ARRHYTHMIA, ELECTROCARDIOGRAPHIC SIGNS, AND PHYSICAL ACTIVITY IN RELATION TO CORONARY HEART RISK FACTORS AND DISEASE. THE TROMSØ STUDY.

by

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To my parents

Lea Juvonen Løchen and Arne Løchen

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This study was carried out at the Institute of Community Medicine and the Institute of Clinical Medicine, University of Tromsø, during the years 1986-94, while I was working as a research fellow and as a senior lecturer.

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Tromsø, July 20, 1994
Maja-Lisa Løchen

LIST OF PAPERS

This thesis is based on the following papers, which will be referred to by their Roman numerals:

I Løchen M-L. The Tromsø study: associations between self-reported arrhythmia, psychological conditions, and lifestyle. Scand J Prim Health Care 1991;9:265-70.

II Løchen M-L, Snaprud T, Zhang W, Rasmussen K. Arrhythmias in subjects with and without a history of palpitations: the Tromsø study. Eur Heart J 1994;15:345-9.

III Løchen M-L, Rasmussen K, Macfarlane PW, Arnesen E. The predictive value of single lead electrocardiogram for myocardial infarction in young and middle-aged men. The Tromsø study. Submitted.

IV Løchen M-L. The Tromsø study: the prevalence of exercise-induced silent myocardial ischaemia and relation to risk factors for coronary heart disease in an apparently healthy population. Eur Heart J 1992;13:728-31.

V Løchen M-L, Rasmussen K. The Tromsø study: physical fitness, self reported physical activity, and their relationship to other coronary risk factors. J Epidemiol Comm Health 1992;26:103-7.

1. AIMS OF THE THESIS

What is the importance of a trivial everyday experience or behaviour such as throbbing of the heart or physical inactivity, and of trivial diagnostic signs such as electrocardiography (ECG) with regard to the epidemiology and prevention of coronary heart disease (CHD)? How should these aspects of life-style and disease be interpreted by the individual, by the physician and by the epidemiologist?

The epidemiological survey in Tromsø 1986-87, with subsequent substudies, provided an opportunity to throw more light on some of these questions. The specific questions asked were:

1. What are the associations between palpitations, coronary risk factors, psychological conditions, lifestyle and arrhythmia?
2. What are the associations between electrocardiographic variables and subsequent myocardial infarction in men?
3. What are the associations between exercise-induced silent myocardial ischaemia and coronary risk factors?
4. What are the associations between physical activity, physical fitness and other coronary risk factors?

2. MATERIALS AND METHODS

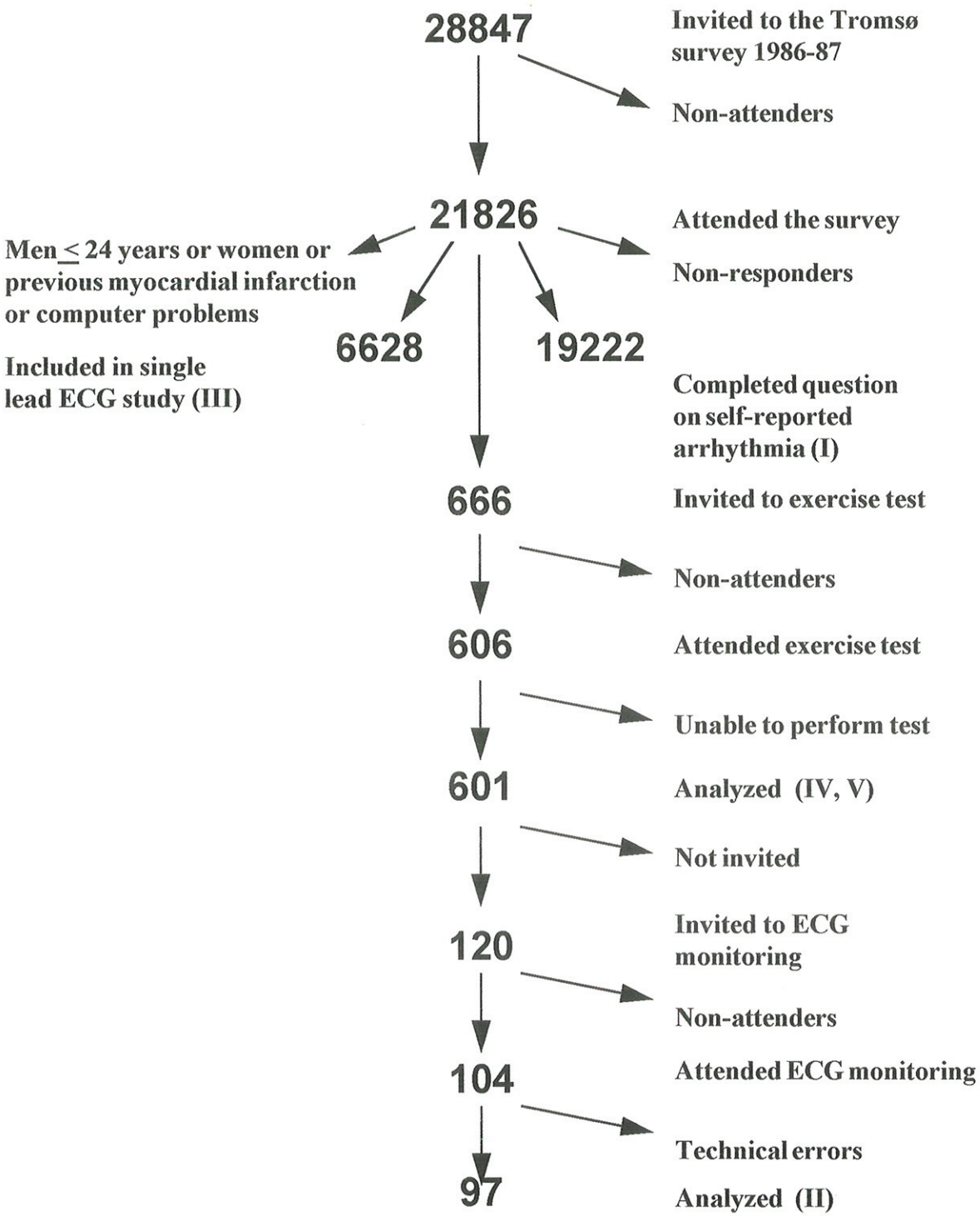
The figure shows a flow chart of subjects of the Tromsø survey and of the three populations included in the subprojects of this thesis. The description of materials and methods follows the same order as the figure. The Roman numerals in the figure refer to the paper numbers in the thesis.

