropped out). Most common side effects included dry mouth, GI disturbances, somnolence, and nervousness.

Advocates of ASDs interpret these results to suggest that long-term use of such medications is safe and efficacious and merits a reevaluation in clinical practice, especially when used as an adjunct to a comprehensive weight-loss program or to prevent weight gain following successful weight loss. Medications might be particularly helpful to patients with severe obesity and medical complications of obesity. Initial experience in such patients, however, has not been encouraging. Medications seem to work best in those who need them the least, that is, motivated patients with mild obesity. Current recommendations are to use medications in selected, highly motivated patients. It is extremely important for patients to be fully informed about the limited efficacy of medications, the risk of side effects, the uncertain long-term effects, and the likely need to continue medications indefinitely before initiating their use. Such realistic information often discourages patients from selecting this approach.

## SURGERY

Although generally considered to be the last resort for the treatment of obesity, over 100,000 obese patients have had surgical therapy. Few controlled trials exist, and the development of rational indications for surgery has been difficult.

Gastric operations are now the procedures of choice. Most popular are the vertical-banded (Mason) gastropasty, in which a smaller stomach pouch is cre-

ated, and gastric bypass procedures. Although both procedures result in significant weight loss, randomized trials comparing the two tend to favor gastric bypass procedures. This is particularly true in patients who consume large amount of sweets. Perioperative mortality averages < 1% but ranges between 0-4% in different centers. Complications are common and include wound dehiscence, peritonitis, nausea and vomiting, vitamin deficiencies, and hair loss. When all necessary reversals, revisions, and patients lost to follow-up are considered, failure rates approach 50%.

Jejunioileal bypass was the first major surgical procedure popularized for the treatment of obesity. This procedure has been abandoned due to numerous complications. Jaw wiring is another surgical treatment that is not recommended. Suction lipectomy, or liposuction, permits the removal of fat from specific areas of the body. Usually performed by plastic surgeons, 5 pounds of fat can be removed with each procedure in an attempt to reshape thighs and waists resistant to more traditional weight-loss and exercise treatments. No advantageous metabolic changes are induced by the procedure.

## SUMMARY

No specific behavioral technique serves as the magic bullet for the very challenging, but medically important task of weight loss. Although many of the most common problems encountered in medical practice can be treated by weight loss alone, only motivated patients should be started on weight-loss programs. Weight-loss treatments vary considerably in terms of risk, cost, and efficacy. For most patients with mild or moderate obesity, a multifactorial approach, including diet, exercise, behavior modification, and social support, can be prescribed. Close patient-provider contact and long-term follow-up with emphasis on exercise are key ingredients for success. Motivated patients with severe obesity should be considered for supervised VLCDs, again emphasizing long-term dietary change, exercise, behavior modification, and social support. The role of medications for the treatment of obesity is increasing but should still be limited to selected, highly informed patients.

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## INTRODUCTION

Thirty-five percent of adults in the United States experience sleep-related symptoms over the course of a year; of this group, half consider the problem a serious one. These figures place insomnia and daytime sleepiness among the most common symptoms seen in primary care practice. Sleep disorders are more than just a large part of clinical practice, however; they represent a major public health problem. Sleepiness impairs work performance and is a major factor in traffic and industrial accidents; sleep-related breathing problems lead to hypertension, cardiovascular disease, and sudden death. In addition, sleep medications themselves carry significant morbidity risks, including falls, daytime anxiety, and worsened sleep apnea. Diagnosis is based on the duration and nature of the symptoms: depression, restless legs, periodic leg movements, substance abuse, circadian-rhythm disturbance, or poor sleep habits that perpetuate the insomnia. Treatment depends on the underlying disorder. Physicians clearly need to be knowledgeable about sleep to treat patients effectively and advocate sound public policy.

### Normal Sleep Physiology

**Adulthood:** The sleep-wake cycle is a complex electrophysiologic process consisting of alternating periods of wakefulness, non-REM (rapid eye movement) sleep, and REM sleep. Each of these periods has characteristic electroencephalogram (EEG), peripheral muscle, and autonomic nervous system patterns. These sleep periods can be documented with a polysomnographic recording (PSG), a procedure that allows sleep clinicians to make specific diagnoses based on electrophysiologic monitoring of EEG; electroculogram; electromyogram of submental and anterior tibialis muscles; respiratory muscles, nasal airflow, ear oximetry; and electrocardiogram.

Sleep has a structure, or architecture, that consists of four stages of non-REM and REM sleep cycles.

The awake EEG consists of low-voltage high-frequency waveforms that become dominated by alpha waveforms as the person becomes drowsy. Stage 1 sleep is defined by the disappearance of the alpha pattern and the establishment of theta waves and slow, roving eye movements. Stage 2 is defined by the appearance of low-frequency high-amplitude discharges and brief high-frequency variable-amplitude discharges on a background of theta waves. Slow waves (high-amplitude low-frequency delta waveforms) herald stage 3 sleep when they make up at least 20% of sleep time and stage 4 sleep when they constitute more than 50% of sleep time. These two stages are known as the deep stages of sleep, since they are associated with high arousal thresholds. REM sleep is a distinct state of sleep characterized by a wake-pattern EEG; skeletal muscle paralysis; and rapid, conjugate eye movements.

With the initiation of sleep, the healthy adult descends through the non-REM stages of sleep within 45–60 minutes before beginning the first REM cycle, which tends to be brief. As the night progresses, less time is spent in slow-wave sleep, and the REM cycles are longer in duration; overall, REM sleep makes up 20–25% of total sleep time. The non-REM–REM cycle typically lasts 90–110 minutes, so there are usually four complete cycles per night.

The timing and duration of sleep are controlled by many factors. Although most adults have some control over when they go to sleep and when they wake up, there is less control over how much sleep they need and when they feel sleepy. Although stimulants such as caffeine and habituation to a state of chronic fatigue can help people cope with inadequate sleep, they must ultimately pay the price of diminished mental efficiency and physical energy. The human range of sleep requirements is enormous, ranging from 3 to more than 12 hours, but the vast majority of people need 6–9 hours.

The body clock, located in the suprachiasmatic nucleus of the hypothalamus, functions as the circadian

pacemaker. It superimposes a rhythm of sleepiness and alertness to days and nights and determines whether specific persons are night owls, morning larks, or somewhere in between. The light-dark cycle and nightly rhythm of melatonin secretion by the pineal gland act synergistically to keep the body clock synchronized with the day-night cycle, allowing alertness during the day and sleepiness at night.

**Childhood:** Newborns spend about 70% of each day asleep, with a high proportion of REM sleep. Circadian patterns do not develop for at least several weeks—or even months. Sleeping through the night is one of the first maturational milestones. Sleep continues to be polyphasic, with daytime naps, until the child is 5 or 6. From ages 5–10, children are normally consummate sleepers with few arousals. Total sleep time decreases gradually though childhood, but for hormonal as well as psychosocial reasons, the quality and quantity of sleep drop sharply with puberty. Sleep can be erratic—brief on some nights and long “recovery” periods on others.

There has been some renewed interest in household arrangements for children's sleep. Anthropologists point out that isolating a newborn in a separate bedroom is almost unique to western cultures and that cosleeping with infants is more natural and may be beneficial. Sleeping alone is a desirable mark of independence, but at what age this should occur varies considerably, depending on the child and the family.

**Old Age:** As people age, sleep tends to be lighter, with more arousals and near disappearance of slow-wave sleep. Sleep onset and final awakening come earlier. Many people experience this change in early midlife; it may progress as they grow older, and waking at 4:00 in the morning is normal for many older persons. Severe insomnia also increases with age, and excessive daytime sleepiness is extremely common as well, possibly the result of a high prevalence of sleep apnea and periodic leg movements, both of which disrupt nighttime sleep.

## CLASSIFICATION OF SLEEP DISORDERS

Sleep disorders are generally grouped into three categories: disorders of initiating and maintaining sleep (insomnias), disorders of excessive daytime sleepiness (hypersomnias), and abnormal sleep behaviors (parasomnias) (Table 27-1).

Every clinician's review of systems should include screening patients for daytime sleepiness and nighttime sleep. Three basic questions give physicians a head start in diagnosing sleep disorders and determining whether they are severe enough to warrant treatment.

- How are you sleeping?
- How much sleep do you get in a typical night?
- How sleepy are you during the day?

**Table 27-1.** The sleepless patient: disorders of initiating or maintaining sleep.

Category	Disorder
Insomnias	Transient and persistent insomnia Chronobiologic disorders Restless legs syndrome Periodic leg movements Mental illness Alcohol and drugs Medical disorders affecting sleep
Hypersomnias	Sleep apnea syndrome Narcolepsy
Parasomnias	Pavor nocturnus (sleep terrors) Nightmares Somnambulism (sleepwalking) REM behavior disorder

Follow-up questions should sharpen the differential diagnosis of specific insomnia disorders.

### The Sleepless Patient: The Insomnias

**Transient and Persistent Insomnia:** These insomnias are among the most common complaints presented to the primary care clinician. It is best to think of insomnia as a symptom rather than a disease or diagnosis. Many factors combine to produce insomnia, which often occurs when a delicate balance is tipped; for example, a constitutionally light sleeper may be fine until he or she enters a period of stress or uses a medication (eg, aminophylline) that has alerting effects. It may be necessary to deal with several causative factors concurrently in order to restore natural sleep cycle.

Temporary insomnia caused by stress, environment (cold, noise, a new baby), acute illness, or pain is easy to identify and usually needs no special intervention. A brief course of sedative-hypnotic medication is occasionally warranted and may reduce the risk of developing long-term insomnia. Travel across time zones brings about a mismatch of the body clock and social clock and induces the transient insomnia known as “jet lag,” which can ruin the first few days of a trip. Shift workers whose shifts change experience the same phenomenon and can suffer severe health and social consequences because of it.

