The effects of smoking cessation intervention in patients with coronary heart disease

- a randomised, controlled trial

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Introduction

The local story before starting the study

Frode Gallefoss MD, PhD, who is now the Superintendent of the Department of Pulmonology at Sørlandet Hospital Kristiansand, had for a long time been interested in helping patients with chronic obstructive pulmonary disease (COPD) and asthma to stop smoking, and he had also been working with the prevention of tobacco in the general population. As a doctor of internal medicine he had been worried about the lack of interest in smoking cessation among doctors treating patients with coronary heart disease, and he wondered why there were so little scientific papers on this topic. He did some power calculations, and found that a randomised trial on the effect of a smoking cessation program in these patients were possible to perform at our hospital. He gathered a steering committee, including the clever nurse Tone Bæck who later was to do most of the intervention and data collections. Frode had no time himself to carry the project as he was in the middle of his own PhD project about the effects of patient education in asthma and COPD. Therefore, I was asked to be the leader of the project. At that time I was working as an internist, and did not have scientific ambitions. But after reading the provisional description of the project I was astonished about the importance of the trial, and agreed to lead the project. Money was raised through an important donation from the Vest-Agder Council for Public Health. In the autumn of 1998, I sat down to write the protocol. Thanks to the recommendations from Frode this was done thoroughly, and most of the scientific questions discussed in this thesis have been prespecified in the protocol. I did not have ambitions regarding a PhD, but we were convinced that the quality of the project should be at a “PhD-level”. During this stage the steering committee had several meetings, and together I believe we managed to create a sound protocol. After the start of the study in the beginning of 1999 all were up to the study nurses, who we knew were clever in their work, and who we thought were able to deliver the intervention as intended. The rest of the story may demonstrate the impact of multidisciplinary co-operation in medicine.

Abstract

Background

Smoking cessation is the most important action to reduce mortality after a coronary event. Largely, it has previously been unknown whether a smoking cessation program applicable in an ordinary clinical setting has any impact on smoking cessation rates in patients with coronary heart disease, and whether such a program is cost effective.

Objectives

To determine whether a nurse managed smoking cessation intervention changes abstinence rates in patients admitted for coronary heart disease, to assess the predictors of smoking cessation, to evaluate whether smoking cessation has any impact on Quality of Life, to estimate the cost effectiveness of the smoking cessation program, and to appraise the feasibility of a fear arousal message.

Design

Randomised controlled trial of usual care compared with individual smoking cessation intervention.

Setting

The cardiac ward in Sørlandet Sykehus, Kristiansand, Norway.

Participants

240 smokers under 76 years of age admitted for myocardial infarction, unstable angina or cardiac bypass surgery. 118 were randomly assigned to the intervention and 122 to usual care (control group). 218 patients (91%) completed the study and were assessable at 12 months follow up.

Methods

The intervention was based on an especially made 17 page booklet and focused on fear arousal and relapse prevention. Individual smoking cessation was delivered by cardiac nurses without special training. The intervention was initiated in hospital, and the participants were telephoned regularly for at least five months. Abstinence rates were determined by self report and biochemical verification.

Survival data from previously published investigations, with life time extrapolation of the survival curves by survival function modelling, were used to be able to estimate the cost effectiveness of the program.

Baseline characteristics were prospectively recorded, and health-related Quality of Life was measured at baseline and at 12 months follow up. The patient satisfaction was assessed at 12 months follow up.

Results

12 months after randomisation 57.0% and 37.3% were abstinent in the intervention and control groups, respectively (95% confidence interval for the difference 6.4 to 33.0). The number needed to treat to get one additional quitter was 5.1 (95% CI 3.0 to 15.6). Assuming all drop outs returned to smoking at 12 months, the cessation rates were 50.0% and 37.0% in the intervention and control group, respectively (95% CI for the difference 0.4 to 25.7).

In a life time perspective, the incremental cost per year of life gained by the cessation program was Euro 280 and Euro 110 in the low and high risk group, respectively (2000 prices). These costs compare favourably to other treatment modalities in patients with coronary heart disease, being approximately 1/25 the cost of both statins in the low risk group and angiotensin converting enzyme inhibitors in the high risk group. In a sensitivity analysis, the costs remained low in a wide range of assumptions.

The participants were in general satisfied with the cessation program, indicating that implementing a fear arousal message is a feasible method.

In multivariate logistic regression analysis, a high level of nicotine addiction was a strong negative predictor of smoking cessation in both the intervention and the control group.

The quitters and sustained smokers had similar improvements in all Quality of Life domains from baseline to 12 months follow up. Further, after adjustment for differences in baseline characteristics, the Quality of Life at baseline was not significantly associated with smoking cessation at 12 months follow up.

Conclusions

A smoking cessation program delivered by cardiac nurses without special training, significantly reduced smoking rates 12 months after hospitalisation for coronary heart disease. The program was very cost effective compared to other treatment modalities in patients with coronary heart disease in terms of cost per life year gained. Among those with a high level of nicotine addiction, more effective cessation programs are needed.

List of publications

1. Quist-Paulsen P, Gallefoss F. Randomised controlled trial of smoking cessation intervention after admission for coronary heart disease. *BMJ* 2003; 327: 1254-1257.
2. Quist-Paulsen P, Bakke PS, Gallefoss F. Predictors of smoking cessation in patients admitted for acute coronary heart disease. *European Journal of Cardiovascular Prevention and Rehabilitation* 2005; 5: 472-478.
3. Quist-Paulsen P, Bakke PS, Gallefoss F. Does smoking cessation improve Quality of Life in patients with coronary heart disease? *Scandinavian Cardiovascular Journal* 2006; 40: 11-16.
4. Quist-Paulsen P, Lydersen S, Bakke PS, Gallefoss F. Cost effectiveness of a smoking cessation program in patients admitted for coronary heart disease. *European Journal of Cardiovascular Prevention and Rehabilitation* 2006; 13: 274-280.
5. Quist-Paulsen P, Gallefoss F. Is fear arousal message feasible in helping cardiac patients to stop smoking? ***Submitted.***

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Background

The history on Tobacco (1)

The origin of the tobacco plants are from America, and they have been used for millenniums by the Indians. Tobacco was introduced to Europeans with Columbus in 1492, and by the late 1600s it was grown on every continent. The appeal of nicotine in these plants quickly gained popularity. Traditionally the tobacco smoke was alkaline, where nicotine as a free base was readily absorbed by the oral mucosa (i.e. cigar smoke and snuff). In the mid-19th century the tobacco leaves were exposed to high temperatures, creating an acidic pH. This generated nicotine salts dissolved in droplets of smoke aerosol, and resulted in milder smoke which could be inhaled. The nicotine could thereby be absorbed by the respiratory epithelium. An ultimate addictive was created: A drug that gave a pleasant feeling with a very rapid increase in blood concentration after inhalation, reaching the brain within seconds. This discovery boosted the cigarette industry, and by the start of the 20th century famous companies like Camel and Lucky Strike spread cigarettes throughout the world.

By the 1920s concerns about a link with lung cancer were growing among physicians, and in 1939 a Science report showed that smokers had a substantially higher mortality rate than non-smokers. During the 1950s evidence that smoking caused serious diseases rapidly developed. By the early 1990s it became evident that passive smoking also was dangerous, and tobacco smoke was regarded as a major pollution problem. Since then, tobacco giant Philip Morris of Marlboro has lost all appeals and will have to pay more than $82 million to the widow of a long-time smoker, many countries have abandoned smoking in public places, and people are beginning to call for a prohibition law.

The prevalence of smoking

Although smoking have been slowly declining in the western Europe and north America during the last thirty years, it increases rapidly throughout the developing world, and up to 70% of the population in several Asian countries are now daily smokers. In the United States, approximately 20% are smoking (2), and in Norway 25% are daily smokers between 16 and 74 years of age (3). A further 11% are occasional smokers (3). Especially in western Europe and north America smoking is now most prevalent among people with a low level of education,. In Norway there are three times as many smokers in this group than in those with a high level of education (3).

The health problems of smoking

World-wide, approximately 5 million people die each year because of smoking (2, 4). Tobacco is regarded as the greatest preventable cause of death (4), and is one of the biggest threats to the world health (4). On average, lifelong smoking shortens the life expectancy with about 10 years (5), and it has been estimated that smoking one cigarette on average reduces life expectancy with 11 minutes (6). A survey of British male doctors showed that the chance of reaching 73 years of age in life long smokers were 42% compared to 78% in life long non-smokers (7). Pulmonary and cardiovascular diseases are the most frequent causes of death in smokers, causing approximately 50% and 35% of smoking related deaths, respectively (2, 4).

Smoking as a risk factor for coronary heart disease

Many observational studies have shown that smoking is an important risk factor for the development of coronary heart disease (8-11). Compared with non-smokers, current smokers have a two- to four-fold higher risk of coronary heart disease and sudden death (9-11). Even as few as one to four cigarettes per day increase the risk of myocardial infarction (12), and smokers are on average ten years younger than non-smokers when they develop myocardial infarction (13).

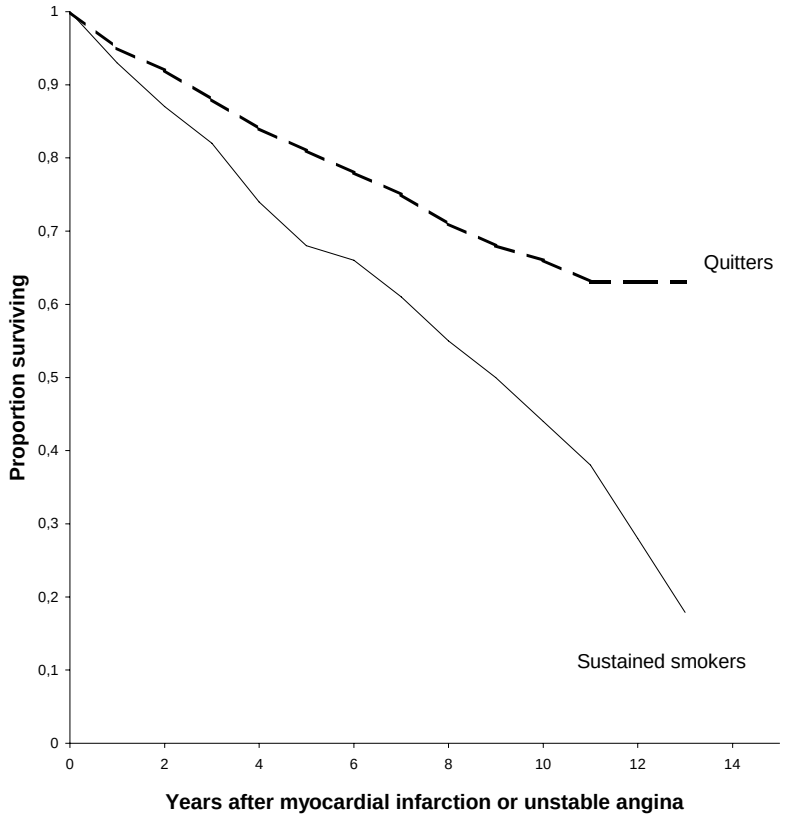
The tobacco smoke is composed of over 4000 components. The pathogenic mechanisms explaining why these components increase the risk of coronary heart disease are not fully understood. Nicotine itself does not seem to increase the risk significantly (14). Experiments have shown the following effects of cigarette smoking, all of which could contribute to coronary heart disease:

- Impaired endothelial function (15), which may be the cause of the increased atherosclerosis found in smokers (8).
- Vastly increased platelet activity, which arises acutely after smoking one cigarette (16).
- Acutely increased vascular resistance with decreased coronary flow velocity (17).
- Acutely vasospasm, including vasospastic angina (18).

The increased risk associated with smoking declines rapidly after cessation, and after two years of abstinence the relative risk nearly equals the risk of non-smokers (19), suggesting that factors other than atherosclerosis per se are involved in the pathogenesis.

Why is it important that patients with coronary heart disease quit smoking?

Smoking cessation after a myocardial infarction or unstable angina is associated with an approximately 50% relative mortality reduction after five years compared to sustained smoking (20, 21). With a longer follow up period, this reduction increases further, as shown by this figure adopted from Daly et. al. (22):



Thirteen years after myocardial infarction or unstable angina only 18% were still alive among those who continued to smoke, compared to 63% among the quitters.

It is both unethical and impossible to randomise patients to continued smoking or smoking abstinence after a coronary event. Therefore, we are left with observational data which is often misleading. Smokers with an acute coronary event tend to be younger and with fewer concomitant cardiac risk factors than non-smokers (13). Therefore, their initial prognosis have been shown to be more favourable than non-smokers ("smokers paradox") (13). Thus, significant differences in mortality between quitters and persistent smokers may take several years to develop. Further, most investigations have not verified quitters biochemically despite the fact that as many as 10-30% do not tell the truth about their smoking behaviour (23), and that many return to smoking within a year (24). All these biases tend to underestimate the effect of smoking cessation. Hence, the benefit of quitting smoking after a coronary event might be even greater than reported.

Why is it so difficult to quit smoking?

Each year about one third of the smokers try to quit, but only approximately 3% of the quit attempts result in sustained (12 months) cessation (25). Ten seconds after inhalation very high level of nicotine reaches the brain, and stimulates the nicotine acetylcholine receptors (25). In response, dopamine is released which speeds reaction times, and improves attention, concentration and problem-solving (14, 25). However, this system adapts after a few hours, and becomes downregulated (14, 25). But after a night sleep the system partly upregulates, and the first cigarette in the morning produces arousal and relaxation. Most regular smokers experience withdrawal symptoms some hours after the last cigarette: Irritability, restlessness, feeling miserable, and impaired concentration (25). These symptoms affect behaviour and are a strong impetus to start smoking again, as all symptoms vanish immediately after inhalation of a cigarette (14, 25). Thus, the withdrawal symptoms are an important cause of the addiction (14, 25).

Population strategies to prevent smoking

The most effective way to reduce the prevalence of smoking is public intervention (26). Evidence exists for the efficacy of the following initiatives: Abandoning smoking in public places and work places, prohibiting tobacco advertisement, educating people about the health hazards of smoking, providing cessation clinics/telephone counseling, and increasing the prices of the tobacco products (26).

What kind of smoking cessation modalities are effective among the general population?

In people without a special incitement for cessation, the sustained quit rates are rather low (i.e. 5%-10%), regardless of the type of intervention. However, several treatment modalities have proven their efficacy: Individual behavioural counseling (27), group behavioural therapy (28), telephone counselling (29), and self-help materials (i.e. booklet, video) (30). Regarding pharmaceutical products, nicotine replacement therapy has been shown to almost double the cessation rates (31), and bupropion might be of similar efficacy (32).

A stage based transtheoretical model has been widely recommended in smoking cessation (33). This model separates individuals into five different stages (precontemplation, contemplation, preparation, action, and maintenance), and different interventions are used in each stadium. Accurate investigation and treatment have to be applied as the individuals progressively go through the different stages (33). This process is rather complicated, and often needs psychologically trained

personnel. Why this method has gained so much popularity is difficult to understand as a systematic review did not find evidence for its efficacy (34). Simple interventions can probably suffice, and not even thorough planning seems necessary as most successful quit attempts may be unplanned (35)!

What kind of smoking cessation modalities are effective among hospitalised patients?

Among hospitalised patients, several trials have shown that intensive intervention started at hospital plus follow-up for at least one month significantly increases quit rates (36). Adding nicotine replacements seem to increase the quit rates further (36), and applying both methods can double the cessation rates compared to usual care (36). Brief interventions during hospitalisation have not shown significant effect (36, 37). The kind of provider does not seem to matter, as smoking cessation intervention delivered by psychologists, physicians, and nurses are all of similar efficacy (38).

Investigations on smoking cessation intervention in patients with coronary heart disease

Randomised investigations on smoking cessation methods in patients with coronary heart disease have obtained mixed results. Several studies of interventions to change lifestyle, where smoking cessation was only part of the intervention, have been performed (39-45). Most of them did not show any significant effect on quit rates (39-42).

Regarding studies only addressing smoking cessation, brief interventions during hospitalisation have been ineffective (46-48). Such brief interventions usually include a firm advice from a physician to stop smoking, information about the health hazards of continued smoking, and self help materials. It is this kind of smoking cessation intervention that is recommended from the Joint European Societies, which includes the European Society of Cardiology (49). It is also the most widely used method to increase quit rates among coronary heart patients (49). Therefore, it is very disappointing that this method does not seem to be of any significant effect (46-48). In a recent paper (49), and in a companion news report in the BMJ (50), more efficient ways to improve quit-rates were asked for.

Before the start of our project, three randomised trials had investigated whether a smoking cessation program with several months of intervention was able to increase the quit rates (51-53). The first was a Lancet paper showing 62% abstainers in the intervention group compared to 28% in the usual care group (51). The intervention principles were rather simple: Patients in the intervention group were told that continued smoking could lead to further heart attacks because it would

narrow the arteries in a manner similar to furring in a pipe, sometimes with complete blockage. Some information on how to quit were sometimes given. The article does not describe the length of the intervention, and unfortunately no biochemically verification of the quitters were performed. Another investigation in patients after coronary arteriography randomised patients to a behavioural smoking cessation program with a mean of four telephone calls during four months, or usual care (52). After 12 months there was a trend toward increased quit-rates in the intervention group, but not reaching standard level of statistical significance (57% versus 48% cessation rates in the intervention and the usual care group, respectively, p0.06). However, the intervention seemed effective in the subgroup with more severe disease. A third study showed a 71% cessation rate in the intervention group compared to 45% in the usual care group one year after admission for myocardial infarction (53). The intervention was delivered by especially trained nurses using social learning theory combined with addiction models. The patients were followed regularly for 4 months. The main drawback of this study was the application of a rather complicated psychological approach, which may be difficult to implement in clinical practice. After our trial was started in 1999, one more investigation has been published. Dornelas et. al. randomised 100 patients after myocardial infarction (54). The intervention was delivered by a psychologist implementing the transtheoretical model, and consisted of bedside counselling followed by regular telephone calls during 6 months. After 12 months 34% and 55% were abstinent in the usual care group and intervention group, respectively ($p<0.05$). The use of the complicated transtheoretical model may be unnecessary in cardiac patients, because most patients suffering an acute coronary event stop smoking while hospitalised (53, paper I). Therefore, they already are in the last stadium of the transtheoretical model (the maintenance stage), and focusing only on relapse prevention seems more logical. Further, the study had a low number of patients, and lack of biochemical verification of the quitters.