2.1 The Tromsø Survey 1986-87 (I)

The first cardiovascular survey in Tromsø was carried out in 1974 and comprised 6595 men born in 1925-54 (1). In the second survey, carried out in 1979-80, 16621 subjects participated. This included men born in 1925-59 and women born in 1930-59, (2,3).

The third survey in 1986-87 was set up by the University of Tromsø and the National Health Screening Service in collaboration with the Tromsø Health Council (3,4). Altogether 28847 subjects in the municipality were invited. This included all men born between 1925 and 1966, all women born between 1930 and 1966, a 10% sample of youths born between 1967 and 1974, and the families (i.e. children born before 1974 and the wives) of the high risk men participating in the family intervention trial. Among the invited subjects, 49 were dead, 1600 had moved out of the area, and 583 were temporarily living outside Tromsø during the screening period, giving 26615 as the eligible

Figure. Flow chart of survey and subproject participants



population. Altogether 21826 persons were examined (179 without invitation), implying that the attendance rate was 81.3% of the eligible population. Attendance increased with age after 20 years and was higher in females than in males.

The procedures were mainly the same as in the first and second surveys (1,2) and in the Norwegian county surveys (5). The survey started in August 1986 in the peripheral parts of the municipality and finished in April 1987 in the city of Tromsø. In the central part of Tromsø the participants were invited according to their day and month of birth, whereas in the rest of the municipality the participants were invited according to their place of residence. One reminder was sent. Enclosed with the letter of invitation was a questionnaire which covered (amongst other things) the following aspects (appendix 1): previous cardiovascular disease including myocardial infarction, symptoms suggestive of angina pectoris, antihypertensive treatment, smoking habits, coffee consumption, and physical activity at work and in leisure time. Analyses of the questions on physical activity have been presented (V). The participants were asked to fill in the questionnaire at home and return it at the examination. All subjects examined completed the questionnaire. The reliability of self-reported myocardial infarction is discussed in chapter 4.4.

The examination comprised checking of the questionnaire for inconsistency, measurements of weight, height, blood pressure and heart rate, taking of one-lead computerized ECG and a nonfasting blood sample. Details of the ECG registration are presented in paper III and in chapter 2.2. The blood samples were analyzed for serum total cholesterol, high density lipoprotein (HDL), triglycerides and gamma glutamyl-transferase. The methods for measurements of height, weight, blood pressure, heart rate and serum lipids have been detailed elsewhere (4). Each person examined was handed a stamped and addressed envelope with a second questionnaire which they were asked to complete at home and return by mail. Altogether 92% of the participants returned the questionnaire which comprised the following main topics (appendix 2): Dietary habits, alcohol intake, previous and present diseases, symptoms including self-reported arrhythmia, diseases in relatives, social conditions, psychological attributes, and use of health care services. The question on self-reported arrhythmia was answered by 19222 participants (88% of all the participants). The results of the analyses of this question and relations to different determinants is presented (I). Validation of the question was performed as presented in

paper II.

2.2 The single lead ECG study (III)

Included in the screening was a one-lead computerized ECG aiming at assessing the predictive value of ECG variables for myocardial infarction in a cohort study (III). The plan was originally to have an ECG registration of all the participants of the survey. However, due to problems in developing suitable software for the computerized registration, only conventional paper registrations were taken in the beginning of the screening. The results of the paper registrations are not included in this work. Only subjects examined after the first ten weeks of the screening had a computerized ECG registration, and all these participants were examined in the central part of the municipality. In paper III only men without previous myocardial infarction and above 24 years at the screening (n=6628) were included, due to the lack of myocardial infarction below this age. Women were excluded from the analyses in paper III because only 12 cases of myocardial infarction occurred in the follow-up period, and this number was considered too low to make valid estimates.

The software for the ECG registration was developed by Spacetec A/S and the computer consultants at the Institute of Community Medicine. The main features of the recording method have been presented (III). The ECG registration was made in a separate room as one of five different stations at the survey. Three drivers from the screening team of the National Health Screening Service were trained to conduct the ECG recording. It was decided to take only lead I in order to make savings in time and money compared to a registration of 12-lead ECG. Another reason for choosing only lead I, was the assumption that this lead would give sufficient information for our purpose which was to use the ECG variables as predictors and not for clinical diagnostics.

Development of software for analyzing the ECGs turned out to be a major challenge. In 1987 contact was made with Peter W. Macfarlane, University of Glasgow, for collaboration, as he had developed a program that might satisfy our needs (6). In February 1988 the first batch of 20 ECGs were successfully analyzed, and a decision was made to analyze all the ECGs at the University Department of Medical Cardiology, Glasgow Royal Infirmary. A magnetic tape with all the data were thereafter sent to Glasgow. The analyzed ECGs were received February 1990. The data file was ready for statistical analyzing in the spring 1991 after correction for logical errors and merging the file to the rest of the Tromsø survey data,

the myocardial infarction register from the hospital and the Death Register at the Statistics Norway. Follow-up procedures are summarized in paper III. Quality control of the computerized ECGs is discussed in chapter 4.4.

The ECG registrations were converted from an ASCII format into common ECG variables (6). In paper III only the predictive value of QRS duration and T wave amplitude are presented, because they were the only variables with significant predictive power after the relatively short follow-up time (3.9 years).

2.3 The exercise test study (IV, V)

A project was set up primarily to examine physical fitness and exercise ECG in relation to other coronary risk factors including self-reported physical activity. All participants were recruited among subjects 20 years or older who attended the Tromsø survey 1986-87 in the central office building in the city of Tromsø; i.e. among about 82% of the participants. This was done to avoid travelling with heavy equipment to the other 18 different examination sites that were used in the survey.

