

Has the California Tobacco Control Program Reduced Smoking?

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Context.—Comprehensive community-wide tobacco control programs are considered appropriate public health approaches to reduce population smoking prevalence.

Objective.—To examine trends in smoking behavior before, during, and after the California Tobacco Control Program.

Design.—Per capita cigarette consumption data (1983-1997) were derived from tobacco industry sales figures. Adult (≥ 18 years) smoking prevalence data were obtained from the National Health Interview Surveys (1978-1994), the California Tobacco Surveys (1990-1996), the Current Population Surveys (1992-1996), and the California Behavioral Risk Factor Survey and its supplement (1991-1997). Trends were compared before and after introduction of the program, with the period after the program being divided into 2 parts (early, 1989-1993; late, 1994-1996).

Main Outcome Measures.—Change in cigarette consumption and smoking prevalence in California compared with the rest of the United States.

Results.—Per capita cigarette consumption declined 52% faster in California in the early period than previously (from 9.7 packs per person per month at the beginning of the program to 6.5 packs per person per month in 1993), and the decline was significantly greater in California than in the rest of the United States ($P < .001$). In the late period, the decline in California slowed to 28% of the early program so that in 1996 an average of 6.0 packs per person per month were consumed. No decline occurred in the rest of the United States, and in 1996, 10.5 packs per person per month were consumed. Smoking prevalence showed a similar pattern, but in the late period, there was no significant decline in prevalence in either California or the rest of the United States. In 1996, smoking prevalence was 18.0% in California and 22.4% in the rest of the United States.

Conclusions.—The initial effect of the program to reduce smoking in California did not persist. Possible reasons include reduced program funding, increased tobacco industry expenditures for advertising and promotion, and industry pricing and political activities. The question remains how the public health community can modify the program to regain its original momentum.

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EARLY PUBLIC HEALTH approaches to reducing population smoking prevalence emphasized interventions aimed at individual smokers.¹ However, the results of numerous studies indicated that too few individuals were reached for such a strategy to effect a measurable reduc-

tion in population smoking prevalence.² The varied successes of several comprehensive, community and statewide tobacco control programs³⁻⁷ led to this approach being widely recommended as the most appropriate way to reduce tobacco use in the United States.^{2,8} Starting in 1989, the California Tobacco Control Program introduced the use of increased tobacco excise taxes to continuously fund a large, coordinated statewide effort to reduce the health costs associated with smoking.⁹

The voter initiative that led to the California Tobacco Control Program clearly specified that the program take a

multipronged or "shotgun" approach to reducing smoking. In addition to imposing an additional tax (\$0.25 per pack), the initiative mandated funding for mass media antitobacco campaigns, local health agencies to provide technical support and monitor adherence to antismoking laws, community-based interventions selected by a competitive grants process, and enhancement of school-based prevention programs. Additionally, it mandated that the program's effectiveness be evaluated.⁹ In this article, we report the longer-term evidence that the California Tobacco Control Program affected smoking behavior.

One problem with assessing the effectiveness of tobacco control programs funded by cigarette taxes is that funding for evaluation research, including population surveys of smoking behavior, becomes available only after the first intervention (imposition of the tax) has occurred. In the United States, surveillance surveys have rarely had designs that provide precise enough estimates of smoking behavior at the state level to allow a sensitive assessment of changes in trends.¹⁰ The research challenge is to reach valid conclusions from the analysis of preprogram trends derived from one set of surveys and postprogram trends from different surveys. Fortunately, another source of data is available from the collection of cigarette excise taxes. All states have such taxes, and the sales reporting methods for tax assessment are uniform. If there is no major change in the average level of consumption per smoker, trends in smoking prevalence should mirror trends in cigarette sales, which would increase the confidence in conclusions based on either analysis.

In this article, we assess trends in per capita cigarette consumption and adult smoking prevalence in California compared with the rest of the United States. The only previous report of the longer-term impact of a statewide tobacco control program indicated that the program

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had an overall impact during its first year of operation.⁵ However, the magnitude of this initial effect was not maintained over the next 4 years. A different pattern was observed for men and women; the rate of decline (trend) in smoking was greater only in men in the second period than it was in the preprogram period.¹¹ If the later trend is not larger than the preprogram trend, then the program can be considered to have lost its effect. Should an ongoing tobacco control program lose its effect, a careful examination of the possible reasons is essential so that appropriate revitalization measures can be taken. Also, it must be considered that counterstrategies used by the tobacco industry may play a role in diminishing a program's effectiveness.

METHODS

Cigarette Sales (Consumption)

The Tobacco Institute reports on monthly tax payments from all packs of cigarettes removed from wholesale warehouses to retail outlets for sale within each state.¹² Data from February 1983 through March 1997 are included in the present analysis. We estimated per capita consumption for a given state in any given year using census estimates for the state population aged 18 years and older. Decade census population data were assumed to reflect the population on April 1, 1980, and April 1, 1990. Supplemental estimates reported from the Current Population Surveys were assumed to reflect the population as of July 1 of each year.^{13,14} To obtain monthly estimates of state populations, we interpolated regression lines fitted to the yearly census data. Since retail outlets appear to stock up in the last month of both the fiscal and calendar years, we removed this source of variation by considering bimonthly averages (for December-January, February-March, etc). The per capita consumption represents the average number of packs removed from wholesale warehouses during a 2-month period divided by the population estimate for the midpoint of the particular time interval. To further deseasonalize the data so that trends over time become more apparent, we applied the SABL procedure (available in the statistical package S-plus¹⁵) to the bimonthly data. The SABL procedure provides robust estimation of seasonal and trend components of a time series, possibly in the presence of nonadditive effects.¹⁶ This procedure was used for both California and the rest of the United States to produce smoothed time-series trend lines indicating changes over time.

A piecewise linear spline regression model was applied to the bimonthly raw

data to further quantify trends. Indicator variables were included to account for the effects of the 6 bimonthly time points. This model allows for changes in the slope at defined points of time.^{17,18} The first cut point was defined as January 1989, when the additional excise tax was imposed in California. The deseasonalized trends suggested that a second cut point occurred in California in mid-1994 and in the rest of the United States in mid-1993, so January 1994 was used to make the analyses consistent. A 2-tailed statistical test yielded a *P* value for differences in slope from one period to the next. Also, from computed SEs for the piecewise slopes, a *z* statistic could be computed to assess (2-tailed) differences in slopes between California and the rest of the United States.

Smoking Prevalence

Surveys.—Smoking prevalence estimates were obtained from several different population-based surveys conducted nationally and in California.¹⁹⁻²⁵ These differed considerably in the methods used, including sample selection, survey mode (face-to-face or telephone), smoking status questions, respondents (self or proxy²⁰), and sampling variability. These issues made combining all the survey estimates and examining trends over time problematic. Therefore, the data from each survey type were first examined separately to establish that they were not contradictory to each other; then they were combined in an

analysis similar to the one used for per capita consumption.

Since 1965, the National Health Interview Surveys (NHIS) have been the surveillance system of choice for smoking prevalence in the United States.^{10,19} Although the NHIS provide only estimates at the regional level, California is the largest state in the Western region. Thus, the NHIS sample sizes for California smoking prevalence estimates are reasonably large. The 1978-1994 NHIS were used for an initial assessment of preprogram and postprogram smoking prevalence trends in California and the rest of the United States. The NHIS conducted before 1978 were excluded, either because they did not include persons as young as 18 years (1976 and 1977 surveys) or because smoking status information was missing for more than 1.5% of respondents (1974 survey). The 1992 NHIS was excluded because it was cancelled suddenly at the midpoint of fieldwork with unknown consequences to response rate and representativeness. The paucity of data points after the start of the California Tobacco Control Program results in insufficient statistical power to precisely evaluate changes in trend or to compare California with the rest of the United States. Nevertheless, we used the piecewise linear regression approach to determine whether these data appeared consistent with the postprogram change in slope identified from the per capita consumption data.

Since 1989, there have been several large-scale population surveys conducted

Table 1.—Survey Data Used for Analysis of Smoking Prevalence (Samples Sizes and Response Rates)*

Year	NHIS†		CTC‡		BRFS/ CATS	CPS	
	California	United States— California	California	California	California	California	United States— California
1978	1178	10 399
1979	2578	21 535
1980	1112	9303
1983	2309	20 109
1985	3572	30 058
1987	5064	39 059
1988	5030	39 203
1990	4898	36 206	65 139 (75)‡
1991	5747	39 029	...	2995 (60)§
1992	21 872 (73)	3982 (62)	(September) 8081	97 856 (89)¶	...
1993	2668	18 360	63 269 (70)	7371 (60)	(January) 8272	96 831 (89)	...
1993	(May) 8151	96 769 (86)	...
1994	2382	17 356	...	8169 (62)
1995	8207 (53)	(September) 5966	77 570	...
1996	78 337 (53)	8165 (49)	(January) 5780	69 375	...
1996	(May) 6041	70 164	...

*NHIS indicates National Health Interview Surveys; CTC, California Tobacco Surveys; BRFS, Behavioral Risk Factor Surveys; CATS, California Adult Tobacco Surveys; and CPS, Current Population Surveys. Ellipses indicate data not applicable. Numbers in parentheses are response rates in percent, where available.