Regarding pharmaceuticals, there are no studies with a long-time follow up period only investigating the effects of nicotine replacements in patients with coronary heart disease (36). On the other hand, they have been incorporated in many of the studies mentioned above (41, 43, 46, 48, 53), and have been found to be safe (55). In a Cochrane review, the authors concluded that nicotine replacements probably increase quit rates if combined with high intensity behavioural intervention (36). Since the start of our project, bupropion has gained popularity. This psychopharmaceutical product has been shown to be safe and effective in patients with coronary heart disease, with a continuos cessation rate of 22% in the bupropion group compared to 9% in the placebo group at 12 months follow up ($p<0.01$) (56). However, its effect in combination with behavioural therapy in patients with heart disease is still unknown.

In conclusion, smoking cessation interventions given briefly during hospitalisation or given as part of a life style intervention program are ineffective regarding quit rates. At start of our study there were some evidence that an individual smoking cessation intervention with several months of follow up had significant

effect, and that such therapy should be combined with nicotine replacements. However, it was unknown whether such a program could be delivered by personnel without special education in smoking cessation, and whether simple intervention principles applicable in an ordinary clinical setting, could be used.

Who manage to quit smoking after a coronary event?

Several investigations have assessed the predictors of smoking cessation in patients with coronary heart disease (46, 53, 54, 58, 60-62). The results have been mixed, but the following predictors have rather consistently been found to be positively associated with smoking cessation:

- Low level of nicotine addiction (46) (most often as assessed by the Fagerstrom index (57)).
- High level of self confidence in smoking cessation (53, 54, 58) (as assessed by the total self efficacy score (59)).
- The severity of the coronary event, i.e. having myocardial infarction as reason for admission (46, 60, 61).
- Having no previous heart disease (62).
- Low level of hostility and depression (60).

Whether a smoking cessation program has any impact on these predictors has not previously been evaluated. A better characterisation of how these predictors are affected in a smoking cessation program may help to improve the intervention, and this was the ambiguous aim for paper II.

Does smoking cessation has any impact on Quality of Life?

As mentioned previously, quitting smoking is the most effective single action to reduce mortality after a coronary event. However, improvement in Quality of Life (QoL) may be equally important (63). Despite some authors claim that smoking cessation improves QoL (2), there are no evidence that this is true. Surprisingly few studies have assessed this question. In the general population, two studies have obtained mixed results (64, 65). In patients with coronary heart disease only one investigation has been performed on this topic (66), and this found that patients who managed to give up smoking after percutaneous coronary intervention improved their health-related QoL to a greater extent than sustained smokers. However, this study included patients without motivation to stop smoking and adequate adjustments for confounders may have been difficult to perform.

Thus, whether smoking cessation in patients with coronary heart disease has impact on QoL is largely unknown. Further, whether QoL life is a predictor of smoking cessation has never been analysed. These questions were the aims of paper III.

Is a smoking cessation program in patients with coronary heart disease cost effective?

After the main result of our trial was published, in our opinion there were no longer doubt that a smoking cessation program with several months of intervention significantly increased smoking cessation rates. Despite this, most hospitals do not provide such programs as part of routine care (49), possibly because they are thought not to be worth their costs. As the therapeutic arena becomes more crowded, and in times of health economic constraints, analyses of treatment costs relative to healthcare benefits are important. Cost effectiveness analyses in terms of cost per year of life saved or gained provide this opportunity, enabling us to compare the various treatment modalities (67). Several cost effectiveness analyses have been published on secondary prevention strategies for cardiovascular disease (68). Regarding smoking cessation intervention, only one study has been performed (69). This 13 year old analysis was based on a study of patients suffering myocardial infarction in the sixties (70), and showed that a smoking cessation program was cost effective compared to other treatment modalities. Whether similar smoking cessation programs after coronary revascularisation or unstable angina also are cost effective have previously been unknown. If a favourable cost effectiveness ratio of our cessation program could be demonstrated, this would serve as an argument for a wider implementation of similar programs.

Objectives

The main objectives of the present study in patients admitted for coronary heart disease were:

- To examine the effect of a smoking cessation program on the smoking cessation rates at 12 months follow-up (paper I).
- To assess the predictors of smoking cessation, and examine whether these could be influenced by a smoking cessation program (paper II).
- To evaluate whether smoking cessation improves health related QoL in a one year perspective (paper III).
- To perform a cost effectiveness analysis of the smoking cessation program (paper IV).
- To examine the patient satisfaction of a smoking cessation program based on a fear arousal message (paper V).

Materials and methods

Participants

All patients admitted to Sørlandet Hospital, Kristiansand for myocardial infarction, unstable angina or postoperative care after coronary bypass surgery were registered (bypass surgery was performed at hospitals in Oslo). Eligible patients had to be under 76 years of age and daily smokers until the start of their present coronary symptoms. Patients with bypass surgery had to be daily smokers until they received the date of operation, and the cause of operation had to be symptomatic coronary artery disease. Patients had to be recovered enough to reliably receive the intervention, had to be able to read Norwegian, and had to live in Vest- or Aust-Agder county. Patients with the following conditions were excluded: Serious illnesses with short life expectancies (cancer, serious chronic obstructive lung disease, serious renal or liver failure), serious psychiatric problems, alcoholism and dementia.

The study was approved by the regional ethics committee, and permission to establish a register of the participants was given by the National Data Supervision Centre ("Data-tilsynet").

Sample size

We aimed to detect a 20% difference between the two groups. With a power of 80% (β=0.2) and α<0.05, 98 patients were needed in each group (χ^2 , two tailed test, Sample Power version 1, SPSS Inc., Chicago). To allow for drop outs, an enrolment of 250 patients was decided. From the patient administration system in DIPS, the numbers of relevant patients available in 1997 were found. We assumed that 50% of available patients were smoking, that 15 % did not want to participate, and that 15% were not fulfilling the inclusion criteria. Thereby, an inclusion period of 2-2.5 years were estimated.

Randomisation

The study nurses recruited patients two to four days after admission. After signing a written informed consent and answering baseline questionnaires (appendix III-VIII), participants were randomly allocated to the control (usual care) or the intervention group. The nurses were given a serially numbered sealed envelope from a secretary who was otherwise uninvolved in the study. The randomisation was in blocks with

varying sizes. The envelopes were made by an external part who also was otherwise uninvolved in the study.

Intervention

Physicians were not involved in the program. Our intention was that all participants in the control and intervention groups should receive the physicians' ordinary quit-smoking messages, and no other special attention from doctors regarding smoking cessation. During the trial the doctors were not informed of the patients' inclusion or randomisation status.

Control group

All cardiac patients, independent of study participation, were offered group sessions conducted by cardiac nurses twice per week, in which the importance of smoking cessation was mentioned. A video shown during these sessions and a booklet handed out to all patients contained general information on coronary heart disease, which included advice to give up smoking. Besides this, the control group received no specific instructions on how to stop smoking.

Intervention group

One of three cardiac nurses consulted the patients one or two times during the hospital stay. The intervention was based on a 17 page booklet especially made for the purpose of the trial (appendix XI). This manual emphasised the health benefits of quitting smoking after a coronary event. Two figures showed the mortality differences between those who continued smoking and those who stopped smoking after myocardial infarction or unstable angina. One of the figures was a bar chart showing 60% risk reduction for death after 5 years if quitting (71), and the other was a linear chart showing that after 13 years 18% of continued smokers were alive compared to 63% of the quitters (22). On the basis of these figures the participants were told that they most probably would suffer a new heart attack if they continued smoking, and that their risk of death would be markedly increased if they continued smoking (fear arousal message).

The booklet also contained chapters on how to prevent relapse, how to stop smoking for those who either continued smoking or relapsed, and how to use nicotine replacements. How to identify and cope with high risk situations for relapse was explained, and action plans for coping with these situations were suggested.

The patients were strongly advised not to smoke during hospitalisation. Those with strong withdrawal urges were advised nicotine replacements (gum or patch). If spouses smoked, they also were asked to quit.

The study nurses initiated telephone contacts two days, one week, three weeks, three months, and five months after hospital discharge. Those with special needs were telephoned monthly thereafter. At six weeks, at the day they were scheduled for the follow up appointment with a physician, all participants in the intervention group had a consultation with the study nurses at the outpatient clinic. The outpatient contacts included positive feedback (e.g. ”Congratulations, you are still free of smoking. That means that you already have a much lesser chance of suffering a new heart attack.”) and relapse prevention. The health benefits of quitting were repeated, and if necessary a fear arousal message was given. Those who either continued smoking or relapsed were offered additional support and advice.

Apart from a one-day course in smoking cessation counselling, the study nursed had no special training in smoking cessation intervention.

Outcome measures

The participants were asked to return 12 months after inclusion for follow up assessment. Patients missing the appointment received a new letter. If they again missed the appointment, they were telephoned and asked to return to the hospital. If they still did not show up, a home visit was suggested.

Paper I - The smoking cessation rates

Patients who stated they were smoking at 12 months follow up were classified as smokers. Those who claimed to be quitters and had a urinary nicotine metabolite concentration < 2.0 mmol/ mol creatinine were classified as non-smokers (clinical decision limit as validated at Sahlgren’s University Hospital). Urinary nicotine metabolite analyses were performed at Sahlgren’s University Hospital, Sweden. The Diagnostic Product Corporation’s Nicotine Double Antibody Method with ^{125}I radioimmunoassay was used, with cotinine as standard for calibration, and calibration values 0.6-85.5 umol/L.

Paper II - The predictors of smoking cessation

The study nurses prospectively recorded medical history and sociodemographic data (appendix III, IV). The participants filled in three questionnaires before randomisation, containing information on smoking habits (appendix V), nicotine addiction (the “Fagerstrom score”, appendix VI) and self-efficacy in smoking

cessation (the "Motivation and Relapse" form, appendix VII). Further details regarding these forms have been explained in paper II. The patients' use of nicotine substitutions and their partners' smoking status were registered at 12 months follow up.

Paper III - The Quality of Life after smoking cessation

Health related QoL was assessed before randomisation and at 12 months follow up. The questionnaire used in the Cardiac Arrhythmia Suppression Trial (CAST) was employed (72). This questionnaire contains a battery of established scales and items, to produce a specific health-related QoL instrument for patients with coronary heart disease. Six QoL dimensions were derived from 24 questions addressing the topics Social function, Physical function, Symptoms, Mental health, Life satisfaction, and Life expectancy (appendix V). The questionnaire has been designed to be especially sensitive to clinical changes in patients with coronary heart disease (73), and has been shown to be reliable, clinically valid, and sensitive to changes over time (73, 74). In an editorial comment the questionnaire was advocated for assessment of health-related QoL in patients with coronary heart disease (75). For further detail regarding the QoL measurements, see paper III.

Paper IV - The cost effectiveness of the smoking cessation program

The incremental cost effectiveness of the program was assessed as the cost per life year gained, and was calculated using the following formula:

(Cost of the program per patient x Number needed to treat to get one additional quitter (NNT)) / Gain in mean discounted life years per patient among quitters compared to sustained smokers.

The variables in this formula were derived as follows:

- The cost of the program was calculated on the basis of Norwegian prices in 2000 and were converted to Euro at the 2000 mean exchange rate (Euro 1=8.1 Norwegian Krones, NOK). The nursing costs were estimated from the average salary of specialised nurses in Norway with more than ten years of seniority (190 NOK/h). The printing costs of the self help material (17 NOK per booklet) were included. The office rental was set to 1500 NOK per square meter per year. The costs of telephoning were calculated using the prices of the telephone company Telenor (0.89 NOK per call + 0.49 NOK per minute). Because the expenses of the program only lasted for less than a year, discounting was not performed regarding the costs. Indirect costs (i.e. time lost from work while participating in the program) were not included in analysis because the intervention after discharge

from hospital were brief and mostly by phone. For further details regarding the estimation of the cost of the program, see paper IV.

- The number needed to treat (NNT) to get one additional quitter from the smoking cessation program was derived from the absolute risk reduction (ARR) in smoking rates in the intervention group compared to the usual care group at 12 months follow up (NNT= 100/ARR).

- The gain in mean discounted life years per patient in quitters compared to sustained smokers were obtained from the differences in integrals between the survival curves of quitters and sustained smokers, and was assessed in a low- and high risk model. In the low risk setting survival data from van Domburg *et. al*. were used. They investigated 985 patients who underwent coronary bypass surgery during the seventies (76), and found an average annual mortality rate of 1.7% at 10 years follow up. This rate of mortality rate was similar to the findings in the Scandinavian simvastatin survival study (4S) of patients with stable coronary artery disease (77). After a median follow up period of 20 years, the relative risk reduction in quitters compared to the sustained smokers was 28% (76), which is lower than in comparable investigations (21). The Kaplan Meier method was used for analysing the survival data for the first 20 years (76), and we extrapolated the survival curves further by using the Gompertz parametric survival function (78).

Patients suffering myocardial infarction or unstable angina were chosen as the high risk group. There are no recent studies with a long follow up period and with proper verification of quitters, investigating the mortality benefit if quitting smoking in these patients. Therefore, a rather old investigation had to be chosen when calculating the survival differences in quitters and sustained smokers in a high risk setting (22). However, the rate of mortality in this study was similar to the annual mortality rate of 4.5% found in a more recent survey of patients after thrombolysis for myocardial infarction (79). Survival curves were estimated for 13 years using life table methods (22), and were estimated in a life time perspective (i.e. 25 years) by assuming the survival thereafter followed an exponential function (the declining exponential approximation of life expectancy (80)).

The differences in integrals between the survival curves of quitters and sustained smokers were then calculated from year i to year i+1, multiplying that by $1/1.05^i$ (5% discounting per year (67)) and summing for i = 1 to 40 in the low risk model and i = 1 to 25 in the high risk model. A short time perspective (five years) was also set up, summing for i = 1 to 5.

See paper IV for further details regarding the estimation of the gain in life expectancies in quitters versus sustained smokers.

Paper V - The patient satisfaction

At 12 months follow up the patient satisfaction was measured by the "Evaluation form" (appendix X), containing questions on the degree the participants felt they were helped by the hospital in quitting smoking and on the satisfaction of this help

Statistical methods

The χ^2 test was used to assess the smoking cessation rates in the control group compared to the intervention group, and to assess other differences between the randomised groups when variables were categorical. The number needed to treat with confidence intervals, were calculated (81). Continuos variables were tested for normal or skewed distribution by the Lilliefors' test. The differences in means (continuos variables) between the randomised groups were assessed with the independent samples T tests and the Mann Whitney U-test for normally and nonnormally distributed data, respectively.

Regarding the evaluation of differences in means (i.e. the QoL- and the patient satisfaction scores) between non-randomised groups (i.e. quitters versus sustained smokers), multivariate linear regression analyses were applied in order to be able to adjust for differences in baseline characteristics.

When analysing differences within groups (i.e. the QoL improvements from admission to 12 months follow up) paired samples t-test and Wilcoxon test for two related samples were used for normally and nonnormally distributed data, respectively.

Univariate and multivariate logistic regression models were used to test the relations between baseline characteristics (covariates/predictors) and the cotinine-validated smoking cessation rates at 12 months (dependent variable). Variables showing significant correlation in univariate analyses were included in the multivariate tests. In the subgroup analyses, interaction terms were added to the logistic regression models to examine if the influence of the covariates on the dependent variables were significantly different in the control group compared to the intervention group (subgroup interaction analysis).

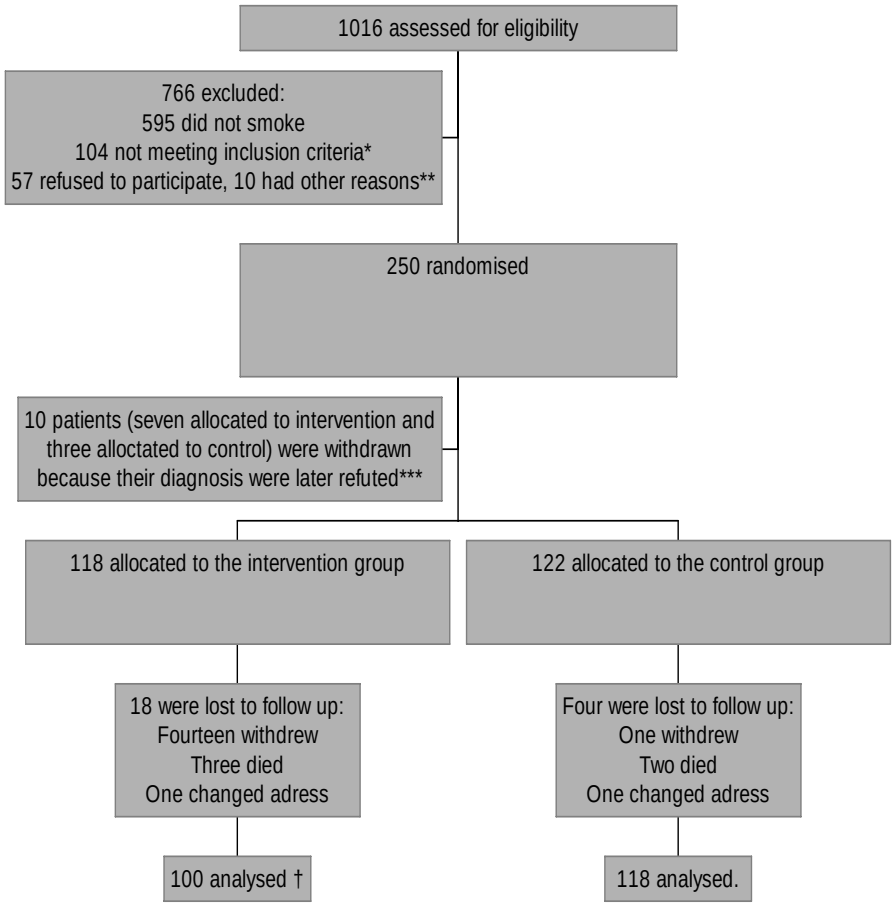
All tests were two-tailed with 0.05 significance level (alpha) and 95% confidence interval. We used SPSS for Windows (version 11 and 12) for all analyses.

