Changes After Quitting Cigarette Smoking

GARY D. FRIEDMAN, M.D., AND ABRAHAM B. SIEGELAUB, M.S.

SUMMARY Changes in cardiorespiratory symptoms and coronary disease risk indicators over an average 11/2-year period were assessed in 9392 persistent cigarette smokers and 3825 persons who quit smoking between two multiphasic checkups. The prevalence of questionnaire-reported chronic cough fell markedly in subjects who quit a one-or-more-pack/day habit (e.g., from 11.2% to 1.8% in white men, \( p < 0.001 \)). However, chest pain, shortness of breath and exertional leg pain showed no consistent improvement in quitters compared with persistent smokers. Weight gain was about 2–3 lbs greater in quitters, but changes in blood pressure were small and not consistent across race-sex groups, nor were there consistent differences between persistent smokers and quitters in trends in vital capacity, cholesterol or prevalence of ECG abnormality. Quitting was associated with increase in serum uric acid levels of about 0.2–0.5 mg/dl and relative falls in hemoglobin, leukocyte count and serum glucose levels, all consistent with smoker-nonsmoker differences previously found in cross-sectional studies. Except for the small increases in weight and uric acid levels, quitting smoking did not appear to increase risk of coronary heart disease by other mechanisms.

BY STUDYING a large group of cigarette smokers who have had repeated multiphasic health checkups (MHCs), we compared those who persisted in the smoking habit with those who quit. We here report the changes observed in cardiorespiratory symptoms and related laboratory findings, emphasizing characteristics related to risk of coronary heart disease.

Subjects and Methods

Since 1964, the automated MHC has been given to about 50,000 persons per year in the Kaiser-Permanente Medical Centers in Oakland and San Francisco. The MHC and the characteristics of the examined population have been described in detail.\(^1\)\(^2\)

For the 41% of patients who received more than one MHC, the intervals between MHCs varied considerably but were almost always at least 1 year.

Smoking habits were assessed by self-administered questionnaire. The version used throughout most of the July 1964–August 1973 study period ascertained current cigarette smoking by the question, “In the past year did you smoke cigarettes?” and past cigarette smoking by the question, “Before 1 year ago did you ever smoke cigarettes?” Starting in April 1972, current cigarette smoking was ascertained by the question “Do you smoke cigarettes now?” and past cigarette smoking by the question, “Have you ever smoked cigarettes for at least 1 year?” To be included in the present study, a subject had to have at least one MHC at which he or she reported both current and past cigarette smoking followed by at least two other MHCs at which current cigarette smoking was either confirmed or denied. Furthermore, the study was limited to the two largest racial groups among the participants, whites and blacks.

A detailed description of the derivation of this study group from all persons receiving MHCs during the 9-year period has been published.\(^3\)

Two smoking groups were defined in this study: persistent smokers and quitters. Persistent smokers, totaling 9392 persons, had at least one MHC at which they reported both current and past cigarette smoking followed by at least two more MHCs at which the question about current cigarette smoking was answered “yes.” Furthermore, they never denied current smoking after first reporting it. Whether pipe or cigar smoking accompanied cigarette smoking was not considered.

Quitters, totaling 3825 persons, also had at least one examination at which they reported both current and past (i.e., established) cigarette smoking. After the last MHC at which current smoking was reported, quitters had at least one MHC at which the question about current smoking was answered “no” and at which there was no pipe or cigar smoking reported or positive response to any quantity of cigarettes smoked. The requirement that subjects have at least three MHCs was made so that we could divide this group into persistent and temporary quitters, according to their smoking habits reported at later MHCs.\(^3\)

This subdivision is not considered in the present report.

An “index MHC” was defined for all subjects. For persistent smokers, the index MHC was the first MHC at which established cigarette smoking was reported. For quitters, the index MHC was the last MHC before quitting at which cigarette smoking was reported. The “first follow-up MHC” was defined as the first MHC after the index MHC. The median date of index MHC was in March 1966 for persistent smokers and in September 1967 for quitters. The median date of first follow-up MHC was in March 1968 for persistent smokers and in September 1969 for quitters. The median interval between the two MHCs was 18 months for both groups. Table I shows the age, sex, and race distribution of the subjects.