In a large multicenter study, the diagnosis of chronic insomnia accounted for 40–88% of patients in medical, psychiatric, and sleep clinics who presented with a primary complaint of insomnia. Chronic insomnia goes by many names: psychophysiologic insomnia, conditioned insomnia, learned insomnia, and primary insomnia. These terms all imply what is known about this condition: it is a chronic ailment that develops over time by operant conditioning to an arousal state that is incompatible with deep, sustained, restful sleep. Chronic insomnia is a diagnosis of exclusion and must be differentiated from the many other causes of persistent sleep disruption (see later discussion). It may develop after a period of sleep disruption caused by

stress, infant care, medical illness, or pain. Unfortunately, after the inciting cause disappears, the insomnia can take on a life of its own. Conditioning mechanisms have been inferred from the observation that patients may have difficulty initiating sleep in the bedroom but sleep normally in the sleep laboratory, a hotel, or even their own living room.

Long-term dependence on sedative-hypnotics is often one result of chronic insomnia, as is incessant sleep deprivation with daytime fatigue and dysphoria. Psychophysiologic insomnia requires a holistic approach that can include medication; sleep-hygiene measures (especially avoiding caffeine, alcohol, and excessive time in bed while awake); and cognitive therapy. Cognitive-behavioral treatment approaches are successful with motivated patients. Such treatment involves techniques to reduce anxiety and initiate behavioral changes that improve sleep hygiene (Table 27-2). Short-term use of sedative-hypnotic medications is appropriate to break the cycle of anxiety, arousal, and insomnia (see the section "Medical Treatment,"). In some patients, the symptoms suggest another disorder that can be targeted separately (eg, depression, restless legs syndrome).

Before initiating treatment, it is helpful to have the patient keep a sleep diary for 2 weeks, documenting sleep onset and wake-up times, rating both sleep quality and daytime fatigue and sleepiness. The sleep diary helps determine the frequency of insomnia and its daytime significance. Many patients report dissatisfaction with their sleep but still function well during the day. These patients should be reassured that nighttime wakefulness is a problem only if daytime somnolence results. Polysomnography is unenlightening for most cases of insomnia and should be reserved for the more refractory cases.

**Chronobiologic Disorders of the Sleep-Wake Cycle:** Disturbances in the circadian timing

of sleep may be only transient, as in jet lag and shift-work syndromes; or chronic, as in delayed-sleep-phase syndrome, advanced-sleep-phase syndrome, and free-running-sleep syndrome. The last is most common in blind persons, who lack the critical input of the light-dark cycle in regulating circadian rhythms. Diagnosis of chronobiologic sleep disorders is based on the understanding that—except for its timing—sleep itself is normal in these conditions. Given the freedom to choose sleep times based only on internal cues of sleepiness, persons with chronobiologic insomnias usually sleep well on weekends or while on vacation. A mismatch between the body clock and the hours during which a person tries to sleep, however, may cause insomnia. Successful treatment can require strategically timed exposure to light that is bright enough to reset the body clock and thus shift the timing of the sleep cycle. The timing of light exposure is critical, based on the fact that bright light in the morning advances the body clock and bright light in the evening delays it. Commercial light boxes are recommended for this purpose (see "Chronobiologic Treatment" later in this chapter). The pineal hormone melatonin, currently sold over the counter in health food stores as a food supplement, may achieve this same goal with greater convenience. For example, a person with delayed-sleep-phase syndrome might take synthetic melatonin (0.5–3 mg) early in the evening, at 8:00 or 9:00 PM—hours before endogenous melatonin secretion begins—to reset the body clock to an earlier hour.

**Case illustration 1:** Greg is a 27-year-old man who presents with a complaint of insomnia. He describes having great difficulty getting up in the morning, which is placing his job in jeopardy because he is chronically late to work. No matter what time he goes to bed, Greg cannot fall asleep until about 2:00 AM and then sleeps through the alarm set for 6:00. His wife has given up trying to wake him, and he has arranged for an answering service to call him, but the ringing telephone does not awaken him either. He is often tired and sleepy during the day, but in the evening gets his second wind just as his wife goes to bed. He craves weekends and vacations, when he sleeps until noon and feels rested.

Greg has delayed-sleep-phase syndrome. Persons with this disorder are extreme night owls—they lack the chronobiologic flexibility to adjust their sleep times according to school, work, and social demands. Origins of delayed-sleep-phase syndrome are probably multifactorial. Sleep timing is delayed, and there are surges of energy in the evening, late sleep onset, and severe morning hypersomnia.

This syndrome is extremely common in teenagers and young adults, in whom the insomnia complaint is usually prolonged sleep onset, with an inability to fall asleep until several hours past midnight. Parents or spouses may describe their frustration with getting the patient out of bed in the morning. Treatment can

**Table 27-2.** Cognitive-behavioral treatment for insomnia.

Essential Cognitive Techniques	Essential Behavioral Changes
Talk about the frustration of not falling asleep and put this into perspective; ie, "decatrophize" being awake at night.	Bed restriction (out of bed if awake for more than 30 minutes; no television, reading, or eating in bed); gradually increase time in bed as sleep improves.
Education about sleep requirements (not everyone needs 8 hours) and daytime napping (all right if midday and less than 1 hour).	No caffeine, alcohol, or nicotine late in the day.
Discuss patients' belief that they are defective in sleep and will always be poor sleepers; reframe concept in hopeful way.	Bright light exposure (especially in the morning) and exercise in afternoon.

involve bright light exposure in the morning (to phase-advance the body clock) and short-term use of a sedative-hypnotic medication to facilitate earlier sleep onset and to make getting up for bright light treatment manageable. Properly timed melatonin intake may also be helpful. Successful management calls for fairly strict adherence to a lifestyle that avoids late-night activity. This last requirement is difficult, especially for teenagers and young adults, with deviations from the early-to-bed schedule resulting in relapses into the delayed-sleep cycle.

**Restless Legs Syndrome (RLS):** This condition comes on with rest and produces an irresistible need to move, stretch, or rub the lower extremities. Severe dysesthesia, sometimes described as “creepy-crawly” sensations, aching, tension, tingling, or prickling, may occur. Most people with RLS also have periodic limb movements during sleep, leading to further sleep disruption. RLS can interfere with plane travel, desk work, reading, and, especially, sleep onset. The syndrome is common, affecting at least 5% of the population, and may be even more common in periodic or subclinical form. It sometimes first appears in females during pregnancy, can run in families, and tends to worsen with age. Many physicians do not think to ask about this symptom and hence fail to recognize it as a source of discomfort and insomnia for their patients.

There is no definitive treatment, although avoidance of caffeine and nicotine, moderate late-afternoon exercise, stretching before bedtime, opioids, L-dopa, and sedative-hypnotics may all be helpful. Carbamazepine and clonidine may also be effective. The syndrome is so distressful for some of its sufferers that there is now a national support group and newsletter (see “Resources” at the end of the chapter).

**Periodic Leg Movements During Sleep (PLMS):** These are repetitive myoclonic movements of the lower extremities that come in bursts lasting from a few seconds to many minutes; they are more common in, but are not limited to, the first half of the night. The movements are usually associated with brief arousals and can lead to nonrestorative sleep and daytime somnolence. The prevalence of PLMS increases with age—5% in people 30–50 years of age, 29% in those 50–65, and 44% in those over 65—and is often seen in metabolic and neurodegenerative diseases. PLMS should be distinguished from nocturnal leg cramps, which are painful, prolonged muscle spasms in the legs; these cramps are often treated with quinine sulfate. Tricyclic antidepressants, lithium carbonate, and withdrawal from benzodiazepines and alcohol can induce or worsen PLMS. It is often asymptomatic, but in its severe form, patients may have sleep-onset insomnia, nonrestorative sleep, or frequent arousals during the night from the more robust myoclonic movements. Not infrequently, the bed partner is the one complaining about the jerking leg movements at night.

Whereas RLS is primarily a symptomatic diagnosis, PLMS is best documented by polysomnography at home or in a sleep laboratory. A PLMS index greater than 5 (muscle jerks per hour) is considered definitive. PLMS responds to some of the same treatments as RLS. Benzodiazepine sedative-hypnotics can improve sleep continuity in PLMS patients, but it does not reduce the number of leg movements. L-dopa may be the treatment of choice, but rebound in the second half of the night or during the following day may make dosing a challenge. Opioids, such as a bedtime dose of codeine, work well in PLMS (as in RLS) but should be reserved for patients with severe symptoms.

**Mental Illness and Insomnia:** Sleep disturbances are among the most common symptoms of mental illnesses, particularly mood disorders. Major depression must always be considered in patients who complain of frequent nighttime awakenings and early morning arousal, particularly when those arousals are accompanied by anxiety and worry. On the other hand, many depressed patients complain of hypersomnia, with fatigue and difficulty waking and getting started in the morning. This change in sleep pattern is especially characteristic of seasonal affective disorder and so-called atypical depression. Both are rather common in young and middle-aged adults.

Mania is frequently accompanied by a reduced need for sleep, making a change in sleep patterns a cardinal diagnostic symptom of the disorder; patients usually do not complain about this change, however. Depressed patients, on the other hand, find their sleep changes very distressing. Sedating, antidepressant medication is the treatment of choice. Doxepin, nortriptyline, trazodone, or nefazodone are all reasonable choices for treating insomnia in depressed patients. Nonsedating antidepressants can also be used, in conjunction with a brief course of a sedative-hypnotic drug for temporary help with sleep. The benzodiazepine can be stopped as the insomnia improves with the resolution of the depression (see Chapter 22).