About 200 persons attended the survey per day. From the middle of September 1986, random samples of four subjects per day were drawn from the database of attenders. The random sample size was increased to seven in the end of October. Invitation to participate in the substudy was sent by mail to 313 men and 350 women. Two reminders were sent, and 294 men and 312 women attended, giving attendance rates of 94% in men and 89% in women (errata). The response rates according to age and sex are given in table 1. For men the response rate varied between 91.8% (50-59 years) and 95.4% (30-39 years). In women the lowest response rate was found in the age group 30-39 years (85.2%), and the highest rate in the age group 50-59 years (97.1%). Causes for non-attendance are presented in table 2.

Participants in the subproject constitute the population for the analyses presented in papers IV and V. The examinations took place between 8 AM and 15 PM from October 1986 to June 1987. The participants met a study secretary and a physician. The examination started with a history of previous disease including previous myocardial infarction and other serious disease. Thereafter peak expiratory flow (Wright peak flow meter), blood pressure measurements, auscultation of carotid arteries for bruits proximally and distally and 12-lead resting ECG recording at 50 mm/s with subjects in the supine position were performed. The exercise test was then done on all subjects except four women and one

man who were unable to perform the test due to disabilities in the musculoskeletal system or serious disease. One woman and eight men were, for safety reasons, examined at the cardiological laboratory at the local hospital. The exercise test consisted of measurement of physical working capacity, exercise ECG and blood pressure measurement after 4 minutes (100 W). The method for the physical fitness (submaximal work load) test is described in paper V. Exercise ECGs were monitored on an oscilloscope, and resuscitation facilities were at hand. ECGs (CH leads V4, V5 and V6) were recorded on paper at 50 mm/s with the subjects sitting on the bicycle before and immediately after the test. The tests were interrupted in all subjects after ten minutes (250 W), unless specific symptoms or signs necessitated a premature break. Nineteen women and 206 men managed to perform the exercise test for ten minutes. Reasons for early discontinuation are shown in table 3.

The ECGs were coded blindly by this author according to the criteria shown in table 4. All the ECGs considered pathological and a random sample of about 100 normal ECGs were thereafter read by a cardiologist (Knut Rasmussen). The overall interobserver agreement was 88%, but the agreement regarding ischaemia and myocardial infarction was 100%. In case of disagreement, the result was settled by mutual consent. The ECG diagnoses are presented in paper IV and in chapter 4.7.3 and 4.7.4. In chapter 4.9 some results concerning peak expiratory flow and carotid auscultation are presented.

2.4 The ECG monitoring study (II)

This subproject aimed at validating the question on self-reported arrhythmia discussed in paper I. We wanted to compare arrhythmia during 24 h ECG monitoring with self-reported arrhythmia or palpitations during the monitoring and the year preceding the monitoring. The study design and methods have been presented (II). The participants were recruited among subjects who attended the exercise test study. Using the same population would give us the opportunity to link data from the two studies. Fifty-eight subjects who answered 'Yes' and 62 who answered 'No' to the question on palpitations were randomly selected for the study. Invitation was sent by mail, and 104 subjects attended among the 115 subjects who were eligible (90.4% of the eligible and 86.7% among the invited). Two reminders were sent. The response rate was 85.7% in men and 88% in women (table 16). Causes for non-attendance according to sex are presented in table 17. Ninety-seven

subjects were analyzed because speed artifacts during ECG tape recording made seven tapes unreadable.

The study was performed from August 1990 to January 1992 at the Cardiological laboratory, Medical Department, University Hospital of Tromsø. Enclosed with the letter of invitation was a questionnaire on some aspects of palpitations (appendix 3). At the visit the participants met a physician (TS, KR, M-LL) and a nurse. The physician checked the questionnaire and obtained information on the arrhythmic sensations in order to obtain a clinical diagnosis. The nurse took a standard resting ECG, and had the responsibility for the 24 h ambulatory ECGs. All the tapes were blindly analyzed by a trained physician (WZ) who had not met the participants and had no knowledge of the group allocation. The printed results were thereafter reviewed in detail by two of us (WZ, M-LL).

3. MAIN RESULTS AND CONCLUSIONS

3.1 Associations between palpitations, coronary risk factors, psychological conditions, lifestyle and arrhythmia (I,II)

Palpitations during the preceding year as reported in a self-administered questionnaire correlated positively with poor self-evaluated health status, psychological problems, and smoking in both genders in the general population. No relationship with the other major coronary risk factors such as serum lipid levels and blood pressure was found. The prevalence of reported palpitations was 12% in males and 17% in females, increasing significantly with age.

Arrhythmia seemed to be a common finding in this general population sample regardless of symptoms. Arrhythmia (at least one episode of ventricular or supraventricular arrhythmia or pauses $\geq$ 1.5s) assessed by 24-hour ambulatory ECG was more common in subjects with reported palpitations during the preceding year (98%) than in those without (74%). No relation was found between recorded arrhythmia and palpitations during ECG monitoring.

3.2 Associations between electrocardiographic variables and myocardial infarction in men (III)

QRS duration and T wave amplitude as measured by computerized ECG of lead I were independent predictors of myocardial infarction in young and middle-aged men without previous myocardial infarction. The relationships remained significant when age, angina pectoris, total- and HDL cholesterol, coffee consumption, systolic blood pressure, anti-hypertensive treatment, smoking and

heart rate were adjusted for. The findings were supported by a dose-response relationship between various categories of QRS duration, T wave amplitude and the outcome measure. T wave amplitude retained its independent association with myocardial infarction in all the analyses. QRS duration was an independent predictor of myocardial infarction when it was included as a categorical variable and as a continuous variable with adjustment for age. In the multivariate model, the relation between QRS as a continuous variable and myocardial infarction was somewhat reduced and became nonsignificant.

3.3 Associations between exercise-induced silent myocardial ischaemia and coronary risk factors (IV)

Based on exercise ECG, the prevalence of silent ischaemia was 2.5% in men and 3.4% in women without previous myocardial infarction. Among coronary risk factors, only systolic blood pressure in men was associated with silent ischaemia.