†Although not published, the NHIS claim a response rate exceeding 86%.

‡For CTC, this is the number of screening interviews completed as a percentage of all households targeted (including telephone numbers for which it was unknown whether the number was that of a residence or a business).

§For BRFS/CATS, this is the product of the household response rate (see CTC) and the interviewee response rate.

¶For CPS, this is the percentage of respondents targeted for smoking supplement interviews for whom the interview was completed. For smoking status, the response was higher because proxy information is included.

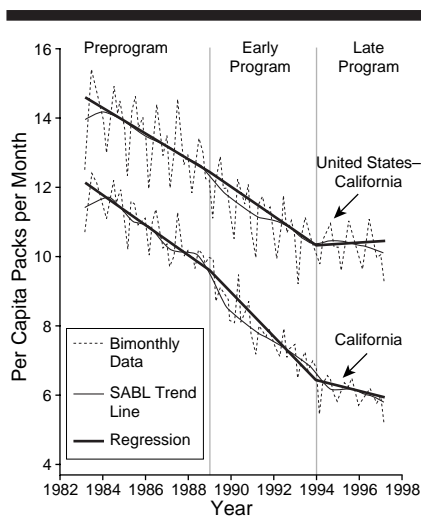


Figure 1.—Trends in monthly per capita adult (≥ 18 years) cigarette consumption in California and the rest of the United States.

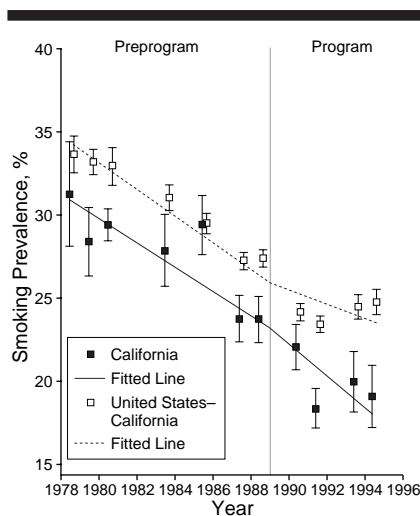


Figure 2.—Trends in adult (≥ 18 years) smoking prevalence in California and the rest of the United States from National Health Interview Surveys data. Error bars indicate SEs.

Table 2.—Summary of Decreases in per Capita Cigarette Consumption*

Period	California		Rest of the United States	
	Rate of Decline, Pack (SE)	Packs/mo	Rate of Decline, Pack (SE)	Packs/mo
Pre-1989 (preprogram)	-0.42† (0.03)	9.7	-0.36 (0.02)	12.5
1989-1993 (early period)	-0.64‡§ (0.03)	6.5	-0.42 (0.03)	10.4
1994-1996 (late period)	-0.17†§ (0.07)	6.0	0.04§ (0.06)	10.5

*The per capita adult (≥ 18 years) cigarette consumption in December 1998, December 1993, and December 1996 were estimated from piecewise linear model.

† $P < .01$, California vs the rest of the United States.

‡ $P < .001$, California vs the rest of the United States.

§ $P < .001$, change from previous period.

in California on a periodic basis. The California Tobacco Surveys (CTS) were the largest of these and specifically funded to evaluate the California Tobacco Control Program. To date, they have been conducted in 1990, 1992, 1993, and 1996. The CTS are random-digit-dialed telephone surveys of households in California.^{20,21} A brief screening interview was conducted with a household adult to enumerate all residents and to obtain demographic information, including age and smoking status. Both self and proxy data from the screening interview were included. The Behavioral Risk Factor Surveys (BRFS) have been conducted in California every year since 1984.²² Beginning in 1991, the sample size was increased, and quality control procedures were established (using California Tobacco Control Program funds) to make this survey a potentially useful tool for assessing trends. Beginning in 1993, a special smoking supplement (modeled after the CTS), the California Adult Tobacco Surveys (CATS), was attached to the BRFS.²³ Finally, the national Current Population Surveys (CPS),^{24,25} conducted in September 1992, January and May 1993, September 1995, and January and May 1996, were de-

signed to provide state-specific estimates. The 1985 and 1989 CPS also had smoking-status questions, but these data were missing from more than 1.5% of respondents, so they were not included in our analyses. The various surveys with sample sizes and response rates (if known) are summarized in Table 1.

Smoking Status.—Respondents to all surveys were asked if they (or the person they were responding for) had smoked at least 100 cigarettes in their lifetime and whether they smoked now. In a few of the more recent surveys (NHIS since 1993, CPS since 1992, and BRFS/CATS since 1994), respondents were asked if they currently smoked “everyday,” “some days,” or “not at all.” The everyday and some days smokers were considered to “smoke now.” The CTS computed smoking prevalence based on the smoke now question. The other surveys also required that smokers report smoking at least 100 cigarettes in their lifetimes before being asked the current smoking question.

Weighting and Variance.—Survey weights, provided with each of the data sets, were constructed to account for the probability that an individual is sampled

and to adjust for differential nonresponse using poststratification procedures. The poststratification procedures for the various surveys were based on different demographic subgroups, and population totals for these subgroups were from different years. Because the demographics of the population changed between 1978 and 1996, data from each survey were standardized (direct method for weighted prevalence) according to sex, age (18-29, 30-39, 40-49, 50-59, and ≥ 60 years), race (white, nonwhite), and educational level (no college, some college). Variance estimates were generated for each estimate (data available from the authors) so that 95% confidence intervals could be computed and so that rates of change in prevalence estimated from each survey during the postprogram period could be evaluated (data available from the authors).

Finally, once it was established that trends from the various surveys were not contradictory, all the data were combined into one piecewise linear regression analysis, using the same model form as for the per capita consumption data. This analysis, though still problematic for all the reasons discussed above, provides a summary of the prevalence trends that can be examined against the per capita consumption data.

RESULTS

Per Capita Cigarette Consumption

Figure 1 shows the bimonthly raw data, the SABL deseasonalized trends, and the fitted trends from the piecewise linear model for monthly per capita cigarette consumption in California and the rest of the United States. Before the California Tobacco Control Program began, the annual rate of decline in monthly per capita cigarette consumption was -0.42 pack, which was significantly ($P < .01$) more rapid (more negative) than the rate of decline of -0.36 pack in the rest of the United States. From January 1989 through December 1993, the annual rate of decline in monthly per capita consumption increased significantly (became more negative) in California, from -0.42 to -0.64 pack ($P < .001$) or by a factor of 52%. There was a slight but insignificant increase in the rate of decline during this period in the rest of the United States. The rate of decline was significantly ($P < .001$) greater in California (by a factor of 52%) than in the rest of the United States during this period. These results are summarized in Table 2. From January 1994 through December 1996, the annual rate of decline in monthly per capita consumption changed significantly ($P < .001$) in California to -0.17 pack, which was only 28%

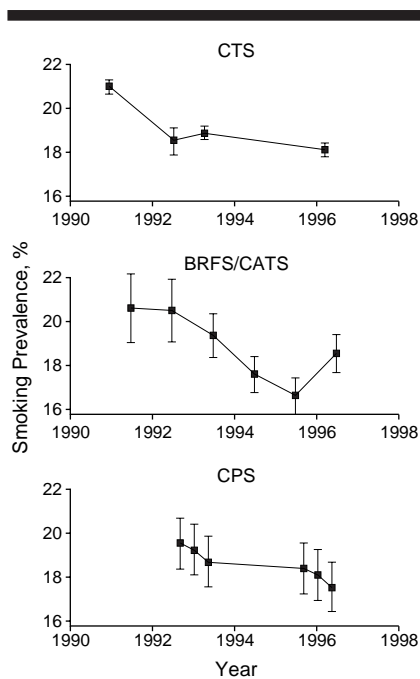


Figure 3.—Top, Trends in adult (≥ 18 years) smoking prevalence in California from California Tobacco Surveys (CTS) data. Middle, Behavioral Risk Factor Surveys and California Adult Tobacco Surveys (BRFS/CATS) data. Bottom, Current Population Surveys (CPS) data. Error bars indicate 95% confidence intervals.

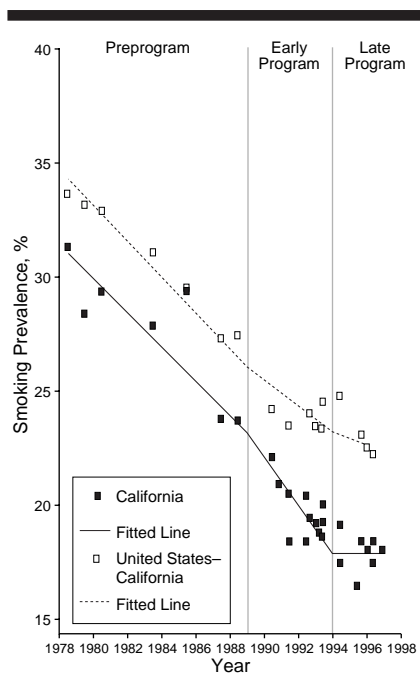


Figure 4.—Trends in adult (≥ 18 years) smoking prevalence in California and the rest of the United States computed from all survey sources combined.