Severe anxiety disorders, including posttraumatic stress disorder, can present with severe insomnia. Nightmares frequently complicate the picture. Treatment is often challenging and may require intensive psychotherapy as well as polypharmacy. Antidepressant medication and clonidine may be helpful with the sleep-related symptoms. (see Chapter 23).

Bereavement is usually accompanied by anxiety and insomnia. Short-term use of benzodiazepines may be very helpful for patients struggling to get through long nights.

**Case illustration 1:** Francine is 47 years old and is seeking her physician's help in dealing with anxiety. She feels restless and fidgety during the day, but she is also tired. She has difficulty concentrating, making decisions, and getting things done, all of which are out of character for her. Although quite fatigued, it can take her an hour to fall asleep. When sleep does come, it is rest-

less and interrupted by many awakenings, filled with worried thoughts.

Francine has major depression, presenting with prominent symptoms of anxiety and insomnia. All patients with these symptoms need to be screened for depression, with a few questions regarding their mood, sense of the future, appetite, and libido. Education, emotional support, and medical treatment with antidepressant medication, perhaps with short-term adjunctive use of a sedative-hypnotic, provide enormous benefit for most patients.

For example, treatment for Francine might start with either a selective serotonin reuptake inhibitor (SSRI) such as sertraline (50 mg every morning) or one of the nonspecific reuptake inhibitors, such as venlafaxine (25 mg three times a day with meals) or nefazodone (100 mg twice a day), with dosage adjustments according to clinical response. For the first few weeks of therapy, a sedative-hypnotic (temazepam, 15 mg at each bedtime, or zolpidem, 5 or 10 mg at each bedtime) should also be prescribed to facilitate sleep until the underlying depression has improved. Some patients get sleepy while taking nefazodone and do not need additional medication for sleep. Fluoxetine and nefazodone can slow the clearance of alprazolam and triazolam, so these drugs should be coadministered with care, although a longer half-life for these benzodiazepines can be beneficial in some cases, such as in patients with end-of-dose rebound insomnia.

**Alcohol and Drugs:** Alcohol has variable effects on sleep patterns, but it generally causes decreased alertness. Like other sedatives, however, alcohol suppresses slow-wave sleep, making sleep lighter. With its short half-life, alcohol also tends to produce a rebound arousal effect in the second half of the night and thus produces less sleep overall.

Other drugs also bring about characteristic sleep changes. Amphetamines and cocaine cause a marked reduction in sleep during acute intoxication and profound hypersomnia during the withdrawal phase. Opioids have variable effects on sleep; they can, of course, improve sleep when used for analgesia. Caffeine causes longer sleep latency (time needed to fall asleep) and increased wakefulness during the night. Some persons may not perceive the effects of caffeine on sleep even when they are documented on sleep EEG; others are very much aware of these effects. Caffeine can even affect sleep when ingested several hours before bedtime.

Both prescription and over-the-counter sedative-hypnotic medications can contribute to rebound cycles of insomnia in the latter part of the night and anxiety the next day. Daytime drowsiness and worsened sleep apnea are other potential problems. Confusion, ataxia, and unexplained falls should cue the physician to consider the presence of alcohol or sedative-hypnotic dependence, especially in elderly patients.

Education about the effects of these substances on sleep may motivate patients to reduce their intake, particularly at times when use may affect sleep. Patients with more severe dependency and abuse problems need referral to specific treatment programs (see Chapter 21).

**Medical Disorders:** Pain, rheumatologic disorders, neuromuscular disease, cardiac disease, pulmonary disease, dyspepsia, inflammatory bowel diseases, and nocturia are all common medical causes of insomnia. Another classic medical syndrome affecting sleep is fibrositis, a condition in which sleep is characterized by alpha-wave intrusion into non-REM sleep. This syndrome produces nonrestorative sleep, in which patients complain of feeling tired despite sleep duration within the normal range. Acquired immunodeficiency syndrome (AIDS) has been associated with daytime sleepiness; a decrease in total slow-wave sleep, with alpha-wave intrusions; increasing arousals; night sweats; and frequent nightmares. Patients with chronic disease are often desperate for good sleep, making adequate nighttime analgesia and sedating antidepressants very welcome.

Acute illnesses often cause diffuse cerebral dysfunction in the frail elderly. The resulting delirium is almost always accompanied by disruption of the sleep-wake cycle and alertness. In many patients, the confusion and sleepiness of delirium are the first clues of illness.

Medications can also cause insomnia and daytime drowsiness. Bronchodilators, activating antidepressants, and steroids, for example, often interfere with sleep; many psychotropics, opioid analgesics, and clonidine can cause daytime drowsiness.

**Neurodegenerative Disease and Sleep:** There is no localized sleep center in the brain; rather, there are several neuronal circuits that function in maintaining sleep or alertness. Diseases that affect diffuse brain functions invariably affect sleep and alertness; Alzheimer's disease (AD) and Parkinson's disease (PD) have been studied more than most others. AD causes the same kinds of changes in sleep as normal aging does, but they are more severe: less clear day-night difference with more daytime and less nighttime sleep. The classic day-night reversal is probably rare, but the trend to many nighttime awakenings is common. This is very stressful for caregivers, who must continue to supervise their charges for safety during these nocturnal wanderings. Sleep disruption is among the most stressful aspects of caring for a demented person at home.

PD patients also have severe sleep problems. Akinesia causes physical discomfort over pressure points that would, in a healthy person, be relieved by tossing and turning in sleep. Parkinsonian medication itself can cause arousal. Furthermore, the intrinsic neurotransmitter and neurodegenerative changes PD causes in the neuronal circuits that are important for sleep can also induce insomnia.

### The Sleepy Patient: Disorders of Excessive Somnolence

Patients are more likely to complain to physicians about insomnia than about hypersomnia, or excessive daytime sleepiness. This is rarely a primary complaint of patients, and uncovering symptoms of excessive daytime sleepiness may take some intuitive detective work on the physician's part. Two questions that can lead to the critical diagnosis of sleep apnea or narcolepsy should be part of every review of systems:

- Do you struggle to stay awake while driving, reading, watching television and movies, or listening to lectures during daytime hours?
- Do you feel tired, fatigued, and lacking in energy during the day, especially in the morning?

If the answer to either question is yes, follow-up questions should be directed at determining if the problem is inadequate nighttime sleep from insomnia, drowsiness from medications, or one of the two serious sleep disorders described in the following section.

#### Sleep-Related Breathing Problems

**1. Snoring**—Apart from being a nuisance to bed partners, snoring may herald serious respiratory obstruction during sleep along a continuum of partial to complete airway closure. Males snore more than females do, although postmenopausal females snore almost as much as men do. Aside from gender, other factors associated with snoring include anatomic narrowing of the airways, body habitus and sleep position (obese, supine), the use of alcohol and sedative-hypnotics, endocrinopathy (hypothyroidism, acromegaly), smoking, and possibly genetic factors. The view of snoring as a mild form of obstructive sleep apnea is supported by the transient drop in blood oxygen saturation and increases in pulmonary and systemic pressure that can occur in snoring. Losing weight, avoiding sedating medications or alcohol, and using appliances to prevent sleeping on the back (eg, a tennis ball sewn to the back of the nightshirt) are warranted in severe cases. Laser surgery to enlarge the oropharynx is increasingly popular, but the long-term effects are not well documented. Because surgery can eliminate the noise of snoring without affecting an associated obstruction, PSG evaluation prior to an operation should be performed to rule out obstructive sleep apnea.

**2. Sleep apnea syndrome**—Obstructive sleep apnea (OSA) is a major cause of cardiovascular morbidity and daytime somnolence in adults. Originally thought of as a relatively rare disturbance in severely obese patients with the classic "Pickwickian syndrome" of somnolence, hypoventilation, and polycythemia, OSA is now known to represent a wide range of severity in upper airway narrowing during sleep that begins earlier in life and is more prevalent than previously thought. In midlife, 2% of women and

4% of men have OSA, with serious daytime sequelae. Nighttime symptoms of OSA include loud snoring (often beginning early in adulthood and worsening progressively with age and increased weight), snorting and gagging sounds, tossing and turning, night sweats, abrupt awakenings with a feeling of choking, and profound sleep disruption. The arousals triggered by the apneic episodes cause such daytime symptoms as fatigue and excessive sleepiness. In mild cases, subjective insomnia may be the chief complaint. Patients are often unaware of the severity of the sleep disruption and may attribute their sleepiness to some other cause, such as working hard. The degree of sleepiness is variable, but it is a key symptom. People may be so accustomed to living with fatigue that they are not fully aware how sleepy they are. Questions about dozing while reading or watching television, nodding off at the wheel, or poor concentration must be directly posed to patients. Untreated OSA leads to hypertension and is probably a significant risk factor for lethal cardiovascular events, including myocardial infarction and stroke. Clinical assessment by PSG either at home or in a sleep laboratory confirms the diagnosis and helps determine the proper setting for positive airway pressure devices, which are the major form of treatment. Surgical uvuloplasty, tracheostomy, and dental appliances designed to keep the tongue from falling back and occluding the airway are other forms of treatment available for those who cannot tolerate continuous or two-stage positive airway pressure therapy (CPAP or bilevel positive airway pressure [BIPAP]). The diagnosis of OSA is an important one to make in terms of both improving the patient's quality of life and preventing serious accidents and cardiovascular disease.