3.4 Associations between physical activity, physical fitness and other coronary risk factors (V)

Physical fitness as tested by bicycle ergometry was positively related to self-reported leisure physical activity and negatively related to work activity in both genders. High and moderate leisure physical activity was considerably more common in men (29%) than in women (9%). Multivariate analyses showed that physical fitness correlated negatively with smoking, physical activity at work and age, and positively with physical activity in leisure and body mass index in both genders. Previous myocardial infarction, HDL cholesterol, and systolic blood pressure were significant predictors of fitness in men. The explained variance varied between 46% and 26% in men and women, respectively. For leisure physical activity, smoking and heart rate were negatively associated with leisure activity in women, and a negative relation with total cholesterol was observed in men. The explained variance was 12% in men and 16% in women. Coronary risk factors seemed to be more firmly linked to fitness than to activity.

4. GENERAL DISCUSSION

4.1 Risk factors

Epidemiologists are careful people and have introduced the risk factor concept which is not to be confused with causes. A risk factor may be defined as an individual characteristic or exposition which increases the individual's risk of a certain disease (7). In addition to etiological and internal factors,

even statistical associations are often called risk factors. Pre-disease states, symptoms and clinical signs may be called risk factors, for example ECG signs, palpitations and arrhythmias. A rather wide definition of a risk factor for CHD may thus be any factor which has been shown in epidemiologic studies to be associated with the incidence of CHD, and an unbiased estimate of this association is what we would like to see (Dag Thelle, personal communication). What then is bias?

4.2 Bias

Bias may be defined as any systematic error in an epidemiologic study that results in an incorrect estimate of the association between exposure and risk of disease (8). Bias should always be considered as a possible alternative explanation of any observed statistical association, and is most effectively dealt with through careful design and conduct of a study. The main precondition for an unbiased estimate of the strength and direction of an association is that the assessments of the exposure and end-point variables are completely independent.

4.3 Selection bias

Selection bias refers to a type of distortion that may result from the way subjects are selected to the study. It is generally difficult to obtain sufficient information to quantify the selection bias, but the direction may be indicated. The main source of selection bias in the present study is non-response, either non-attendance to the screening and the substudies or non-response to the questionnaires in the main survey. The best way to avoid non-response bias is to increase the response rate, and in the third Tromsø survey both the attendance rate (81%) and the response to questionnaires was high (100% to questionnaire 1, 92% to questionnaire 2) (3). We have no direct information about non-participants in the Tromsø survey 1986-87. Death or incidence rates from CHD were higher in non-attenders than in attenders in the first Tromsø study (Dag Thelle, personal communication), the Oslo study (9), the Norwegian County study (10) and in a Swedish cohort (11). An exception may be the youngest non-attenders whose distribution of health related variables were the same as among the attenders in the Nord-Trøndelag study (12). The Swedish study also showed lower socioeconomic status among the non-participants (11). These findings indicate that persons with adverse coronary risk factor levels are more prevalent among non-attenders than among attenders. The same tendency for smoking and respiratory symptoms was found in a study from the second Tromsø survey (13).

The excess coronary mortality among non-attenders may also reflect an unwillingness to seek medical care in the event of illness. Selection bias may therefore underestimate or not influence the true association between palpitations, coronary risk factors and other determinants in paper I. It does not seem likely that the relationships are overestimated as a consequence of selection bias.

In the single lead ECG study (III) only 73.7% of the attenders had an ECG registration, due to the software problems described above (chapter 2.2). There is no reason to believe that the ECG variables or the incidence of myocardial infarction differed in those with and without a registration, and the results should therefore be representative of the whole population of attenders.

The age and sex distributions of attenders to the exercise test study and to the ECG monitoring study are presented in chapters 2.3 and 2.4, respectively. A comparison of some coronary risk factors between the attenders and the non-attenders to the exercise test study is shown in table 5. Female non-attenders were significantly younger than the attenders. From chapter 2.3 we know that the attendance rate was somewhat lower among women than among men. Except from the sex distribution and the age difference among the female groups, the attenders and non-attenders were comparable. The response rate was high in both studies and particularly among men in the exercise test study. The high male response is in contrast to the response to the main screening. Men were perhaps more interested than women to have their fitness tested. This impression was confirmed during the study, since many men seemed to have a higher motivation compared to women for trying their hardest. No significant differences in coronary risk factors were found between attenders and non-attenders to the ECG monitoring study (table 18).

With respect to the findings and conclusions in the present work, the selection bias due to non-attendance is considered to be a minor problem.

4.4 Information bias

Information bias is a distortion due to systematic measurement error, and results from differences in the way data are obtained from the groups studied (8). Systematic errors are potentially serious, since the bias they cause may lead to invalid conclusions being drawn. Random errors, on the other hand, give rise to reduced precision, but can only work in the direction of underestimating the true association (14).

Self-administered questionnaires were used to classify subjects into various categories. The data quality depends on the often imperfect memory of individuals, and the extent to which they want to tell the truth. Recall bias can either exaggerate or underestimate the estimate of an association. Self-reported information of risk factors or diseases should whenever possible be documented through other sources of data. We had the opportunity to compare some of the question responses with alternative examinations, and found that the reproducibility of the response about self-reported arrhythmia was fairly high (II). Further validation of this issue revealed a significant relationship between subjectively reported and objectively verified arrhythmia (II). The relatively small number of participants in the study, however, makes this result somewhat questionable. The gross validity of the responses to questions about physical activity has been discussed in relation to the significant association between leisure activity and physical fitness (V, chapter 4.8.4). Few of the participants were aware of any hypothesized relation between the variables in this study. The misclassification of subjects is therefore likely to be randomly distributed in different groups, resulting in an underestimate of the true association.