Table 3.—Summary of Decreases in Smoking Prevalence*

Period	California		Rest of the United States	
	Rate of Decline, % (SE)	Smoking Prevalence, %	Rate of Decline, % (SE)	Smoking Prevalence, %
Pre-1989 (preprogram)	-0.74 (0.12)	23.3	-0.77 (0.09)	26.2
1989-1993 (early period)	-1.06†‡ (0.17)	18.0	-0.57 (0.14)	23.3
1994-1996 (late period)	0.01‡ (0.21)	18.0	-0.28 (0.26)‡	22.4

*Adult (≥ 18 years) smoking prevalence in December 1998, December 1993, and December 1996 were estimated from piecewise linear model.

† $P < .05$, California vs the rest of the United States.

‡ $P < .001$, change from previous period.

of the rate of decline identified for January 1989 through December 1993 and only 40% of the preprogram rate of decline. In the rest of the United States, the annual rate of change in monthly consumption halted altogether (only 0.04 pack), which was a significant change from the earlier period ($P < .001$). The rate of decline in California, although considerably diminished, was still significantly ($P < .01$) greater than the essentially zero decline in the rest of the United States for this period.

In December 1988, before the California Tobacco Control Program began, monthly per capita cigarette consumption, 9.7 packs, was less than the 12.5 packs for people in the rest of the United States, by a factor of 22%. In December 1996, the per capita consumption of 6.0 packs was 43% less than the 10.5 packs seen in the rest of the United States.

Cigarette Smoking Prevalence

Change From Preprogram.—The NHIS data from California and the rest of the United States are presented in Figure 2. The rate of decline in California before the start of the California Tobacco Control Program was -0.72% (SE, 0.19%) per year, which was not statistically different from the rate of decline in the rest of the United States, which was -0.79% (SE, 0.10%) per year. After 1988, the rate of decline in California increased (more negative) to -0.98% (SE, 0.35%) per year. This 36% increase in the rate of decline was not statistically significant because there were too few estimates to provide sufficient precision. In the rest of the United States, the rate of decline was -0.42% (SE, 0.20%) per year, but the decrease (less negative) from the earlier rate of decline was also not statistically significant. The overall rate of

decline in the rest of the United States from 1978 to 1994 was -0.67% (SE, 0.07%) per year, and in California it was -0.79% (SE, 0.11%) per year.

Changes During Program Period.

Figure 3 gives the standardized smoking prevalence estimates with 95% confidence intervals from the various surveys conducted in California in the postprogram period. The top panel presents CTS estimates. The decline ($\pm 95\%$ confidence interval) from $20.9\% \pm 0.5\%$ in 1990 to $18.9\% \pm 0.5\%$ in 1993, $-0.85\% \pm 0.30\%$ per year, was significantly greater ($P < .001$) than the rate of decline of $-0.22\% \pm 0.17\%$ per year from 1993 to a prevalence of $18.1\% \pm 0.4\%$ in 1996.

The middle panel of Figure 3 shows the standardized smoking prevalence estimates from the BRFs/CATS. In 1991, the prevalence estimate was $20.5\% \pm 1.6\%$, which decreased to $17.6\% \pm 0.8\%$ by 1994; this represents a rate of decline of $-0.99\% \pm 0.59\%$ per year. By 1996, the prevalence estimate was $18.5\% \pm 0.9\%$, which was a rate of increase of $0.47\% \pm 0.60\%$ per year from 1994. The difference between the rate of decline in the early period and the rate of increase in the later period was statistically significant ($P < .001$).

The bottom panel of Figure 3 shows the standardized CPS data for California. For example, smoking prevalence was $18.7\% \pm 1.1\%$ in May 1993 and $17.5\% \pm 1.1\%$ in May 1996, which represented a rate of change of $-0.39\% \pm 0.55\%$ per year, which was not statistically different from zero.

In summary, the CTS data indicate a slower rate of decline in the later period as compared with the earlier period, the BRFs/CATS indicate a decline in the early period and an increase in the later period, and the CPS showed no significant change in the later period.

Combined Analysis.

Since data from the California surveys did not contradict the observation that a decline occurred in the early period that was not maintained later, the data from all of them, including the NHIS, were combined into a single analysis similar to the one performed on the per capita cigarette consumption data. Figure 4 shows all the data points and the resulting fitted regression lines, and Table 3 presents the rates of decline and prevalence estimates derived from the model. Before the California Tobacco Control Program began in 1989, smoking prevalence declined at about the same rate in California (-0.74% per year) and the rest of the United States (-0.77% per year). The rates of decline were not statistically different, but prevalence in California was below that for the rest of the United States. The rate of decline increased (became more negative) significantly

Table 4.—Funding for the California Tobacco Control Program and the Advertising and Promotion of Cigarettes in California*

Expenditures Targeted at Tobacco Use in California, \$ Millions ³¹								
Budget Category	Fiscal Year							Total, 1989-1996
	1989-1990	1990-1991	1991-1992	1992-1993	1993-1994	1994-1995	1995-1996	
Mass media	14.3	14.3	16.0	15.4	12.9	12.2	6.6	91.7
Local lead agency	35.6	35.4	14.5	17.8	13.5	16.4	10.2	143.4
Competitive grants	3.3	49.7	1.1	27.5	15.1	10.9	9.7	117.3
Local schools	32.6	32.6	24.3	23.3	19.6	16.8	15.3	164.5
Actual Totals	85.8	132.0	55.9	84.0	61.1	56.3	41.8	516.9

Expenditures by the Tobacco Industry in California, \$ Millions ³²								
Budget Category	Calendar Year							Total, 1989-1995
	1989	1990	1991	1992	1993	1994	1995	
Advertising	111	114	112	99	94	89	82	701
Incentive to merchants	100	102	116	151	156	168	187	980
Promotional items	122	149	207	252	332	210	201	1473
Other	28	34	31	22	22	17	19	173
Totals	361	399	466	524	604	484	489	3327

*Data are from Balbach et al³¹ and the US Federal Trade Commission.³² Dollar amounts are not adjusted for inflation.

($P < .001$) in California after the program began, whereas in the rest of the United States it did not. As a result, the rate of decline from 1989 through 1993 was significantly greater ($P < .05$) by a factor of nearly 90% in California (-1.06% per year) than in the rest of the United States (-0.57% per year). After 1993, the rate of decline in California and in the rest of the United States was not significantly different from zero, and in both instances, the change in the rate of decline was significantly less ($P < .001$) than in the preceding period. Obviously, these late program trends were less than the preprogram rates of decline.

From the fitted model (Table 3), adult smoking prevalence in December 1988 was 11% lower than in the rest of the United States, and by December 1996 it was 20% lower.

COMMENT

Analysis of trends in per capita cigarette consumption indicates that the start of the California Tobacco Control Program in 1989 was associated with a 50% more rapid rate of decline that was unique to California. After 1993, the rate of decline in per capita consumption in California slowed to less than one third of the rate observed from 1989 through 1993 and to less than one half of the rate of decline observed before the program began. However, this post-1993 rate of decline was still significantly more rapid in California than in the rest of the United States, for which the decline in consumption halted.

The smoking prevalence trends from the combined survey data are fairly consistent with the changes observed in per capita consumption. The initiation of the program was associated with a 36% increase in the rate of decline of smoking prevalence, which was nearly twice the rate of decline identified for the rest of

the United States. However, from 1994 through 1996, there was no identifiable decline in smoking prevalence either in California or the rest of the country. In California, smokers may be reducing their consumption rather than quitting, while it appears that in the rest of the United States they are doing neither.

It is important to the future of tobacco control in general and to the California Tobacco Control Program specifically to hypothesize why the loss of the early program success occurred. Additional analyses will be required to fully understand the influences of various factors. Did the program lose its effectiveness because it failed to introduce new and innovative approaches to interest the population in tobacco control, or did it suffer from countermeasures used by the tobacco industry? The fact that the tobacco industry lowered prices for premium brands of cigarettes in 1993²⁶ could be at least partly responsible. Also, it is possible that lower funding for the Tobacco Control Program or increased expenditures by the tobacco industry for advertising and promotion played a role. Finally, the tobacco industry engaged in a variety of political activities, which may have influenced the level of commitment of the state administration and legislature to the California Tobacco Control Program. These possibilities will be examined in some detail below.