Obstructive apneas also occur in children. Enlarged tonsils and adenoids are usually responsible, but craniofacial abnormalities and obesity can be the underlying causes. As in adults, loud snoring, restless sleep, and observed pauses in breathing make up the picture. Children with obstructive apnea may not complain of sleepiness; instead, they may manifest daytime irritability, decreased concentration, or declining school performance. Some children have even been misidentified as intellectually impaired. Nocturnal enuresis may be another symptom of OSA in children. Parents should be asked about snoring and breath-holding during sleep. Consultation with an ear, nose, and throat specialist is advised whenever OSA is suspected. Tonsillectomy (if the underlying cause) is usually completely curative. Other causes of obstructive apnea can be treated with CPAP. Surgical approaches such as tracheostomy or craniofacial reconstruction may be necessary in rare cases.

**Central apnea** is defined as the cessation of airflow for at least 10 seconds with no ventilatory effort. Patients with predominantly central apnea tend to complain more of insomnia with frequent awakenings than of the hypersomnolence that is so typical of pa-



tients with obstructive apnea. Arterial oxygen desaturation usually occurs in central apneas, but serious cardiovascular sequelae are less common than in obstructive apnea. Predisposing factors to central sleep apnea are congestive heart failure (mechanism unknown), nasal obstruction, and neurodegenerative diseases that can affect the central nervous system's respiratory control or induce profound hypoventilation from respiratory muscle weakness.

Central apneas are often seen in sleeping neonates, especially premature infants and can be fatal (eg, sudden infant death syndrome [SIDS]). The cause of SIDS remains a tragic mystery; obstructed breathing has long been suspected as an important etiology in at least some cases, but it is far from proven. There is currently an ongoing public information campaign to encourage mothers to avoid placing infants face down (prone), especially in soft bedding. Epidemiologic assessments will determine whether this public health intervention is effective.

*Case illustration 3:* Jim, who is 64, visits his primary care physician for a follow-up of his hypertension treatment. His wife has accompanied him to the office to ask whether there is any medical explanation for her husband's fatigue. Close questioning reveals that the fatigue predates the hypertension treatment and is not clearly attributable to the medication. The tiredness is accompanied by true sleepiness; Jim can fall asleep anytime during the day while reading or driving. He minimizes the problem, yet he acknowledges having difficulty with concentration and memory. He falls asleep easily after getting into bed at night, but his wife describes him as a restless sleeper who snores loudly.

Jim has obstructive sleep apnea. The clinical clues are his snoring, daytime sleepiness, and hypertension. Referral to a sleep disorders specialist should help confirm the diagnosis and provide a review of the best treatment options.

**Narcolepsy:** Narcolepsy is a syndrome consisting of four primary symptoms: excessive daytime sleepiness, cataplexy and, less frequently, sleep paralysis and hypnagogic hallucinations. It occurs in approximately 1 in 2000 people. Narcolepsy often begins in the teens or early twenties but mid- and late-life onset can occur. Males are affected more commonly than are females. A formal diagnosis is frequently not made until 5 or 10 years after the onset of symptoms. If the syndrome is not diagnosed, patients are typically perceived as lazy and unmotivated. Genetic factors play an important role in the disorder, with at least two genes involved, one of them HLA (human leukocyte antigen)-related. The cardinal symptom is sleepiness that comes on suddenly and irresistibly in what are called "sleep attacks." Low-grade, persistent sleepiness that affects concentration, thinking, and memory may also occur. Sleep episodes may be brief (several minutes to an hour), but the person usually awakens feeling more alert, and the next sleep episode usually does not come on for at least an hour. When unrecog-

nized, the disorder leads to poor school and work performance, social stigma, and accidents.

**Cataplexy**, the brief, sudden loss of muscle tone leading to slurred speech, clumsiness, leg weakness, or complete collapse, is triggered by strong emotional reactions such as laughter or anger. **Sleep paralysis** is transient immobility on awakening, often accompanied by the vivid hallucinations of REM dreaming, all while the patient is lying in bed perfectly alert. The spells are brief, lasting several minutes at most. Narcolepsy can impair nighttime sleep with frequent awakenings, vivid nightmares, and intense, realistic (hypnagogic) hallucinations that precede sleep onset. The hallucinations are usually visual but can involve any sensory modality.

Patients should have a formal evaluation by a sleep disorders specialist before treatment for narcolepsy is initiated. The diagnosis usually requires documentation of sleep-onset REM by PSG. Excessive daytime somnolence is treated with scheduled daytime naps and medication. CNS stimulants, most commonly dextroamphetamine (5–60 mg a day), methamphetamine (20–25 mg/day) or methylphenidate (10–90 mg/day), improve daytime function. Cataplexy and sleep paralysis can be treated with REM-suppressant drugs such as tricyclic antidepressants. Joining a narcolepsy support group can help patients cope with the disorder's psychologic sequelae, which result from the social and occupational stigma of having little control over sleep onset.

### Patients With Bizarre Nighttime Behavior: The Parasomnias

Parasomnias are perhaps the least common sleep disorders and their diagnosis can be difficult, although they should be considered whenever bizarre nighttime behavior is present.

**Pavor Nocturnus:** Sleep terrors (pavor nocturnus) are very disconcerting to parents but are usually quite benign. The child (usually aged 3–6) awakens with a scream and appears terrified, with signs of autonomic arousal: eyes bulging, heart racing, sweating. Although episodes usually last a few minutes, they can go on for half an hour. Attempts at comfort are to no avail. Finally, the child falls asleep. In the morning, the child is typically amnesic about the episode or may have a fragmentary memory of a bad dream. Sleep terrors involve partial arousals from stage 4 (deep) sleep. Reassurance of the parents is the usual treatment; in persistent night terrors, however, benzodiazepines may be justified.

**Nightmares:** True nightmares occur in REM sleep and involve a narrative story people can often relate. Nightmares are usually a transient problem, presumably triggered by stressful personal events. Persistent nightmares are a serious concern, however, and may require referral to a mental health specialist.

**Somnambulism:** Like sleep terrors, sleepwalking is a partial arousal from stage 4 sleep. Occasional

sleepwalking is very common in childhood and may follow a period of stress or sleep deprivation. The main concern is accidental injury, and protective measures, such as placing gates in front of a stairwell, may need to be taken.

**REM-Behavior Disorder (RBD):** In this syndrome, loss of the normal REM sleep muscle atonia leads to dream-enactment behavior. The diagnosis is made in patients with sudden bursts of excited, intense, sometimes violent, activity during sleep. The syndrome may be subtle, in the form of leg movements and talking, or dramatic, in the form of punching, kicking, grabbing, strangling, running, and moving about the bedroom. Dreams of an intense, violent nature are typical. RBD is seen frequently in toxic or metabolic delirium, but most persistent forms of the syndrome occur in old age and are presumed to be idiopathic, ischemic, or neurodegenerative in etiology. Lesions or dysfunction in the pons are responsible for the loss of inhibition to spinal motor-neurons. Treatment of RBD with clonazepam is highly effective.

## TREATMENT

Managed health-care systems vary considerably in their coverage of sleep disorder evaluations. The routine use of expensive polysomnographic evaluations for most cases of insomnia is unwarranted and is appropriately limited by most insurance plans. Nonetheless, the timely and accurate diagnosis of sleep apnea syndrome and other severe sleep disorders can have powerful effects in maintaining health, and physicians should advocate for their coverage. The optimal assessment and treatment of sleep disorders can also decrease unnecessary office and emergency department visits and reduce drug use.

### Medical Treatment

Treating insomnia with medication should involve specific target goals, such as shorter sleep latency, delayed wake-up, or fewer nocturnal awakenings. Insomnia of recent onset should be treated with the expectation that short-term therapy will be effective. Sedative-hypnotic drugs should be used in conjunction with a sleep-hygiene program to maximize efficacy and reduce both the dosage and the duration of treatment (see Table 27-1). These medications should not be used in pregnancy. Patients with insomnia secondary to depression, pain, substance abuse, medication, or circadian-rhythm disorder should also receive treatment for the primary cause of the insomnia.

**Benzodiazepines:** These are the most commonly prescribed sleeping medications.

**1. Efficacy and safety—**All benzodiazepines have sleep-promoting effects, although only five are currently marketed as sedative-hypnotics. These drugs work well for short-term treatment of insomnia; tolerance to their sleep-promoting effects can develop

quickly, and some authorities recommend avoiding long-term use. Some patients, however, especially those with an anxiety component to their insomnia, may benefit from long-term use. Benzodiazepines alter sleep structure, reducing both REM and slow-wave sleep, but the clinical significance of this is uncertain. They are generally safe for younger adults, even in overdose, although combining them with alcohol and other depressants can produce potentially catastrophic synergistic effects. In older individuals, the safety profile is less benign; amnesia, ataxia, confusion, and worsening sleep apnea may develop.

**2. Pharmacokinetics—**Choosing a benzodiazepine on the basis of half-life involves ranking the target goals. Short-acting drugs such as triazolam are useful for the treatment of sleep-onset insomnia, but many individuals have rebound insomnia in the second half of the night or anxiety the following day. Longer-acting drugs such as flurazepam may work better for middle-of-the night insomnia, but some persons have morning hangover effects. Longer-acting drugs can be particularly troublesome in the elderly, in whom drug accumulation magnifies toxic effects. Temazepam and estazolam have an intermediate half-life and represent reasonable compromises for patients with sleep-maintenance insomnia who get hangover effects from longer-acting drugs.

Patients with sleep apnea, severe respiratory disease, gait and balance problems, or alcohol abuse should not be given these drugs. Doses should be kept low for the elderly and for patients with hepatic insufficiency. Rebound insomnia can complicate withdrawal from these drugs, causing patients to return to their use.

