Influence of cigarette smoking on morbidity and mortality after myocardial infarction

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Cigarette smoking is strongly related to the development of atherosclerosis, including coronary artery disease, and it has a number of other potentially adverse effects on the cardiovascular system. Some of these are mediated through the effect of nicotine on thrombosis, arrhythmias,¹ and platelet function.^{2,3} Others are mediated through the effect of raised carboxyhaemoglobin and nicotine levels which may jeopardise left ventricular function⁴ as well as lowering the ventricular fibrillation threshold.⁵

Most early survivors of myocardial infarction have extensive underlying coronary atherosclerosis. The major determinant of prognosis after myocardial infarction, at least for the first one or two years, is the extent of damage to the myocardium. Is it reasonable therefore to expect cessation of smoking to improve prognosis after myocardial infarction or are the potential benefits negated by irreversible atherosclerosis and myocardial scarring?

Considerable data are available to answer this question. It should be stressed at the outset that there have been no randomised control trials of stopping smoking after myocardial infarction. This raises the question of the comparability of those who stop smoking after myocardial infarction with those who continue. Nevertheless, the extent and consistency of available evidence, and the lack of contradictory evidence, are notable. Even without such evidence a randomised control trial of smoking cessation would be ethically dubious and impractical to perform.

Subsequent cigarette smoking habits and mortality

Although cigarette smoking at the time of initial myocardial infarction does not appear to influence subsequent prognosis,^{6,7} there is substantial evidence that smoking habits after myocardial infarction have an important influence on subsequent morbidity and mortality.

The Coronary Drug Project⁸ and the Newcastle⁹ and Scottish¹⁰ clofibrate trials found a higher mortal-

ity in smokers at entry to their studies compared with non-smokers. The coronary mortality ratio of smokers to non-smokers in the Coronary Drug Project was 1.25 while the ratio for sudden deaths, deaths from fatal and further non-fatal myocardial infarction combined was 1.33 and 1.70 for the Newcastle and Scottish smokers compared with non-smokers.

It is important to note that the Coronary Drug Project and the Newcastle and Scottish clofibrate studies related subsequent morbidity and mortality to smoking at entry to the study. In these three studies entry occurred at a variable time after infarction and in some cases not for several months. In the case of the Coronary Drug Project a few years may have elapsed before entry. These studies were not designed specifically to measure the effect of postmyocardial infarction cigarette smoking on subsequent morbidity or mortality. This and the wide variation in time of entry to the study may have resulted in a less precise measure of benefit.

In a study designed to measure the effect of subsequent smoking on cardiovascular and all-causes mortality, Salonen¹¹ recorded smoking habits six months after initial myocardial infarction. All-causes and cardiovascular mortality were significantly higher in smokers compared with non-smokers with risk ratios of 1.7 and 1.6, respectively, over a three year period.

Wilhelmsson *et al.*,¹² in a study of 564 male patients who survived a first myocardial infarction and whose smoking habits were measured three months after entry, reported a halving in cardiovascular mortality and in subsequent non-fatal myocardial infarction over a two year period in those who stopped smoking. In a further paper from this group published in this issue, Åberg *et al.*¹³ confirmed the deleterious effects of continued cigarette smoking on subsequent morbidity and mortality over an eight year period among 1306 male patients. In this later study smoking status was measured three months after first myocardial infarction. While there was apparently relatively little change in smoking status during the subsequent

follow-up period, such changes were possible, so that Åberg and his colleagues may be underestimating the benefits of stopping smoking.

Mulcahy *et al.*,⁷ in a similar study of men under 60 years who had survived a first myocardial infarction, where smoking habits were noted up to death or last follow-up, found that initial smokers who survived over a four year period had achieved a mean reduction of 80% in number of cigarettes smoked per day compared with 40% reduction in decedents. In a subsequent report¹⁴ we noted a 50% lower mortality in smokers who stopped over a five year period compared with smokers who continued. Unlike Wilhelmsson *et al.*,¹² we noted no significant difference in subsequent non-fatal events. Smoking experience was measured at last follow-up.

Daly *et al.*¹⁵ reported on 378 male initial smokers who had survived a first myocardial infarction by at least two years and who had their subsequent smoking experience measured at this point. They found that the all-causes mortality ratio in smokers who continued to smoke compared with smokers who stopped was 1.8 over a period of 15 years. Significant differences in cardiovascular and coronary mortality were also noted between the two groups. According to these authors the adverse effect of smoking became more pronounced over the 13 year period.

Pohjola *et al.*¹⁶ followed 648 male and female patients for four years and measured their smoking one year after entry to the study. For both sexes the coronary mortality risk of smokers compared with smokers who stopped was 2.3.

The Framingham group¹⁷ showed a 62% reduction in all-causes mortality over six years in those who stopped compared with those who continued to smoke after myocardial infarction. Smoking status was measured at the next follow-up visit after the myocardial infarction.