Results

Paper I - The smoking cessation rates

Patients were recruited from February 1999 to September 2001. The following figure shows the participant flow through the trial:

Figure 1. Participant flow



* Sixty-two patients were older than 75 years, four did not live in a nearby community, five did not read Norwegian, seven had dementia, nine had psychiatric problems, nine had other serious illnesses and eight were alcoholics.

** Seven died before randomisation, one had no telephone, and two were registered too late.

*** All had normal coronary angiograms.

† All received the allocated intervention

Most patients (85%) received more telephone calls than the intended minimum of five. The mean total time devoted to each patient was approximately 2.5 hours, including time to fill in questionnaires for the purpose of the trial. A third of the participants used nicotine replacements.

At 12 months follow up, the intervention group had a statistically significant 20% increased cessation rate compared to the control group (NNT 5). Due to a higher drop out rate in the intervention group, in an intention to treat analysis the difference in quit rates between the groups was 13% (NNT 8, still statistically significant).

The groups showed similar smoking cessation rates while in hospital and at six weeks' follow up.

Paper II - The predictors of smoking cessation

When analysing the control and intervention groups combined, a high level of nicotine addiction, a low level of self confidence in quitting, and having previous coronary heart disease all were significant negative predictors of smoking cessation in a multivariate analysis. We speculate whether our smoking cessation program was especially important among patients with no previous coronary heart disease and another diagnosis than myocardial infarction, although the differences between groups did not reach level of significance in subgroup interaction analyses. A high level of nicotine addiction was the strongest negative predictor in both groups. Our results indicate that the level of nicotine addiction can be assessed with one simple question; are you smoking the first cigarette in the morning within 30 min of waking? This is easier than applying the Fagerstrom Questionnaire which covers eight items of various aspects of smoking behaviour.

Paper III - The Quality of Life after smoking cessation

Even when applying a questionnaire especially designed to be sensitive for changes in patients with coronary heart disease, we did not find that the health-related QoL improved with smoking cessation in a one year perspective. Further, the QoL was not a significant predictor of smoking cessation.

Paper IV - The cost effectiveness of the smoking cessation program

Due to the vast uncertainties associated with cost effectiveness analyses we aimed to do conservative assumptions, thereby calculating a maximum cost per life year

gained by the program. The only important cost of the program was the 2.5 hours of a nurse's working time. This one time investment gave a much lower cost effectiveness ratio than pharmaceuticals which have to be taken every day for years (i.e. 1/25 the cost of both statins in the low risk group and angiotensin converting enzyme inhibitors in the high risk group).

Paper V - The patient satisfaction

Compared to the control group, participants in the intervention group stated they had got significantly more information on the tobacco's effect on the heart, that the hospital had helped them significantly more in quitting smoking, and that they were significantly more satisfied with the help they had got from the hospital in quitting smoking (question 2, 5 and 6 from the "Evaluation form", appendix X). On a scale from 1 (very unsatisfied) to 5 (very satisfied), the participants in the intervention and control group had a mean score of 4.1 and 2.9, respectively, on these three questions. These differences were not due to increased cessation rates in the intervention group, as both sustained smokers and quitters scored significantly higher in the intervention group than in the control group on all the three questions.

Discussion

Methodological considerations

The study design and the smoking cessation rates

A randomised controlled trial (RCT) is the gold standard in evidenced based medicine (82, 83), but does encompass some problems. Many RCTs only include a highly selected group of patients, thereby making the results difficult to implement in daily practice. We wanted to create a study which was applicable and feasible in an ordinary clinical setting. Therefore, we had to include most smoking patients with coronary heart disease, and the intervention principles had to be simple. Of eligible patients, 19% (n=57) refused to participate. Most of these patients did not want to quit smoking. A wish to stop smoking was a prerequisite for inclusion in the trial. The applicability of our results could have been further strengthened by also inviting these patients to participate. On the other hand, working with participants not motivated to quit smoking is most probably ineffective, and might have been demoralising for the study nurses.

Ideally a RCT should be double blinded (83), but due to the type of intervention blinding was not possible in our trial. We tried to compensate for this potential bias by not telling the participants in which group they were allocated. Still, the intervention group may have regarded themselves as a positively selected group of patients, which could possibly have led to increased cessation rates in this group. However, also the control group may have attained this feeling as many of them actually believed they were in a group with active treatment due to the standard information about the importance of smoking cessation given to all cardiac patients in the ward. Not giving informed consent could have abolished these biases, but according to the Declaration of Helsinki this is undesirable (83).

It was not possible to separate the two groups in the ward. Therefore, the control group may have been "contaminated" by patients in the intervention group passing along important messages from the booklet. Increased focus on smoking cessation among staff members due to the present trial may also have increased the possibility that the control group received a better standard of care than usual. The lack of difference in the smoking cessation rates at hospital discharge and at 6 weeks follow up may support this point of view.

A drop out rate of less than 10% was lower than in comparable studies (53, 54), and half of the assumed when writing the protocol. Such a low drop out rate increases the applicability of the study. However, the drop out rate was higher in the

intervention group than in the control group, and this may have been a result of the intervention itself. When the participants withdrew they gave various reasons for withdrawal: Some changed their mind during hospitalisation because they did not want to take part in a study after all, some did not want to quit smoking after all, and some were sure they could stay free of smoking without help from others. At time of withdrawal, half of the patients stated they had stopped smoking (not verified biochemically). In an intention to treat analysis, counting all drop outs as continued smokers, the intervention group still had a statistically significant increased cessation rate compared to the control group, but the difference of 13% was rather small.

Biochemical validation of the smoking status is important because some patients do not tell the truth about their smoking behaviour (23). There are several methods to validate smoking cessation (84, 85). CO measurements in breath or blood are inadequate in detecting occasional and light smokers (86). Cotinine measurements in serum, saliva or urine is the most widely used method. Cotinine is a metabolite from nicotine, and all nicotine products (i.e. patches, gum, snuff) will increase the cotinine concentration above the cut-off value for non-smokers. The half life of cotinine is approximately 20 hours, and a person who uses nicotine replacements may have an increased cotinine concentration for several days after cessation (84, 85). Thus, patients using snuff or nicotine replacements were asked to stop this for 10 days. However, this was difficult to accomplish, and on many occasions we had to collect the urine samples after shorter time periods. Thiocyanate measurements would have abolished this bias, because thiocyanate is not increased in patients using snuff or nicotine replacements as it derives from the cyanide in the tobacco smoke. However, thiocyanate measurements has been reported to have a relatively low specificity due to its presence in various nourishments (87). After smoking cessation its concentration remains elevated for up to a month (88), but in our study this would not have posed problems since only two of the quitters stated they stopped smoking between 6 and 12 months of follow up. Retrospectively, using thiocyanate measurements might have decreased the number of false positive values.

It could be debated whether continuos abstinence from hospital discharge until 12 months follow up rather than point prevalence cessation rates at 12 months follow up, and whether a longer follow up period would have been more adequate when assessing the cessation rates. However, in our trial 93% of the quitters at 12 months follow up stopped smoking while hospitalised, and others have found that return to smoking after one year is very rare (24). Therefore, we believe our methods in assessing the cessation rates were adequate.

The search for predictors

In paper II, we tried to evaluate the predictors of smoking cessation using multiple logistic regression models. It is debated what to include in this type of analysis (89). Including too many variables may turn a variable that is not associated with the outcome at all into an independent risk factor, and factors that are closely related may

both loose level of significance (89). It is tempting to do many tests, varying the inclusion of risk factors, and thereby torture the data until they speak. Ideally, every test and method should be planned a priori and stated in the protocol. This is often done regarding the primary outcome, but not regarding the secondary outcomes as was the case in our protocol. We believe only a limited set of clearly unrelated baseline characteristics being significant at the 0.05 level in univariate tests should be included in a multivariate test, and have practised this throughout. Further, if finding a statistically significant independent risk factor/predictor in a multivariate analysis, we have tried not to imply causality (i.e. grey hear is a significant independent risk factor for coronary heart disease, but not a causal factor).

We also tried to analyse whether the effect of the smoking cessation program differed between subgroups. This kind of analysis is performed in order to assess whether the treatment suits the patient you are currently dealing with (i.e. is the cessation program effective in a patient who still smokes when admitted for his second myocardial infarction). There are no consensus regarding such analyses (90), and the CONSORT statement includes only a few lines on subgroup analysis (91). The problem of multiplicity arises. If you test ten associations, and the null hypothesis is true in all associations, the probability of finding one significant difference by chance is 0.4 if a significance value of 0.05 is used in each test ($1-[1-0.05]^{10}$) (92). Therefore, it is important that every test is prespecified in the protocol, that all performed tests are clarified in the paper (not only those with significant results), and that proper statistical adjustments for multiplicity are performed. This have often not been the case, and serious false conclusions have been made because of this (i.e. aspirin is ineffective after ischemic stroke in woman, and tamoxifen is ineffective in woman with breast cancer aged <50 years) (90). The only reliable statistical approach in subgroup analyses is to test for a subgroup-treatment interaction (90). This approach requires widely differing risks of an outcome with or without intervention to reach level of significance (90), and trials are seldom powered to perform subgroup analyses with acceptable type II error (β) (90). On basis of these limitations regarding subgroup analyses, we first approached the predictors in the whole set of data and subsequently analysed a limited number of them in subgroup analyses. Power problems may have been the reason why we did not find any significant differences between subgroups on cessation rates in the intervention versus the control group. But the the assessment of this was probably too ambiguous, and the results can at best be regarded as hypothesis-generating.

The Quality of Life assessment

It is impossible to randomise patients to either smoking cessation or continued smoking. Assessments of QoL improvements in quitters versus sustained smokers are therefore difficult to perform, as the results may be biased by unmeasured confounders (i.e. patients not able to quit smoking may be a negatively selected group with low potential for QoL improvements). Multiple regression analyses have commonly been performed in order to adjust for differences in baseline

characteristics in non-randomised groups. However, adjustment for all confounders is impossible (89), as exemplified by observational data showing that vitamins and estrogens reduce cardiovascular mortality, both which have later been refuted by randomised trials (93, 94). In contrast to the only other investigation on this topic in cardiac patients (66), we only included patients motivated to quit smoking. We may therefore have been more successful in adjusting for differences in baseline characteristics, and this could explain why our results contradict the findings by Taira et. al. (66).

Significant improvements in physical symptoms in quitters compared to sustained smokers may take several years to develop, and it is possible that our negative results regarding health-related QoL improvements were due to a too short follow up period.

The cost effectiveness analysis

The differences in integrals between the survival curves of quitters versus sustained smokers translates to the gain in life expectancy if quitting smoking, and the cost per life year gained (the cost effectiveness ratio) can easily be calculated. The main problem of calculating the cost effectiveness of the smoking cessation program was the lack of high quality survival curves in quitters and sustained smokers after a coronary event. As explained in the introduction, observational studies on this topic tend to underestimate the benefit of smoking cessation. Another problem is the short follow up period of most studies. Therefore, methods to estimate survival curves in the life time perspective are necessary, but unfortunate these methods are hampered by uncertainties (78).

The patient satisfaction

Research on communicating messages arousing fear have shown that such messages are effective when they are accompanied by education on how to reduce the health threat (95). However, the use of fear arousal message is controversial, and our trial has been criticised by health psychologists stating it may provoke defensive responses and emotions such as denial of personal risk, hostility, anger and anxiety (96, 97). In paper V we seem to have refuted these believes, as the participants in general were very satisfied with the cessation program. However, due to the following reasons it can be debated whether the patient satisfaction scores specifically measured the participants' feelings regarding the fear arousal message: First, the patient satisfaction was recorded 12 months after the introduction of the fear arousal message, at which point the anger and hostility, if previously present, might have been forgotten. Second, we did not ask the participants specifically how they evaluated the fear arousal message. Other elements in the program may have outweighed the negative feelings around the fear arousal message. Third, the delivery of the message was probably important. The study nurses tried to give the message

with empathy, and in a positive way (i.e. if you manage to give up smoking, your risk of suffering another heart attack will be cut down to the half compared with continued smoking.). Hence, some of the patients may not have recognised that they actually received a fear arousal message.

Discussion of the main findings

> *"Everything should be made as simple as possible, but not simpler." Albert Einstein.*

Approximately 80% of the participants did not smoke during the initial hospitalisation, and over 90% of the quitters at 12 months follow up stopped smoking while hospitalised. Others have found similar figures (53). Thus, smoking cessation programs in patients with coronary heart disease only have to focus on relapse prevention. Complicated approaches are probably not necessary. Because the intervention and the control group showed similar smoking cessation rates while in hospital and at six weeks' follow up, we speculate that a long follow up period was the most important element in the program. This point of view is supported by the fact that most smoking cessation trials showing an effect on quit rates in hospitalised patients have implemented a long follow up period (36, 51-53), and on the contrary that studies with brief interventions have been ineffective (37, 46-48).

We believe there are three important elements in smoking cessation programs in cardiac patients:

1. You have to inform on the health hazards of continued smoking, thereby increasing the patients' motivation to stay free of smoking ("fear arousal message"). It may be important to repeat this information as patients may forget and deny.

2. You have to follow-up the patients for several months. Someone has to care about the patients and their smoking behaviour. The patients need to know that someone is going to call them, and ask whether they are still free of smoke.

3. We believe most patients, especially those smoking within 30 min of waking, should be offered nicotine replacements and/or bupropion. Further studies are needed to determine the efficacy of this strategy.

The relatively high inclusion rate make our results applicable to an ordinary clinical setting. The simple intervention principles, without especially educated personnel, should make the program feasible to every cardiac ward.

Direct comparison of the cost effectiveness of the smoking cessation program against the various medications used in patients with coronary heart disease is not straightforward, because the prices of the pharmaceuticals have fallen since their respective analyses were performed. But all of the cost effectiveness analyses on pharmaceuticals have discounted the costs by approximately 5% per year (98-100). Further, most of them have tried to estimate the exact cost per life year gained (98-101), and not a maximum cost like we did. Therefore, we are confident that the smoking cessation program is very cost effective compared to other treatment modalities in patients with coronary heart disease.

Why is the easiest, cheapest and most effective method to reduce mortality not part of routine care in most cardiac wards? Is working with this topic too "low technology", and associated with too low prestige? Is it due to too few resources in marketing compared to the pharmaceutical industry? Anyway, we hope the results presented in this thesis will be helpful in increasing the implementation of effective smoking cessation programs in cardiac clinics, and thereby saving lives world-wide.

Source of data

1. Bolliger CT, Fagerstøm KO (eds). The Tobacco Epidemic. Prog Respir Res. Basel, Karger, 1997, vol 28, pp 1-11.

2. Schroeder SA. What to Do With a Patient Who Smokes. *JAMA* 2005;294:482-87.

3. Statistisk Sentral Byrå: www.ssb.no/emner/03/01/royk. Accessed March 13, 2006.

4. Edwards R. The problem of tobacco smoking. *BMJ* 2004;328:217-19.

5. Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ*. 2004;329:1519

6. Shaw M, Mitchell R, Dorling D. Time for a smoke? One cigarette reduces your life by 11 minutes. *BMJ*. 2000; 320: 53.

7. Philips AN, Wannamethee SG, Walker M, Thomson A, Smith GD. Life expectancy in men who have never smoked and those who have smoked continuously: 15 year follow up of large cohort of middle aged British men. *BMJ* 1996;313:907-908.

8. Rigotti NA, Pasternak RC. Cigarette smoking and coronary heart disease. *Cardiology clinics* 1996;14:51-68.

9. Jonas MA, Oates JA, Ockene JK, *et.al.* Statement on smoking and cardiovascular disease for health care professionals: AHA Medical/Scientific Statement. *Circulation* 1992;86:1664-1669.

10. Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years' observations on male British doctors. *BMJ*. 1994;309:901-11.

11. Phillips AN, Wannamethee SG, Walker M, Thomson A, Davey Smith G. Life expectancy in men who have never smoked and those who have smoked continuously: 15 year follow up of large cohort of middle aged British men. *BMJ*. 1996;313:907–908.

12. Willet WC, Green A, Stampfer MJ, *et. al*. Relative and absolute excess risk of coronary heart disease among women who smokes cigarettes. *N Engl J Med* 1987;317:1303-9.

13. Jorgensen Stig, Kober L, Ottensen M, Torp-Pedersen C, Videbaek J, Kjoller E. The Prognostic Importance Of Smoking Status At The Time Of Acute Myocardial Infarction In 6676. *J Cardiovasc Risk* 1999; 6:23–27.

14. Benowitz NL. Cigarette smoking and nicotine addiction. *Med Clin North Am* 1992;76:415-37.

15. Celermajer DS, Sorensen KE, Georgakopoulos D. Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent dilatation in healthy young adults. *Circulation* 1993;88:2149-55.