The validation of self-reported myocardial infarction is discussed in paper III. In the study we assessed the risk of developing myocardial infarction according to QRS duration and T wave amplitude in men without previous myocardial infarction. In our preliminary analyses we excluded 130 subjects with self-reported myocardial infarction, and during follow-up 93 events of myocardial infarctions occurred. We then decided to merge the self-reported information on previous myocardial infarction with the local hospital diagnostic register, and found that 11 of the 93 subjects with a new event of myocardial infarction during follow-up actually had had a myocardial infarction without reporting it on the questionnaire. The linkage resulted in a reduction of new events from 93 to 82 (11.8%) which weakened some of the estimates of the association between QRS duration and myocardial infarction. The misclassification of subjects based on only self-reported information would thus have resulted in exaggerated estimates in this study.

We decided to classify the computerized ECGs with a QRS duration exceeding 119 msec according to traditional ECG diagnoses (III). The possibility for observer bias was eliminated because Peter W. Macfarlane, University of Glasgow, was unaware of the event status of those with a prolonged QRS duration. He performed the coding on

ECG paper printed out on his system after receiving the identity codes from us. This procedure also gave us the possibility to validate the computer with regard to accuracy in assessing QRS durations. In total 65 ECGs were evaluated, 49 were grouped according to different diagnoses, and 16 (24.6%) had a normal QRS duration. These erroneous computer estimates were thereafter altered to a normal from an abnormal value, and it is assumed that all of the other QRS durations less than 120 msec were correct. Otherwise they would have shown up as wide QRS complexes which would have been detected. On two occasions the QRS duration of some ECGs were altered without changing their classification. It was not possible to find out why the ECGs with a normal QRS duration had found their way into the group of 62 ECGs. Noise generated by instability, poor contact within electrical circuits or external electromagnetic field interference may be part of the explanation. The misclassification of subjects based on only computerized information would have resulted in exaggerated estimates regarding the predictive value of QRS duration.

Observation or expectation bias might arise from the observer's knowledge of the participants in a study. In this study the results of the arrhythmia interpretations could be suspected to be biased (II). Wei Zhang was unaware of the hypothesis of the study and performed all the preliminary analyses. When this author looked at all print-outs together with her, we were both blinded with regard to identity and group allocation of the subjects. Observation bias is therefore not considered to be a problem in this study.

The exercise study was an opportunity to validate the reproducibility of some of the responses to the survey questions. Questions about self-reported previous myocardial infarction and angina pectoris were posed on both occasions, but unfortunately this was not done for physical activity. For myocardial infarction in both sexes and angina pectoris in women, the reproducibility was perfect (table 6). As presented in table 7, the number of ECG diagnoses of myocardial infarction were reduced in both sexes as compared to the questionnaire response. Six men answered 'yes' to the question about angina pectoris at the survey, compared to eight at the exercise test, but only two men had ischaemia verified during the exercise test. Table 6 also shows the mean systolic blood pressure measurements at the survey and at rest before the exercise test. As expected, there was a higher resting systolic blood pressure at the exercise test. It is likely that the difference is due to both the

less standardized measurement conditions before the exercise test, and the subjects being somewhat tense before the ergometric test.

A major source of bias in cohort studies concerns the loss of subjects to follow-up. The Register of all deaths in the Statistics Norway (former Central Bureau of Statistics) has been evaluated as nearly complete (15). Some subjects with non-fatal myocardial infarction may have been lost to follow-up due to at least two different reasons (III): There may have been subjects with myocardial infarction who did not attend the local hospital. Furthermore, there may have been some migrated subjects with a myocardial infarction. The migration rate is lowest among the oldest who are at the highest risk of having a myocardial infarction. Probably few cases are lost to follow-up which is considered to be a minor problem in this study.

4.5 Confounding

The term confounder is used by epidemiologists to describe a covariate that is associated with both the outcome variable of interest and a primary independent variable or risk factor (16). Confounding can have a very important influence, possibly even changing the direction of an association.

There are a number of methods for control of confounding in the design or in the analysis of an epidemiological study. In papers III and IV restriction of admissibility criteria was used at the design stage, and stratification and multivariate statistical modelling were used in the analysis. Restriction may limit the generalizability of the results, and makes it difficult to determine the magnitude and the direction of the confounding unless data are collected among those excluded. We excluded respondents who reported previous myocardial infarction on the questionnaire because their ECG changes would have strengthened both the risk estimates in paper III, and the prevalence of silent ischemia and associations to other risk factors in paper IV, and we wanted to study those without previous myocardial infarction.

Age is a strong confounder, and all papers show age-stratified findings and results of multivariate analyses where age is one of the independent variables. One exception is paper II, but all the reported findings were tested for age trend which was not found to be a confounder in the study. In paper III all common coronary risk factors were taken into account in the multivariate models for estimating the predictive value of QRS duration and T wave amplitude for myocardial infarction. It is worth noting that the

multivariate-adjusted estimates for relative risk did not differ much from the age-adjusted estimates. This indicates that the common risk factors were not strong confounders for the association between the ECG variables and subsequent myocardial infarction.

4.6 Palpitations and arrhythmia

4.6.1 Relations to coronary heart disease

Palpitations, the subjective awareness of the action of one's own heart, is a common symptom occurring in many normal individuals, as indicated by the present data (I). There is some uncertainty about the prognostic significance of this symptom in the population. Some studies have stated that patients complaining of palpitations have a higher incidence of complex arrhythmias and cardiac disease, suggesting that they are at increased risk of myocardial infarction or fatal arrhythmia (17,18). Others find that palpitations are not an independent risk factor (19), but the study is biased by a small sample size (n = 109), a young population (mean age 43.3 years) and a short follow-up period (3.5 years). A follow-up study of the participants of paper I might provide more insight into the predictive strength of palpitations in a population.

We found that although the majority of the population have some kind of arrhythmia, only a minority have complex or serious arrhythmia (II). Previous studies also suggest that complex arrhythmias occur infrequently in asymptomatic young and middle-aged subjects, but the prevalence increases with age (20-4). Controversy exists regarding the prognostic implications of arrhythmia in apparently healthy subjects, since one recent study found an increased risk (25), and others did not (26,27). Objections against these studies relates partly to the inclusion of soft end-points, such as angina pectoris, among cardiac events (25), and partly to the lack of power to answer the question on predictive value (26,27). Our single lead ECGs may be analyzed with respect to arrhythmia, and a prospective study of these data may contribute to the discussion on the predictive value of arrhythmia.