There were several tobacco control strategies that were emphasized during the early phase of the California Tobacco Control Program. One was support for the adoption of ordinances at the local level that restricted or banned smoking in indoor workplaces and public places. The percentage of indoor workers reporting smoke-free workplaces increased during the early years of the program but continued to increase even more later.²¹ California Assembly Bill 13 was enacted in

January 1994, and it prohibited smoking statewide in 1995 in all indoor workplaces except bars, taverns, and casinos. If smoke-free workplaces encourage smokers to reduce their consumption or quit, the effect on per capita consumption and prevalence should have been evident throughout the entire program period. Another important element of the very early California Tobacco Control Program was a well-funded and effective media campaign.²⁷ Antismoking television ads focused on the duplicity of the tobacco industry and the dangers of secondhand tobacco smoke. Funding for the media campaign was vetoed by the governor in 1992 and later restored,²⁸ but it was reinstated at a lower level than previously (Table 4). Also, the administration has been accused of "watering down" the antismoking advertising.²⁷

Economic theory and empirical data have suggested that cigarette price is a major determinant of smoking behavior.^{10,29} However, recent data suggest that when tobacco control programs are in place, the price elasticity of demand may be altered (S. Emery, E. A. Gilpin, J. P. Pierce, unpublished data, 1998).³⁰ In 11 of the 14 states that participated in the American Stop Smoking Intervention Study (ASSIST), where there was a decrease in the real price of cigarettes from 1992 to 1994 (which spanned the date when the tobacco companies lowered the price of cigarettes), per capita cigarette consumption did not increase as economic theory would predict.³⁰ In the remaining 3 ASSIST states, the increase in consumption was very minimal. In the non-ASSIST states (excluding California), all showed a decrease in the real price of cigarettes from 1992 to 1994, and over half showed the expected increase in per capita consumption. A recent analysis of changes in cigarette price and per capita consumption in Cali-

fornia showed that when the excise tax increase went into effect the percentage change in per capita consumption (12.2%) closely matched what economic theory would predict from the resultant change in cigarette price (11.8%) (S. Emery, E. A. Gilpin, J. P. Pierce, unpublished data, 1998). The increased tax was the first element of the California Tobacco Control Program implemented, and as additional programs were introduced, the expected relationship between price and consumption disappeared. Importantly, per capita consumption decreased 8.5% from 1993 to 1994, when the price decrease would have predicted a 4.9% increase. The price of cigarettes has remained stable from 1993 through 1996. These results suggest that price alone cannot be responsible for the loss of effect of the California Tobacco Control Program.

The level of funding for the California Tobacco Control Program has varied over the course of the program.³¹ Expenditure data for the Health Education Account (which funds the Tobacco Control Program) are shown in Table 4 (top) for the line items of mass media, local lead agencies, competitive grants and school programs, and other expenses.³¹ The funds allocated for administration and evaluation, which averaged about 5% of the total budget each year, are not included. There is variation over time, which suggests that money from 1 year was brought forward to the next, particularly in the category of competitive grants. From fiscal year 1989-1990 to fiscal year 1992-1993, the average annual expenditure was \$85.5 million, or \$3.35 per capita per year (considering a population of 25.5 million people in California >12 years old). However, beginning with fiscal year 1993-1994, there was a marked reduction in program funding. The annual average was \$53.0 million, or \$2.08 per capita, which was a reduction of 40% from the early years of the program. This reduction in the level of effort aimed at reducing smoking in California is a possible explanation for the loss of program effect.

Concurrent increases in the amount of money the tobacco industry spent to promote cigarette use may have exacerbated the problem. The lower portion of Table 4 shows the estimated amount spent for each of several line-item categories as reported to the Federal Trade Commission.³² Traditional print media and billboard expenditures constitute the advertising category. We combined the categories for "coupons," "retail value added," and "specialty item distribution" into one category labeled "promotional items." The category "incentives to merchants" includes the Federal

Trade Commission category that they designate as "promotional allowances," which covers expenditures to encourage wholesalers and retailers to stock and promote particular cigarette brands. We assumed that the tobacco industry did not specifically target California with its marketing dollars and that California received a share of the industry's national promotion and advertising effort in proportion to its population (approximately 10%). This assumption is likely to be conservative, since the tobacco industry may have specifically increased their promotional efforts in California to counteract the Tobacco Control Program when there were early indications that it was having an impact.^{7,33} Furthermore, the data on expenditures for advertising and promotion are for manufactured cigarettes only and do not include other tobacco products, such as cigars. The amount spent on advertising has decreased over time, but the amount spent on incentives to merchants has increased markedly, as has the budget allocation for promotional items. From 1989 to 1993, it is estimated that the tobacco industry spent an average of \$437 million annually, or \$17.14 per capita, in California; thereafter, it spent an average of \$525 annually, or \$20.59 per capita, an increase of 20% from the earlier period. In the earlier period, the industry outspent the program by approximately \$5 to \$1 (\$17.14 to \$3.35 per capita), and in the period from 1993 to 1996, it outspent the program by nearly \$10 to \$1 (\$20.59 to \$2.02 per capita).

The cuts made by the administration and legislature in the California Tobacco Control Program budget appeared to be about the same in each budget category of the Health Education Account, except in 1995-1996, when the expenditure for the media program was halved. The decision by the administration to divert funding for the program could not have been justified on the basis that the program was considered to be performing above expectations. The 1993 interim assessment of the program suggested that since early indications demonstrated that the program was having an effect on smoking behavior, this effect needed to be increased by 50% more in order for the program to meet its goal for the year 2000.³⁴ The goal was to decrease adult smoking prevalence by 75% within a 12-year period. Further, the decision to reduce expenditures for the program was made in the face of active lobbying by health advocacy organizations and lawsuits against the administration brought by the American Lung Association, Americans for Nonsmokers' Rights, the American Cancer Society, and the American Heart Association.

Recently, a set of internal memoranda from the Tobacco Institute surfaced. These internal memoranda, written in 1990, outlined a strategic plan for combatting the California Tobacco Control Program.³⁵ The plan called for lobbying the California legislature to intervene, encouraging and supporting minority organizations to oppose the program, convincing the health services director to pull or modify media messages that reflected poorly on the industry, and encouraging the governor to intercede against the program. There is evidence that these strategies were used and met with some success. As mentioned previously, the governor initially vetoed the media budget in 1992, although he reconsidered following significant public pressure.²⁸ Antismoking media funding was reduced by 50% for 1995-1996, and anti-industry media spots were short-lived.²⁷ Furthermore, tobacco industry campaign contributions to legislature candidates, other elected officials, political parties, and political party committees totaled over \$1.5 million in 1995-1996; this was a 70% increase compared with the level of such contributions in the 1993-1994 election cycle.³¹ On a per legislator basis, members of the California legislature received twice as much money as did members of the US Congress, even though California is not a tobacco-producing state.³¹ The slowing of the decline in smoking in recent years may well be a result of these political counterstrategies by the tobacco industry.

The California Tobacco Control Program has confirmed findings from earlier studies³⁻⁷ that large health promotion programs can have a major influence on smoking behavior. Similar programs have been initiated in Massachusetts (1993), Arizona (1995), and Oregon (1996). Furthermore, the Robert Wood Johnson Foundation and the Centers for Disease Control and Prevention have provided limited support for the development of similar tobacco control programs in other states.^{36,37} Only the relatively well-funded Massachusetts program³⁸ has been in effect long enough to potentially confound the results of our analyses. However, Massachusetts represents a small percentage of the US population, so it was not surprising that a reanalysis of the data without it did not change the results.

In conclusion, the California Tobacco Control Program clearly lost its original positive effect on reducing smoking, which must be of considerable concern to the public health movement. In this article, we have discussed some of the factors that might have been associated with the loss of effect. The Tobacco Institute memoranda³⁵ revealed that the

tobacco industry decided early on to actively oppose any potentially effective tobacco control efforts. Traynor and Glantz³⁹ and Heisner and Begay⁴⁰ have outlined the political difficulties faced in developing and maintaining an effective tobacco control program in such a climate. Despite active industry opposition and political influences, it is urgent that the public health community determine how the California Tobacco Control Program can be modified to regain its original momentum.

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Dr Fouts is completely on target. The nidus of the pathology is among the permanent members of the UN Security Council and especially in Washington, DC, which has initiated and been a key player in shaping the atomic age. The concept of nonproliferation was flawed from the outset. The superpowers retained the right to keep testing and modernizing weapons notwithstanding their huge arsenals and their qualitative and quantitative dominance. At the same time, the nonweapons states were expected permanently to forswear any intent of acquiring nuclear military capabilities. Such a double standard could not be permanently enforced. The options were 2: either eradicate the malignant virus, nuclearism, or witness its spread. The events in South Asia confirm the bankruptcy of nuclear apartheid policies.

From the vantage point of our long-range national security, nuclear weapons offer nothing except a dire threat. With the United States the strongest nation on earth by every conceivable military, economic, and scientific measure, legitimization of nuclear weapons detracts rather than bolsters our safety. They are potential equalizers, enabling the weak to inflict unacceptable damage on the strong. Nuclear know-how is everywhere. Terrorists are aware that a nuclear bomb affords "the cheapest and biggest bang for the buck." No blackmail would be as compelling as holding an entire city hostage. Few societies are more susceptible to their malevolent consequences than the rich, urbanized, highly developed, industrialized North, foremost the United States. It is already the object of growing global resentment and envy. One may surmise that it will increasingly be the target for terrorist acts.

Physicians for Social Responsibility and the International Physicians for the Prevention of Nuclear War have long argued that nuclear abolition is a "number one public health priority." Political leaders respond not to historic imperatives but to the clamor of their constituencies. Only an upsurge of public opinion in favor of nuclear abolition will sway Washington to reconsider a flawed, immoral, and dangerous course.