16. Pittilo RM, Clarke JM, Harris D, *et al*. Cigarette smoking and platelet adhesion. *Br J Haematol* 1984;58:627-32.

17. Quillen JE, Rossen JD, Oskarsson HJ, *et al*. Acute effect of cigarette smoking on the coronary circulation: Constriction of epicardial and resistance vessels. *J Am Coll Cardiol* 1993;22:642-47.

18. Sugiishi M. Cigarette smoking is a major risk factor for coronary spasm. *Circulation* 1993;87:76-9.

19. Rosenberg L, Kaufman DW, Helmrich SP, *et al.* The risk of myocardial infarction after quitting smoking in menn under 55 years of age. *N Engl J Med* 1990;322:213-17.

20. Wilson K, Gibson N, Willan A, Cook D. Effect of smoking cessation on mortality after myocardial infarction: meta-analysis of cohort studies. *Arch Intern Med* 2000;160:939-44.

21. Critchley JA, Capewell MD. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease. *JAMA* 2003; 290(1): 86-97.

22. Daly LE, Mulcahy R, Graham IM, Hickey N. Long term effect on mortality of stopping smoking after unstable angina and myocardial infarction. *BMJ* 1983;287:324-6.

23. Woodward M, Tunstall-Pedoe H. Biochemical evidence of persistent heavy smoking after a coronary diagnosis despite self-reported reduction: analysis from the Scottish Heart Health Study. *Eur Heart J* 1992; **13**:160-165

24. Hunt W, Barnet L, Branch L. Relapse rates in addiction programs. *Journal of Clinical Psychiatry* 1977;(27):455

25. Jarvis MJ. Why people smoke. *BMJ* 2004;328:277-9.

26. Jamrozik K. Population strategies to prevent smoking. *BMJ* 2004;328:759-762.

27. Lancaster T, Stead LF. Individual behavioural counselling for smoking cessation. *The Cochrane Databse of Systematic Reviews* 2005;(2): CD001292.

28. Stead LF, Lancaster T. Group behavioural therapy programmes for smoking cessation. *The Cochrane Databse of Systematic Reviews* 2002;(3): CD001007.

29. Stead LF, Lancaster T, Perera R. Telephone counselling for smoking cessation. *The Cochrane Databse of Systematic Reviews* 2001;(2): CD002850.

30. Lancaster T, Stead LF. Self-help interventions for smoking cessation. *The Cochrane Databse of Systematic Reviews* 2005;(3): CD001118.

31. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. Nicotine replacement therapy for smoking cessation. *The Cochrane Databse of Systematic Reviews* 2004;(3): CD000146.

32. Jorenby D. Clinical efficacy of bupropion in the management of smoking cessation. Drugs 2002;62 Suppl 2:25-35.

33. Prochaska JO, DiClemente CC, Norcross JC. In search of how people change. Applications to addictive behaviors. *Am Psychol* 1992;47:1102-14

34. Riemsma RP, Pattenden J, Bridle C, Sowden AJ, Mather L, Watt IS, Walker A. Systematic review of the effectiveness of stage based interventions to promote smoking cessation. *BMJ* 2003;326:1175-77.

35. West R, Sohal T, "Catastrophic" pathways to smoking cessation: findings from national survey. *BMJ* 2006;332:458-69.

36. Rigotti NA, Munafo MR, Murphy MF, Stead LF. Interventions for smoking cessation in hospitalised patients. *The Cochrane Databse of Systematic Reviews* 2001;(2): CD001837.

37. Hajek P, Taylor TZ, Mills P. Brief intervention during hospital admission to help patients to give up smoking after myocardial infarction and bypass surgery: randomised controlled trial. *BMJ* 2002;324:87-9.

38. Mojica WA, Suttorp MJ, Sherman SE, Morton SC, Roth EA, Maglione MA, *et.al.* Smoking-cessation interventions by type of provider: a meta-analysis. *Am J Prev Med* 2004;26:391-401.

39. Van Elderen-van Kemenade T, Maes S, van den Broek Y. Effects of a health education programme with telephone follow-up during cardiac rehabilitation. *Br J Clin Psychol* 1994;33:367-78.

40. Jolly K, Bradley F, Sharp S, Smith H, Thompson S, Kinmonth AL, et al. Randomised controlled trial of follow up care in general practice of patients with myocardial infarction and angina: final results of the Southampton heart integrated care project (SHIP). The SHIP Collaborative Group. *BMJ* 1999;318:706-11.

41. Campbell NC, Ritchie LD, Thain J, Deans HG, Rawles JM, Squair JL. Secondary prevention in coronary heart disease: a randomised trial of nurse led clinics in primary care. *BMJ* 2002;324:87-9.

42. Murchie P, Campbell NC, Ritchie LD, Simpson JA, Thain J. Secondary prevention clinics for coronary heart disease: four year follow up of a randomised controlled trial in primary care. *BMJ* 2003;326:84-90.

43. DeBusk RF, Miller NH, Superko HR, Dennis CA, Thomas RJ, Lew HT, et al. A case-management system for coronary risk factor modification after acute myocardial infarction. *Ann Intern Med* 1994;120:721-9.

44. Carlsson R, Lindberg G, Westin L, Israelsson B. Influence of coronary nursing management follow up on lifestyle after acute myocardial infarction. *Heart* 1997;77:256-9.

45. The Vestfold Heartcare Study Group. Influence on lifestyle measures and five-year coronary risk by a comprehensive lifestyle intervention programme in patients with coronary heart disease. *Eur J Cardiovasc Prev Rehabil* 2003;10:429-37

46. Hajek P, Taylor TZ, Mills P. Brief intervention during hospital admission to help patients to give up smoking after myocardial infarction and bypass surgery: randomised controlled trial. *BMJ* 2002;324:87-9.

47. Moreno Ortigosa A, Ochoa Gomez FJ, Ramalle-Gomara E, Saralegui Reta I, Fernandez Esteban MV, Quintana Diaz M. Efficacy of an intervention in smoking cessation in patients with myocardial infarction.. *Med Clin (Barc)* 2000;114:209-10. (In Spanish.)

48. Rigotti NA, McKool KM, Shiffman S. Predictors of smoking cessation after coronary artery bypass graft surgery. Results of a randomized trial with 5-year follow-up. *Ann Intern Med* 1994;120:287-93.

49. Reimer W S, de Swart E, de Bacquer D, Pyørælæ K, Keil U, Heidirch J, *et. al*. Smoking behaviour in European patients with established coronary heart disease. *Eur Heart J* 2006;27:35-41.

50. Kmietowicz Z. More than half of smokers go on smoking after coronary events. *BMJ* 2005;331:862.

51. Burt A, Thornley P, Illingworth D, White P, Shaw TR, Turner R. Stopping smoking after myocardial infarction. *Lancet* 1974;1:304-6.

52. Ockene J, Kristeller JL, Goldberg R, Ockene I, Merriam P, Barrett S, et al. Smoking cessation and severity of disease: the coronary artery smoking intervention study. *Health Psychol* 1992;11:119-26.

53. Taylor CB, Houston-Miller N, Killen JD, DeBusk RF. Smoking cessation after acute myocardial infarction: effects of a nurse-managed intervention. *Ann Intern Med* 1990;113:118-23.

54. Dornelas EA, Sampson RA, Gray JF, Waters D, Thompson PD. A randomized controlled trial of smoking cessation counselling after myocardial infarction. *Prev Med* 2000;30:261-8.

55. Working Group for the Study of Transdermal Nicotine in Patients With Coronary Artery Disease. Nicotine replacement therapy for patients with coronary artery disease. *Arch intern Med* 1994;154:989-95.

56. Tonstad S, Farsang C, Klaene G, Lewis K, Manolis A, Perruchoud AP, et al. Bupropion SR for smoking cessation in smokers with cardiovascular disease: a multicentre, randomised study. *Eur Heart J*. 2003;24(10):946-55.

57. Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction* 1991;86(9):1119-27.

58. Hasdai D, Garratt KN, Grill DE, Mathew V, Lerman A, Gau GT, *et al*..Predictors of smoking cessation after percutaneous coronary revascularization. *Mayo Clin Proc*. 1998;73(3):205-9.

59. Yates AJ, Thain J. Self-efficacy as a predictor of relapse following voluntary cessation of smoking. *Addictive Behaviors* 1985;10(3):291-8.

60. Brummett BH, Babyak MA, Mark DC, Williams RB, Siegler IC, Clapp-Channing N, *et. al*.. Predictors of smoking cessation in patients with a diagnosis of coronary artery disease..*J Cardiopul Rehab* 2002;22:143-7.

61. Ockene J, Kristeller JL, Goldberg R, Ockene I, Merriam P, Barrett S. Smoking cessation and Severety of Disease: The Coronary Artery Smoking Intervention Study. *Health Psychol* 1992, 11(2), 119-126.

62. Attebring MF, Hartford M, Hjalmarson A, Caidahl K, Karlsson T, Herlitz. Smoking habits and predictors of continued smoking in patients with acute coronary syndromes. *J Adv Nurs* 2004; 46(6):614-23.

63. Probstfield JL. How cost-effective are new preventive strategies for cardiovascular disease? *Am J Cardiol*. 2003;91:22G-27G.

64. Mitra M, Chung MC, Wilber N, Klein Walker D. Smoking status and quality of life: a longitudinal study among adults with disabilities. *Am J Prev Med*. 2004;27:258-60.

65. Tillmann M, Silcock J. A comparison of smokers' and ex-smokers' health-related quality of life. *J Public Health Med*. 1997;19:268-73.

66. Taira DA, Seto TB, Ho KK, Krumholz HM, Cutlip DE, Berezin R, Kuntz RE, Cohen DJ. Impact of smoking on health-related quality of life after percutaneous coronary revascularization. Circulation. 2000;102:1369-74.

67. Kupersmith J, Holmes-Rovner M, Hogan A, Rovner D, Gardiner J. Cost-effectiveness analysis in heart disease, part I: general principles. *Prog Cardiovasc Dis* 1994; 37: 161-84.

68. Probstfield J L. How cost-effective are new preventive strategies for cardiovascular disease? *Am J Cardiol* 2003;91(suppl):22G-27G.

69. Krumholz HM, Cohen BJ, Tsevat J, Pasternak RC, Weinstein MC. Cost-Effectiveness of a Smoking Cessation Program After Myocardial Infarction. *JACC*. 1993; 22(6): 1697-702.

70. Åberg A, Bergstrand R, Johansson S, Ulvenstam G, Vedin A, Wedel H, et al. Cessation of smoking after myocardial infarction. Effects on mortality after 10 years. *Br Heart J* 1983; 49: 416-22.

71. Sparrow D, Dawber TR. The influence of cigarette smoking on prognosis after a first myocardial infarction. A report from the Framingham study. *Journal of Chronic Diseases* 1978;31:425-32.

72. The CAST Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. New Engl J Med. 1989;321:406-12.

73. Wiklund I, Gorkin L, Pawitan Y, Schron E, Schoenberger J, Jared LL, Shumaker S. Methods for assessing quality of life in the Cardiac Arrhythmia Suppression Trial (CAST). *Qual Life Res*. 1992;1:187-201.

74. Brooks M M, Gorkin L, Schron EB, Wiklund I, Campion J, Ledingham MS. Moricizine and quality of life in the cardiac arrhythmia suppression trial II. *Control Clin Trials*. 1994;15:437-49.

75. Fallowfield L. Quality of life assessment in patients with heart disease. *Br Heart J*. 1993;69:371-2.

76. Van Domburg RT, Meeter K, van Berkel DFM, Veldkamp RF, van Herwerden LA, Bogers JJC. Smoking cessation reduces mortality after coronary artery bypass surgery: A 20-year follow-up study. *J Am Coll Cardiol* 2000; 36:878-883.

77. Strandberg TE, Pyorala K, Cook TJ, Wilhelmsen L, Faegeman O, Thorgeirsson G, *et al.* Mortality and incidence of cancer during 10-year follow-up of the Scandinavian Simvastatin Survival Study (4S). *Lancet* 2004; 364:771-777.

78. Lawless JF. Statistical Models and Methods for Lifetime Data. 2nd edition. New Jersey (USA): Wiley, Hoboken, 2003.

79. Baigent C, Collins R, Appleby P. ISIS-2: 10 year survival among patients with suspected acute myocardial infarction in randomised comparison of intravenous streptokinase, oral aspirin, both or neither. *BMJ* 1998; 316:1337-1343.

80. Beck JR, Kassirer JP, Pauker SG. A convenient approximation of life expectancy (the "DEALE"). I. Validation of the method. *Am J Med*. 1982; 73:883-897.

81. Altman DG. Confidence intervals for the number needed to treat. *BMJ* 1998;317:1309-12.

82. Devereaux PJ, Yusuf S. The evolution of the randomized controlled trial and its role in evidence-based decision making. *Journal of internal medicine* 2003;254:105-13

83. Pocock SJ. *Clinical trial: A practical approach*. Suffolk: Wiley; 1983.

84. Gilbert DD. Chemical analyses as validators in smoking cessation programs. *Journal of Behavioral Medicine* 1993;16:295-308.

85. Idle JR. Titrating exposure to tobacco smoke using cotinine--a minefield of misunderstandings. *Journal of Clinical Epidemiology* 1990;43:313-7.

86. Lando HA, McGovern PG, Kelder SH, Jeffery RW, Forster JL. Use of carbon monoxide breath validation in assessing exposure to cigarette smoke in a worksite population. *Health Psychology* 1991;10:296-301

87. Foss OP, Lund-Larsen PG. Serum thiocyanate and smoking: interpretation of serum thiocyanate levels observed in a large health study. Scand J Clin Lab Invest 1986;46:245-51.

88. Støa KF. Studies on thiocyanate in serum. Doctoral thesis. University of Bergen, 1957.

89. Brotman DJ, Walker E, Lauer MS, O'Brien RG. In search of fewer independent risk factors. *Arch intern med*. 2005;165:138-45

90. Rothwell PM. Subgroup analysis in randomised controlled trials: importance, indications, and interpretation. *Lancet* 2005;365:176-86.

91. Altman DG, Schultz KF, Moher D, *et. al,* for the CONSORT Group. The revised CONSORT statement for reporting randomised trials: explanation and elaboration. *Ann Intern Med* 2001;134:663-94.

92. Schulz KF, Grimes DA. Multiplicity in randomised trials I: endpoints and treatments. *Lancet* 2005;365:1591-95.

93. Hulley S, Grady D, Bush T, Furberg C, Herrington D, Riggs B, Vittinghoff E. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) Research Group. .*JAMA* 1998;280:605-13.

94. Bønaa K. H., Njølstad I., Ueland P. M., Schirmer H., Tverdal A., Steigen T., Wang H., Nordrehaug J. E., Arnesen E., Rasmussen K., the NORVIT Trial Investigators. Homocysteine Lowering and Cardiovascular Events after Acute Myocardial Infarction. *N Engl J Med*; published at www.nejm.org on Mar 12, 2006.

95. Witte K, Allen M. A Meta-analysis of fear appeals: Implications for effective public health campaigns. *Health Educ Behavior* 2000; 25: 591-615.

96. Fonteyn M E. A nurse led smoking cessation intervention increased cessation rates after hospital admission for coronary heart disease. *Evidence-Based Nursing* 2004; 7:46.

97. Bundy C. Fear is arrhythmogenic. *bmj.com*, 3 Dec 2003.

98. Johannesson M, Jønsson B, Kjekshus J, Olsson A, Pedersen TR, Wedel H. For the Scandinavian Simvastatin Survival Study Group. Cost effectiveness of simvastatin treatment to lower cholesterol levels in patients with coronary heart disease. *N Engl J Med* 1997; 336:332-336.

99. Gaspoz JM, Coxson PG, Goldman PA, Williams LW, Kuntz KM, Hunink MGM, *et al*. Cost effectiveness of aspirin, clopidogrel, or both for secondary prevention of coronary heart disease. *N Engl J Med* 2002; 346:1800-1806.

100. Malik, IS, Bhatia VK, Kooner JS. Cost effectiveness of ramipril treatment for cardiovascular risk reduction. *Heart* 2001; 85:539-543.

101. Probstfield J L. How cost-effective are new preventive strategies for cardiovascular disease? *Am J Cardiol* 2003; 91(suppl):22G-27G.

Erratum

Appendix

	Norwegian title	English title
Appendix I	Registreringsskjema	Registration form
Appendix II	Informert samtykke	Informed consent
Appendix III	Inklusjonsskjema	Inclusion form
Appendix IV	Personopplysninger	Personal information
Appendix V	Røykestatus	Smoking habits
Appendix VI	Fagerstrøm	The Fagerstrom score
Appendix VII	Motivasjon og Tilbakefall	Motivation and Relapse
Appendix VIII	Livskvalitet	The Quality of Life
Appendix IX	Sluttskjema	End of study
Appendix X	Evalueringsskjema	Evaluation form
Appendix XI	En hjelp til røykeslutt -for deg som virkelig trenger det	A help to smoking cessation - for you who really needs it
Appendix XII	Paper I-V	Paper I-V

Appendix I-X

Pasient nr: Skjema nr. 2

Registreringsskjema. TOBAMI

NAVN, PERSONNUMMER OG ADRESSE :

<Pas.etikett klistres her>

Fylles ut av visittgående lege :

Aktuell diagnose (en av fem) : hvert

	Ja	Nei
1. Hjerteinfarkt ?		
2. Ustabil angina pectoris:		
1. Nyoppstått angina ?		
2. Betydelig forverring av kronisk angina ?		
3. Hvileangina ?		
3. ACB-opr. ? (Må ha kommet direkte fra operasjonsstedet.)		

Andre opplysninger (sett kryss for spørsmål) :

	Ja	Nei
4. Tidl. kjent coronarsykdom ?		
5. Tidl. infarkt ?		
6. Daglig røyker fram til aktuelle sykdom ?		