With regard to the variables studied, symptoms also were assessed by self-administered questionnaire, the reliability of which has been evaluated.\(^4\) Blood

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716
pressures were measured manually from July 1964 to March 1967, then usually by the Air Shield automated sphygmomanometer from March 1967 to January 1970, and by the Godart device from January 1970 through the rest of the study period. Comparison of these measurement methods revealed no large or consistent differences except for the tendency of the Air Shield device to give diastolic readings about 10 mm higher than the other methods. To avoid a biased comparison due to differing examination times, only the systolic blood pressure data are presented.

The ECGs consisted of six leads (aVR, aVL, aVF, V1, V3, and V5) and were read by cardiologists and coded onto standard forms. Pulmonary function was measured by the Collins spirometer before May 1966 and by the Wedge spirometer thereafter. The change in machines had no appreciable effect on total vital capacity, but the 1-second value (FEV1.0) was consistently higher with the Wedge spirometer. Data analysis concerning FEV1.0 was therefore restricted to the period when the Wedge spirometer was used. Blood cells were counted using the Fisher hemocytometer before November 1968 and the Coulter counter thereafter. To avoid artifacts due to changes in measuring devices, analyses of hemoglobin and leukocyte count were restricted to subjects with index and follow-up MHCs using the Fisher hemocytometer. This reduced the number of subjects by about one half. Serum chemistries were measured by an AutoAnalyzer from July 1964 to March 1969, by an AutoChemist from April 1969 to January 1972, and by an SMA 12/60 analyzer thereafter. The initial AutoAnalyzer continued to be used to measure serum glucose levels during the time the AutoChemist was used to measure the remaining chemistry values. Again, to avoid artifacts due to changes in measuring devices, the analyses of serum cholesterol and uric acid were restricted to the approximate half of subjects whose MHCs of interest both used the AutoAnalyzer.

A detailed comparison of persistent smokers and quitters at the index MHC has been published. Data Analysis and Presentation

For dichotomous (yes-no) variables, such as questionnaire responses and presence or absence of electrocardiographic abnormality, the changes in persistent smokers from index to first follow-up examination were compared with those in quitters in the four race-sex groups, black and white men and women. The statistical significance of the smoker-quitter difference was assessed in the following manner. For each age decade subgroup, a two-by-two table was set up showing the numbers of subjects who changed between MHCs, either improving (going from positive to negative response) or worsening (going from negative to positive response); on one axis was smokers vs quitters and on the other was improved vs worsened. The Mantel-Haenszel test was performed on these two-by-two tables, summarized across all age groups. The crude overall percentages with positive responses are shown in tables 2 and 3. Adjusting these percentages for age had virtually no effect on the findings.

For quantitative variables, mean values at an MHC and mean changes between MHCs were adjusted for age by the indirect method. Statistical significance of differences between age-adjusted means was assessed by the t test using the method of Chiang to calculate standard errors.

The quitters and persistent smokers were divided into those who reported smoking less than one pack per day and those who reported smoking one pack or more at index MHC. Complete findings are shown for chronic cough, but for other characteristics we do not present tabular data for subjects smoking less than one pack at index MHC or for blacks, who were relatively few in number. The findings for these omitted groups that are markedly different from those for whites smoking at least one pack per day are mentioned. In tables 3, 4 and 5, the numbers of subjects correspond closely to those for whites who smoked at least one pack of cigarettes per day as given in table 2, except for small differences due to a few subjects with missing data on at least one MHC.

Results

Symptoms

Chronic Cough

The presence of chronic cough was assessed by the question, "In the past 6 months have you had a cough
### Table 2. Prevalence and Mean Change (%) in Reported Chronic Cough Between Multiphasic Health Checkups in Persistent Smokers and Quitters According to Amount of Cigarettes Smoked at Index Multiphasic Health Checkup*