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Proximal Myopathy Associated With Inhaled Steroids

To the Editor: A number of undesirable systemic effects have been reported with the use of inhaled corticosteroids, including cataracts, suppression of the hypothalamic-pituitary-adrenal axis, osteoporosis, and possibly glaucoma.^{1,2}

Report of a Case. A 37-year-old woman who had a seizure disorder that was well controlled with carbamazepine therapy began to use a beclomethasone nasal inhaler, 42 µg of drug per nasal pump unit, 1 to 2 sprays in each nostril twice daily, for

the treatment of seasonal allergic rhinitis. Four weeks later, the patient began to experience tightness in her thighs and increased fatigability when she climbed stairs or walked distances that were previously readily tolerated. Findings on a general medical examination were normal. Neurologic evaluation revealed a mild to moderate reduction in strength (4½/5 range) in neck flexion and bilaterally in shoulder abduction and hip flexion. Muscle weakness had not been noted in the past. Findings on the remainder of the neurologic examination, including muscle strength elsewhere, cranial nerve function, and deep tendon reflexes, were within normal limits. Erythrocyte sedimentation rate, thyroid function test results, cortisol and serum creatine kinase levels, and antinuclear antibody titer were normal. Serum carbamazepine level was in the midtherapeutic range. Electromyography (EMG) showed normal resting activity, without fibrillations or trains of myotonic potentials. Voluntary contraction of proximal muscles showed an increased recruitment pattern and diminished average size of units, consistent with myopathy. The possibility of steroid-induced myopathy was considered, and the use of inhaled steroids was discontinued. At the time of follow-up 1 month later, the patient was free of exercise-induced muscle tightness and reported normal exercise tolerance and strength. Examination revealed full motor strength, and repeat EMG showed normal unit morphology and recruitment. Without steroids, she has had no recurrence of weakness for 2 years.

Comment. Clinicians may need to consider proximal myopathy among the possible undesirable systemic adverse effects that can occur with the use of inhaled corticosteroids.

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1. Garbe E, Suissa S, LeLorier J. Association of inhaled corticosteroid use with cataract extraction in elderly patients. *JAMA*. 1998;280:539-543.
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CORRECTIONS

Authors' Names Omitted: In the Letter entitled "Acetaminophen and Risk Factors for Excess Anticoagulation With Warfarin," published in the August 26, 1998, issue of *THE JOURNAL* (1998;280:697), authors Daniel E. Singer, MD, and Steven J. Skates, PhD, were omitted.

Incorrect Figure Key: In the Original Contribution entitled "National Use and Effectiveness of β-Blockers for the Treatment of Elderly Patients After Acute Myocardial Infarction," published in the August 19, 1998, issue of *THE JOURNAL* (1998;280:623-629), there was an incorrect figure key. On page 627, the key to Figure 1 should have read "Prescribed Use, % (No.) of States."

Author's Name Omitted: In the Original Contribution entitled "Has the California Tobacco Control Program Reduced Smoking?" published in the September 9, 1998, issue of *THE JOURNAL* (1998;280:893-899), Arthur J. Farkas, PhD, was omitted from the byline.

Reducing Treatment Delay and Improving Diagnostic Accuracy for Patients With Acute Stroke

To the Editor: After reading the thoughtful and provocative study by Dr Schriger and colleagues,¹ we came away unsure about the generalizability of their findings. The authors carefully showed that the accuracy of computed tomographic (CT) interpretation depended on the difficulty of scan type. The average correct score for interpretation ranged from 98% for “easy hemorrhages” to only 48% for “difficult acute infarctions.” They also provided the frequency distribution of scan types shown to the physician readers. To evaluate the external validity of their results, however, we need to know the distribution of scan types for the general population of patients with acute stroke, not just the patients in their protocol library.

The estimate of overall accuracy hinges on this information. If easier scans are more heavily represented in practice than in the study population, then physicians’ overall accuracy in CT interpretation is likely better than what the authors report. On the other hand, if difficult scans are more common in practice, accuracy would be worse than reported. Do the authors know whether the distribution of scan types in their study mirrors the mix seen among patients with stroke presenting to a typical emergency department?

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1. Schriger DL, Kalafut M, Starkman S, Krueger M, Saver JL. Cranial computed tomography interpretation in acute stroke: physician accuracy in determining eligibility for thrombolytic therapy. *JAMA*. 1998;279:1293-1297.

To the Editor: “Timely and appropriate treatment of acute stroke” will not be delivered to patients until neurologists, the stroke specialists, receive adequate training in neuroimaging during residency or through fellowships. This is what is really “missing from this picture,” in answer to the question asked in the Editorial in *THE JOURNAL* issue devoted to stroke.¹

Most neurologists on both sides of the Atlantic have relinquished their responsibility to interpret images of the brain to neuroradiologists and even to general radiologists. The ominous consequences of this behavior are already felt in the new thrombolytic therapies for stroke, which requires simple but accurate interpretation of a brain CT scan to rule out hemorrhage and early infarction. The Food and Drug Administration has approved the general use of recombinant tissue-type plasminogen activator (rtPA) in stroke in the United States even in hospitals without a neuroradiologist. Because only 1800 neuroradiologists serve 6000 US hospitals, most “emergency” head CT scans will be interpreted

by neurologists or general radiologists. Yet, the results of 2 recent consecutive surveys of American Academy of Neurology members indicate that most US neurologists do not feel qualified to interpret neuroimaging studies (40% for CT and 55% for MRI) and most believe that currently neurology residents received insufficient training in neuroimaging (76% in the first survey and 80% in the second) (unpublished data, J. Masdeu, MD, 1996). The report by Dr Schriger and colleagues² provides statistical confirmation of the inaccuracy of CT interpretation by neurologists (as well as radiologists and emergency physicians); only 40% of neurologists and 52% of radiologists achieved 100% sensitivity for identification of hemorrhage.

On the European side, the European Cooperative Acute Stroke Study (ECASS) was marred by 109 (17.4%) major protocol violations; the majority of these were CT exclusions (n = 66), including major signs of early infarction (n = 52).³ ECASS recommended postponing general use of rtPA in Europe and remarked, “Neurologists (and neuroradiologists) who plan to participate in further trials of thrombolytic therapy . . . must be trained . . . on pathomorphological signs provided by early CT.” Furthermore, ECASS stated that “measures have to be taken to reduce these adverse results (brain hemorrhage and mortality rates), with additional information about occlusion site, collaterals, and remaining perfusion,” using modern imaging techniques, such as magnetic resonance imaging diffusion and perfusion, and echoplanar magnetic resonance angiography.

Contrary to the above recommendations, the American Board of Medical Specialties has recently approved a new certificate of added qualifications (CAQ) in neuroradiology restricted to general radiologists and effectively excluding neurologists from eligibility.⁴ Neurologists thus excluded from the CAQ in neuroradiology can still be credentialed in neuroimaging by the American Society of Neuroimaging (ASN). That only 20 neurologists decided to take the ASN 1998 written examination attests to the inadequate training in neuroimaging currently available for neurologists in the United States.

Before new thrombolytic therapies can gain widespread use in the treatment of stroke, the treating neurologists must be em-

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Edited by Margaret A. Winker, MD, and Phil B. Fontanarosa, MD, Senior Editors.

powered with advanced neuroimaging skills and not just CT interpretation.

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1. Fontanarosa PB, Winker MA. Timely and appropriate treatment of acute stroke: what's missing from this picture? *JAMA*. 1998;279:1307-1308.
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To the Editor: Dr Schriger and colleagues¹ have done a major disservice by incorrectly concluding that board-certified general radiologists are incapable of identifying intracranial hemorrhage on cranial CT and therefore are unable to determine eligibility for thrombolytic therapy.

Their sample of 36 board-certified general radiologists is flawed. There is no documentation to indicate that those chosen to participate represent an appropriate sample of the type of general radiologist that would be responsible for interpreting emergency cranial CTs in most clinical situations. Their data are therefore suspect, and the authors are correct in stating that "our convenience sampling method is the main threat to the external validity of the study." More than a threat, this limitation destroys any credibility to their conclusion.

No one would argue that the additional training of a neuro-radiologist makes that individual's skills in that specialized area of imaging better than most general radiologists. But this study did not prove that general radiologists who interpret emergency cranial CT examinations on a regular basis are not qualified to recognize the findings of an acute stroke.

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1. Schriger DL, Kalafut M, Starkman S, Krueger M, Saver JL. Cranial computed tomography interpretation in acute stroke: physician accuracy in determining eligibility for thrombolytic therapy. *JAMA*. 1998;279:1293-1297.

To the Editor: Drs Fontanarosa and Winker¹ contend that several key components are missing in the current stroke treatment picture. Reducing time from stroke onset to initiation of thrombolytic therapy must address 2 sources of delay. The first, patient-generated delay, requires further research to determine reasons and to identify educational strategies to prevent delays. Public health service organizations are eagerly implementing programs to reeducate the public as to the emergency nature of strokes. The second source of delay, that generated by the hospital, can be reduced by organizational interventions, including computer automation.

Evaluating the quality of stroke services provided by a hospital can be a difficult task for the public as well as for hospital administrators. Therefore, I suggest the following stroke service scorecard to gauge an institution's stroke service infrastructure. Does the hospital have:

- emergency department, computed tomographic (CT) scanning, and radiologist interpretation services on a 24-hour-a-day basis?