Dato:_____

Underskrift:_____

Skjemaet skal ligge mellom kurve- og medikament-arket i alle pasientpermer på 2C og MINT.
Det fylles ut på alle pasienter med diagnosene:

-akutt hjerteinfarkt
-ustabil angina pectoris
-ACB-operasjon.

Visittgående lege fyller ut skjemaet på **første ordinære visitt etter innkomst** (ikke ettermiddagsvisitt eller helgevisitt), og markerer dette med et kryss på kurvearket. (**TOBAMI.....X)**

Etter at skjemaet er fylt ut legges det i posthyllen til TOBAMI på avdelingen.

Participant number:
Registration form. TOBAMI

< Participant label >

The physicians going rounds are to fill in the following:

The present diagnosis (one of five):

	Yes	No
1. Heart attack?		
2. Unstable angina:		
1. Recent development of angina?		
2. Exacerbation of chronic angina?		
3. Angina at rest?		
3. Coronary bypass surgery?		

Some other information (one mark per question):

	Yes	No
4. Previous coronary heart disease?		
5. Previous heart attack?		
6. Daily smoker until the present diagnosis		

Date:_____

Signature_____

Vest-Agder Sentralsykehus
Medisinsk avdeling.

VEST-AGDER FYLKESKOMMUNE

Undersøkelse om røykevaner hos pasienter med hjertesykdom.

Til deg med hjertesykdom.

Vi gjør for tiden en undersøkelse om hjertepasienters røykevaner som vi håper du er villig til å være med på.

Alle som er med i undersøkelsen må svare på noen enkle spørsmål, og komme til kontroll om ett år.
Halvparten av dem som er med i undersøkelsen får spesiell hjelp til å slutte å røyke i form av samtaler med sykepleier.
All informasjon vil bli behandlet konfidensielt og kun brukt til forskning.
Hvis du ombestemmer deg, og ikke ønsker å være med, kan du når som helst trekke deg uten å måtte oppgi noen grunn.

Det er fint hvis du vil skrive under på at du er villig til å delta i undersøkelsen.

På forhånd takk.

_____ _____

Lege. Sykepleier.

Navn :
Adresse :
Postnr. :
Poststed :
Tlf.privat :
Tlf. mobil :
Tlf. arbeid :
Vil være med på undersøkelsen: Ja ☐ Nei ☐ Dato : _____ Signatur : _____

Vest-Agder Central Hospital
Medical Department.

VEST-AGDER FYLKESKOMMUNE

Investigation about smoking habits in patients with coronary heart disease

For you with coronary heart disease.

We hope you are willing to participate in this investigation about smoking habits in patients with heart diseases.

You have to answer some questions, and attend at the 12 months follow up appointment.
Half of the participants in this investigation are going to receive special help in quitting smoking from a study nurse.
All the information in this study will be treated strictly confidentially.
At any time you may withdraw from the trial without further notice.

We hope you are able to join the study and need your written consent.

With regards,

_____ _____

Doctor. Nurse.

Name:
Address:
Telephone number:
I participate in the investigation: Yes ☐ No ☐ Date : _____ Signature : _____

Pas.nr.: «Pasnr» Skjema nr. 4

<Pas.etikett>

Inklusjonsskjema. TOBAMI

Fylles ut av prosjektsykepleier på alle pasienter som røyker og er ≤75 år med AMI, UAP eller ACB :
Inklusjonskriteriene (se baksiden for detaljert beskrivelse) :

	Ja	Nei
1. Daglig røyker fram til aktuelle coronare hendelse ? (Hvis nei skal resten ikke fylles ut)		
2. ≤75 år ?		
3. Adresse Vest- Agder eller Aust-Agder ?		
4. Snakker norsk ?		
5. Orientert for tid, sted og situasjon ?		
6. Har ikke alvorlig psykiatrisk sykdom eller alvorlig alkoholisme ?		
7. Har ikke uhelbredelig kreft eller annen alvorlig organsykdom ?		
8. Har AMI, UAP eller ACB ?		
Sett kryss i kun en «Ja»-rubrikk :		
8.1. Akutt hjerteinfarkt ? Ja ☐ Nei ☐		
8.2. Ustabil angina pectoris :		
1 Nyoppstått ? Ja ☐ Nei ☐		
2. Kronisk med forverring ? Ja ☐ Nei ☐		
3. Hvileangina ? Ja ☐ Nei ☐		
8.3. ACB-opr ? Ja ☐ Nei ☐		
9. Ønsker å slutte å røyke ?.		
10. Har erklært skriftlig at de ønsker å være med i studien ?.		

	Ja	Nei
11. Inkludert i TOBAMI ? (Må ha svart «Ja» på alle spørsmål fra 1-10)		
12. Inklusjonsdato :		
13. Hvis nei : Spesifiser årsak :		

Fylles ut på alle inkluderte pasienter :

14. Randomisert til: (Sett ett kryss i ruten som passer)	Intervensjon	Kontroll

Participant number:

< Participant label>

The inclusion form. TOBAMI

This form is to be filled in by the study nurse on all patients who smoke and are≤75 years of age, and are suffering a heart attack, unstable angina or coronary bypass surgery:

	Yes	No
9. Daily smoker until the start of the present coronary symptoms		
10.≤75 years of age?		
11.Living in Vest- Agder or Aust-Agder?		
12.Are speaking Norwegian?		
13.Oriented regarding the time, the place, and the situation?		
14.Does not have serious psychiatric disease, or alcoholism?		
15.Does not have incurable cancer or serious organ damages?		
16.Does suffer from heart attack, unstable angina or coronary bypass surgery?		
Only mark one question:		
8.1. Acute heart attack? Yes ☐ No ☐		
8.2. Unstable angina:		
1 Recent development? Yes ☐ No ☐		
2. Chronic with exacerbation? Yes ☐ No ☐		
3. Angina at rest? Yes ☐ No ☐		
8.3. Coronary bypass surgery? Yes ☐ No ☐		
9. Does have motivation to quit smoking?		
10. Have signed the written informed consent?		

	Yes	No
11. Included in TOBAMI ? (Yes on all questions 1-10)		
12. Inclusion date:		
13. If not included, specify why:		

14. Allocation:	Intervention	Control

Pas. nr : «Pasnr» Skjema nr. 5

<Pas.etikett>

Personopplysninger. TOBAMI.

Fylles ut på alle som er inkludert i TOBAMI.

1.

Kvinne ☐	
Mann ☐	

	Ja	Nei
2. Gift eller samboer ?		
3. Hvis ja : Røyker livsledsageren		
4. Skilt eller separert ?		
5. Enke (mann) ?		
6. Barn ?		

7. Utdannelse (sett ett kryss på det som passer):

1. Grunnskole	☐
2. Videregående	☐
3. Yrkesskole/fagbrev/ fagutdanning/handelsskole	☐
4. Høyskole	☐
5. Universitet	☐

8. Arbeidssituasjon (sett ett kryss på det som passer):

1. Uføretrygdet	☐
2. Arbeidsledig	☐
3. Pensjonert	☐
4. Hjemmeværende	☐
5. I arbeid	☐

9. Tidligere sykdommer :

Antall år med coronarsykdom (sett et kryss i ruten som passer) :

1. Nyoppstått.	☐
2. <1 år.	☐
3. 1-5 år.	☐
4. 6-10 år.	☐
5. >10 år.	☐

10. Alkoholforbruk (sett et kryss i ruten som passer) :

1. Aldri	☐
2. < 1 drink/pils/glass vin pr dag.	☐
3. 1-3 d/p/v pr. dag.	☐
4. >3 d/p/v pr. dag.	☐

	Ja	Nei
11. Røykfritt hjem ? (Må ut for å røyke.)		
12. Røykfri arbeidsplass ? (Må ut for å røyke.)		

13. Hvor mange prosent er røykere av de pas. bor sammen med? (Sett ett kryss i ruten som passer) :

0-20	20-40	40-60	60-80	>80

14. Hvor mange prosent av arbeidskollegaene røyker ?

0-20	20-40	40-60	60-80	>80

15. Antall tidl. røykestopp-forsøk	

Aktuelle innleggelse:

	Ja	Nei
16. Røykt under innleggelsen ?		

17. Nikotin-abstinens under innleggelsen ? (Sett ett kyss. Se baksiden for forklaring):

Ingen	Lite	Middels	Mye	Svært mye

18. Hvis infarkt : Max. Troponin I	
19. Antall døgn innlagt	
20. Antall døgn på MINT	

	Ja	Nei
21. Fått antirøykebudskap av lege under innleggelsen ?		
22. Hvis ja: Hvor mange ganger ?		
23. Sett hjerte-filmen ?		
24. Fått brosjyre om AMI/UAP/ACB ?		

Participant number:

< Participant label>

The personal information form. TOBAMI.

This form is to be filled in on all patients included in TOBAMI.

1.

Woman	☐
Mann	☐

	Yes	No
2. Married or living with a partner ?		
3. If yes: Does the spouse smoke?		
4. Divorced or separated?		
5. Widow(er)?		
6. Children?		

7. Education (only one mark):

1. Primary school	☐
2. High school	☐
3. Vocational school	☐
4. Higher education or university	☐

8. Working situation (only one mark):

1. Infirmity	☐
2. Unemployed	☐
3. Retired	☐
4. Working at home	☐
5. Employed	☐

9. Previous diseases:

The number of years with coronary heart disease (only one mark):

1. Recently discovered	☐
2. <1 year	☐
3. 1-5 years	☐
4. 6-10 years	☐
5. >10 years	☐

10. Alcohol consumption (only one mark):

1. Never	☐
2. < 1 beverage per day	☐
3. 1-3 beverage per day	☐
4. >3 beverage per day	☐

	Yes	No
11. Smoking not allowed at home?		
12. Smoking not allowed at work?		

13. The percentage of smokers at home?				
0-20	20-40	40-60	60-80	>80

14. The percentage of smokers at work?				
0-20	20-40	40-60	60-80	>80

15. Number of previous quit-attempts?	

The present hospitalisation:

	Yes	No
16. Smoked during the hospitalisation?		

17. Nicotine-abstinence during the hospitalisation:

None	Little	Medium	Much	Very much

18. If AMI: Max. Troponin I	
19. The number of days in hospital	
20. The number of days in the intensive care unit	

	Yes	No
21. Received advise not to smoke from a doctor?		
22. If yes: How many times?		
23. Seen the heart movie?		
24. Received booklet on AMI/UAP/CABG?		

Pas. nr.: «Pasnr»	Skjema nr. 6
<Pas.etikett>	

Røykestatus. TOBAMI.

Fylles ut på alle som er inkludert i TOBAMI.

1. Type tobakk (sett kryss på det som passer):

1. Ferdig sigaretter.	
2. Rullings	
3. Pipe.	
4. Sigarer/sigarillos.	

2.

1. Antall sig./sigarer dgl. i gj.snitt i den tiden pas. har røykt.	
2. Hvis pipe: Ant. pakker tobakk pr. uke	

3.

Antall år pas. har røykt.	

4. Hvis ferdigsigaretter/rullings:

Med filter ☐ Uten filter ☐

5.	Ja	Nei
Har pasienten røykt siste 24 t. før innleggelsen ?		

6.

Antall sig./piper/sigarer dgl i gj.snitt siste uka.	

Participant number: < Participant label>

Smoking habits. TOBAMI.

1. Type of tobacco:

1. Pre-made cigarettes	
2. Self-rolling cigarettes	
3. Pipe	
4. Cigar	

2.

1. Mean number of cigarettes/cigars per day since started smoking	
2. If pipe: The number of packs of tobacco per week	

3.

The number of years of smoking	

4. If cigarettes:

With filter ☐ Without filter ☐

5.	Yes	No
Smoked the last 24 hours before admission?		

6.

Mean number of cigarettes/cigars/pipes per day the last week before admission?	

Pas. nr.: «Pasnr» Skjema nr. 7

<Pas.etikett>

Fagerstrøm

Sett ring rundt det som passer:

1. Hvor mange sigaretter røyker du pr. dag ?	0-15 16-24 25 eller mer	0 1 2
2. Hvor lang tid etter at du våkner tenner du din første sigarett ?	Innen 30 min Etter 30min	1 0
3. Røyker du mer om morgenen enn senere på dagen ?	Ja Nei	1 0
4. 1. Hvis du røyker ferdigsigaretter : Hvilket nikotininnhold har sigarettmerket du røyker ? 2. Hvis du ruller egne sigaretter eller røyker pipe: Hvilken type tobakk bruker du ? (Alle typer hylser med filter reknes også som ekstra mild)	≤ 0,8 mg 0,9-1,2 mg > 1,2 mg Ekstra mild (<2 mg). Mild (2-2,5 mg). Sterkere type (>2,5 mg).	0 1 2 0 1 2
5. Har du problemer med å la være å røyke i situasjoner der røyking er forbudt (kino, teater, mm.) ?	Ja Nei	1 0
6. Røyker du selv om du er så syk at du nå og da er sengeliggende ?	Ja Nei	1 0
7. Hvilken sigarett er det vanskeligst å unnvære ?	Morgenens første ? En senere på dagen ?	1 0
8. Inhalerer du røyken ?	Alltid Iblant Aldri	2 1 0

Participant number: < Participant label> **The Fagerstrom score**

1. Number of cigarettes per day ?	0-15 16-24 25 or more	0 1 2
2. The time to the first cigarette in the morning?	Within 30 min After 30min	1 0
3. Do you smoke more in the morning than later in the day?	Yes No	1 0
4. 1. If you smoke pre-made cigarettes: What is the nicotine content per cigarette? 2. If you role your own cigarettes or smoke a pipe: What type of tobacco do you use?	≤ 0,8 mg 0,9-1,2 mg > 1,2 mg Extra mild (<2 mg). Mild (2-2,5 mg). Stronger type (>2,5 mg).	0 1 2 0 1 2
5. In places where smoking is prohibited, do you have problems in not to smoke?	Yes No	1 0
6. Do you smoke when you are confined to bed because of sickness?	Yes No	1 0
7. Which cigarette is hardest to avoid?	The first one in the morning? One later in the day?	1 0
8. Do you inhale the smoke?	Always Sometimes Never	2 1 0

Pas. nr.: «Pasnr» Skjema nr. 8

<Pas.etikett>

Motivasjon og Tilbakefall. TOBAMI.

Hvor sikker er du på at du vil unngå å røyke i følgende situasjoner ?
(Sett ett kryss for hvert spørsmål.)

1. Når jeg føler meg utålmodig ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

2. Når jeg venter på noe eller noen ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

3. Når jeg føler meg frustrert ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

4. Når jeg er bekymret ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

5. Når jeg er sulten eller har lyst på noe godt ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

6. Når jeg ønsker å bli i godt humør ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

7. Når jeg ønsker å få tiden til å gå ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

8. Når noen tilbyr meg en sigarett ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

9. Når jeg drikker alkohol ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

10. Når jeg føler meg utilpass ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

11. Når jeg er i en situasjon der jeg føler røyking er en del av mitt selvbilde ?

Usikker	Litt usikker	Hverken eller	Litt sikker	Sikker	Meget sikker	Svært sikker
☐	☐	☐	☐	☐	☐	☐

12. Hvor sikker er du på at du er røykfri om 1 år ?

(Sett et kryss i rute 1-10. 1=jeg er i tvil om jeg er røykfri. 10=helt sikker).

I tvil Helt sikker

1	2	3	4	5	6	7	8	9	10

13. Hvor sterkt ønsker du å slutte å røyke for alltid ?

(Sett et kryss i rute 1-10. 1=jeg er i tvil om jeg virkelig ønsker å slutte. 10=veldig sterkt.).

I tvil Veldig sterkt

1	2	3	4	5	6	7	8	9	10

Participant number:

<Participant label>

Motivation and Relapse. TOBAMI.

How confident are you in resisting smoking in the following situations?
(Only one mark per question)

1. When I feel impatient?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

2. When I am waiting for someone or something ?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

3. When I fell frustrated ?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

4. When I am worried ?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

5. When I am hungry or want something good to eat?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

6. When I want to cheer up?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

7. When I am trying to pass time?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

8. When someone offers me a cigarette ?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

9. When I am drinking an alcoholic beverage ?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

10. When I feel uncomfortable

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

11. When I am in a situation in which I feel smoking is a part of my self-image?

Completely unsure	A bit unsure	Neither sure nor unsure	A bit sure	Sure	Very sure	Absolutely sure
☐	☐	☐	☐	☐	☐	☐

12. How confident are you in being free of smoking one year ahead ?

Not confident — Absolutely confident

1	2	3	4	5	6	7	8	9	10

13. How much do you want to stop smoking forever?

In doubt — Very much want to

1	2	3	4	5	6	7	8	9	10

Pas. nr.: «Pasnr»

Dato:

<Pas.etikett>

Livskvalitet

1. Hvor ofte i løpet av den <u>siste måneden</u> har din helse begrenset din <u>sosiale aktivitet ?</u>
(Som f.eks. besøk hos venner eller nære slektninger.)