Jenkins *et al.*,¹⁸ reporting from the Western Collaborative Group study, found a significant ($p = 0.02$) reduction in cigarette smoking among 220 angina or infarct patients who had not suffered a further myocardial infarct during the follow-up period compared with 67 comparable patients who suffered a fatal or non-fatal myocardial infarct.

Vlietstra *et al.*¹⁹ reported a four year coronary mortality risk ratio of 1.6 in 2689 men with arteriographically confirmed coronary atherosclerosis who continued to smoke compared with 984 similar subjects who had stopped.

Examination of the data from the various studies specifically designed to measure the effect of subsequent smoking on long term outcome suggests that a mean ratio of about 2.0 measures the magnitude of the benefit for smokers who stop in terms of all-causes and coronary mortality compared with those who con-

tinue. This benefit exists to a greater or lesser degree irrespective of stratification of patients into different age and risk groups, and whether survival follows a first or subsequent myocardial infarct.

No study designed to test the effect of subsequent smoking on mortality in survivors of myocardial infarct has reported negative results.

Cigarette smoking habits and subsequent morbidity

While it is likely that benefit in terms of subsequent mortality is to be achieved from stopping cigarette smoking after myocardial infarction, there is less substantial evidence that subsequent morbidity as measured by recurrence of non-fatal myocardial infarction may be improved.^{14,17} The Coronary Drug Project⁸ found a non-fatal myocardial infarction ratio of 1.29 in those who continued to smoke compared with those who stopped. Wilhelmsson *et al.*¹² found a significant reduction in non-fatal myocardial infarction in their patients and in their latest study¹³ the cumulative non-fatal reinfarction rate over five years was 20% in stopped smokers compared with 30% in continued smokers ($p < 0.0001$). In all studies where subsequent myocardial infarctions, both fatal and non-fatal, were aggregated a significant reduction is apparent.

Cigarette smoking and chronic angina

Habitual cigarette smoking is associated with increased levels of carboxyhaemoglobin. There is evidence that a raised carboxyhaemoglobin will aggravate exercise-induced angina and increase ST-T changes in the exercise electrocardiogram of patients with angina.²⁰ Smoking may thus add to the disability of those with postinfarction angina. The Coronary Drug Project⁸ reported a postmyocardial infarction angina ratio of 1.23 in those who continued compared with those who stopped smoking. No other published information is available on the influence of smoking on postinfarction angina.

One report is available about the effect of follow-up cigarette smoking on survival in patients with chronic angina.²¹ This report from Framingham concludes that the risk of myocardial infarction, fatal and non-fatal combined, in subjects with chronic angina without a history of previous myocardial infarction is adversely affected by continuing the cigarette smoking habit.

Other effects of smoking

Authors who reported on mode of death in relation to subsequent smoking found no difference in the ratio of sudden death to deaths from fresh myocardial

infarction between smoking and non-smoking decedents.^{17 22}

It also seems that the adverse effect of smoking after infarction is independent of other primary and secondary risk factors, including Type A personality, hyperlipidaemia,¹⁸ hypertension, and severity of first myocardial infarction.⁷

Not all reports commented on the dose related effect of smoking during follow-up. Salonen¹¹ found that the adverse effects of follow-up smoking in coronary patients were dose related. Vlietstra *et al.*,¹⁹ in their arteriography study, also noted a dose related effect. In our five year study¹⁴ we noted no difference in mortality between those who stopped smoking and those who reduced their smoking volume by more than half, thus suggesting benefits from reducing the volume of smoking.

According to some authors, survivors who stop smoking have the same long term prognosis as survivors who have never smoked or who are ex-smokers at the time of the initial attack.^{7 16}

Pipe and cigar smoking

Little information has been published about the effect of pipe and cigar smoking on outcome in survivors of myocardial infarction. In a recent study we have noted no adverse effects of pipe smoking on survival but we found the same adverse effect in habitual cigar or cheroot smokers as in those continuing to smoke cigarettes.²³ These results will require confirmation by further studies.

Cigarette smoking, drugs, and surgery

Other forms of intervention which may benefit survivors are the use of beta adrenergic blocking agents and coronary artery bypass surgery. There is increasing evidence from results of large scale trials that beta blockers may improve survival after myocardial infarction,²⁴ at least for a year or two after recovery, but much remains to be learnt about timing and mode of administration, and particularly about the selection of cases for long term beta blockade.

Evidence favouring the role of other drugs in improving survival, including anticoagulants and platelet-active drugs, is insufficient to justify their routine use.²⁵ Irrespective of decisions about drug usage to improve survival, the cessation of cigarette smoking should be mandatory in all cases.

Cessation of smoking should be advised after coronary artery bypass surgery and plays a crucial part in pre- and postoperative management. Coronary artery bypass surgery in selected cases, such as those with left main stem or three vessel disease, may lead to improved survival,²⁶ but no information is available

about the effects on subsequent morbidity and survival of continuing smoking in surgical patients. Patients who continue to smoke after surgical treatment for peripheral vascular disease have shown significant postoperative deterioration compared with those who stop smoking.²⁷ It seems reasonable and prudent to postulate that improved survival after coronary bypass surgery may be prejudiced by continuing the smoking habit.