- organizational structures such as a stroke team, a stroke unit (physical or virtual), a measurable stroke outcome improvement program to be accomplished over time, and a stroke research program?

- information on its "door-to-vein" time (the time that it takes to start stroke treatment after the patient arrives in the emergency department) and on its long-term stroke-related death and disability rate?

Positive responses to these questions demonstrate that the institution has invested time and resources to organize its stroke service. However, these services are so expensive that few hospitals can afford them. Therefore, a regional system of stroke care, parallel to that of trauma care, may be required.

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1. Fontanarosa PB, Winker MA. Timely and appropriate treatment of acute stroke: what's missing from this picture? *JAMA*. 1998;279:1307-1308.

To the Editor: Dr Pancioli and colleagues¹ report a study on public perception of stroke warning signs and knowledge of risk factors; this is important to decrease the hospital arrival time for effective stroke treatment. The American Heart Association Stroke Council recommends thrombolytic therapy within 180 minutes of symptom onset.²

We are conducting an observational study on the time to presentation of stroke patients. Preliminary data at 7 months confirm that the gaps in the public's knowledge of stroke can contribute to a significant delay in admission to the hospital.

We enrolled 397 consecutive patients with stroke (176 men and 221 women; mean \pm SD age, 76 \pm 13 years); 347 (87.4%) had ischemic stroke and 50 (12.6%) had hemorrhagic stroke as assessed by cranial CT. The median time from symptom onset to arrival in the emergency department was 180 minutes (mean [SD], 413 [734] minutes; range, 1-5760 minutes). A total of 196 patients (49.4%) presented after 180 minutes. Many of these patients were older than 75 years; 138 (70.4%) of 196 patients presenting after 180 minutes were older than 75 years, while of all 397 patients, 262 (66.0%) were older than 75 years.

Based on the National Institute of Neurological Disorders and Stroke definition¹ of important warning signs of a stroke, we found that 95 (52.2%) of 182 patients with at least 1 established warning sign arrived after 180 minutes and 36 (37.9%) of 95 patients with 2 or more warning signs arrived after 180 minutes.

Like Pancioli and colleagues, we analyzed risk factors and found that 108 (54.0%) of 200 patients had a history of hypertension and 22 (53.7%) of 41 patients had diabetes. Fifty-one (52.0%) of 98 patients with 1 or more previous episodes of acute cerebrovascular disease arrived after 180 minutes.

There are several possible explanations for these delays: delay in deciding to seek medical attention, delay because of transportation from home to the hospital, or delay due to inability of elderly patients or patients living alone to communicate their

complaints.³ The lack of knowledge of stroke warning signs and risk factors can be an important reason for prolonged hospital arrival time.

In Italy, thrombolytic therapy is not yet approved for acute ischemic stroke. Public programs for better education of both patients and physicians should be encouraged and promoted in anticipation of this therapeutic option.

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1. Pancioli AM, Broderick J, Kothari R, et al. Public perception of stroke warning signs and knowledge of potential risk factors. *JAMA*. 1998;279:1288-1292.
2. Adams HP Jr, Brott TG, Furlan AJ, et al. Guidelines for thrombolytic therapy for acute stroke: a supplement to the guidelines for the management of patients with acute ischemic stroke: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke*. 1996;27:1711-1718.
3. Alberts MJ, Bertels C, Dawson DV. An analysis of time of presentation after stroke. *JAMA*. 1990;263:65-68.

In Reply: Drs Mines and Rosenzweig point out that the case mix of easy and difficult scans will greatly influence average performance and that our subjects' average performance may not be representative of these subjects' average performance on a series of scans of patients presenting in the community. While we suspect that intracerebral hemorrhages that are difficult to diagnose are less common in community practice than in our study, our study did not include other forms of intracranial bleeding (small epidural hematomas, chronic subdural hematomas, and subarachnoid bleeds) that may be more challenging to detect. Our collection of acute cerebral infarction scans included "easy" ones seldom seen in patients who meet criteria for thrombolysis (presenting within 3 hours of symptom onset). Therefore, the average difficulty of the infarctions shown to our subjects was likely much lower than the average difficulty of infarction scans seen in practice. Overall, we are uncertain whether our scan pool's case mix was harder or easier than that of a series of actual cases seen in the community. As an initial investigation, our study focused on characterizing the physicians' ability to interpret various kinds of scans rather than on measuring exact "actual practice" performance. We agree that better characterization of actual performance would be worthwhile.

Dr Campa uses our data plus American Academy of Neurology surveys of neurologists to lament that neurologists have mistakenly abandoned their role in CT interpretation, and this should be remedied. We stress that CT scans must be rapidly interpreted by qualified readers regardless of their specialty training.

Dr Stolz is concerned that results of our article will be misinterpreted to imply that general radiologists cannot read cranial CT scans in stroke patients. Our article repeatedly notes that there were qualified readers within each specialty. The problem is that board certification in the 3 specialties we tested does not guarantee that an individual physician is competent in this specific task. Patients need to know that whoever reads their scan is well

qualified to do so regardless of that physician's specialization. Our article highlights the importance of developing methods that ensure that scans are accurately read in a timely manner by qualified readers. We support any educational or systematic actions that foster this goal.

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In Reply: In our study, individuals whose age, race, and sex matched the population of patients with acute stroke in our region had a striking lack of knowledge of stroke warning signs and potential risk factors.

We have reported a similar lack of knowledge among stroke patients.¹ In that study, of the 125 patients with stroke who had documented times of symptom onset, there was no significant difference in hospital arrival within 3 hours between those who knew a sign, symptom, or risk factor of stroke and those who did not.

Dr Villa and colleagues provide data from a larger group of stroke patients in Italy. Particularly interesting are their correlations between the number of warning signs present, the presence of stroke risk factors, and the subsequent time to hospital arrival.

Studies such as these may help to identify independent, knowledge-based predictors of time to arrival. Defining such predictors of arrival time will be useful in creating educational programs designed to reduce delays to presentation.

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1. Kothari R, Sauerbeck L, Jauch M, et al. Patients' awareness of stroke signs, symptoms, and risk factors. *Stroke*. 1997;28:1871-1875.

In Reply: Dr Campa and Dr Palma express concerns similar to those raised in our Editorial: how to achieve the optimal balance between reducing time to initiation of acute treatment for patients with stroke and how to minimize the likelihood of adverse events from therapy.

We agree with Campa that all physicians involved in decision making regarding therapeutic interventions for patients with acute stroke should have training and expertise in interpretation of neuroimaging studies as well as with the clinical examination and management of patients with acute neurologic conditions. Neurologists play a critical role in the management of patients with stroke, and their prompt availability and early involvement have been shown to be related to better functional outcomes and shorter

hospitalizations.¹ Indeed, in proceedings from a National Institutes of Health–sponsored symposium, Grotta² recommends that the components of a primary stroke center should include a stroke team, with “a neurologist or other physician with stroke expertise on call within 15 minutes, either on site or by telemedicine.” However, in 2 previous studies of patients with stroke, the mean time from the patient’s arrival in the emergency department until evaluation by a neurologist was reported to be 123 minutes³ and 3.6 hours.⁴ Such delays, especially if coupled with delays in obtaining a CT scan, could reduce the efficacy of thrombolytic therapy, even if patients arrive at the hospital soon after symptom onset.

Palma reiterates the importance of having an organized plan and a multidisciplinary approach for responding effectively to the patient with acute stroke and also presents a scoring system for evaluating the infrastructure for stroke services provided by a hospital. Several points, such as time to initiation of stroke treatment⁵ and availability of an inpatient stroke unit,⁶ have been shown to be related to improved patient outcomes. However, the evidence for other items he proposes for evaluating quality is not so clear: Is 24-hour availability of a radiologist necessary if other physicians with expertise in neuroradiology are available? Does presence of a research program indicate a better stroke service if all the other criteria are met? Regardless, any such scoring system requires further study and validation with patient outcomes before it can be adopted as a relative measure of quality of stroke care among institutions or used in decisions regarding regionalization of care for patients with stroke.

Phil B. Fontanarosa, MD
Margaret A. Winker, MD
Senior Editors, *JAMA*

1. Davalos A, Castillo J, Martinez-Vila, et al. Delay in neurological attention and stroke outcome. *Stroke*. 1995;26:2233-2237.
2. Grotta JC. Acute hospital care: resource utilization. In: Marler JR, Jones PW, Emr M, eds. *National Institute of Neurological Disorders and Stroke, Proceedings of a National Symposium on Rapid Identification and Treatment of Acute Stroke*. Bethesda, Md: National Institutes of Health; August 1997:87-89. NIH publication 97-4239.
3. Bratina P, Greenberg L, Pasteur W, Grotta JC. Current emergency department management of stroke in Houston, Texas. *Stroke*. 1995;26:409-414.
4. Menon SC, Pandey DK, Morganstern LB. Critical factors determining access to acute stroke care. *Neurology*. 1998;51:427-432.
5. Donnan GA, Davis SM, Chambers BR, et al. Streptokinase for acute ischemic stroke with relationship to time of administration. *JAMA*. 1996;276:961-966.
6. Stroke Unit Trialists' Collaboration. Collaborative systematic review of the randomized trials of organized inpatient (stroke unit) care after stroke. *BMJ*. 1997; 314:1151-1159.