4.6.2 Impact of depression and coffee

The characteristics of the episodes of palpitations listed in table 19 may give clues to the differentiation of palpitations in various subgroups. In many subjects the awareness of palpitations is probably related to psychological factors and lifestyle, and palpitations may be seen as part of a syndrome which is usually closer linked to depression and lifestyle than to coronary risk factors (I). Furthermore, no significant relations with coronary risk factors were

found for objective arrhythmia (table 20), except for high coffee consumption (> 5 cups per day) which was more prevalent in the arrhythmia group than in those without arrhythmia. No significant difference in coffee consumption was found, however, between those with and without serious arrhythmia (table 21). The only difference was for peak expiratory flow which was significantly lower in the serious arrhythmia group, indicating a possible malignant prognosis in this group as further discussed in chapter 4.9.1.

Previous reports have linked excess caffeine intake to an increased prevalence of supraventricular and ventricular arrhythmia (28,29). It has furthermore been shown that depression may participate in the formation of severe ventricular arrhythmia in otherwise healthy men (30). In addition to inducing true arrhythmias and palpitations, depression may also lead to a better recognition of true arrhythmia and cause even more anxiety. It may well be that many reporters of palpitations have a somewhat nervous constitution (31), and are likely to drink a lot of coffee to relieve their symptoms. Optimal clinical management of such patients thus primarily requires a consideration of psychological conditions and lifestyle habits, particularly coffee consumption.

4.7 Electrocardiographic signs

4.7.1 Utilization in epidemiologic studies

ECG is a useful, practical, inexpensive test of the function of the heart. Although it has come under increasing pressure in recent years from other techniques such as echocardiography, there is still an important role for this simple, non-invasive test. It can be rapidly recorded with portable equipment, and is therefore feasible in population studies, particularly after the introduction of computerized methodology, as demonstrated in our study (III).

For consistent interpretation and comparability, measurement and coding techniques must be standardized. The standardized coding scheme in widest use for epidemiological applications is the Minnesota Code (32,33). Its primary purpose was to improve the comparability of ECG classification and thus the consistency of the assessment of CHD in different populations. The code has been widely used in large population studies, and the prognostic significance of various ECG signs for CHD and mortality has been extensively validated (34-41).

4.7.2 Single lead electrocardiography

The single lead ECG study was performed in order to try to develop a more feasible and simple way of using ECG as a predictor

for coronary heart disease compared to the Minnesota code (33) in large population surveys.

The Minnesota code describes any ECG in terms of a number of discrete numerical codes, which pertain to most aspects of the 12-lead ECG (42). Various feature patterns are observed or measured, and a set of rules are used to categorize the patterns into a given set of usually mutually exclusive diagnostic classes. The rules can be expressed as a branching decision tree which uses the presence or absence of various features in each decision node to determine which branch of the decision tree to follow. The main limitation of the Minnesota code is its complexity. Another limitation may be that the majority rule should be applied (42). The rule states that a codable item has to be present in the majority of available beats in a lead. This means that the code does not necessarily utilize all the information in the lead. A third limitation is that only more or less pathological features are coded, and that the interpretation is based on patterns and not single electrocardiographic items or variables such as amplitudes or durations alone. It is difficult to find a precise cut-off point between normal and pathological values of a certain ECG variable. There may thus, as a supplement, be a need for a system which takes into account the whole variation of the ECG variables in order to improve the use of ECG for prediction of CHD. Our findings suggest, albeit a short follow-up period, that ECG of lead I may improve the prediction in population groups where stratification into risk categories is important (III). Computerized single lead ECG may be an alternative or a supplement to the 12-lead ECG categorized according to the Minnesota code. The usefulness of our findings in individual patient management may at present be limited, as this study does not provide a rationale for routinely obtaining serial ECGs in asymptomatic persons. Single lead ECG is included in the fourth Tromsø Survey (1994-95), but the results should also be verified in other populations.

4.7.3 Twelve lead electrocardiography

The prevalences of myocardial infarction and other diagnoses as categorized by ECG are presented in table 8. Eight men and one woman in the exercise study had self-reported myocardial infarction (IV, V), while the prevalence as diagnosed by ECG is slightly different. As expected for this age group, the prevalence of abnormal resting ECG was higher in men than in women. In men, the most common ECG abnormalities at rest were myocardial infarction, possible

left ventricular hypertrophy and conduction de- fects. In women, ventricular arrhythmia and ST or T wave changes were most common. Serum cholesterol was higher for both genders with an abnormal resting ECG (table 9). The men with pathological 12 lead ECG findings obviously had more severe heart disease than the women. This is further indicated by the lower physical fitness and peak expiratory flow (table 9), and higher resting blood pressure as measured before the exercise test (table 10) in these men compared to those with a normal ECG. Men with a pathological resting ECG also had a lower exercise heart rate response (chronotropic incompetence) (table 11) compared to those with a normal ECG. Chronotropic incompetence probably increases the risk of CHD (43-6).

4.7.4 Exercise electrocardiography

Silent ischaemia is defined as objective evidence for myocardial ischaemia in the absence of angina or equivalent symptoms (47), and has been found to be an independent predictor of CHD in men (48-50). It remains uncertain whether medical or surgical treatment in asymptomatic men prolongs survival significantly (51).

CHD has traditionally been regarded as a disease of men, but it is also the leading cause of death in women. Most epidemiological studies of CHD and its risk factors, including ECG abnormalities, have been conducted in men. Prospective studies of silent ischaemia in women have to our knowledge not been performed. There is now a general agreement regarding the need for more studies in women because we want to know whether the present recommendations for preventing, investigating and treating CHD are equally appropriate in both sexes (52). The most important contribution of our exercise ECG study may therefore be that women are included (IV).