**Zolpidem:** An imidazopyridine, this drug is structurally unrelated to benzodiazepines but shares some characteristics with them. Its short half-life (1.4–3.8 hours) makes it most suitable for patients with sleep-onset or initial sleep maintenance problems. Zolpidem preserves the natural sleep architecture, providing at least a theoretical advantage. The dose is 5–10 mg at bedtime, and patients should be cautioned to get into bed shortly after taking the drug because of its quick onset. Precautions similar to those for the benzodiazepines apply to zolpidem, although tolerance and withdrawal problems may be less likely.

**Sedating Antidepressants:** Many clinicians believe drugs such as trazodone, nefazodone, doxepin, and amitriptyline are better choices for long-term treatment of severe insomnia than are benzodiazepines, although this has not been well studied. The theoretic advantages include less impairment of nighttime breathing and slow-wave sleep. Treatment of underlying depression in many insomnia patients provides another advantage. Tolerance to the sedating effects of these drugs develops in many patients, but some clinicians believe this is slower to develop than with benzodiazepines. Side effects of these drugs are

numerous, and special care must be taken in elderly patients, especially with amitriptyline and its potent anticholinergic effects.

**Barbiturates:** Similar to benzodiazepines in mechanism and efficacy, these drugs are extremely dangerous in overdose, and tolerance develops quickly.

**Antihistamines:** Diphenhydramine is sedating and is found in many over-the-counter preparations. It is generally safe and effective for short-term use, although tolerance develops very quickly after nightly ingestion. Diphenhydramine has some anticholinergic properties and can cause confusion and urinary retention in elderly persons.

**Alternative Substances:** Since L-tryptophan was taken off the market because some preparations were found to cause eosinophilic myalgia syndrome, various homeopathic and folk remedies have become popular. Teas and capsules containing valerian root extracts may be the most effective of these alternative substances, but safety and efficacy studies have not been done. The pineal hormone melatonin, which is secreted at night in decreasing amounts as people age, clearly has sleep-promoting effects in many people. It is currently under intensive investigation. Although considered an experimental drug by the FDA, it is marketed as a food supplement at health food stores and has attracted a large following. Patients should be cautioned that melatonin remains an experimental drug and is a naturally occurring hormone with potential neuroendocrine, immunologic, and reproductive effects, although it appears to be remarkably benign in short-term use. Commercial preparations may contain 0.5–5 mg of melatonin per capsule (sometimes in combination with vitamins). The most effective dose is unknown and may vary from person to person. Doses in the 0.5–10 mg range are reasonable, although doses above 0.5 mg produce plasma levels greater than would occur naturally and thus engender more concern as to long-term effects. Preparations containing “pineal extracts” should be avoided in favor of synthetic melatonin.

### Chronobiologic Treatment

Sleep-wake-cycle disorders can be treated with scheduled exposure to bright natural or artificial light. Patients with advanced-sleep-phase syndrome need a corrective phase delay with exposure to bright light in the evening. Bright light exposure must be carefully timed so that the circadian pacemaker is phase-shifted to move sleep propensity to a later time. For the more common delayed-sleep-phase syndrome, patients need to force themselves awake in the early morning (between 6:00 and 7:00 AM) to receive appropriately timed light exposure. The first few mornings are very difficult, but after several 30- to 60-minute light exposures, patients can begin falling asleep before midnight and wake up in time for morning classes or work. Light fixtures for treating these syndromes are available from numerous commercial vendors. The

Society for Bright Light and Biological Rhythms (see “Resources” at the end of the chapter) can provide a list of vendors and more details about using bright light exposure in treating sleep disorders and winter depression. Melatonin may also be useful in treating these disorders; this remains an experimental modality for the time being.

### Psychosocial Treatment

Sleep problems in children and adolescents usually affect the rest of the family, causing sleep loss in parents and siblings who may suffer—in some respects—as much as the patient. Because misinformation and inappropriate blaming may confound the problem, the disturbed sleep of such patients needs to be addressed as a problem for the whole family.

Modifying family routines may be helpful. Good sleep hygiene, including well-maintained bedtime rituals such as bathing, story-telling, and rocking a small child can facilitate the winding-down process that is an important prelude to sleep. Occasionally a child becomes overdependent on a particular routine (eg, repeated drinks of water every time he or she wakes up), and parents must set limits. After an expected period of protest, most children relinquish unnecessary attention. These benign disruptions must be differentiated from the more serious panic that some children experience with separation. For this latter kind of anxiety, parental access through the night may be necessary, at least for a time.

In adolescence, sleep is often shortened at both ends. In the evening, there are the demands of homework, telephone socializing, and family life. In the morning, high school schedules often begin quite early, sometimes preceded by an even earlier bus ride. For many teenagers, the morning includes a formidable grooming routine. Add to this the increasing tendency for teenagers to take part-time jobs after school, and the result is an epidemic of chronic sleep deprivation that is an increasing societal concern. Weekend sleeping may recover some of the lost sleep, but it tends to produce a phase delay in circadian rhythms that makes it more difficult to fall asleep during the week. In one experiment, high school students increased their IQ scores by 20 points after a week in which they systematically extended their sleep times.

In counseling teenagers, some flexibility and compromise are usually most effective. Adding naps during the day may improve alertness. A warning about the dangers of driving while sleepy, intoxicated—or both—is important. Chronobiologic interventions, such as light therapy, may be needed to counteract extremely delayed sleep. Outside the office, informed and politically active physicians may be able to influence public policy to help alleviate the problem, such as adopting sensible work rules for teens and scheduling school activities at reasonable hours.

Adults with sleep complaints need to feel that their health-care providers take the problem seriously and

understand the effect the disorder has on their lives. At the same time, clinicians can reassure the insomnia patient without severe daytime sleepiness that the problem of nighttime awakenings is more a nuisance than a serious health problem. Educating patients about appropriate sleep hygiene and cognitive measures helps them regain some sense of control over their symptoms (see Table 27-2). Persons with more persistent insomnia or those who appear to have severe psychologic distress as a result—or cause—of the sleep disturbance may warrant evaluation by a mental health specialist.

Sleep disorders in adult patients can, of course, affect others. Partners or caregivers of patients with severe sleep disorders may need both emotional support and education about the nature of the sleep disturbance. Understanding the problem can help them reduce their frustration and enable them to support the patient in following treatment recommendations.

## Resources

The National Sleep Foundation, at 1221 Robertson Boulevard, third floor, Los Angeles, CA 90048, publishes an outstanding newsletter for physicians and other health-care providers.

The RLS Foundation, at 1904 Banbury Road, Raleigh, NC 27608, offers support, including a newsletter, for persons with RLS.

The Society for Bright Light and Biological Rhythms, at 10200 West 44th, Suite 304, Wheat Ridge, CO 80033 [Telephone (303) 424-3697; fax (303) 422-8894], offers information on treating chronobiologic sleep disorders and seasonal affective disorder. Names of vendors of clinical bright light fixtures are also available through the organization.

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## INTRODUCTION

Cigarette smoking is the leading preventable cause of death in the United States, responsible for an estimated 419,000 deaths per year, or one in every five deaths. Physicians must take care of the health consequences of their patients' tobacco use. It is equally important for them to prevent smoking-related disease by treating their patients' smoking habits.

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## PATTERNS OF TOBACCO USE

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Cigarette smoking in the United States and its health consequences are largely 20th-century phenomena. Smoking was uncommon before 1900, rose rapidly in the first half of the century, and peaked in 1965, when 40% of adult Americans smoked cigarettes. Since then, smoking rates have declined, reflecting growing public awareness of the health risks of tobacco use. By 1994, adult smoking prevalence had fallen to 25.5%. This decline is primarily attributable to smoking cessation. Smoking initiation rates have not decreased since 1980, despite a concurrent fall in smoking prevalence. In both sexes, smoking starts during childhood and adolescence; 90% of smokers begin to smoke before the age of 20.

These aggregate data conceal dramatic differences in the smoking patterns of men and women that have led to substantial gender differences in smoking-related disease mortality. American women did not take up smoking in large numbers until World War II, three decades later than men. When smoking prevalence

peaked in 1965, only 32% of adult women smoked, compared with 50% of men. After 1965, smoking rates fell four times faster in men than in women. The result has been a convergence in the smoking rates of men and women. By 1994, 23.1% of adult women and 28.2% of men smoked cigarettes.

In the United States, smoking is more closely linked to education than it is to age, race, occupation, or any other sociodemographic factor. Educational attainment is a marker for socioeconomic status, and these data indicate that smoking is a problem that is becoming concentrated in lower socioeconomic groups.

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## HEALTH CONSEQUENCES OF TOBACCO USE

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Cigarette smoking increases the overall mortality and morbidity rates of both men and women. In both sexes, smoking is a cause of cardiovascular disease (including myocardial infarction and sudden death); cerebrovascular disease; peripheral vascular disease; chronic obstructive pulmonary disease; and cancers of the lung, larynx, oral cavity, and esophagus.

Lung cancer, once a rare disease, has increased dramatically. Since 1955, it has been the leading cause of cancer death in men, and by 1986, lung cancer surpassed breast cancer to become the leading cause of cancer death in women. Cigarette smokers also have higher rates of cancers of the bladder, pancreas, kidney, stomach, and uterine cervix. Smoking interacts with alcohol to increase the risk of laryngeal, oral cavity, and esophageal cancers. Tobacco also interacts with occupational exposures such as from asbestos to greatly increase cancer risk.