<table>
<thead>
<tr>
<th>Finding</th>
<th>White men</th>
<th></th>
<th></th>
<th>White women</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 1 pack/day</td>
<td>≥ 1 pack/day</td>
<td></td>
<td>&lt; 1 pack/day</td>
<td>≥ 1 pack/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Persistent smokers</td>
<td>Quitters</td>
<td>Persistent smokers</td>
<td>Quitters</td>
<td>Persistent smokers</td>
<td>Quitters</td>
</tr>
<tr>
<td>Index MHC (I)</td>
<td>6.3</td>
<td>3.4</td>
<td>17.6</td>
<td>11.2</td>
<td>6.3</td>
<td>3.4</td>
</tr>
<tr>
<td>First follow-up MHC (F)</td>
<td>5.4</td>
<td>2.0</td>
<td>16.8</td>
<td>1.8</td>
<td>5.4</td>
<td>2.0</td>
</tr>
<tr>
<td>Yes → No</td>
<td>3.7</td>
<td>2.2</td>
<td>7.6</td>
<td>10.1</td>
<td>3.7</td>
<td>2.2</td>
</tr>
<tr>
<td>No → Yes</td>
<td>2.8</td>
<td>0.9</td>
<td>6.9</td>
<td>0.6</td>
<td>2.8</td>
<td>0.9</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-0.9</td>
<td>-1.3</td>
<td>-0.7</td>
<td>-9.5†</td>
<td>-0.9</td>
<td>-1.3</td>
</tr>
</tbody>
</table>

*Minor inconsistencies due to rounding.
†p < 0.001.
‡p < 0.05.

Abbreviations: MHC = multiphasic health checkup; I = index MHC; F = first follow-up MHC.

### Table 3. Initial Prevalence and Mean Change (%) in Symptoms and Electrocardiographic Findings Among White Male and Female Persistent Smokers and Quitters Who Smoked One Pack/day or More at Index Multiphasic Health Checkup

<table>
<thead>
<tr>
<th>Finding</th>
<th>White men</th>
<th></th>
<th></th>
<th>White women</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 1 pack/day</td>
<td>≥ 1 pack/day</td>
<td></td>
<td>&lt; 1 pack/day</td>
<td>≥ 1 pack/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Persistent smokers</td>
<td>Quitters</td>
<td>Persistent smokers</td>
<td>Quitters</td>
<td>Persistent smokers</td>
<td>Quitters</td>
</tr>
<tr>
<td>Exertional dyspnea</td>
<td>24.0</td>
<td>15.1</td>
<td>37.9</td>
<td>22.1</td>
<td>24.0</td>
<td>15.1</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-8.9</td>
<td>-4.8</td>
<td>-11.8</td>
<td>-5.4</td>
<td>-8.9</td>
<td>-4.8</td>
</tr>
<tr>
<td>Exertional chest pain</td>
<td>17.6</td>
<td>11.9</td>
<td>22.1</td>
<td>13.7</td>
<td>17.6</td>
<td>11.9</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-3.2</td>
<td>-3.2</td>
<td>-3.5</td>
<td>-5.0</td>
<td>-3.2</td>
<td>-3.2</td>
</tr>
<tr>
<td>Any chest pain</td>
<td>38.3</td>
<td>27.9</td>
<td>41.2</td>
<td>28.9</td>
<td>38.3</td>
<td>27.9</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-6.7</td>
<td>-5.9</td>
<td>-7.6</td>
<td>-7.9</td>
<td>-6.7</td>
<td>-5.9</td>
</tr>
<tr>
<td>Exertional leg pain</td>
<td>5.9</td>
<td>4.7</td>
<td>7.8</td>
<td>6.6</td>
<td>5.9</td>
<td>4.7</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-1.3</td>
<td>-1.6</td>
<td>0.3</td>
<td>0.2</td>
<td>-1.3</td>
<td>-1.6</td>
</tr>
<tr>
<td>Electrocardiographic abnormality</td>
<td>13.5</td>
<td>12.5</td>
<td>14.3</td>
<td>10.8</td>
<td>13.5</td>
<td>12.5</td>
</tr>
<tr>
<td>Net change (F – I)</td>
<td>-1.2</td>
<td>-0.6</td>
<td>-0.9</td>
<td>-1.0</td>
<td>-1.2</td>
<td>-0.6</td>
</tr>
</tbody>
</table>

Abbreviations: MHC = multiphasic health checkup; I = index MHC; F = first follow-up MHC.
almost every day?” Of all characteristics studied, this symptom showed the most marked difference between persistent smokers and quitters with respect to degree of change, particularly in white men. The difference was largely confined to the one-or-more-pack/day smokers, who initially had a higher prevalence of cough than the less-than-one-pack/day smokers (table 2).