Ikke i det hele tatt	Noen få ganger	Av og til	Ganske ofte	For det meste	Hele tiden
☐	☐	☐	☐	☐	☐

2. Alt i alt, hvordan vil du bedømme ditt sosiale liv <u>den siste måneden</u> ?
Jeg har vært :

Svært fornøyd	Litt fornøyd	Verken fornøyd eller misfornøyd	Litt misfornøyd	Svært misfornøyd
☐	☐	☐	☐	☐

3. Har du følt deg frisk nok til å gjøre det du ønsket å gjøre <u>den siste måneden</u> ?

Ja, absolutt	For det meste	Problemer med helsen har begrenset meg på noen viktige områder	Jeg har bare vært frisk nok til å ta vare på meg selv	Jeg har trengt hjelp til det meste av det jeg ønsket å gjøre
☐	☐	☐	☐	☐

4. Hvor lenge (hvis i det hele tatt) har <u>helsen begrenset deg</u> i hver av følgende aktiviteter:

	Ikke begrenset meg i det hele tatt	Begrenset meg i mindre enn 3 mndr	Begrenset meg i mer enn 3 mndr
Kraftig fysisk aktivitet som tunge løft, løping eller fysisk krevende idrett.	☐ 1	☐ 2	☐ 3
Moderat fysisk aktivitet som å flytte et bord, bære handleposer, e.l.	☐ 1	☐ 2	☐ 3
Gå opp en bakke eller noen få trapper.	☐ 1	☐ 2	☐ 3
Gå 500 m på flat mark.	☐ 1	☐ 2	☐ 3
Spise, kle på seg eller gå på toalettet	☐ 1	☐ 2	☐ 3

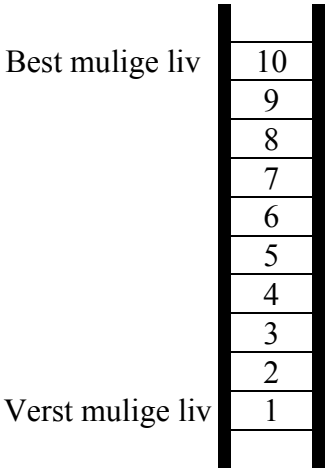
5. Personer med hjertesykdom får av og til noen av følgende symptomer. Sett et kryss i hver rad for å angi hvor ofte du har hatt disse symptomene <u>siste måned.</u>

SYMPTOMER	Hver dag	Flere dager i uka	Ca. en gang pr.uke	2-3 ggr. pr. måned.	Ca. en gang pr. måned	Ikke i det hele tatt.
Trøtthet	☐	☐	☐	☐	☐	☐
Svimmel	☐	☐	☐	☐	☐	☐
Glemsk	☐	☐	☐	☐	☐	☐
Hjertebank	☐	☐	☐	☐	☐	☐
Brystsmerter	☐	☐	☐	☐	☐	☐
Konsentrasjonsvanskeer	☐	☐	☐	☐	☐	☐
Tung pust	☐	☐	☐	☐	☐	☐
Synsforstyrrelser	☐	☐	☐	☐	☐	☐
Søvnvansker	☐	☐	☐	☐	☐	☐

6. For hvert av spørsmålene nedenfor ønsker vi at du markerer det svaret som <u>passer best</u> med hvordan du har følt deg <u>siste måned.</u>

SYMPTOMER	Hele tiden	For det meste	Ganske ofte	Av og til	En sjelden gang	Ikke i det hele tatt
Hvor ofte har du følt deg nervøs eller urolig den siste måneden ?	☐	☐	☐	☐	☐	☐
Hvor ofte har du følt deg rolig og avslappet den siste måneden ?	☐	☐	☐	☐	☐	☐
Hvor ofte har du vært lei deg eller mismodig den siste måneden ?	☐	☐	☐	☐	☐	☐
Hvor ofte har du følt deg lykkelig den siste måneden ?	☐	☐	☐	☐	☐	☐
Hvor ofte i løpet av den siste måneden har du følt deg så nedtrykt at ingenting kunne få deg i godt humør ?	☐	☐	☐	☐	☐	☐

7. Denne stigen symboliserer livets stige. Øverste trinn representerer det best mulige liv for deg. Nederste trinn representerer det verst mulige liv for deg.



1. På hvilket trinn føler du at du står for tiden ?

FOR TIDEN (1-10) ☐

2. På hvilket trinn sto du for ett år siden ?

FOR ETT ÅR SIDEN (1-10) ☐

3. Hvis du tenker framover, på hvilket trinn tror du at du står om fem år ?

OM FEM ÅR (1-10) ☐

Participant number:

Date:

< Participant label>

The Quality of Life

1. How much of the time during the past month has your health limited your social activities (like visiting with friends or close relatives)

None of the time	A little of the time	Some of the time	A good bit of the time	Most of the time	All of the time
☐	☐	☐	☐	☐	☐

2. All things considered, how satisfied have you been with your social life during the last month?

Very satisfied	Fairly satisfied	Neutral or mixed feelings	Somewhat satisfied	Very satisfied
☐	☐	☐	☐	☐

3. Did you feel healthy enough to do the things you wanted to do during the last month?

Yes-definitely so	For the most part	Health problems limited me in some important ways	I was only healthy enough to take care of myself	I needed someone to help me with most or all of the things I had to do
☐	☐	☐	☐	☐

4. For how long (if at all) has your health limited you in each of the following activities?

	Not limited me at all	Limited me for 3 months or less	Limited me for more than 3 months
Vigorous activities, like lifting heavy objects, running, or participating in strenuous sports	☐ 1	☐ 2	☐ 3
Moderate activities, like moving a table, carrying groceries or bowling	☐ 1	☐ 2	☐ 3
Walking uphill or climbing a few flights of stairs	☐ 1	☐ 2	☐ 3
Walking one block	☐ 1	☐ 2	☐ 3
Eating, dressing, bathing, or using the toilet	☐ 1	☐ 2	☐ 3

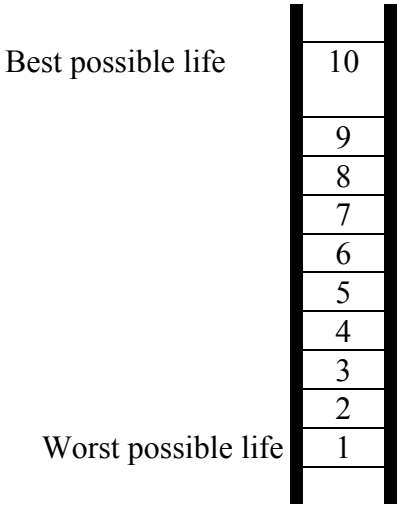
5. How often did you experience the following symptoms during the last month?

SYMPTOMS	Every day	Several days a week	About once a week	2-3 times a month	About once a month	Not at all
Tired	☐	☐	☐	☐	☐	☐
Dizzy	☐	☐	☐	☐	☐	☐
Forgetful	☐	☐	☐	☐	☐	☐
Irregular heart beats	☐	☐	☐	☐	☐	☐
Chest pain	☐	☐	☐	☐	☐	☐
Problems concentrating	☐	☐	☐	☐	☐	☐
Shortness of breath	☐	☐	☐	☐	☐	☐
Blurred vision	☐	☐	☐	☐	☐	☐
Trouble sleeping	☐	☐	☐	☐	☐	☐

6. For each of the following questions, please mark the answer that comes closest to the way you have been feeling during the past month

SYMPTOMS	All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
How much of the time have you been a nervous person?	☐	☐	☐	☐	☐	☐
How much of the time have you felt calm and peaceful?	☐	☐	☐	☐	☐	☐
How much of the time have you felt down-hearted and blue?	☐	☐	☐	☐	☐	☐
How much of the time have you been a happy person?	☐	☐	☐	☐	☐	☐
How often have you felt so down in the dumps that nothing could cheer you up?	☐	☐	☐	☐	☐	☐

7. Here is a ladder representing the ”ladder of life”.
The top of the ladder represents the best possible life for you, The bottom of the ladder represents the worst possible life for you.



1. On which step of the ladder do you feel you stand at present time? PRESENT TIME (1-10) ☐

2. On which step would you have stood five years ago? FIVE YEARS AGO (1-10) ☐

3. Thinking about your future, on which step do you think you will stand about five years from now? FIVE YEARS FROM NOW (1-10) ☐

Pas. nr.: «Pasnr»	Skjema nr 14
<Pas.etikett>	Dato:

Sluttskjema. TOBAMI

Fylles ut på alle inkluderte pasienter ved sluttkontrollen.

Hendelser siste året (Fra utskrivelsen til sluttkontrollen etter ca. et år) :

1.	Ja	Nei
1. Død ?		
2. Hvis død :		
Cardial årsak ?		

2. Drop out ?		
Hvis ja, spesifiser årsak :		
3. Antall sykehuskonsultasjoner.		
4. Antall telefonsamtaler.		
5. Antall polikliniske konsultasjoner.		
6. Antall AMI.		
7. Antall sykehusinnleggelser pga hjertesykdom.		
	Ja	Nei
8. ACB operert ?		
9. PTCA ?		
10. Fått diagnosen hjertesvikt ?		

11. Røykte pas. ved 6-ukers-ktr. ?		
12. Påstår pas. at han/hun har sluttet å røyke ved sluttktr. ?		

Hvis ja :

13. S-Cotinine-verdi :	
14. Når sluttet pas. å røyke ? (Sett ett kryss på det som passer):	
1. Under innleggelsen ved inklusjonen	☐
2. <1 mnd etter utskrivelsen	☐
3. 1-3 mndr etter utskrivelsen	☐
4. 3-6 mndr etter utskrivelsen	☐
5. 6-12 mndr etter utskrivelsen	☐

Hvis pas. fremdeles røyker :

15. Hvor mange sig./piper el. sigarer Røyker pas. daglig ved sluttkontrollen ?	

Til alle:

16. Antall tilbakefall ? (Def.: Ant. perioder med daglig røyking.)	

	Ja	Nei
17. Er hjemmet røykfritt ved sluttktr. ?		
18. Røyker livsledsager ved sluttktr. ?		

19. Ant. min. brukt på pas.konsult.:	
1. Under innleggelsen	
2. Etter utskrivelsen	
20. Nikotinsubstitusjon:	
1. Ant. uker med plaster	
2. Ant. uker med tyggegummi	
3. Ant. uker med annen type	

Participant number:	
<Participant label>	Date:

End of study. TOBAMI

Events from discharge until 12 months follow up:

1.	Yes	No
1. Dead?		
2. If yes:		
Cardiac cause?		

2. Drop out?		
If yes, the cause of withdrawal:		
3. Number of hospital admissions:		
4. Number of telephone calls from study nurse:		
5. Number of out-patient visits with study nurse:		
6. Number of AMI:		
7. Number of admissions due to heart disease:		
8. Bypass surgery?		
9. Percutaneous coronary intervention?		
10. Diagnosed heart failure?		

11. Smoked at 6 weeks?		
12. Stopped smoking at 12 months (end of study?		

If yes:

13. U-Cotinine:	
14. When did the participant stopped smoking?:	
1. During the initial hospitalisation	☐
2. <1 month after discharge	☐
3. 1-3 months after discharge	☐
4. 3-6 months after discharge	☐
5. 6-12 months after discharge	☐

If still smoking:

15. Number of cigarettes per day:	

16. Number of relapses with daily smoking?	

	Yes	No
17. Smoking forbidden at home?		
18. Does the spouse smoke at end of study?		

19. Number of minutes used in the intervention:	
1. During hospitalisation:	
2. After discharge:	
20. Nicotine replacements:	
1. Number of weeks with patches:	
2. Number of weeks with gums:	
3. Number of weeks with another kind:	

Pas. nr.: «Pasnr»

Dato:

<Pas.etikett>

Evalueringsskjema. TOBAMI.

Vi ønsker at du svarer så ærlig du kan på følgende spørsmål. (Sett ett kryss for hvert spørsmål.)

1. Hvordan synes du det siste året har vært i forhold til tidligere år når det gjelder din hjertesykdom ?

Svært mye verre	Ganske mye verre	Hverken bedre eller verre	Ganske mye bedre	Svært mye bedre
☐	☐	☐	☐	☐

2. I hvor stor grad har du fra sykehuset fått informasjon om røykens virkning på hjertet ?

Svært liten grad	Liten grad	Middels grad	Høy grad	Svært høy grad
☐	☐	☐	☐	☐

3. I hvilken grad har legen anbefalt deg å slutte å røyke ?

I svært liten grad	I liten grad	I middels grad	I høy grad	I svært høy grad
☐	☐	☐	☐	☐

4. I hvilken grad har annet helsepersonell anbefalt deg å slutte å røyke ?

I svært liten grad	I liten grad	I middels grad	I høy grad	I svært høy grad
☐	☐	☐	☐	☐

5. I hvilken grad har du blitt hjulpet av sykehuset til å slutte røyke ?

I svært liten grad	I liten grad	I middels grad	I høy grad	I svært høy grad
☐	☐	☐	☐	☐

6. Hvor fornøyd er du alt i alt med hjelpen du har fått fra sykehuset til å slutte å røyke ?

Svært lite fornøyd	Lite fornøyd	Middels fornøyd	Veldig fornøyd	Svært fornøyd
☐	☐	☐	☐	☐

7. I hvilken grad har du blitt hjulpet av familie og venner til å slutte å røyke ?

I svært liten grad	I liten grad	I middels grad	I høy grad	I svært høy grad
☐	☐	☐	☐	☐

Til deg som har sluttet å røyke:

8. Mener du at du hadde vært røykfri i dag hvis du ikke hadde fått hjelp fra sykehuset ?

Ja, helt sikkert	Ja, sannsynligvis	Vet ikke	Nei, sannsynligvis ikke	Nei, helt sikkert ikke
☐	☐	☐	☐	☐

Participant number:

Date:

<Participant label>

Evaluation form. TOBAMI.

Please be as honest as possible when answering the following questions (only one mark per question):

1. Compared to previously, how do you feel the last year has been regarding your heart disease?

Very much worse	Quite much worse	Neither worse nor better	Quite much better	Very much better
☐	☐	☐	☐	☐

2. Regarding the tobaccos effect on your heart, to what degree have you been informed about this from the hospital?

Very low degree	Low degree	Neither low nor high	High degree	Very high degree
☐	☐	☐	☐	☐

3. To what degree did the doctor recommend you to stop smoking?

Very low degree	Low degree	Neither low nor high	High degree	Very high degree
☐	☐	☐	☐	☐

4. To what degree have other health personnel recommended you to stop smoking?

Very low degree	Low degree	Neither low nor high	High degree	Very high degree
☐	☐	☐	☐	☐

5. To what degree do you feel you have been helped from the hospital in quitting smoking?

Very low degree	Low degree	Neither low nor high	High degree	Very high degree
☐	☐	☐	☐	☐

6. All in all, how satisfied are you with the help you have received from hospital in quitting smoking?

Very little satisfied	Little satisfied	Medium satisfied	Much satisfied	Very much satisfied
☐	☐	☐	☐	☐

7. To what degree do you feel you have been helped from family and friends in quitting smoking?

Very low degree	Low degree	Neither low nor high	High degree	Very high degree
☐	☐	☐	☐	☐

For you who have managed to stop smoking:

8. If you had not received any help from the hospital in smoking cessation, do you believe you had been free of smoking today?

Yes, absolutely	Yes, probably	Do not know	No, probably not	No, absolutely not
☐	☐	☐	☐	☐

Appendix XI

VEST-AGDER FYLKESKOMMUNE

En hjelp til røykeslutt

- for deg som virkelig trenger det

INNHOLDSFORTEGNELSE

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1. KJÆRE

Du har nettopp gjennomlevd et hjerteinfarkt, et kraftig anfall med hjertekrampe eller en hjerteoperasjon. Dette oppleves dramatisk av de fleste og mange tenker:

- Hva med framtidsutsikter?
- Hvordan blir livet etter dette?
- Hva kan jeg gjøre og hva må jeg unngå?
- Hvilken hverdag får jeg?

Få tilstander er så grundig utforsket av medisinsk ekspertise som hjertesykdom. Vi vet i dag svært mye om hvordan vi kan hjelpe deg. All utredning, medisinering og behandling har som hovedmål at du skal unngå å oppleve det samme igjen. Vi som jobber med dette merker at den medisinske utviklingen gir gode resultater. Behandlingen er betraktelig bedre i dag enn for bare få år siden!
Mange røykere opplever det samme som deg. Hjerteinfarkt er fremdeles en typisk røykesykdom.

Men nå til det positive: Du ønsker å slutte å røyke!
Din største motivasjon er sannsynligvis at du ikke ønsker å oppleve det samme igjen. **Røykeslutt er det viktigste du kan gjøre får å unngå nye hjerteanfall**. Ingen av de medisiner eller behandlinger vi rår over i dag gir bedre effekt enn røykestopp når det gjelder å forebygge det du nettopp har gjennomlevd. Så selv med dagens høyteknologiske medisin må vi innrømme at det du selv kan gjøre er viktigere enn det vi kan gjøre for deg. Det betyr ikke at det vi gjøre er uvirksomt. Nei, det er tvertimot meget effektivt. Det er bare det at røykestopp vanligvis er dobbelt så effektivt som medisinene når det gjelder å forebygge nye hjerteanfall.

Nå har vi aldri sagt at røykestopp er enkelt. Nettopp derfor engasjerer vi oss og vil hjelpe deg med den motivasjonen du

har. Vi vet litt om hva de som slutter å røyke sliter med. Kan vi hjelpe deg til å bli røykfri, har vi hjulpet deg på beste måte.

Røykestopp er ikke enkelt, men det er viktig

Vi håper denne brosjyren og den hjelp du ellers får ved Vest-Agder Sentralsykehus kan motivere til å leve videre uten tobakk.

Lykke til !

Hilsen

Tone Bæck, sykepleier.

Finn Tore Gjestvang, seksjonsoverlege, hjerteavd.

Eva Borøy, sykepleier.

Petter Quist Paulsen, ass.lege.

Dette heftet er utarbeidet av:

Tone Bæck
Frode Gallefoss
Finn Tore Gjestvang
Eva Borøy
Petter Quist Paulsen

2. LITT OM TOBAKK OG HELSE-SKADER

Vi vet i dag at halvparten av de som røyker dør for tidlig pga. røyking. Røyking kan føre til forskjellige kreftsykdommer, spesielt i lunger, munnhule, spiserør, magesekk, nyrer og urinblære. Men den fører først og fremst til karsykdommer som hjertekrampe, hjerteinfarkt og hjerneslag.

3. RØYKING OG HJERTESYKDOM

Det som kommer nå er hard kost for mange. Men som helsepersonell føler vi plikt til å legge fakta på bordet, og fortelle hvor skadelig røyken er for hjertet.
Mange holder seg friske i mange år selv om de røyker. Noen får aldri røyke-relatert sykdom.
Dessverre har du fått hjertesykdom. Det er et slags bevis på at hjertet ditt ikke tåler røyken.