Tables 8 and 12 present the exercise ECG findings and relations to coronary risk factors, respectively. In table 8 subjects with previous myocardial infarction were included, which gave slightly different prevalences for ischaemia compared to paper IV. The prevalences of ischaemia and nonspecific ST/T wave changes were quite similar in both genders. Table 12 shows a higher systolic blood pressure for men with an abnormal compared to those with a normal exercise ECG. The results in paper IV further indicate that silent ischaemia may have different importance in women compared to men as indicated by different relations to coronary risk factors. In men, silent ischaemia was positively related to systolic blood pressure and coronary risk score, while no such relations were found in

women. This points to the necessity of discussing different consequences of ECG findings according to gender.

4.7.5 Ambulatory electrocardiography

Ambulatory ECG is a complex procedure. An inherent problem is the almost inevitable occurrence of many artifacts, resulting from a failure of one or more of the components of the system. A better knowledge of these artifacts and a stricter study design could probably have reduced our problems leading to the exclusion of as much as seven tape recordings from the analyses (II).

The main finding in our study was that cardiac arrhythmia, irrespective of symptoms, is very common in a population, although the experience of palpitations the preceding year defines a group with significantly more arrhythmia than those without such palpitations. A common finding is not necessarily normal, but our findings are in accordance with others, concluding that most arrhythmias are normal, probably with the exception of some complex and rare arrhythmias (53).

A somewhat surprising finding was that the clinical evaluation of those with reported palpitations the previous year did not improve our capability of identifying those with serious arrhythmia. This may indicate that arrhythmias can be judged more objectively in those reporting palpitations on a questionnaire than from a subsequent clinical evaluation.

Ambulatory ECG is recommended as the optimum way for evaluation of palpitations (53). In many cases no abnormality can be seen at the time of the symptom (II). In contrast, it is well known that subjects may have severe arrhythmia without being aware of them. We therefore suggest that serious concomitant symptoms such as syncope should be present before further clinical investigation including 24-hour ambulatory ECG is performed.

Reduced heart rate variability as a measure of cardiac autonomic activity is an independent risk factor of cardiac death in patients following myocardial infarction (54, 55), and in healthy subjects heart rate variability is influenced by age, sex, smoking and physical activity (56). It is thought that decreased heart rate variability results from a relative decrease in parasympathetic activity in relation to sympathetic activity, and that this facilitates arrhythmogenesis (57). It would be of interest to study the relation between heart rate variability, arrhythmia and palpitations, and we are currently planning such a study, utilizing the same tape recordings as in the present study.

4.8 Physical activity, physical fitness and coronary heart disease

4.8.1 The benefit of physical activity

Physical activity is a behaviour defined as any bodily movement produced by skeletal muscles that results in energy expenditure (58). The literature on the role of physical activity in the primary prevention of coronary heart disease is overwhelming and presents a wide variety of methods. Numerous epidemiologic reviews have concluded that physically active people are at lower risk of CHD than those who are inactive (59-61). The risk of death from CHD is less in men whose jobs are strenuous than in those who do light work (62). However, in industrialized countries, so few occupations could now be described as "heavy" that interest is centering mainly on the protective effect of leisure-time physical activity. We found that fitness increased with increased leisure activity and decreased with work activity (V), which supports that leisure activity is the most important activity dimension today.

In a recent meta-analysis a consistent, independent relation was found between physical activity and CHD in men. A relative risk of 1.37 (95% confidence interval 1.27-1.48) was found when physical inactivity was compared with moderate and heavy activity (61). Since the relative risk was less than two, the strength of the association must be considered weak compared to other risk factors, such as hypercholesterolemia and smoking. Data on the type, intensity, duration, and frequency of activity needed to protect against CHD are limited and inconsistent and require further clarification (63-8).

Physical activity has been found to be associated with several other coronary risk factors, such as blood pressure (69), serum cholesterol (70), HDL cholesterol (71,72), and smoking (73). We found that smoking emerged as a strong negative predictor of leisure activity in women, and a negative relation with cholesterol was found in men (V). Evidence for other mechanisms is growing, including direct effects on atherosclerosis and the coronary artery anatomy (74,75), haemostatic function (76), fibrinolytic response (77), platelet aggregability (78), glucose tolerance and insulin sensitivity (79,80). Exercise increased the threshold for ventricular fibrillation in exercising rats (81). Furthermore, activity lowers the resting heart rate in humans (4), and low heart rate is associated with lower mortality (82). In our study physical activity was negatively related to heart rate in women. The low level of leisure activity in women, and the relation between leisure activity, smoking and other atherogenic

behaviours and risk factors (V, 83) justifies the importance of public health strategies which are directed particularly against the female population.

4.8.2 The benefit of physical fitness

Physical fitness is a set of attributes that relates to the ability to perform physical activity. One health-related component of physical fitness is cardiovascular fitness which relates to cardiorespiratory endurance (58). Since physical activity is more difficult to quantify than fitness, the latter has gained popularity both in the assessment of cardiovascular function and as a predictor for CHD. There are some indications that fitness is a more powerful predictor of CHD than activity. The relative risk of CHD in epidemiologic studies comparing the least to the most fit categories ranges from 1.2 to 4.8 (84). All major published studies suggest a favourable long-term outcome in fit compared with unfit subjects, regardless of how fitness was measured (85-91).

Although the genetic component of fitness has been suggested to be at least 40% (92), this leaves about 60% of the variation attributable to other causes, particularly physical activity. Our study confirms the significant association between fitness and leisure activity, and suggest that coronary risk factors are more closely related to fitness than to activity (V).

4.8.3 The risk of physical activity and physical fitness

It is an apparent paradox that regular exercise protects against coronary heart disease, while an acute bout of strenuous exercise is associated with an increased risk of arrhythmia, myocardial infarction and sudden cardiac death. It is, however, important to distinguish absolute risk from relative risk. Based on eight population studies in men aged 35-64 years it appears that sudden cardiac death associated with physical activity occurs between 0.00 and 2.00 per 100000 person-hours of exercise (93). This is up to 100 times greater than the rate of sudden cardiac death during nonexertion. In a review of 22 studies it was concluded that about 15% of fatal and non-fatal coronary events were associated with moderate or heavy exercise (94).