Sale of Goods From Physicians' Offices

To the Editor: It was gratifying to read the American Medical Association’s statement on the sale of non–health-related goods from physicians’ offices, generally condemning this practice. However, the sale of certain “health-related” items should also be scrutinized.

Anecdotally, I have noticed the sale of high-dose vitamin and mineral supplements from physicians’ offices. The excess mortality observed in the Alpha-Tocopherol Beta Carotene Cancer

Prevention Study² and the Beta-Carotene and Retinol Efficacy Trial³ in groups receiving vitamin supplementation for chemoprevention should give pause not only to physicians who recommend supplements in the absence of demonstrated benefit from well-designed randomized controlled trials but also to those who peddle these products.

Andrea Lyman, MD, MSCM
New York, NY

1. Council on Ethical and Judicial Affairs, American Medical Association. Sale of non–health-related goods from physicians’ offices. *JAMA*. 1998;280:563.
2. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. The effects of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *N Engl J Med*. 1994;330:1029-1035.
3. Omenn GS, Goodman GE, Thornquist MD, et al. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease. *N Engl J Med*. 1996;334:1150-1155.

In Reply: Dr Lyman raises an issue about which the Council on Ethical and Judicial Affairs is also concerned. In fact, the council is currently drafting guidelines that will address the practice of selling health-related goods. The council hopes to submit a report on this topic with recommendations regarding the ethical limits of such practices at the 1999 annual meeting of the American Medical Association. In the meantime, the council is encouraged to learn that individual physicians are also wary of the increasing prevalence of physicians who sell health-related goods from their offices.

Robert M. Tenery, Jr, MD
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American Medical Association
Chicago, Ill

Evaluating Reporting and Abstracts of Clinical Trials

To the Editor: We congratulate Dr Scherer and Ms Crawley¹ for the first published study to use the Consolidated Standards of Reporting Trials (CONSORT) statement as an instrument to systematically evaluate the quality of the reporting of clinical trials. However, we have some methodological concerns regarding this study.

The authors did not prospectively estimate the sample size that would have resulted in adequate power, and they fail to emphasize the low power as an important limitation, especially since the study in general failed to find significant differences in regard to its hypotheses. The authors mention that “it is possible that our sample size was insufficient to detect subtle changes in reporting,” suggesting incorrectly that there was adequate power to detect meaningful changes. Power was further limited by the fact that proportions of certain potential confounders were significantly different across journals; comparisons were made separately for each journal, further lowering the sample size. For example, *Ophthalmology* had the largest sample size of 51 articles, 28 of which had structured abstracts and 23 did not. If a criterion was met in 50% of the articles without structured abstracts and if a 25% difference (ie, 50% greater or lesser than that in ar-

ticles without structured abstracts) was considered to be meaningful, then the study had only 34% power to detect this difference, even without correcting for multiple comparisons. For comparisons in the *Archives of Ophthalmology*, the power was even lower because the sample size was only 26.

Scherer and Crawley do not state whether both raters rated each article. If so, the method for achieving a consensus rating should have been mentioned; if not, the interrater reliability of the operationalized version of the CONSORT criteria should have been established.

The authors also fail to consider the possibility that the limitations, especially the low power, may have accounted for the lack of significant differences. We hope that this interesting question will be addressed by a subsequent study that has a larger sample size and that addresses the limitations of the study. We also hope that the CONSORT statement will be used extensively as an instrument to monitor the reporting of clinical trials and to explore hypotheses related to it.

Rajnish Mago, MD
Paul Crits-Christoph, PhD
 University of Pennsylvania
 Philadelphia

1. Scherer RW, Crawley B. Reporting of randomized clinical trial descriptors and use of structured abstracts. *JAMA*. 1998;280:269-272.

In Reply: For our study and in using CONSORT descriptors¹ as a “gold standard” for assessing reporting of randomized clinical trials, we developed and pretested our instrument using randomized controlled trials in ophthalmology reported in journals or published in years not included in our study. We have not tested the instrument using reports of clinical trials in other disciplines or for interrater reliability and welcome suggestions and comments from Drs Mago and Crits-Christoph as they test our instrument. Indeed, at their request, we have forwarded a copy of our data collection instrument and instructions to them.

Mago and Crits-Christoph are correct in that we had insufficient sample size in our study to determine definitively whether use of structured abstracts results in better text reporting. We did not mean to imply that there was sufficient sample size. When the editors of 2 ophthalmology journals decided at about the same time to change the required abstract format from nonstructured to structured, we chose to use this natural experiment to evaluate changes in text reporting following structured abstract use. We described reporting of randomized controlled trials using CONSORT descriptors and reported some small but inconsistent differences in text reporting following use of structured abstracts. As mentioned in our article, we analyzed reports from journals separately because of differences among journals. Also, because of the number of observations, we did not feel confident in stating whether the differences we observed were real or caused by chance.

Based on our findings, increasing the sample size is problematic; adding trials reported in additional ophthalmology journals (or even in other general or specialty journals) is not feasible because of potential differences between journals. While it

may be possible to increase the sample size by adding reports published earlier or later in the same journals, it then becomes even more critical to control for other changes over time that may affect reporting of trials. For example, publication of the CONSORT statement in 1996 may have influenced reporting of randomized trials before its use was required by editors.

Roberta W. Scherer, PhD
Barbara Crawley, MS
 University of Maryland School of Medicine
 Baltimore

1. Begg C, Cho M, Eastwood S, et al. Improving the quality of reporting of randomized controlled trials: the CONSORT statement. *JAMA*. 1996;276:637-639.

Clinical Crossroads: A 24-Year-Old Woman With Anorexia Nervosa

To the Editor: Dr Halmi's¹ discussion of a 24-year-old woman with anorexia nervosa illustrates the risk that psychiatry, as it cleaves to a certain kind of “remedicalization,” may lose the patient as a person. This loss would reflect what has been decried as the shift from a “brainless” to a “mindless” psychiatry.² The loss of the voice of the patient would be ironic as medicine rediscovers the place in clinical care of the patient's experience and the patient-physician relationship.³

Fortunately, in the conference during which Halmi offered her consultation, the patient is allowed to speak along with the physicians. In her eloquent part of the printed record, she gives evidence of understanding her disease:

- curiosity about her eating disorder—where does it come from?—and its relationship to her mood disorder;
- doubt that her own thoughts, feelings, and wishes are valid, with consequent difficulty in negotiating expectations with others (especially parents) and vulnerability in all relationships, including those with clinicians;
- lost feeling of partnership and feeling “done to” rather than working with;
- an extreme sense of worthlessness, of not having a “right to a life,” sensing that she is slowly dying, able to mobilize her wish to live and take care of herself only when she comes close to death;
- intense identification with her illness, even doubting there is anything to her apart from it (“We're always going to be together”) and appearing to find little meaning in anything else.

The patient also shows considerable self-observation, especially with regard to the dramatic shift that occurs in her thoughts, feelings, and sense of herself (“I feel I'm not myself anymore”) that comes when her weight goes below a certain point (33 kg [73 lb]).

While Halmi emphasizes cognitive-behavioral therapy to help the patient learn to correct maladaptive thinking, the patient's own words cry out that any therapy must start with a recognition of her actual situation—existential, relational, and developmental. To say she “steadfastly refuses to increase her weight,” while true on one level, misses an opportunity to state her dilemma empathically. She is a bright young adult, captured by an

illness that has stolen her sense of self (or betrayed its weakness), but that she wants to understand and master.

Gordon Harper, MD
Children's Hospital
Harvard Medical School
Boston, Mass

1. Halmi KA. A 24-year-old woman with anorexia nervosa. *JAMA*. 1998;279:1992-1998.
2. Eisenberg L. Mindlessness and brainlessness in psychiatry. *Br J Psychiatry*. 1986; 148:497-508.
3. Reiser SJ. The era of the patient: using the experience of illness in shaping the missions of health care. *JAMA*. 1993;269:1012-1017.

To the Editor: Dr Halmi¹ presents a full survey of epidemiologic studies, discusses possible genetic factors, and speculates about the role of neurotransmitter anomalies.

Where is the discussion of psychosocial factors, emotional factors, and psychodynamics, the neuropsychobiological matrix, "body image," and the like?

