good health care and by the awareness that health care spending is sapping our country of vitality and options.

We do have limits. We also have remarkable abilities to heal. We need to claim, to proclaim, to reclaim the basics—science, education, respect, and trust.

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REFERENCES

1. Brandeis LD: Business—A Profession. Boston, Mass, Small, Maynard & Company, 1914
2. Thomas Jefferson, Declaration of Independence, July 1776
3. Beeson PB: On becoming a clinician, *In* Reynolds R, Stone J (Eds): On Doctoring. New York, NY, Simon & Schuster, 1991, p 178

Getting Some Breathing Room—Persuading Patients to Stop Smoking

ABOUT 35 YEARS AGO when I was participating in a summer internship in chronic disease research methods sponsored by the California State Health Department, a discussion took place about how to "prove" that cigarette smoking was responsible for the development of chronic obstructive pulmonary disease (COPD). Randomized controlled clinical trials were relatively new, and concerns ranged from whether there were sufficient data to suggest that any controlled trial was necessary to whether any trial was ethical. Could one, with what was known at that time, ethically randomly assign consenting adults to take up smoking or not provide all patients with the contradicting information that smoking was or, as some suggested at that time, was not harmful? Little was said or known about how difficult it was to actually get subjects to stop smoking. Most of the studies of that era related to highly motivated people who had just suffered a major illness—mostly men with stomach ulcers or myocardial infarctions. Primary prevention was rarely mentioned.

In one sense Browner and co-workers in their report elsewhere in this issue of the journal have attempted, using data from the Multiple Risk Factor Intervention Trial (MRFIT), to do such a study in the only way ethically possible.[1] As part of a randomized trial designed to reduce the risk of heart disease in what most would agree was a high-risk group, the investigators took advantage of data being obtained to assess heart disease to evaluate the effects of cigarette smoking cessation on the change in lung function. More than 12,800 men aged 35 to 57 years were enrolled in the MRFIT study. These men were randomly assigned to either an intensive integrated program of smoking cessation or usual care. Of those entering the trial, 6,347 were smokers for whom acceptable repeated pulmonary function measurements were obtained during the course of the trial. In general, acceptable pulmonary function data were available only for the latter half of the six-year follow-up study. Thus, the analysis was based on the change in pulmonary function as measured by the forced expiratory volume in one second (FEV_1) expressed as an annual rate of decline over two to four years. Unfortunately, for the hypothesis to be tested in terms of pulmonary function change, the subjects most susceptible to the effects of cigarette smoking (those with diagnosed chronic respiratory disease) were systematically excluded from the sample. Yet, even after excluding the group likely to be most sensitive to the effects of smoking cessation—that is, those with COPD whose pulmonary function is declining fastest[2]—the investigators interpret their findings as showing a beneficial effect of stopping smoking.

The authors are appropriately cautious in discussing the limitation of their findings; nevertheless, the results are important in adding to the evidence that, no matter when a patient stops smoking, the effect, at least in terms of nonmalignant respiratory disease, is beneficial. These results were recently summarized by the United States Surgeon General's 1990 Report entitled *The Health Benefits of Smoking Cessation.*[3] A relatively large body of evidence from cross-sectional, cohort, and interventional studies mostly indicates less morbidity and, in some cases, greater levels of pulmonary function among former smokers than among those who continue to smoke.

The pathophysiologic correlates of these epidemiologic findings support some postulated mechanisms for the measured responses. Generally, the earliest lesions found in smokers' lungs involve the small airways. When these are noted there may be no changes in pulmonary function as measured with the FEV_1. Changes in mucus production, however, brought about by the direct irritant effects of cigarette smoke on goblet cells in the larger airways, may result in partial obstruction, which will reduce the FEV_1. In fact, it is this mucous hypersecretion that is initially affected by a reduced exposure to cigarette smoke, with the subsequent decline in goblet cell number and a reduced production of mucus.

In humans the effect of reduced smoking on the production of mucus can be measured by reduced sputum volumes both in general population samples and in those with chronic bronchitis. A corresponding improvement in pulmonary function in patients with severe COPD can be detected within three months of stopping smoking.[4] Similarly, in normal populations (adjusted for pack-years of smoking), an improvement of about 5% in the FEV_1 is noted in both men and women after three years of smoking cessation (the interval between measurements). As long as the subjects continue not to smoke, they maintain that 5% difference from those who continue to smoke.[5] Some studies have found a reduced rate of decline in the FEV_1 in former smokers; others have found that, once the initial improvement has occurred, the decline continues at a rate similar to that in current smokers. Although it is clear that the initial improvement is maintained, few studies to date have been carried out long enough to show with certainty that, once the initial improvement is taken into account, further slowing of the rate of decline in the FEV_1 occurs. Without detracting from the importance of the MRFIT findings, I should note that the study suffers from this same difficulty of not having observed the patients long enough to be certain that the effect on the FEV_1 is not all due to the immediate effect of stopping smoking rather than an ongoing slowing of the rate of decline.

As previously mentioned, the Surgeon General's report on the health benefits of stopping smoking refers to several studies, contributing to a similar conclusion that for nonmalignant respiratory diseases, stopping smoking is beneficial. In addition, that same volume reports on a wide body of data that speak to a variety of health benefits of quitting smoking that extend beyond concerns related to the development of COPD. As indicated by the Surgeon General, "Cigarette smoking is the most important preventable cause of death in our society . . . and is responsible for approximately 390,000 deaths each year in the United States, or more than one of every six deaths."[3] Such statistics, in fact, do not consider all the effects related to smoking.