Smoking is associated with many pregnancy complications. It is a cause of low birth weight (<2500 g) in infants. Babies born to smokers weigh, on average, 200 g (7 oz) less than the babies of nonsmoking moth-

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Selected portions of this chapter are based on material written by the author and Lela Polivogianis, MD, for a chapter entitled, "Smoking Cessation" in: *Primary Care of Women*. Carlson et al (editors). Mosby-Year Book, Inc, 1995.

ers. This is primarily attributable to intrauterine growth retardation (IUGR), although smoking in pregnancy also increases the risk of preterm delivery. Smoking is the major known cause of IUGR in the developed world, responsible for an estimated 30% of cases. Other adverse pregnancy outcomes linked to smoking are miscarriage (spontaneous abortion), stillbirth, and neonatal death. Placenta previa, abruptio placenta, and bleeding occur more often in smokers than nonsmokers, suggesting that smoking impairs placental function.

Smoking during pregnancy affects children even after birth. Sudden infant death syndrome is two to four times more common in infants born to mothers who smoked during pregnancy. Cognitive deficits and developmental problems in childhood have also been linked to maternal smoking during pregnancy, although the relationships are not clearly causal. The adverse health effects of smoking extend to reproductive function before pregnancy. Smoking has been associated with reduced fertility in both men and women, though a causal link remains to be established. Smoking also increases the risk of serious cardiovascular disease among women using oral contraceptives. Compared with nonsmokers, women smokers who also use oral contraceptives have a substantially increased risk of myocardial infarction, subarachnoid hemorrhage, and stroke.

There is debate about whether smoking increases a woman's risk of developing postmenopausal osteoporosis. Some, but not all, studies link smoking with reduced bone mass and osteoporosis. There is also conflicting evidence about whether smokers have a higher risk of osteoporotic fracture than nonsmokers. Women who smoke are thinner and have menopause 1–2 years earlier than nonsmokers, both of which are risk factors for osteoporosis. Smoking may also have an antiestrogen effect.

Smokers have higher rates of peptic ulcer disease, poorer ulcer healing, and higher recurrence rates than nonsmokers. They are more susceptible to upper respiratory infections and have more cataracts than nonsmokers. Smokers have more prominent skin wrinkling than nonsmokers, an association independent of sun exposure. The majority of residential fire deaths are caused by smoking.

There is no safe level of tobacco use. Smoking as few as one to four cigarettes per day increases the risk of myocardial infarction and cardiovascular mortality. Smoking cigarettes with reduced tar and nicotine content does not protect against the health hazards of smoking.

The health hazards of smoking are not limited to those suffered by smokers. Nonsmokers are harmed by chronic exposure to environmental tobacco smoke (ETS). The children of parents who smoke have more serious respiratory infections during infancy and childhood, more respiratory symptoms, and a higher rate of chronic otitis media and asthma than the children of nonsmokers. Nonsmoking women whose hus-

bands smoke have a higher lung cancer risk than nonsmoking women whose husbands do not smoke. A 1993 Environmental Protection Agency report identified ETS as a source of carcinogens, responsible for approximately 3000 lung cancer deaths per year in US nonsmokers. Passive smoke exposure also appears to increase nonsmokers' risk of coronary heart disease.

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## HEALTH BENEFITS OF SMOKING CESSATION

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Epidemiologic data demonstrate that smoking cessation has health benefits for men and women of all ages. Even those who stop smoking after the age of 65 or who quit after the development of a smoking-related disease derive benefit. Smoking cessation decreases the risk of lung cancer and other cancers, heart attack, stroke, chronic lung disease, and peptic ulcer disease. After 10–15 years of abstinence, overall mortality rates for smokers approaches that of those who never smoked. The cardiovascular risk reduction occurs more rapidly than the risk reduction for lung cancer or overall mortality. Half of the excess risk of cardiovascular mortality is eliminated in the first year of quitting, whereas for lung cancer, 30–50% of the excess risk is still evident 10 years after quitting and some excess risk remains after 15 years.

The benefits of stopping smoking translate into a longer life expectancy for former smokers, compared with continuing smokers. The degree to which former smokers benefit from cessation depends on their previous lifetime dose of tobacco, their health status at the time of quitting, and the elapsed time since quitting. Smokers who benefit the most are those who quit when they are younger, have fewer pack-years of tobacco exposure, and are free of smoking-related disease. The health benefits of smoking cessation far exceed any risks from the small weight gain that occurs with cessation. The benefits of smoking cessation were summarized in the *1990 Surgeon General's Report on Smoking*.

Smoking cessation reverses the hazards of smoking to the fetus. Women who stop smoking before pregnancy or during the first 3–4 months of gestation have infants who weigh the same as those born to women who never smoked. Pregnant smokers who stop smoking at any time up to the 30th week of gestation have infants with higher birth weights than women who smoke throughout pregnancy. Reducing daily cigarette consumption without quitting smoking is of less benefit in preventing low birth weight.

## SMOKING CESSATION

Approximately half of living Americans who ever smoked have quit smoking. According to surveys, a

majority of the remaining smokers would like to stop smoking and have made at least one serious attempt to do so.

Surveys of former smokers reveal how and why smokers stop smoking. The reason most often cited by former smokers for stopping smoking is fear of illness. Awareness of health risks is not sufficient to motivate smoking cessation, however. Over 90% of current smokers know that smoking is harmful to health, yet they continue to smoke. Many smokers rationalize that they are immune to the health risks of smoking until these risks become personally salient. Current symptoms (eg, cough, breathlessness, chest pain), even if they represent minor illness rather than the onset of a smoking-related disease, stimulate change in smoking behavior more powerfully than does fear of future disease. Illness in a family member may also motivate smoking cessation. Another frequently cited reason for quitting is growing social pressure from work, family, and friends not to smoke.

In surveys, 90% of former smokers say that they quit on their own. Most quit abruptly ("cold turkey"), although smokers can progressively reduce daily cigarette intake in preparation for quitting. A variety of programs are available to smokers who seek help. The best evidence for efficacy supports behavior modification programs; hypnosis is poorly evaluated, and acupuncture is not effective for smoking cessation.

The majority of former smokers did not succeed in stopping on their first try. About 30% of smokers who attend formal programs are not smoking 1 year later; most resume smoking within 3 months. Behavioral scientists liken smoking cessation to a learning process rather than to a discrete episode of will power. Smokers learn from mistakes made during a prior attempt at quitting, thereby increasing the likelihood that the next attempt will succeed. Psychologists have identified a series of cognitive stages through which smokers pass as they move toward nonsmoking: (1) initial disinterest in quitting; (2) thinking about health risks and contemplating quitting; (3) actively preparing to quit within the next month; (4) currently taking action to stop smoking; and (5) maintained nonsmoking (see Chapter 16.)

## MANAGEMENT OF BARRIERS TO SMOKING CESSATION

### Nicotine Withdrawal Syndrome

Cigarettes and other tobacco products are addicting. Evidence summarized in the 1988 *Surgeon General's Report on Smoking* identified nicotine as the addictive drug in tobacco that is capable of creating tolerance, physical dependence, and a withdrawal syndrome in habitual users. Nicotine withdrawal symptoms include (1) cravings for a cigarette; (2) irritability, anxiety, impatience, and anger; (3) difficulty concentrating; (4) excessive hunger; and (5) sleep dis-

turbance. These symptoms begin within a few hours of the last cigarette, are strongest during the first 2–3 days after quitting, and gradually diminish over 2–3 weeks. The severity of nicotine withdrawal is variable and related to the level of prior nicotine intake. Other than craving for a cigarette, the symptoms are non-specific, and many smokers fail to recognize them as nicotine withdrawal.

The discomfort of nicotine withdrawal is one reason why smokers fail in their efforts to stop. Nicotine replacement therapy relieves the symptoms, but pharmacological treatment is not mandatory, because mild symptoms can be managed with behavioral methods.

### Behavioral Factors

The attractiveness of smoking is attributable to more than nicotine dependence. Smoking is also a habit, a behavior that has become an integral part of a daily routine. Smokers come to associate cigarettes with enjoyable activities, such as finishing a meal or having a cup of coffee. These actions trigger the desire for a cigarette in smokers who are trying to quit. Smokers also use cigarettes to cope with stress and negative emotions such as anger, anxiety, loneliness, or frustration. Quitting smoking represents the loss of a valuable coping tool.

Behavior modification strategies address these barriers to quitting smoking and are effective in aiding smoking cessation. Smokers monitor their cigarette intake to identify cues to smoking, change their habits to break the link between the trigger and smoking, and learn to anticipate and handle the urges to smoke that do occur. The skills can be taught in formal group programs or packaged into booklets or videotapes for at-home use.

### Weight & Smoking Cessation

Smokers weigh 5–10 lb. less than nonsmokers of comparable age and height. When smokers quit, 80% of them gain weight. The average weight gain of 5 lb. (2.3 kg) poses a minimal health risk, especially when compared with the benefits of smoking cessation. Women gain more weight than men; their average weight gain in one national sample was 8 lb. (3.8 kg). Though many smokers fear large weight gains, only about 10% gain more than 25 lb. after cessation. Heavier smokers (>25 cigarettes/day) gain more than lighter smokers. The mechanism is incompletely understood, but a nicotine-related decrease in metabolic rate and possibly increases in food intake appear to be largely responsible. The weight gain occurs in both sexes, but survey data reveal that it concerns female smokers more than male smokers, presumably because women experience greater cultural pressure to be slender. Although weight gain has been considered a trigger to relapse, some studies show that successful abstainers gain more weight than relapsers. Smokers who use nicotine gum gain less weight than those who quit with a placebo, though weight gain may just be

delayed until after gum use stops. Use of the nicotine patch does not reduce weight gain. Behavior modification approaches to avoiding weight gain have not been successful.