David R. Metcalf, MD
Philipsburg, Mont

1. Halmi KA. A 24-year-old woman with anorexia nervosa. *JAMA*. 1998;279:1992-1998.

In Reply: Dr Harper believes my discussion illustrates remedicalization of psychiatry. I have always made the assumption that psychiatry is part of medicine and that all of medicine needs to listen to the voice of the patient. Ms K eloquently stated her overwhelming sense of ineffectiveness, worthlessness, and depression. That did not need to be restated. Ms K also expressed feeling safe and comfortable with her present treatment team, which implies they have empathetically recognized her dilemma and have respected her voice. This team requested (I did not offer) my consultation after their efforts for more than a year dealing with her actual existential, relational, and developmental situation had less effect than they had wished. The question then became what else can we do? Ms K stated "a lot of my treatment involves trying to make things positive." This is an indication that the treatment team is addressing Ms K's overwhelming sense of ineffectiveness (except in dieting) and worthlessness. In her maturation to the age of 24 years, Ms K did not have the experiences she personally needed to become self-confident and secure. Her therapy must and is focusing on developing these positive experiences. Garner and Bemis¹ developed cognitive-behavioral therapy for anorexia nervosa with great empathy and concern for the anorectic patient. This technique can be used to deal with both developmental and interpersonal issues.²

Dr Metcalf is disappointed that I did not more fully discuss psychosocial and psychodynamic factors. Editing decisions are made jointly by the editors and discussant and take into account severe space limitations. For my part, I did not encourage expanding further on those topics because of the extensive coverage they have had in the literature. I refer readers to a superb discussion of psychosocial aspects by DiNicola³ and psychodynamics by Vitousek and Manke.⁴

Katherine A. Halmi, MD
Weil Medical College of Cornell University
White Plains, NY

1. Garner DM, Bemis KM. Cognitive therapy for anorexia nervosa. In: Garner DM, Garfinkel PE, eds. *Handbook of Psychotherapy for Anorexia Nervosa and Bulimia*. New York, NY: Guilford; 1985:107-146.
2. Kleifield EI, Wagner S, Halmi KA. Cognitive-behavioral treatment of anorexia nervosa. *Psychiatr Clin North Am*. 1996;19:715-734.
3. DiNicola VA. Anorexia multiform: self-starvation in historical and cultural context. *Transcultural Psychiatr Res Rev*. 1990;27:165-286.
4. Vitousek K, Manke F. Personality variables and disorders in anorexia nervosa and bulimia nervosa. *J Abnorm Psychol*. 1994;103:137-147.

Abolishing Nuclear Weapons

To the Editor: While it is important to condemn the irresponsible behavior of Pakistani and Indian leaders for testing nuclear weapons, thereby increasing the risk of nuclear war, as Dr Lown and colleagues¹ point out, the real blame for this unhappy state of affairs rests squarely on the shoulders of the United States and the other nations with nuclear weapons. Over the last 50 years, these nations have sent the world mixed messages. For instance, the United States has promoted nuclear abstinence for others while continuing its own unilateral nuclear arms race, even 7 years after the end of the Cold War. The prestige and power associated with possessing nuclear weapons have not gone unnoticed by other countries.

As the one world superpower and the first country to develop and use nuclear weapons, the United States should take the lead in bringing them under control before their distribution becomes even more widespread. In his recent book, Jonathan Schell² records interviews with civilian leaders, scientists, and retired high-level military officers from the United States, Russia, and Europe who are convinced that the time is ripe for the world to eliminate nuclear weapons. If this opportunity is missed, it is likely that these terrible weapons will eventually be possessed by "rogue" states and terrorist groups who could and likely would use them to cause destruction beyond imagination. The United States is not exempt from this threat. We have only to consider the World Trade Center and Oklahoma City bombings to get an idea how this might happen.

As guardians of the health of society, physicians can support the process of nuclear weapon abolition by speaking out to their friends, patients, and congressional representatives. Saving the world from nuclear destruction should be our number one public health priority.

David W. Fouts, MD
Lutherville, Md

1. Lown B, Chazov EI, Foege WH, Ul-Majeed S, Reddy RJ. An appeal for nuclear sanity: an open letter to: Prime Minister Atal Behari Vajpayee of India, Prime Minister Nawaz Sharif of India. *JAMA*. 1998;280:467.
2. Schell J. *The Gift of Time: The Case for Abolishing Nuclear Weapons*. New York, NY: Henry Holt; 1998.

In Reply: Nuclear weapons are instruments of genocide. Their possession is a crime against humanity. No reason of state can ever justify their use, which would inflict indiscriminate mass murder, injure generations yet unborn, and despoil an already degraded environment. We have defeated Hitler, yet have succumbed to his vicious philosophy. In going nuclear, India and Pakistan have extended the reach of barbarism.

Dr Fouts is completely on target. The nidus of the pathology is among the permanent members of the UN Security Council and especially in Washington, DC, which has initiated and been a key player in shaping the atomic age. The concept of nonproliferation was flawed from the outset. The superpowers retained the right to keep testing and modernizing weapons notwithstanding their huge arsenals and their qualitative and quantitative dominance. At the same time, the nonweapons states were expected permanently to forswear any intent of acquiring nuclear military capabilities. Such a double standard could not be permanently enforced. The options were 2: either eradicate the malignant virus, nuclearism, or witness its spread. The events in South Asia confirm the bankruptcy of nuclear apartheid policies.

From the vantage point of our long-range national security, nuclear weapons offer nothing except a dire threat. With the United States the strongest nation on earth by every conceivable military, economic, and scientific measure, legitimization of nuclear weapons detracts rather than bolsters our safety. They are potential equalizers, enabling the weak to inflict unacceptable damage on the strong. Nuclear know-how is everywhere. Terrorists are aware that a nuclear bomb affords "the cheapest and biggest bang for the buck." No blackmail would be as compelling as holding an entire city hostage. Few societies are more susceptible to their malevolent consequences than the rich, urbanized, highly developed, industrialized North, foremost the United States. It is already the object of growing global resentment and envy. One may surmise that it will increasingly be the target for terrorist acts.

Physicians for Social Responsibility and the International Physicians for the Prevention of Nuclear War have long argued that nuclear abolition is a "number one public health priority." Political leaders respond not to historic imperatives but to the clamor of their constituencies. Only an upsurge of public opinion in favor of nuclear abolition will sway Washington to reconsider a flawed, immoral, and dangerous course.

Bernard Lown, MD
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Brookline, Mass

Proximal Myopathy Associated With Inhaled Steroids

To the Editor: A number of undesirable systemic effects have been reported with the use of inhaled corticosteroids, including cataracts, suppression of the hypothalamic-pituitary-adrenal axis, osteoporosis, and possibly glaucoma.^{1,2}

Report of a Case. A 37-year-old woman who had a seizure disorder that was well controlled with carbamazepine therapy began to use a beclomethasone nasal inhaler, 42 µg of drug per nasal pump unit, 1 to 2 sprays in each nostril twice daily, for

the treatment of seasonal allergic rhinitis. Four weeks later, the patient began to experience tightness in her thighs and increased fatigability when she climbed stairs or walked distances that were previously readily tolerated. Findings on a general medical examination were normal. Neurologic evaluation revealed a mild to moderate reduction in strength (4½/5 range) in neck flexion and bilaterally in shoulder abduction and hip flexion. Muscle weakness had not been noted in the past. Findings on the remainder of the neurologic examination, including muscle strength elsewhere, cranial nerve function, and deep tendon reflexes, were within normal limits. Erythrocyte sedimentation rate, thyroid function test results, cortisol and serum creatine kinase levels, and antinuclear antibody titer were normal. Serum carbamazepine level was in the midtherapeutic range. Electromyography (EMG) showed normal resting activity, without fibrillations or trains of myotonic potentials. Voluntary contraction of proximal muscles showed an increased recruitment pattern and diminished average size of units, consistent with myopathy. The possibility of steroid-induced myopathy was considered, and the use of inhaled steroids was discontinued. At the time of follow-up 1 month later, the patient was free of exercise-induced muscle tightness and reported normal exercise tolerance and strength. Examination revealed full motor strength, and repeat EMG showed normal unit morphology and recruitment. Without steroids, she has had no recurrence of weakness for 2 years.

Comment. Clinicians may need to consider proximal myopathy among the possible undesirable systemic adverse effects that can occur with the use of inhaled corticosteroids.

Andrew G. Herzog, MD, MSc
Beth Israel Deaconess Medical Center
Boston, Mass

1. Garbe E, Suissa S, LeLorier J. Association of inhaled corticosteroid use with cataract extraction in elderly patients. *JAMA*. 1998;280:539-543.
2. Cumming RG, Mitchell P, Leeder SR. Use of inhaled corticosteroids and the risk of cataracts. *N Engl J Med*. 1997;337:8-14.

CORRECTIONS

Authors' Names Omitted: In the Letter entitled "Acetaminophen and Risk Factors for Excess Anticoagulation With Warfarin," published in the August 26, 1998, issue of *THE JOURNAL* (1998;280:697), authors Daniel E. Singer, MD, and Steven J. Skates, PhD, were omitted.

Incorrect Figure Key: In the Original Contribution entitled "National Use and Effectiveness of β-Blockers for the Treatment of Elderly Patients After Acute Myocardial Infarction," published in the August 19, 1998, issue of *THE JOURNAL* (1998;280:623-629), there was an incorrect figure key. On page 627, the key to Figure 1 should have read "Prescribed Use, % (No.) of States."

Author's Name Omitted: In the Original Contribution entitled "Has the California Tobacco Control Program Reduced Smoking?" published in the September 9, 1998, issue of *THE JOURNAL* (1998;280:893-899), Arthur J. Farkas, PhD, was omitted from the byline.