Røyking fører til :

1. Tette blodårer til hjertemuskelen

Blodårene rundt hjertet forsyner hjertemuskelen med blod. Røyking fører til at veggen i disse blodårene blir tykkere, og at det dermed blir mindre plass til blodet. Dette er forandringer som kommer over flere år, og det kalles åreforkalkning. Når blodkarene går nesten tett får man hjertekrampe (angina pectoris) og når de går helt tett får man hjerteinfarkt.

Figur 1

Åreforkalkning
Nesten tett kar fører til hjertekrampe

2. Klebrige blodplater

Blodplatene sørger for at blodet levrer seg og at det dannes blodpropp. Straks etter man har tent en røyk påvirkes blodplatene i kroppen slik at de blir mere klebrige. De kan da feste seg til den fortykkede åreveggen i blodkarene, og tette igjen disse. Skjer det i blodårene rundt hjertet, får man et hjerteinfarkt.

Figur 2

Åreforkalkning med blodpropp
Helt tett kar fører til hjerteinfarkt

3. Hjerterytmeforstyrrelse

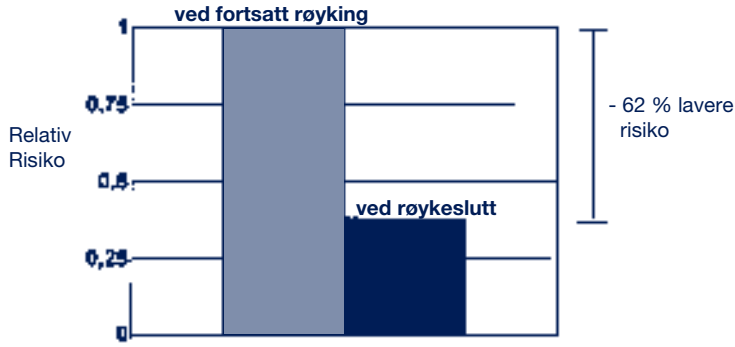
Nikotinen i tobakken påvirker hjertet slik at pulsen øker med 10-15 slag per minutt. Noen hjertemedisiner som gis etter hjerteinfarkt skal senke pulsen for at hjertet skal gå roligere. Virkningen av disse medisinene oppveies av fortsatt røyking. Man ser også av og til at røyking utløser hjerterytmeforstyrrelser. Disse kan være alvorlige.

4. LITT STATISTIKK TIL DIN FORDEL

Det er flere ting som kan bidra til den type hjertesykdom du nå opplever. Vi vet f.eks. at røyking, arvelige forhold, høyt kolesterol og høyt blodtrykk øker risikoen.
Det er selvfølgelig dumt hvis du har fått din hjertetilstand pga. røyking. Men når du nå først har fått det, er det viktig at du har en risikofaktor som kan fjernes. Hvis du tar bort din risikofaktor "røyking", så er dine leveutsikter dramatisk endret sammenliknet med hvis du fortsetter å røyke. En lang rekke både norske og internasjonale undersøkelser viser at effekten av røykestopp hos personer med hjertesykdom er meget god. Denne **effekten kommer med en gang** man slutter å røyke. Hvis du slutter å røyke har du allerede etter 1-2 år **40-60% mindre risiko for nytt hjerteinfarkt og død** enn om du fortsetter. Ingen av medisinene vi gir deg har tilnærmelsesvis så god effekt. De fleste medisiner vi gir i dag reduserer din risiko for nytt hjerteinfarkt med dødelig utgang de neste 5 årene med bare ca 30-40%

Figur 3

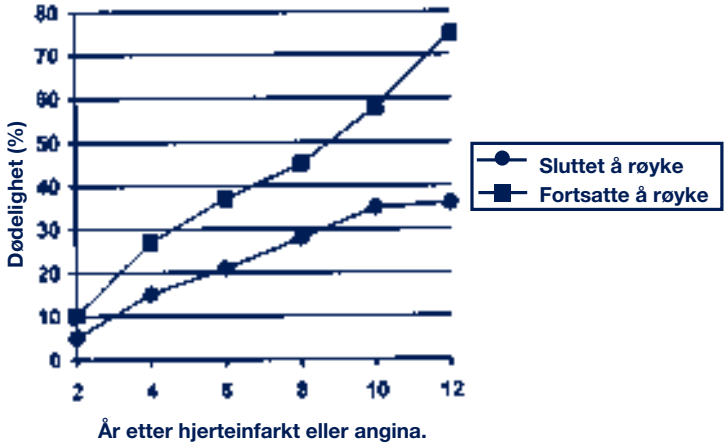
Risiko for død 5 år etter hjerteinfarkt



Jo lengre du holder deg røykfri, jo større blir effekten. Det viser denne figuren:

Figur 4

Forløpet etter hjerteinfarkt eller hardt angina-anfall.



Denne undersøkelsen viste at hele 63 % av dem som sluttet å røyke levde etter 13 år. Av dem som fortsatte å røyke var 18 % i live etter 13 år.

5. LITT OM NIKOTIN-AVHENGIGHET

Nikotin skaper avhengighet. Det kan være flere forhold som gjør det vanskelig å slutte å røyke. Det kan være at du blir rastløs og urolig når du kutter ut røyken. Kroppen blir vant til nikotinen og reagerer når nikotinnivået synker (fysisk abstinens). Det kan også være at du lengter etter «magadraget» fordi man setter pris på den umiddelbare effekten av nikotinen som treffer hjernen 7 sekunder etter at røyken er pustet inn (psykisk abstinens).

Det er altså to typer nikotinabstinens

1. Fysisk abstinens

Symptomer:
Hodepine, tretthet, skjelving, søvnproblemer, konsentrasjonsvansker, svettetokter, svimmelhet, irritasjon, angst, depresjon.

Disse symptomene er verst de første 2-3 dagene etter røykestopp. Deretter avtar de gradvis, og etter 2- 4 uker er den fysiske abstinensen over. Når du leser dette, har du gjennomlevd de mest plagsomme fysiske abstinensproblemer i forbindelse med din røykeslutt.
Du er altså allerede over den fasen som de fleste opplever som mest plagsom. Det du da må kjempe mot er psykiske abstinensplager.

2. Psykisk abstinens

Røykesug er et eksempel på psykisk abstinens. Hjernen din vil lenge huske hvor godt det var med en røyk. Du vil derfor ofte få lyst på røyk, selv lenge etter at den fysiske abstinensen er over. Noen drømmer om «magadraget» mange år etter at de har sluttet å røyke. Husk da at røykesuget ofte er veldig impulspreget («må ha en røyk akkurat nå») og situasjonsbetinget, og pleier å forsvinne etter bare 3-4 minutter.

6. HAR DU VÆRT RØYKFRI UNDER INNLEGGELSEN ?

I så fall er halve jobben gjort ! Den verste abstinensfasen er over. Hvis du ikke har vært plaget med fysiske abstinenssymptomer på sykehuset, får du det sannsynligvis heller ikke! (Du kan imidlertid fremdeles bli plaget med situasjonsbetinget røykesug.)
Du kan nå konsentrere deg om å unngå tilbakefall.

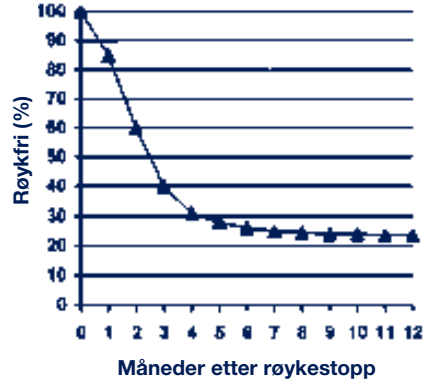
7. HVORDAN UNNGÅ TILBAKEFALL?

7.1 Det aller viktigste:

Prøv ALDRI «bare én sigarett».

Undersøkelser viser at hvis du bare tar en sigarett, er sjansen for tilbakefall stor selv etter mange år!
Det er altså mye som tyder på at en bør tenke: «En gang røyker, alltid røyker». En sigarett på julaften, nyttårsaften eller 17.mai er ofte første skritt på vei mot tilbakefall.

Denne figuren viser hvor mange som forblir røykfrie etter røykestopp:



Vi ser at etter 4 måneder flater kurven ut. Dvs. at greier du de fire første månedene, er sannsynligheten stor for at du vil mestre dette.

7.2 Andre viktige momenter

Det er gjort flere hundre internasjonale undersøkelser om røykestopp. Her følger en liste over det som har vist seg å være viktigst for å unngå tilbakefall. Ikke alt vil passe for deg, men kanskje finner du noen punkter som vil kunne hjelpe deg.

1. Hvis din livsledsager røyker er det vanskelig å lykkes. Prøv å overtale partneren til å slutte sammen med deg. Ta gjerne han/hun med på samtalene med sykepleier.

2. Lag røykeforbud i ditt eget hjem. Da blir det lettere både for deg og dine nærmeste å forbli røykfrie.

3. Noe av det første du bør gjøre når du nå har sluttet å røyke, er å bli klar over i hvilke situasjoner fristelsen er størst. I mange situasjoner vil du føle røykesug. Hvis du får tankene over på noe annet varer røykesuget bare i ca. 3-4 minutter. Gå igjennom denne listen og se hvilke situasjoner som passer best med dine røykevaner.

Jeg får ekstra lyst på røyk

når jeg begynner dagen.
etter at jeg har spist.
når jeg drikker kaffe.
når jeg drikker alkohol.
når jeg er sammen med andre på fest.
når jeg tar en pause.
når jeg er stresset eller har behov for å roe meg ned.
når jeg må vente på noe eller noen.
når jeg kjører bil.
når jeg snakker i telefonen.
når jeg kjeder meg.
når jeg synes jeg fortjener en belønning.
når jeg er stresset.
når jeg er engstelig.
når jeg er deprimert.
når jeg føler meg «likeglad».

Tenk så igjennom hva du skal gjøre når røykesuget kommer. Stikkordet er: **Få tankene over på noe annet.**

7.3 Noen andre tips:

- Flytt deg fysisk vekk fra den situasjonen du er i når røykesuget kommer.
  Lag f.eks. en liste over alle ting som må gjøres i huset, i hagen, med bilen eller med en hobby. Når røykesuget kommer tar du fram listen, og går i gang med oppgavene.
- Ikke bli sittende når du har spist. Bryt opp og gjør noe annet.
- Unngå stressituasjoner de første ukene.
- Unngå alkohol de første vanskelige ukene. Motstandskraften svekkes når du drikker, og de fleste risikerer å sprekke
- Drikk mindre kaffe. Forbrenningen av kaffe (i leveren) settes kraftig ned når du slutter å røyke. Du kan få symptomer på koffein-forgiftning hvis du ikke reduserer inntaket. Disse symptomene er ofte helt like nikotinabstinensen! Du bør redusere inntaket av kaffe/te med minst 50%.
- Fjern alle røykesaker og alt som kan minne om tobakk hjemme, i bilen og på jobben.
- Fortell familie, venner og kollegaer at du har sluttet. Da risikerer du ikke så lett å bli tilbudt en sigarett. Samtidig blir det vanskeligere å begynne igjen.
- Legg gjerne pengene til side som du før brukte på tobakk. Bestem deg for noe å bruke dem til den dagen du er ferdig med avvenningen (f.eks. etter 1 år). Det blir mye penger !
- Pass på vekten. De fleste legger på seg 2-3 kg i starten. Apetitten øker og forbrenningen nedsettes. Forsøk å spise sunt. Prøv å unngå småspising. Mosjon nedsetter røykesuget. Gå gjerne turer o.l. Det er fint for hjertet ditt uansett!

8. HAR DU RØYKT UNDER INNLEGGELSEN ?

- **Sett en sluttdato** sammen med sykepleier innen 3-4 uker etter utskrivelsen.
- **Trapp gradvis ned nikotinforbruket**. Det er stor forskjell på å stupe fra 10-meteren og fra bassengkanten. Du skal ned på bassengkanten før du jumper uti.
  Bytt til tobakk med mindre nikotininnhold. De første 4-5 dagene kan du ellers røyke som før. Antakeligvis røykte du noe mindre enn vanlig på sykehuset. Ikke røyk flere sigaretter enn da du var innlagt. Etter ca. en uke trapper du ned på antall sigaretter. Bestem deg på forhånd hvilke sigaretter du skal kutte ut. Det kan da være lurt å kutte ut «de beste» først. Det er de som er vanskeligst å være foruten, og det er lurt hvis de ikke står igjen til slutt.
- Samtidig kan du prøve å vente 15 minutter hver gang du har lyst på røyk.
- Gjør klar det antallet sigaretter du skal røyke pr. dag, og legg disse i en egen eske.
- Lag «rene» røykesituasjoner. Dvs. du skal ikke lese, se på TV, snakke i telefon, kjøre bil osv. når du røyker. Du skal heller ikke røyke sammen med andre. På denne måten vil du automatisk røyke mindre, og du gjør noe aktivt for å bryte din røykevane.

Når sluttdatoen er der, kan du følge anbefalingene som under punkt 7.

9 TILBAKEFALL

Ikke se for mørkt på dette. Mange har flere tilbakefall før de endelig blir røykfrie. Tilbakefall er en del av prosessen med å bli røykfri ! Prøv å ta lærdom av det, og tenk igjennom hva som gikk galt.

- Hvor skjedde det, -i hvilken situasjon ?
- Hva fikk deg til å begynne igjen?
- Hvem fikk deg til å begynne igjen ?
- Hva kan jeg gjøre annerledes neste gang?

Avtal så en ny sluttdato sammen med sykepleier. Trapp gradvis ned nikotinforbruket og forsøk på nytt.

De største seire blir sjeldent vunnet etter det første slaget.

Johan Falkberget.

10 NIKOTINPREPARATER

Nikotinpreparater avgir nikotin til blodet, og demper derved abstinensplager i forbindelse med røykeslutt. Preparatene får du kjøpt uten resept på apotek.

Mange klarer å slutte uten bruk av nikotinpreparater. Hvis du ikke har røykt under innleggelsen er du over den verste perioden med fysiske abstinenssymptomer, og vil bare ha nytte av nikotinpreparater hvis du får plager med situasjonsbetinget røykesug.

Du må ALDRI røyke samtidig som du bruker nikotinpreparater. Da kan du få for mye nikotin i blodet, og det kan være uheldig for hjertet.

Vi vil anbefale nikotinpreparater hvis du er mye plaget med røykesug eller har mye av de fysiske abstinenssymptomene.

10.1 For deg med situasjonsbetinget røykesug

Nikotintyggegummi anbefales

Forsøk først med laveste styrke (Nicorette 2 mg eller Nicotinell 2 mg). Ved manglende effekt kan du øke til høyeste styrke (Nicorette 4 mg eller Nicotinell 4 mg).
Ta en tyggegummi når du føler røykesug. Hvis du på forhånd vet at røykesuget vil komme, bør du ta den noen minutter før. Det tar 30 minutter før du får maksimalt nikotinnivå i blodet.

Slik skal de tygges

Tygg langsomt til smaken kjennes sterkt (10-15 ganger). La deretter tyggegummien hvile i munnen i ca. 1 minutt eller til smaken avtar. Tygg langsomt 10-15 ganger til, og la igjen tyggegummien hvile før du fortsetter på samme måte.
Svelg minst mulig under tyggingen (Nikotinen tas opp fra slimhinnen i munnen).
Etter ca. 30 minutter er alt nikotinen tygget ut av tyggegummien.

Bruk ikke mer enn 8-12 tyggegummier pr. dag. Ta bare en om gangen.

10.2 For deg med fysisk abstinens

Nikotinplaster anbefales.

Dette gir en jevn tilførsel av nikotin til kroppen.

- Hvis du har sluttet å røyke under innleggelsen eller røyker mindre enn 20 ferdigsigaretter daglig kan du begynne med middels styrke:

Nicorette 10 mg/16 t. eller Nicotinell 14mg/24 t.

Hvis du fremdeles har symptomer kan du øke til høyeste styrke (se nedenfor).

Etter 2-3 uker kan du skifte til svakeste styrke :

Nicorette 5 mg/16 t. eller Nicotinell 7mg/24 t.

Etter ytterligere 2-3 uker kan du slutte med plaster.

- Hvis du ikke har sluttet å røyke under innleggelsen og røyker mere enn 20 ferdigsigaretter daglig eller røyker rulletobakk :

Begynn med høyste styrke nikotinplaster:

Nicorette 15 mg/16 t. eller Nicotinell 21 mg/24 t.

Etter 2 uker skiftes til middels styrke. Etter ytterligere 2 uker skiftes til laveste styrke, som du kan bruke i 1-2 uker.

Nicorette plaster tas av hver kveld, og det settes på et nytt neste morgen. Nicotinell plaster skiftes hver morgen. Plasteret settes på hårfri, tørr og ren hud. Det presses i 10-15 sekunder mot huden. Stedet hvor plasteret settes varierer fra dag til dag for å unngå hudirritasjon.

10.3 Bivirkninger

Ved for mye nikotin: Blekhet, svetting, økt spyttdannelse, kvalme, oppkast, diaré, hodepine.
Ved plaster: Lokale hudreaksjoner.
Ved tyggegummi: Hikke (ved for rask tygging), irritasjon i munn og svelg og magesmerter. Tyggegummien kan feste seg til tannproteser og gjøre skade.

Det er viljen som det gjelder
Viljen frigjør eller feller
Henrik Ibsen

Utarbeidet ved Sørlandet Sykehus Kristiansand HF

R&M REKLAMEBYRÅ AS TRYKK: STAV JOHANSEN TRYKKERI

VEST-AGDER FYLKESKOMMUNE

A help to smoking cessation

- for you who really needs it

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1. Dear,_____

You have just suffered a heart attack, unstable angina or coronary bypass surgery. You may have found the disease dramatic, and you may think:

- What about the future?
- How will the rest of my life be?
- What can I do myself, and what do I have to avoid?
- What will my daily life be like after this?