**Exertional Dyspnea**

Exertional dyspnea was considered present if the subject answered “yes” to either of two questions about “severe shortness of breath” in the past 6 months — either “with your usual work or activity” or “that makes you stop after climbing 10-14 steps or a short hill.” Most groups studied showed a net improvement between the index and first follow-up examination. Surprisingly, persistent smokers showed more improvement (expressed as net change in percentage) than the quitters (table 3). To determine whether this discrepancy might be due to greater weight gain among the quitters, the analysis was repeated, omitting all subjects who gained more than 2 lbs between examinations. The findings were affected very little by this restriction in the study groups — the prevalence in white male persistent smokers was initially 25.0%, with a net decrease of 9.9%; in quitters, the prevalence was 15.7%, with a net decrease of 6.4%. The corresponding percentages in the white women were 38.9%, with a decrease of 14.2%, and 26.7%, with a decrease of 12.9%.

**Exertional Chest Pain**

Exertional chest pain was considered present if the subject answered “yes” to either of two questions about “bad pain or pressure or tight feeling in your chest... in the past year... which was brought on by exertion or walking fast or uphill, and which left after a few minutes rest” or “... that forced you to stop walking.” Most groups studied showed slight reduc-
tions in prevalence of this complaint between index and first follow-up MHC (table 3). A notable exception were black female quitters who had smoked at least one pack per day, in whom the prevalence of exertional chest pain rose from 14.5% to 23.2% between the two MHCs. The only statistically significant difference between the changes among persistent smokers and quitters was among white females who smoked less than one pack per day; however, the net changes in these two groups were small, −2.5% and −4.1%, respectively (p < 0.05).

Any Chest Pain

In addition to the two questions about exertional chest pain, we asked seven other questions about non-exertional types of chest pain that have been associated with cigarette smoking. We tabulated the prevalence of a positive response to at least one of the nine chest pain questions. Slight reductions in prevalence were noted in almost all groups studied, but the groups did not differ significantly from one another (table 3). The notable exception was a large and statistically significant increase in prevalence among black female quitters who had smoked at least one pack per day (23.2% to 36.2%, p < 0.01).

Exertional Leg Pain

Exertional leg pain was assessed by the question “In the past year have you often had pain in your legs that forced you to stop walking and left after a few minutes rest?” The prevalence of this symptom generally showed little change between MHCs, and there were no statistically significant differences between smokers and quitters with respect to such changes (table 3).

Physiologic Measurements

Electrocardiographic Abnormality

Presence of some electrocardiographic abnormality was indicated by the absence of the diagnosis “no significant abnormality.” The prevalence of abnormality changed very little between examinations in whites, and there were no statistically significant differences between persistent smokers and quitters in any race-sex group (table 3). However, quitters among the blacks who smoked one or more packs per day showed rather marked increases (men 6.5%, women 11.5%), while the corresponding persistent smokers did not (men +0.1%, women no change).

Total and 1-second Vital Capacity

Mean total vital capacity (FVC) and 1-second vital capacity (FEV₁) showed only slight changes between index and first follow-up MHCs (table 4). There were no significant differences in these changes between persistent smokers and quitters.

Weight

A general tendency for weight to increase between examinations was noted, but the average increase was about 2–3 lbs greater in the quitters than in the persistent smokers. Large gains in weight among the quitters were not the rule but were more common than in persistent smokers (table 6). Among those who had smoked at least one pack per day, about 24% of white male and female quitters gained 10 lbs or more, and only about 4% gained at least 20 lbs.

Systolic Blood Pressure

Systolic blood pressure tended to increase between examinations in men and decrease in women, except for a slight increase in white female quitters who had smoked at least one pack per day. The increase in white men was significantly greater in quitters than in persistent smokers (p < 0.05), while among corresponding black women, the decrease was significantly greater in quitters (p < 0.05). In general, the changes in blood pressure, both systolic and diastolic, were small and inconsistent across the race-sex groups, with no consistent distinction between persistent smokers and quitters.