The best approach to the problem may be a change in attitude. Smokers can learn that although some weight gain is likely, the amount is less than they fear. Accepting a small increase in weight until smoking cessation is secure is a better strategy than attempting to stop smoking and lose weight simultaneously. The chance of a large weight gain may be reduced by avoiding high-calorie snacks and increasing physical activity during smoking cessation. Nicotine gum may provide a way to delay and possibly avoid weight gain.

### Social Support

Smokers with nonsmoking spouses are more likely to quit than smokers whose partners smoke. Smokers whose efforts to stop are supported by partners, family, and friends are more likely to succeed than smokers without this support. This is especially true for women smokers. Those who live with smokers can ask them to restrict smoking to outdoor areas or to limited areas of the home in order to provide a smoke-free area in the home. Formal cessation programs provide an additional source of social support.

### Mood Disorders

Stopping smoking represents a loss for many smokers. Cigarettes have been reliable "companions" as well as coping tools. Transient sadness is common and requires no special treatment. Acknowledgment that this is normal can be helpful. There is, however, growing evidence of an association between smoking and mood disorders. Smokers have more depressive symptoms than nonsmokers and are more likely to have a history of major depression. Depressed smokers are less likely to stop smoking than nondepressed smokers. There are case reports that smoking cessation precipitated depression in smokers with a history of major depression and that resuming smoking restored mood. These observations suggest that some smokers use nicotine to regulate mood. Clinicians should be alert to the possibility of depression in smokers. If present, it should be treated before cessation is attempted. Smokers with a history of depression should be watched for the reemergence of symptoms during smoking cessation.

### Substance Abuse

Smokers use more drugs—including coffee and alcohol—than nonsmokers. Because alcohol is frequently an ingredient in relapse situations, smokers attempting to quit are commonly advised to avoid alcohol temporarily after quitting. Smoking increases caffeine excretion. Smokers who stop should be advised to reduce caffeine intake to avoid increased blood levels and jitteriness. There is a high rate of smoking among abusers of alcohol, cocaine, and

heroin. Depression and substance abuse should be considered as potential comorbid disorders in smokers who repeatedly try and fail to quit.

## THE PHYSICIAN'S ROLE

The optimal way to avoid the health hazards of tobacco is never to smoke. Because smoking behavior begins so early, preventing smoking is particularly the task of physicians who care for children and adolescents. The challenge for physicians taking care of adults is smoking cessation. Physicians have the opportunity to intervene with smokers, because each year, they see an estimated 70% of the smokers in the United States. Primary care physicians have the advantage of repeated contact with smokers. Physicians have the additional opportunity of seeing smokers at times when symptoms have made them concerned about their health and therefore more likely to change their smoking behavior. For example, the diagnosis of coronary artery disease motivates behavior change. Approximately one third of smokers stop smoking after a myocardial infarction, and this proportion can be increased with brief counseling delivered by a physician or nurse. For women, pregnancy encourages smoking cessation. Approximately 30% of female smokers stop smoking while pregnant, though many resume smoking after delivery. Other smoking-related conditions may also provide "teachable moments" when smokers are more receptive to advice to stop smoking.

Although physicians report advising most patients to stop smoking, fewer than half of smokers recall having ever received this advice. Providing brief advice to stop smoking to all patients seen in the office increases patients' smoking cessation rates, as was demonstrated in a randomized controlled trial of British general practitioners. Although advice alone is effective, randomized controlled trials in general medicine and family practices have demonstrated that supplementing advice with brief counseling is more effective. Programs shown to be effective consist of brief structured counseling during which the physician asks the patient to set a quit date, provides written materials, offers nicotine replacement, and schedules a follow-up visit. Cost-effectiveness analyses demonstrate that counseling smokers in office practice is as cost-effective as, or more so, than other accepted medical practices and that counseling smokers as part of prenatal care is cost-saving.

### SMOKING CESSATION COUNSELING STRATEGY FOR OFFICE PRACTICE

Detailed evidence-based clinical guidelines for smoking cessation treatment by primary care pro-



viders have been developed by the Agency for Health Care Policy and Research. These guidelines recommend that the primary care of adults should include a routine assessment of smoking status in all patients, strong advice to all smokers to quit, and assistance for those smokers who are ready to stop. The National Cancer Institute has organized the common elements of effective smoking cessation counseling programs into a four-step protocol intended to require only a few minutes of an office visit (Box 18-1).

1. **ASK:** Physicians should routinely ask all patients at every visit whether they smoke cigarettes. Those who smoke should be asked whether they are interested in quitting smoking. This permits the physician to determine the smokers' readiness to change. Categorizing smokers in this way is a clinically useful approach that helps the physician to determine what counseling strategy is appropriate and to set achievable goals for that encounter. Although the clinician's overall goal is to assist the smoker to stop permanently, a realistic goal for a single office visit is to move the smoker to the next stage of readiness to stop smoking.
2. **ADVISE:** Regardless of a smoker's degree of interest in quitting, it is the physician's responsibility to deliver clear advice to each smoker

about the importance of stopping smoking. The message should be strong and unequivocal; for example, "Quitting smoking now is the most important health advice I can give you." If appropriate, advice should be tailored to the clinical situation, either current symptoms or family history. For example, smokers can be informed that they will have fewer colds, less asthma, or healthier babies if they stop smoking. Advice is more effective when phrased in a positive way; for instance, emphasizing the benefits to be gained from quitting rather than the harms of continuing to smoke.

3. **ASSIST:** The third step is to assist the smoker in quitting smoking. The physician's approach should vary according to the smoker's readiness to stop smoking.

*If the smokers are interested in quitting smoking,* the physician should ask whether they are ready to set a "quit date," a date within the next 4 weeks when they will stop smoking. If so, the date should be recorded in the chart and on material given to them to take home. The physician should discuss with the patients what approach is most likely to be successful. A stepped-care model of smoking treatment can guide the physician in making this recommendation. For smokers making a first attempt to quit, the

**Box 18-1.** Smoking cessation counseling protocol for physicians.

1. **ASK**—about smoking at every visit.
  - a. "Do you smoke?"
  - b. "Are you interested in stopping smoking?"
2. **ADVISE**—every smoker to stop.
  - a. Make advice clear: "Stopping smoking now is the most important action you can take to stay healthy."
  - b. Tailor advice to the patient's clinical situation (symptoms or family history).
3. **ASSIST**—the smoker in stopping smoking.
  - a. For smokers ready to quit
    - (1) Ask smoker to set a "quit date."
    - (2) Provide self-help material to take home.
    - (3) Consider nicotine replacement therapy.
    - (4) Consider referral to a formal cessation program.
  - b. For smokers not ready to quit
    - (1) Discuss advantages and barriers to cessation, from smokers' viewpoint.
    - (2) Provide motivational booklet to take home.
    - (3) Advise smoker to avoid exposing family members to passive smoke.
    - (4) Indicate willingness to help when the smoker is ready.
    - (5) Ask again about smoking at the next visit.
4. **ARRANGE**—follow-up visits.
  - a. Make follow-up appointment 1 week after quit date.
  - b. At follow-up, ask about smoking status.
  - c. For smokers who have quit:
    - (1) Congratulate!
    - (2) Ask smoker to identify future high-risk situations.
    - (3) Rehearse coping strategies for future high-risk situations.
  - d. For smokers who have not quit:
    - (1) Ask: "What were you doing when you had that first cigarette?"
    - (2) Ask: "What did you learn from the experience?"
    - (3) Ask smoker to set a new "quit date."

Source: Adapted, with permission, from Glynn TJ, Manley MW: *How to Help Your Patients Stop Smoking: A National Cancer Institute Manual for Physicians*. US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute, Division of Cancer Prevention and Control. NIH Publication No. 89-3064, 1989. [Available free by calling 1-800-4-CANCER.]

physician should assist them in quitting on their own; this can be done most simply by providing a take-home booklet containing standard behavioral modification strategies (Box 18-2). Alternatively, this information can be provided by office staff or on a videotape. The physician should be prepared to discuss management of barriers to cessation if the smokers ask.

Nicotine replacement therapy should also be considered. Although it is especially appropriate for smokers likely to have severe nicotine withdrawal symptoms, it has been demonstrated to benefit smokers of all types. Smokers who suffered nicotine withdrawal symptoms on previous attempts to quit or who smoke more than 25 cigarettes daily, have their first cigarette within 30 minutes of awakening, or are uncomfortable when forced to refrain from smoking for more than a few hours are likely to suffer nicotine withdrawal.

More intensive treatment is indicated for smokers who have been unsuccessful in previous attempts to quit. The option at this second level is referral to a formal smoking cessation program, with or without prescription of nicotine replacement therapy. Smoking cessation programs provide intensive training in behavioral smoking cessation skills combined with social support from the counselor and other group members. Combinations of nicotine replacement and a behavioral counseling program are more effective than either one alone.

*For smokers not interested in quitting or not ready to set a quit date*, the physician should ask them what they consider to be the benefits and harms of smoking. From an understanding of the patients' perspective, the physician can provide missing information about health risks and correct misconceptions about the process of smoking cessation. The discussion should focus on short-term benefits rather than distant risks, and the physician should be prepared to discuss common barriers to smoking cessation. The clinician should advise the smokers not to ex-

pose family members to passive smoke (eg, not to smoke inside the home if nonsmokers are present), provide a take-home booklet about smoking cessation, and indicate future availability to help them when they are ready to quit.

4. **ARRANGE:** Randomized trials have demonstrated that arranging follow-up visits to discuss smoking increases the success of physician counseling. Smokers should be asked to return shortly after the quit date to monitor progress; this is especially important for smokers using nicotine replacement.