Thus, the public health message is clear. Given that more than 60% of the adult population in the United States has smoked at some time, efforts to help people stop smoking and to remain nonsmokers will have a significant impact on the health of the nation. It is imperative that primary care professionals play a vital role in this effort and have accurate information on what can be expected in smoking patients. Their smoking patients will need objective evidence beyond "the statistics" that giving up smoking will have an affect on them. Thus, if pulmonary function is measured as part of the cessation plan both before and several months after quitting, that improvement will be manifest. Because pulmonary function is a strong predictor of morbidity and mortality not only from COPD but also from cardiovascular diseases, practitioners can honestly tell patients that smoking cessation and the consequent improved lung function will lessen their risk.

Clearly, taking the time to explain to patients the importance of stopping smoking will reduce the time available to give to other sick patients in the waiting room. But because as many as 20% of the patients are there because of smoking-related conditions, an effective campaign would surely make all of our work a little easier. The potential of such a campaign to reduce costs to the national health care system would give us all (including our patients) a little more breathing room.

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REFERENCES

1. Browner WS, DuChene AG, Hulley SB: Effects of the Multiple Risk Factor Intervention Trial smoking cessation program on pulmonary function—A randomized controlled trial. West J Med 1992 Nov; 157:534-538

2. Postma DS, De Vries K, Koeter GH, Sluiter HJ: Independent influence of reversibility of air-flow obstruction and nonspecific hyperreactivity on the long-term course of lung function in chronic air-flow obstruction. Am Rev Respir Dis 1986; 134:276-280

3. The Health Benefits of Smoking Cessation: A Report of the Surgeon General—US Dept of Health and Human Services publication No. (CDC) 90-8416. Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1990

4. Buist AS, Nagy JM, Sexton GJ: The effect of smoking cessation and modification on pulmonary function: A 30-month follow-up of two smoking cessation clinics. Am Rev Respir Dis 1979; 120:953-957

5. Dockery DW, Speizer FE, Ferris BG Jr, Ware JH, Louis TA, Spiro A: Cumulative and reversible effects of lifetime smoking on simple tests of lung function in adults. Am Rev Respir Dis 1988; 137:286-292

Caffeine Under Examination—A Passing Grade

DURING THE PAST FEW DECADES, biomedical researchers have studied caffeine more intensively than almost any other substance, naturally occurring or synthetic. Yet, when all the data have been analyzed, caffeine seems to be remarkably safe when used at current levels of consumption. In clinical research, interest in caffeine has generally been focused on coffee, with tea and caffeine-containing soft drinks receiving considerably less attention.

Why are medical investigators so attracted to research on coffee? Unlike a synthetic drug, coffee is relatively easy to study from an ethical point of view. Most people drink coffee, and administering or withholding it does not raise important ethical issues. There is also considerable interest in coffee among both the general public and the scientific community. As a consequence, investigators can initiate studies on coffee secure in the knowledge that scientific journals will welcome their findings, be they negative or positive.

Despite extensive study, misconceptions about coffee prevail among health care practitioners. When questioned on their beliefs, physicians will generally agree that coffee increases the heart rate, causes cardiac arrhythmias, elevates the blood pressure, increases sympathetic nervous system activity, and is associated with a higher incidence of coronary heart disease. The available literature as reviewed by Chou in the current issue of this journal does not substantiate any of these beliefs, however.[1] Although it is true that the ingestion of coffee or caffeine initially causes a small increase in blood pressure (with a slight reduction in heart rate) and an increase in plasma catecholamine levels, tolerance develops rapidly, and these effects disappear after a few days of regular coffee use.

So why do people continue to take a dim view of coffee? One reason is that clinical studies, mostly of a case-control design, have frequently reported its use to be harmful. On the other hand, tea has escaped any association with human disease, implying that coffee contains something other than caffeine to account for its apparent adverse effects. But a search for other potentially harmful ingredients in coffee has failed to explain the differences reported between tea and coffee.

It is conceivable that coffee may only be a marker for disease and may not be the actual offending agent. Chou and others have noted that regular coffee drinkers possess other risky life-style traits, including cigarette smoking, poor dietary habits, high stress, and greater alcohol use.[1,2] In contrast, tea drinkers are often portrayed as quietly sipping tea to relax and take a break from life's hectic pace. When coronary risk factors are examined,[3] the habits of tea drinkers are generally the opposite of coffee drinkers. Unless such differences are taken into account in the design of studies, coffee may be erroneously implicated in the pathogenesis of a variety of conditions including cancer and coronary artery disease.

For example, coffee drinking and cigarette smoking have both been associated with an increased incidence of coronary heart disease.[4-6] Most of the findings linking coffee to coronary heart disease, however, come from case-control studies.[7] In matching coronary artery disease patients to controls, researchers attempt to adjust the data analysis for other coronary risk factors. But not all risk factors can be quantitated precisely, and atherogenic behavior common to heavy coffee users may lead to potentially biased results. Thus, if respondents underestimate their cigarette use, then any residual risk of coronary artery disease from smoking may be attributed to coffee consumption. Unfortunately, small "statistically significant" increases in risk (odds ratios $<2:1$) tend to be widely reported by the media and are often portrayed as proving causation.

Longitudinal studies are less susceptible to bias from confounding variables than those using a case-control design. As noted by Chou, none of six recently reported longitudinal studies found any association between coffee use and pancreatic carcinoma.[1] Similarly, a recently completed meta-analysis of longitudinal studies found no link between coffee consumption and coronary artery disease.[7]

Coffee may not be everyone's favorite drink. It may cause some people to feel jittery and nervous, and others may have trouble sleeping when they drink coffee in the evening. We should be careful to keep our concerns about coffee in perspective, however, and avoid linking its use to perceived