Blood Tests

Serum Cholesterol Level

In white men and women, serum cholesterol showed a greater net increase in persistent smokers than in

| Table 6. Age-adjusted Percentages of White Male and Female Persistent Smokers and Quitters Who Gained at Least 10 Pounds and at Least 20 Pounds Between Index and First Follow-up Multiphasic Health Checkup According to Amount Smoked at Index Multiphasic Health Checkup |
|---|---|---|---|---|---|
| Group and quantity smoked at index MHC | Persistent smokers | | Quitters | | |
| | Total (n) | Percent gaining | | Total (n) | Percent gaining |
| | | ≥ 10 lb | ≥ 20 lb | | ≥ 10 lb | ≥ 20 lb |
| White men | | | | | | |
| < 1 pack/day | 928 | 9.8 | 1.0 | 613 | 13.1 | 3.1 |
| ≥ 1 pack/day | 1959 | 10.8 | 0.9 | 585 | 24.6 | 4.3 |
| White women | | | | | | |
| < 1 pack/day | 1718 | 6.5 | 1.6 | 1092 | 12.5 | 2.5 |
| ≥ 1 pack/day | 2205 | 10.5 | 1.6 | 448 | 23.7 | 4.5 |

Abbreviations: MHC = multiphasic health checkup.
quitters (table 5). This pattern was not consistent in blacks. The mean changes were generally small.

**Serum Glucose 1 Hour After Challenge**

Among smokers of one or more packs per day, the mean glucose levels generally rose in persistent smokers and fell in quitters (table 5); however, among black female quitters, there was virtually no change. There was no consistent pattern in the smokers of less than one pack per day. The mean changes were generally small.

**Serum Uric Acid Levels**

Although the changes were small, persons who quit smoking more than one pack per day experienced an increase in the mean uric acid level, while those who persisted, except for black women, experienced a decline (table 5). The smokers of fewer cigarettes did not show a consistent pattern.

**Hemoglobin**

Mean hemoglobin levels rose in all groups. Persistent smokers showed more change than quitters (table 5).

**Leukocyte Count**

The mean leukocyte count showed a decline in all study groups (table 5). Quitters consistently showed a greater decline than persistent smokers.

**Discussion**

Our finding of a decrease in chronic cough after quitting smoking corresponds to what has been frequently reported in the past. Little information exists about the effects of smoking cessation on other symptoms generally regarded as smoking-related. We previously studied in detail the relation of cigarette smoking to chest pain as reported by questionnaire, and the evidence generally suggested a causal connection to both exertional and nonexertional types of chest pain. Thus, we were surprised at the absence of a clear-cut difference in the degree of improvement shown by quitters and persistent smokers. The lack of a relative improvement in exertional dyspnea and exertional leg pain after quitting smoking is also counter to the commonly held view that smoking causes or aggravates these symptoms, although removal of a causal factor does not necessarily reverse its effect.

Our data on symptoms should be viewed in light of the previous study of the reliability of the questionnaire used. Some of the apparent improvement in both persistent smokers and quitters may be due to a much greater tendency for "yes" answers to be changed to "no" than for the reverse to happen. For example, 21.1% of "yes" answers by men to one of the questions on exertional dyspnea were changed to "no" when it was posed again 30 minutes later, but only 1.9% of "no" answers were changed to "yes." If 24% answer "yes," a net improvement of 3.6% (21.1% x 24% yes - 1.9% x 76% no) would result from unreliability alone. It is not known whether the same degree of change in response occurs if the interval is over a year rather than 30 minutes for patients with unchanged symptoms.