Very much science has been performed regarding heart diseases, and we know a lot about how to help you. All kinds of investigation, medication and treatment for your condition aim at avoiding another heart attack . Treatment has improved greatly during the last years. Still, many smokers experience the same as you. Heart disease is still a typical smoking-related disease.

But now to the positive part: You wish to stop smoking. Your increased motivation has emerged probably because you do not want to experience this one more time. **Quitting smoking is the most important action to avoid another attack of heart disease.** None of the medications or treatment modalities we have today are as effective as smoking cessation in order to prevent the condition you now have survived. So, although today's high-tech medicine is of great importance, we have to admit that what you can do by yourself is more important than what we can do for you. This does not mean that our high-tech medicine is ineffective. On the contrary, it is very effective. However, quitting smoking is twice as effective in preventing another heart attack!

Many find it difficult to stop smoking. That is why we want to help you. We have some knowledge about the problems of smoking cessation, and if we can give you some advice to stay free of smoking, we know that we have helped you in the best way.

We hope this booklet, and the help you receive from Sørlandet Hospital, can motivate you to stay free of smoking.

Good luck!

Sincerely,

Tone Bæck, nurse.

Finn Tore Gjestvang, Chief consultant, Dep. of heart diseases.

Eva Borøy, nurse.

Petter Quist-Paulsen, physician.

This booklet was written by: Tone Bæck, Frode Gallefoss, Finn Tore Gjestvang, Eva Borøy, and Petter Quist-Paulsen.

2. About the health effects of tobacco

We know today that half of those who smoke dies many years ahead because of smoking. Smoking is the cause of many cancers, especially in the lung, oesophagus, stomach, kidney, and urinary bladder. But it also leads to blood vessel diseases like heart attack, angina, and stroke.

3. Smoking and heart disease

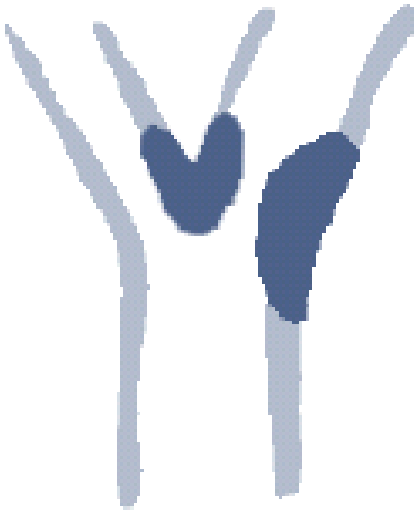
Some of you my find the following hard to accept. But as health care providers we feel it is our duty to give you all the facts about the health hazard of continued smoking. Many people stay healthy for years despite smoking and may never develop smoking-related disease. Unfortunately, you have now got heart disease. This may support the fact that your heart does not tolerate smoking.

Smoking leads to

Occluded vessels to the heart muscle

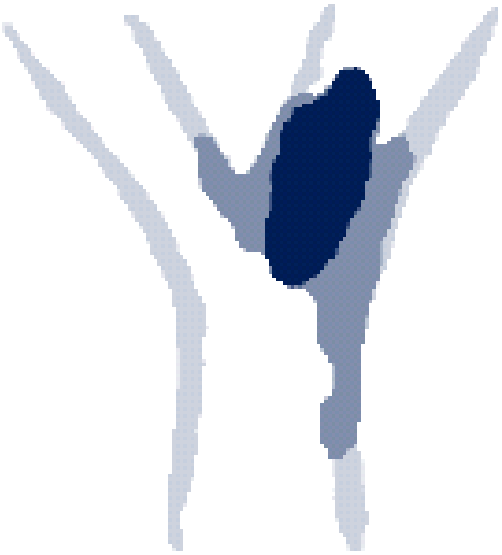
The vessels around the heart supplies blood to the heart muscle. Smoking increases the thickness of the vessel wall, and during several years of smoking the vessel becomes narrowed and regional blood flow decreases. This is called atherosclerosis. When the vessels narrows, angina subsequently develops, and when they occlude, a heart attack arises.

A narrowed blood vessel leads to angina.



Sticky platelets

The platelets accomplish blood coagulation, and the development of a blood clot. Immediately after smoking, the platelets become more sticky. They may then adhere to the diseased blood vessel wall and cause a blood clot. If this happens in the coronary arteries, a heart attack develops.



An occluded vessel leads to a heart attack

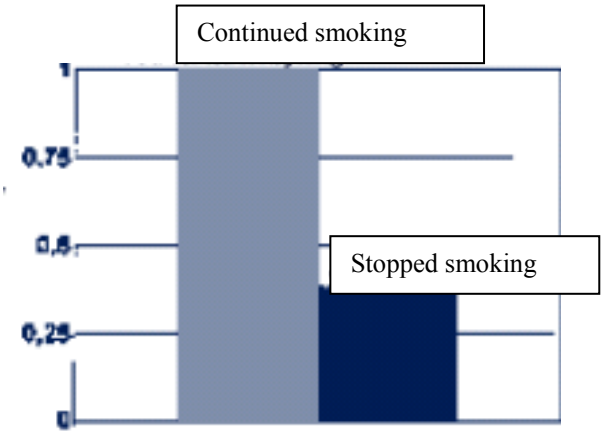
Heart rhythm disturbance

Nicotine in the tobacco increases the pulse with about 10-15 beats per minute. Some medications prescribed after a heart attack works by slowing down the pulse. Therefore, smoking may outweigh the effect of these medications. Further, the nicotine may also lead to heart rhythm disturbances, some of which may be dangerous.

4. Some statistics to your advantage

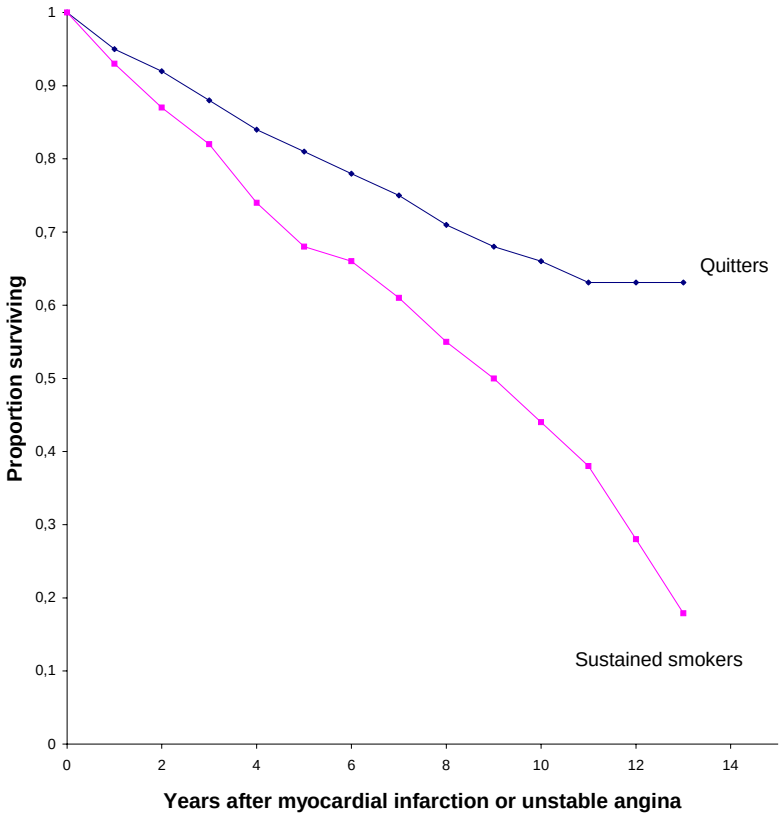
Many factors like high cholesterol level, high blood pressure, inheritance and smoking may contribute to the heart disease you now are suffering from. What's important about smoking as a cause of heart disease, is that it can be totally removed! If you manage to quit smoking, your life expectancies will increase dramatically compared to continued smoking. Many investigations have shown that smoking cessation is very effective in patients with heart disease, and that the effect is evident shortly after cessation. If you manage to stay free of smoking your chance of dying because of heart disease is reduced by approximately 50% after one or two years of abstinence. None of the pharmaceuticals you receive are comparable to this effect.

Risk of death 5 years after a heart attack



This figure shows a 60% risk reduction for death five years after a heart attack in patients who managed to stop smoking compared to patients who continued to smoke

The longer you manage to stay free of smoking, the larger is the effect on mortality, as shown by this figure:



This investigation found that 13 years after a heart attack or unstable angina 63% were still alive in the group that managed to quit smoking, compared to only 18% among those who continued smoking.

5. About nicotine dependence

Nicotine causes dependence, and that is why many find it difficult to quit smoking. You may feel irritable, restless, or miserable after cessation because your body has got used to the nicotine, and reacts with abstinence when nicotine concentration declines. This is related to physical dependence. You may also long for the pleasant feeling the nicotine creates when it reaches the brain some seconds after inhalation, and this is related to psychological dependence.

1. Physical abstinence

Symptoms:

Headache, tiredness, shivering, sleep disturbance, concentration impairment, dizziness, irritability, anxiety, depression.

These symptoms are most severe the first 2-3 days, and then gradually declines over the next 2-4 weeks. When you read this, you already have lived through the most troublesome physical abstinence. In other words, the most unpleasant period has already passed! What you now have to fight against is the psychical dependence.

2. Psychological dependence

For a long time, your brain will remember the pleasant feeling that may arise after inhalation. Therefore, you may often experience a desire for smoking in many situations, even long after the physical dependence has vanished. Some people struggle with this urge for several years after cessation. When this urge develops, you have to remember that it is only a momentary feeling, and that it will go away after a few minutes.

6. Have you stayed “smoke free” during hospitalisation?

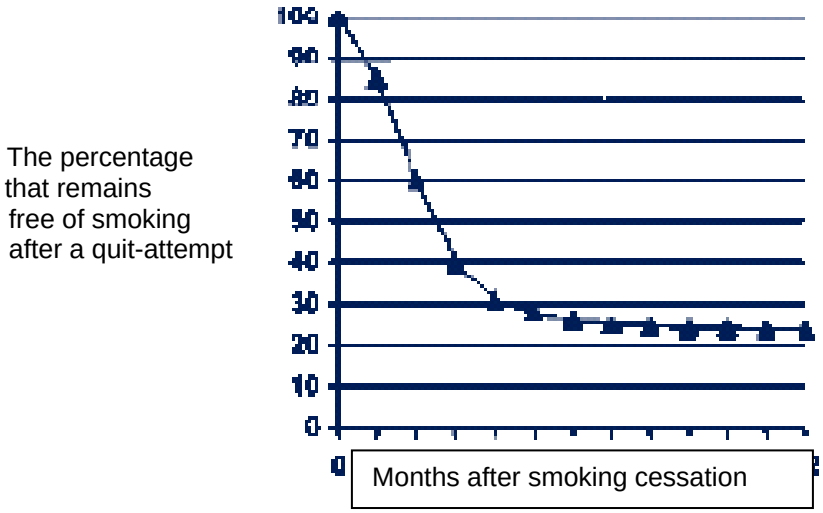
That is excellent, half of the job has already been done! Most of the abstinence has already passed. If you have not experienced physical abstinence during hospitalisation, you most probably never will suffer from this. Now, you can fully concentrate on relapse prevention.

7. How to prevent relapse?

The most important: NEVER try "just one cigarette".

Studies have shown that if you smoke just one cigarette, the chance of relapse is large even after several years of abstinence. You have to think: "One time smoker, always smoker." Only one cigarette at Christmas eve, New Years eve, or at your birthday may well start regular smoking again.

The following figure shows that relapse to smoking is rather infrequent after four months of abstinence:



If you manage to stay free of smoking the first 4-5 months, you probably will succeed!

Some other important elements

Several investigations on how to stop smoking have been performed. Maybe you will find some of the following elements helpful in staying free:

1. If your spouse is smoking, it is difficult to succeed. Try to convince he/her to quit along with you. Your spouse is welcome to join the consultations with the nurse who is going to help you to stay free.
2. Abandon smoking in your own house. Then it will be easier for both you and your family to stay free.
3. You might find it wise to think through the situations when your smoking urge is at its highest. If you manage to think of something else, or keep occupied with something else, the urge to smoking will disappear after 3-4 minutes. Others have found that resisting smoking may be difficult in the following situations. Maybe some of these situations also pose problems for you:

<u>It is difficult to resist the urge to smoke</u>

when I start the day.

when I drink coffee.

after eating.

when I talk in the phone.

when I drink an alcoholic beverage.

when I deserve an award.

when I am on a party.

when I am taking a break.

when I drive a car.

when I am bored.

when I feel anxiety.

when I am depressed.

when I don't care about nothing.

when I am stressed or want to calm down.

when I am waiting for someone or something.

What will you do when the urge arises in these situations? Most important is probably to move you mind and think of something else, and NEVER try "just one cigarette".

Some more advises

- Remove yourself physically away from the place where the urge develops. You may want to make a list of things you have to do in the house, in the garden, or with a hobby. When the urge arises, go ahead with the tasks!
- After eating a meal, go on and do something else in stead of stay seated.
- Avoid stressing situations the first couple of weeks.
- Avoid alcohol the first weeks.
- Drink less coffee. Nicotine increase the metabolism of coffee when smoking. Therefore, symptoms of coffein intoxication, which may be very similar to nicotine withdrawal symptoms, may develop if you do not reduce the amount of coffee!
- Remove all smoking-related advises in the house.
- Tell your family and friends that you have stopped smoking. Then, they probably will not offer you a cigarette, and it may be easier to stay free.
- You may want to put away the money you are saving by not smoking, and use them on something nice. It's a lot of money!
- Watch your weight. Many increases 2-3 kg in weight. Try nor to eat between meals. Exercise will help, and is good for your heart!

8. Have you smoked during the hospitalisation?

- Set a quit-date together with the nurse within 3-4 weeks after discharge.
- Gradually lower the number of cigarettes per day. You have nevertheless probably smoked a bit less than usual while hospitalised. Do not increase the amount to the previous level when you come home! After one week you should reduce the number of cigarettes per day. It is often wise to remove “the best ones” first (i.e. the cigarette after dinner). They are the most difficult to avoid, and should not be the last to go!
- Try to wait for 15 minutes every time you want a cigarette.
- Lay aside the cigarettes you are allowed to smoke in a day.
- Develop “pure smoking situations”. In other words, you shall only smoke when smoking, and not read, watch television, talk in the phone etc. at the same time.
- When you reach the quit-date, you are advised to follow the recommendations in chapter 7.

9. Relapse

Try not to be depressed about this. Most people that at last manage to quit smoking have relapsed several times. Relapsing is part of the process of quitting! Try to learn from it. What went wrong?

- Where did it happen?
- What got you started?
- Who got you started?
- What can I do better next time?

Arrange a new quit-date with the nurse, start gradually reduction of the number of cigarettes per day, and have try again!

10. Nicotine replacements

Nicotine replacements deliver nicotine to the blood, and thereby reduces the physical abstinence after smoking cessation. The products can be bought at pharmacies and in grocery stores without prescription from a doctor.

Many people manage to quit smoking without nicotine replacements. If you have stayed free of smoking while hospitalised, the period with a high level of physical abstinence has already passed, and you only need nicotine replacements when you experience situation-related urge.

You should not smoke while using the replacements because of the risk of high level of nicotine concentration in the blood, which could be dangerous to your heart.

We recommend the use of replacements if you either have problems with physical abstinence or with situation-related urge.

If you experience situation-related urge

Nicotine gum is recommended

First you can try the gums with the lowest nicotine content (Nicorette 2 mg or Nicotinell 2 mg). If this is ineffective, you may tray a gum containing 4 mg of nicotine. When the urge to smoke develop, you take a gum. If you in advance know that the urge will turn up, you should take a gum some minutes before the situation arises. It takes 30 minutes of chewing before the highest level of nicotine in the blood is reached.

How to chew the gums

Chew slowly until the taste becomes strong (10-15 times). Then put the gum aside in the mouth for approximately one minute, or until the taste disappear. Then repeat the process. The nicotine is released to the blood via the mucous membrane, and after about 30 minutes there

are no nicotine left in the gum. You should not use more than 8-12 gums per day, and only one at a time.

For you with physical abstinence

Nicotine patch is recommended

A steady supply of nicotine to the blood is delivered by the nicotine patch.

1. If you have stopped smoking while hospitalised, or if you are smoking less than 20 cigarettes per day, you can

- Start with a patch with medium concentration: Nicorette 10 mg/16 h or Nicotinell 14 mg/24 h.
- If you still have nicotine withdrawal symptoms, you can change to the patch with the highest concentration (see below).
- After 2-3 weeks, change to the lowest concentration: Nicorette 5 mg/16 h or Nicotinell 7 mg/24 h.
- After another 2-3 weeks you can stop with the patches.

2. If you have smoked during hospitalisation, and are smoking more than 20 cigarettes per day:

- Start with the patch with the highest concentration:
- Nicorette 15 mg/16 h or Nicotinell 21 mg/24 h.
- After 2 weeks change to the medium strength, and after another 2 weeks to the lowest strength which you can use for 1-2 weeks.

You have to take off the Nicorette patch every evening, and apply another one in the morning. The Nicotinell patch has to be exchanged every morning. The patches must be applied on dry and clean skin which is free of hair. You have to press it against the skin for 10-15 sec. To avoid skin-irritation you should vary the places for application.

Side effects

To much nicotine: Pallor, sweating, increased salivation, nausea, diarrhoea, headache.

Patches: Local skin irritation.

Gums: Hiccup, irritation in the mouth and throat, abdominal pain. The gum may stick to, and injure tooth-prosthesis.