*If smokers are not smoking at the follow-up visit*, they should be congratulated but warned that continued vigilance is necessary to maintain abstinence. The level of nicotine withdrawal symptoms should be assessed, and if indicated, treated. To prevent relapse to smoking, patients should be asked to identify future situations in which they anticipate difficulty remaining abstinent. The physician can help to plan and rehearse coping strategies for these times. Further follow-up visits or telephone calls should be offered.

*If smokers have not been able to remain abstinent*, the physician's role is to redefine an experience that the smokers feel has been a failure into a partial success. They can be told that even one day without cigarettes is the first step toward quitting and reminded that it takes time to learn to quit, just as it took time to learn to smoke. To help smokers learn from the experience, the physician should ask in detail about the circumstances surrounding the first cigarette smoked after the quit date. They should be asked what they learned from the experience that can be used for the next attempt to quit. Finally, these smokers should be asked whether they are ready to set a new quit date.

### Office Organization

Smoking counseling in an office need not and should not be limited to the physician's actions. A system-wide approach is at least as effective as a

**Box 18-2.** Selected smoking cessation guidelines for physicians and patients.

#### For Physicians

1. *How to Help Your Patients Stop Smoking*. A National Cancer Institute Manual for Physicians.
2. *Smoking Cessation*. Clinical Practice Guideline No. 18. Agency for Health Care Policy and Research. AHCPR Publication No. 96-0692, April 1996.
3. *Clinical Opportunities for Smoking Intervention—A Guide for the Busy Physician*. National Heart Lung and Blood Institute.
4. *Family Physician's Guide to Smoking Cessation*. American Academy of Family Physicians.
5. *A Healthy Beginning Counseling Kit*. American Lung Association.

#### For Patients

1. *Clearing the Air*. National Cancer Institute. (Order from 1-800-4-CANCER).
2. *Smart Move*. American Cancer Society.
3. *Freedom from Smoking for You and Your Family*. American Lung Association.
4. *Freedom from Smoking for You and Your Baby*. American Lung Association.

physician-focused model, and it reduces the burden on a busy physician. In this model, the patients' smoking status is assessed before the physician sees them. The staff member who checks weight or blood pressure asks about smoking and labels the chart to remind the physician to discuss smoking. Simple reminder systems like this increase the amount of time physicians spend counseling smokers. The physician's prime role is to provide advice to stop smoking and ask the patient to set a quit date. Office personnel build on physician advice to provide counseling, medication instruction, or referrals to outside programs.

## PHARMACOLOGICAL TREATMENT

### Nicotine Replacement Therapy

The rationale of nicotine replacement is to supply nicotine in a form other than tobacco in order to block the symptoms of nicotine withdrawal. Nicotine replacement permits the smoker to break the smoking habit first and taper off nicotine later. Three forms of nicotine replacement are currently approved for use in the United States: nicotine polacrilex (a gum), a transdermal skin patch, and a nicotine nasal spray (Table 18-1). The first two produce relatively constant blood levels of nicotine, a substantially different pattern from the fluctuating nicotine levels produced by cigarette smoking. The nasal spray produces nicotine levels that more closely resemble those produced by smoking. The nicotine supplied by the gum, patch, or nasal spray is sufficient to block nicotine withdrawal symptoms but not to reproduce the pleasures of smoking.

Randomized placebo-controlled trials demonstrate that nicotine gum, patch, and nasal spray all reduce nicotine withdrawal symptoms and increase smoking cessation rates. A meta-analysis of nicotine patch trials found that the patch more than doubled smoking cessa-

tion rates, compared with a placebo, though the cessation rates in individual trials varied widely. A meta-analysis of nicotine gum demonstrated its effectiveness in special clinics treating smokers but not when used in medical settings. The effectiveness of all products depends on what instruction and counseling accompany it. This is particularly true for the gum and nasal spray, which require careful instruction for proper use. Compliance is less of a problem with the nicotine patch. The most effective use of any product, however, requires that the physician provide the smoker with concurrent behavioral counseling of some type that teaches how to break the cigarette habit.

Nicotine replacement has been used safely, even in smokers with stable coronary artery disease. Contraindications to nicotine replacement include acute or recent cardiovascular events (myocardial infarction, unstable angina, or serious arrhythmias). Anecdotal reports of myocardial infarction in patients who were smoking while using the patch were not borne out by more careful study, but smoking is contraindicated in patch users; it increases blood nicotine levels and, more importantly, is not a pattern of behavior that leads to smoking cessation. The gum is contraindicated in patients with temporomandibular disease; the patch is contraindicated in patients with widespread skin eruptions. The safety of any product in pregnancy is not established. Nicotine replacement is almost certainly safer than smoking cigarettes, but medicolegal concerns have limited its use. Clinicians who have used it reserve it for use in patients who have failed a behavioral smoking cessation program and prescribed it only after documenting a risk-benefit discussion with the patient.

**Transdermal Nicotine Patch:** The nicotine patch contains a reservoir of nicotine that is released at fixed doses that are absorbed through the skin. Each of the four products available is more effective than placebo in relieving withdrawal symptoms and pro-

Table 18-1. Nicotine replacement products.

Brand Name	Dosage per Day	Recommended Duration of Use
<i>Transdermal Nicotine Patch</i>		
Habitrol	21 mg/24 h	4 weeks
	14 mg/24 h	2 weeks
	7 mg/24 h	2 weeks
Nicoderm CQ	21 mg/24 h	4 weeks
	14 mg/24 h	2 weeks
	7 mg/24 h	2 weeks
Nicotrol	15 mg/16 h	8 weeks
Prostep	22 mg/24 h	4 weeks
<i>Nicotine Gum</i>		
Nicorette CQ 2 mg	9-12 pieces/day*	2-3 months
	(maximum 30)	(maximum 6)
Nicorette CQ 4 mg	9-12 pieces/day	2-3 months
	(maximum 20)	(maximum 6)
<i>Nicotine Nasal Spray</i>		
Nicotrol NS	1-2 doses/h (maximum 8/day)	3 months

\* Chew as needed or 1 piece every 1-2 h while awake.

moting smoking cessation. No studies have directly compared different patches; therefore, there are insufficient data to recommend any one patch as most effective or safest. The most common side effect is local skin irritation, which rarely requires discontinuation of treatment and can be treated with topical steroids. Vivid dreams, insomnia, and nervousness have also been reported; they can be managed by removing the patch at bedtime or using a lower-dose patch.

The smoker applies the first patch on the morning of the quit day and applies a new patch to rotating skin sites each morning afterward. Three products are intended for 24-hour use; the fourth is removed after 16 hours (eg, at bedtime). Smokers started on the patch should be monitored after 1 week to assess level of withdrawal symptoms and to screen for smoking during patch use. Some patches are available in three sizes to permit tapering, and data suggest that patch use continue for 2 months. Lower starting doses are recommended for patients weighing under 100 lb. or smoking fewer than 10 cigarettes/day. Long-term dependence on the nicotine patch appears to be uncommon. Four nicotine patches (Habitrol, Nicoderm, Nicotrol, and Prostep) have been available by prescription since 1972; in 1996 two of them (Nicoderm and Nicotrol) were granted permission to be sold as over-the-counter products.

**Nicotine Gum:** Nicotine gum is available without prescription in 2-mg and 4-mg strengths. Careful instruction in proper chewing technique is essential for it to be effective and to avoid side effects; for this reason, compliance is more problematic than with the nicotine patch. The gum should not be chewed like regular gum. A piece is chewed only long enough to release the nicotine, producing a peppery taste, then placed between the gums and buccal mucosa to allow for nicotine absorption. After 30 minutes, it is discarded. No liquid should be drunk while the gum is in the mouth, and acidic beverages (eg, coffee) should be avoided for 1–2 h before gum use. Side effects are common but minor; they include those related to nicotine (nausea, dyspepsia, hiccups, dizziness) and to chewing (sore jaw, mouth ulcers). The product is approved for use as needed to handle urges to smoke; however, its effect is slower than smoking. Most pa-

tients chew fewer than the recommended 9–12 pieces daily. Consequently, some clinicians use fixed-dose schedules (eg, chewing one piece every hour) to achieve adequate blood nicotine levels to prevent withdrawal. Approximately 5–10% of gum users develop long-term dependence on the gum.

**Nicotine Nasal Spray:** A nicotine nasal spray became available in 1996. Nicotine is rapidly absorbed from the nasal mucosa, reaching a peak within 15 minutes, then declining over 1–2 hours. The variable pattern of nicotine exposure produced from repeated use of the nasal spray resembles that produced by smoking cigarettes, raising concerns that the nasal spray will have a greater potential for producing dependence than has been observed with other nicotine replacement products. It is superior to placebo in randomized controlled trials, but its high incidence of side effects (nose and throat irritation, watery eyes, sneezing and cough) and concerns about its potential for producing dependence are likely to limit its appeal, except in smokers who have failed with other nicotine replacement products. Nicotine nasal spray is not recommended for smokers with asthma or chronic rhinitis or sinusitis, and careful instruction is required for its proper use.

### Other Pharmacological Agents

Clonidine, a centrally acting alpha-adrenergic agonist, is used to treat craving for psychoactive drugs other than nicotine. In randomized, placebo-controlled trials, both oral and transdermal clonidine reduced withdrawal symptoms, but there is inconsistent evidence that either product increases smoking cessation rates in the long term. Clonidine is not approved for use as an aid for smoking cessation.

There is no evidence to support the use of tranquilizers or benzodiazepines for smoking cessation. The growing awareness of the link between smoking and mood disorders has stimulated interest in the use of antidepressants to treat smoking in individuals with a history of depression. Until more is known, it is appropriate to use these agents to treat depression in a smoker and to continue their use when the patient attempts to stop smoking. Whether they have any role in smokers who are not currently depressed is not known.

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