The accuracy of questionnaire data on smoking habits is also of concern. We have previously attempted to validate the questionnaire by a follow-up interview of a sample of examinees at the time of the MHC. Briefly, with regard to current cigarette smoking, all 73 patients who reported current smoking on the questionnaire confirmed this at interview. Similar confirmation was given by 42 of 43 who denied current smoking on the questionnaire. This apparently excellent agreement still may not reflect the true situation if the patient does not wish to reveal his or her actual smoking status. This has been of special concern for smoking intervention clinics or studies in which the patients are expected to stop smoking and wish to please the therapist. Thus, objective measures that are relatively specific and accurate indicators of smoking status, such as the serum thiocyanate level or expired-air carbon monoxide concentration, have been used recently to validate smoking questionnaire data. Results from the San Francisco clinic of Multiple Risk Factor Intervention Trial (MRFIT) indicate that where both objective tests gave concordant results implying that the subjects were smokers, 73 of 74 subjects reported smoking at interview. Where both objective tests gave concordant results implying non-smoking, 41 of 49 subjects reported non-smoking. However, the remaining eight subjects who reported smoking were "marginal smokers" in that they reported not inhaling, smoking less than nine cigarettes per day or not smoking within 24 hours of testing. Because the most likely motive for inaccuracy in the MRFIT or our setting would be to deny smoking when it existed, the finding that only one of 74 apparent smokers did not admit to the habit suggests that the subjective reports are generally accurate. We have not had the opportunity to validate our MHC smoking questionnaire with these objective tests but plan to do so for a future study.

Because of bronchial irritation and obstruction by excess mucus production, smoking has been associated with impairment in the forced expiratory lung volumes, which improve after smoking is stopped. Perhaps because this group of smokers had less impairment in pulmonary function than some others that have been studied, we could not detect an improvement in forced respiratory lung volumes in quitters compared with persistent smokers.

Weight gain after quitting smoking is often believed to be large, but this notion often derives from small series of patients or anecdotal material. A large-scale study of British physicians found an average weight gain of 4.3 lbs after quitting. In another study, follow-up of 224 men and women who quit smoking showed that 68% of the group gained weight, and in two-thirds of these, the gain was 15 lbs or less. We found that both persistent smokers and quitters showed an average gain in weight between MHCs that was 2–3 lbs greater in quitters. Quitters with large gains in weight were in the minority.
Because smokers often have slightly lower mean blood pressures than nonsmokers and because cessation is often accompanied by a weight gain, blood pressure might also be expected to rise after quitting. The findings on smokers followed longitudinally have been conflicting, however. A relatively greater increase in blood pressure was found in quitters in the Normative Aging Study but not in either the Evans County Study or the Framingham Study. Our results seem to perpetuate the discrepancy, because they were inconsistent across race-sex groups, with statistical significance achieved for opposite findings. The changes were, in general, rather small.

Our failure to find relative changes in the prevalence of electrocardiographic abnormality confirms a previous report that the frequency of abnormal findings was not substantially affected by 1 year of abstinence resulting from a smoking-withdrawal program.

The changes in the blood test findings tended to conform to previous cross-sectional comparisons of smokers and nonsmokers in our study population. The relative decrease (actually, a decrease or smaller increase) in serum cholesterol level in whites after quitting was consistent with the higher level in white male smokers than in nonsmokers. This consistency was true also for serum glucose level, although in the cross-sectional data, alcohol consumption appeared to explain at least part of the smoker-nonsmoker difference. The changes in serum uric acid level were very small, but the levels in persistent smokers tended to decrease, while those in quitters tended to increase, corresponding to the lower levels in male smokers than in nonsmokers. For the same reason, the considerably greater fall in leukocyte count and slightly smaller rise in hemoglobin in quitters was not unexpected.

Striking differences between persistent smokers and quitters were already present at the index MHC in some of the characteristics. The most notable were the lower mean weight and lung volumes, the higher prevalence of symptoms, and the higher mean leukocyte count in the persistent smokers initially. A comparison of these and other characteristics at baseline was previously reported. Cross-classification by quantity of cigarettes smoked indicated that some of the larger baseline differences were not attributable to the fact that most persistent smokers tended to be heavier smokers. We were also concerned about bias possibly being introduced by the wording of the question used to determine current smoking: “In the past year did you smoke cigarettes?” Some persons reporting current smoking in this way at index MHC may already have quit during the past year, and this may have been true for a greater proportion of quitters than of persistent smokers. If so, some of the baseline deviations of quitters from persistent smokers could be attributable to quitting that had already taken place. However, analysis of pre-index MHCs in those quitters who had them and reported smoking at that earlier time indicated that most of the smoker-quitter differences were also present at that time and were not, therefore, attributable to substantial unrecognized quitting before the index MHC.