Approach to smoking control in coronary patients

We are aware from previous studies that the most important factor in achieving cessation of smoking is the antismoking advice of a doctor.^{28 29} It is clear from the secondary prevention studies cited that the advice of a doctor is particularly effective after a life threatening event such as a myocardial infarction. Other approaches to cessation, such as smoking clinics, television programmes, hypnotism, and acupuncture fail to achieve the same results and are clearly only aids to reinforce the motivation of the subject.

Long term cessation rates of 50% or more have been reported after myocardial infarction in centres where adequate rehabilitation and secondary prevention services are provided, and where the physician and the medical team are committed to smoking control among their patients. We have reported a cessation rate in coronary patients for each of the five three year periods from 1961 to 1975.³⁰ Cessation rates of 46% in 1961 to 1963 had increased to 58% by 1973 to 1975. More recent experience during the three years period 1978 to 1981 has shown an increase in the last follow-up cessation rate to 70%. We attribute this very high cessation rate to the influence of a dedicated rehabilitation team and to the provision of a special rehabilitation and secondary prevention follow-up clinic. We must also attribute some of the improved response to changing attitudes towards smoking and health in the community.

A number of psychological and personal factors are known to influence success in stopping smoking. Those with higher levels of education are more likely to stop.^{31 32} Married people are more successful than single people or those who are divorced. We find that impairment of self image, anxiety, and poor motivation as measured by standard psychological tests and semistructured interview are adverse features. These attributes have also been noted by others,³¹ though there are wide variations in the reported psychological and personality factors which discriminate smokers who stop from those who fail to stop.^{33 34}

Failure of compliance in response to advice about return to work and adherence to exercise, dietary, and drug regimens is also, in our experience, a pointer to

poor compliance in relation to smoking advice. An awareness of these adverse factors is important if we are to achieve optimum success in encouraging patients to stop smoking. The likelihood of permanent success is greater in older patients, in lighter rather than heavier smokers, non-inhalers rather than inhalers, in those who use filter tips, in those who have previously stopped or tried to stop, and particularly in those subjects who are well motivated.^{31 35}

In patients with myocardial infarction, intervention in relation to smoking must start from the time of admission to the coronary care unit. All health personnel concerned with the care of the infarct patient can play a part in counselling and influencing the patient. The coronary care nurse has a role as have the resident medical staff, physiotherapist, dietitian, social worker, and above all the physician or cardiologist in charge of the patient. Printed material containing information on the importance of cigarette smoking as a risk factor, on possible mechanisms, and on techniques of stopping smoking, is invaluable. Maintaining contact with the patient after discharge by providing rehabilitation and secondary prevention clinics can be crucial in long term management. This also applies to cessation techniques in healthy subjects.³¹

The importance of social support in smoking cessation is clear.³⁶ Social support includes the co-operation and encouragement of relatives and friends, including the avoidance of smoking in the company of the subject. The spouse has an important role—nagging and “policing” lead to poor cessation results while encouragement, support, and co-operation evoke good results.³⁷

Patients who cannot stop immediately should receive a sympathetic and understanding professional response. They should be advised about means of reducing the volume of smoking and of generating a conflict about the habit which may eventually lead to cessation.

Patients must be advised about the management of withdrawal effects and about the risks of resuming the habit. Here again a follow-up service is invaluable. Weight gain after cessation can be prevented by the counselling and supervision of a dietitian.³⁸ Other withdrawal effects, such as depression, boredom, and insomnia, can be alleviated by encouragement and reassurance, by increasing physical exercise, and occasionally and on a temporary basis, by the use of tranquillisers and sedatives.

Encouraging results have been reported by the use of nicotine chewing gum³⁹ as a substitute for cigarettes. In the more intractable cases such ancillary techniques may be advantageous but in the majority of patients success is achieved by close contact between a well motivated patient and a doctor and medi-

cal team committed to the importance of smoking control.

Conclusions

A mortality ratio of about 2.0 in favour of survivors who stop smoking has been consistently reported in secondary prevention studies of myocardial infarction. No careful study has been published which would contradict these findings. This mortality ratio is close to the all-cause mortality of 2.0 in favour of non-smokers noted by Hammond⁴⁰ among 1 078 894 men and women in the age group 35 to 64 years drawn from the apparently healthy American population. It is also close to the ratio somewhat above 2.0 in favour of non-smokers noted by Kahn⁴¹ in 250 000 US veterans. It is possible that the intervention of a myocardial infarction does not appreciably alter a subject's susceptibility to the adverse effects of cigarette smoking.

A coronary mortality risk ratio of 2.0 is a highly significant finding in patients with coronary heart disease because of their fourfold risk of early death compared with the general population. It is also important in relation to the management of patients because of the uncertainties of benefit to be derived from intervention in relation to other risk factors, including hyperlipidaemia, hypertension, obesity, and exercise, and the paucity of information about the benefits of multifactorial risk factor intervention.

Stopping cigarette smoking may be the most effective single means of intervention currently available to us in the management of patients with established coronary heart disease.

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