One of the primary reasons for recommending that cigarette smokers discontinue the habit is the hope that by doing so they will reduce their risk of coronary heart disease. Thus, it was important to determine whether other risk factors would change in an unfavorable direction when smoking ceased. Compared with persistent smokers, the mean weight of quitters increased, but not by a large amount. Only for the small fraction of quitters whose weight increased greatly does the potential gain in risk associated with quitting become sufficient to be considered seriously against the potential benefits of discontinuing the cigarette habit. Uric acid, which has uncertain status as a causal risk factor, also increased slightly, suggesting slightly higher risk. Small but unfavorable changes in blood pressure were noted in male quitters, but in view of the inconsistencies among race-sex groups in our data and in other studies, the importance and generality of these findings is questionable. Other changes in risk indicators were generally either favorable or absent. A prudent policy might be to encourage all smokers to quit but routinely recheck weight and blood pressure 6 months to 1 year later, encouraging the patient to return earlier if he or she notes substantial weight gain. An earlier recheck would be indicated if the patient was already hypertensive while a smoker.

In the present study the duration of follow-up after quitting smoking was relatively short, averaging about 1.5 years. Thus it is possible that changes in coronary heart disease risk factor status, different from those we observed, would occur later. Even more important is the need to determine in this study population the degree to which quitting smoking is associated with a decreased incidence of major manifestations of coronary heart disease, particularly mortality. Thus, long-term follow-up is planned to compare the development of coronary heart disease events in persistent smokers and quitters, taking into account the differences between these groups noted at baseline.

Acknowledgment

The data were originally collected under research projects directed by Morris F. Collen, M.D., from multiphasic laboratories directed by Derek Crawford, M.D., and Robert Feldman, M.D. Computer programming was performed by Gary Lee, B.S., and Della Mundy, M.L.S., provided editorial assistance.

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Optimal Diagnosis in Acute Myocardial Infarction

A Cost-effectiveness Study

PEER GRANDE, M.D., CLAUS CHRISTIANSEN, M.D., ASGER PEDERSEN, M.D.,
AND MERETE SANVIG CHRISTENSEN, M.D.

SUMMARY The predictive value of a diagnostic test estimates the likelihood for presence or absence of disease in a patient with a positive or negative test result (PV pos or PV neg). We evaluated the predictive values of serum activities of the heart-specific creatine kinase isoenzyme MB (CK-MB), aspartate aminotransferase, lactate dehydrogenase, CK, and ECG in 401 consecutively admitted patients suspected of acute myocardial infarction (AMI). The study showed that CK-MB (PV pos \(= 0.98\), PV neg \(= 1.00\)) was better than the other enzymes (single as well as serial) and ECG, evaluated both separately and in combinations. In all cases of AMI CK-MB was positive within 17 hours from admission. Replacement of the standard enzymes with CK-MB provides a faster and safer diagnosis of AMI and reduces hospitalization time considerably for patients without AMI.

THE BALANCE between increased health care costs vs mortality and morbidity makes cost-effectiveness studies necessary. As laboratory services increase the costs in this field must be spent wisely. Although the technical reliability of a laboratory test is important, only the diagnostic validity and consequences for patients can justify its use.

Acute myocardial infarction (AMI) is one of the most common causes of death and one of the most frequent causes of hospitalization in the Western world, so many attempts have been made to improve its diagnosis. Detection of myocardial damage by increasing serum concentrations of enzymes as creatine kinase (CK), aspartate aminotransferase (ASAT), and lactate dehydrogenase (LDH) are now widely used to diagnose AMI. Recent studies indicate that measurements of the CK isoenzyme MB (CK-MB) in serum are more sensitive and specific in diagnosis of AMI. However, several of these reports do not meet the criteria for establishing the true diagnostic efficacy of CK-MB. The comparative patient group should consist of patients suspected of AMI and the establishment of the true diagnosis should be done independently of the results of CK-MB measurements. Furthermore, only a few of these studies answer the clinically important problem of the likelihood of AMI if a positive or negative CK-MB
Changes after quitting cigarette smoking.
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