One study allowed for simultaneous evaluation of the benefits of habitual physical activity as well as the risks of sudden cardiac death during activity (95). An increased risk of sudden cardiac death was found at any level of habitual activity compared to periods of inactivity. A particularly exaggerated risk during exercise compared to other times of the day was found among the men who less frequently engaged in high-intensity

activity (relative risk 56) compared to habitually active men (relative risk 5).

The transiently increased risk of sudden cardiac death during activity is countered by a lower overall risk for habitually active subjects (93). The risk should not preclude participation for benefits of a physically active lifestyle.

4.8.4 Measurement of physical activity and physical fitness

As is true for most complex and frequent activities, the amount of physical activity is difficult to measure with precision. It is not readily dichotomized, such as smoking versus nonsmoking, nor does it conform to a readily measured continuum, such as cholesterol (59). Unfortunately, no methods are generally accepted, so it has become the custom for investigators to devise their own standard, often with scant reference to validity, and still less to comparability with other studies. At least 30 different measurement techniques have been described (96). Many of these are complex instruments that are associated with excessive costs and time commitments that make them impractical in epidemiological studies.

The question (97) used in the Tromsø Study (paper V, appendix 1) has been widely used in other Scandinavian studies (98-100), and has been shown to have predictive power with regard to coronary heart disease mortality (100). In addition to the question on activity level (paper V, appendix 1), another question was included (appendix 2) which covered intensity rather than level of physical activity. The intensity question was: "How often do you perform physical activity for at least 20 minutes, leading to sweating and breathlessness?" "Never - Weekly - Several times per week - Daily". The question did not distinguish between leisure and work activity. Table 22 shows the distribution of the whole screened population according to the different questions. The activity level question (appendix 1) placed as much as 50% and 66% of men and women, respectively, in the "walking" category, while the intensity question (appendix 2) gave a more even distribution of the three lowest categories of intensity. Total cholesterol, HDL cholesterol and smoking showed a linear trend according to the levels of the activity level question. For the categories of the intensity question, however, there was a U-shaped curve for many of the risk factors, with a more risky profile in the category with the hardest physical activity. This is probably due to subjects with hard physical intensity at work in this category, having more atherogenic risk factors. A more suitable question for our next survey would be to

distinguish between leisure and work physical activity in the intensity question, which would probably give a more linear distribution of the risk factors associated to intensity. To avoid the large proportion of participants, particularly women, in the "walking" category of the activity level question, one may include both an intensity dimension and a gender dimension in the third category of the question (Training, gardening or housework at least four hours per week, hard enough to work up a sweat or breathlessness). It has been shown that failure to account for hard domestic work may result in women being misclassified as inactive when just the opposite may be true (101).

Cardiorespiratory power (VO_2max) has been the main component of physical fitness measured in population studies, generally in terms of maximum oxygen uptake (91). This reflects the maximum functional capacity of the integrated performance of the heart, lungs, vascular system, and muscle tissue.

The direct measurement of VO_2max is seen as the gold standard (102), but is contraindicated in field studies by both technical and safety considerations. Other simpler tests have therefore been developed as a proxy for oxygen uptake, such as heart rate, maximal or submaximal workload (V), measured on a bicycle ergometer or a treadmill. There is a need for methods for measuring fitness with acceptable precision in population surveys, but with a lower requirement to the level of technology than those currently in use.

Studies relating questionnaire data regarding physical activity to measured fitness have reported correlations between total leisure time activity and fitness ranging from near zero to over 0.6 (103). This range reflects both the variability in the quality of questionnaires and the fact that only activity above customary intensity for the individual, and dynamic aerobic activity at that, will have a cardiorespiratory training effect. We found correlation coefficients of 0.18 in men and 0.39 in women (V). The sex difference may be due to the fitness test being maximal in most women, but only in few men, as discussed in chapter 2.3.

In paper V it is concluded that fitness should be added as an important variable in addition to activity in future studies on the relationship between exercise habits and disease. For population studies, however, expensive fitness tests involving heavy equipment are impractical on a large scale. We therefore primarily need short, easily understood questions on physical activity which also cover the dimension of physical fitness and training effect better than previously.

4.9 Additional results

Some of the data that were collected in the exercise study are not included in the papers. They are presented and briefly discussed in this chapter.

4.9.1 Peak expiratory flow

Reduced lung function, expressed as forced vital capacity, forced expiratory volume or peak expiratory flow has been associated with increased risk of CHD morbidity and mortality and all-cause mortality in several studies (104-6). The associations persisted after adjustment for smoking habits.

We measured peak expiratory flow in the exercise test study. A multiple regression analysis of peak expiratory flow, as measured before the exercise test, with several coronary risk factors as independent variables is shown in table 13. In both sexes age and physical fitness were significantly associated with lung function, in addition to smoking habits in men. The relations to other risk factors were trivial. Age adjusted correlation coefficients of peak expiratory flow and fitness were 0.33 and 0.30 ($p < 0.0001$) for men and women, respectively (data not shown), which corresponds to previous findings (107). Peak expiratory flow may thus serve as a marker of physical fitness.

Due to high correlations between different tests for lung function (108), they can probably to some extent be used interchangeably. Peak expiratory flow is more effort-dependent than other tests for lung function and might provide a measure of overall vitality and state of health. The test is simple, inexpensive and convenient to include in screening programmes.

4.9.2 Carotid auscultation

Previous studies have revealed a high predictive value of carotid bruit for carotid atherosclerosis and ischaemic stroke in patient populations (109,110). We performed auscultation of the carotid arteries before the exercise test study with the original intention to validate the findings with ultrasonography. Due to small numbers the evaluation study was not performed, but tables 14 and 15 present the prevalence of carotid bruit and some associations with survey and exercise test characteristics. The prevalence of carotid bruit was 1.4% and 1% in men and women, respectively. The age range in those with carotid bruit was 31-61 years in men and 49-55 years in women. In women age, total cholesterol, relative weight and blood pressures measured before and during the exercise test were higher in those with carotid artery bruit. No significant associations with coronary risk factors were found in men. It has been shown that when

there is greater than 25% stenosis of the carotid artery, a bruit may become audible by auscultation (109). Our findings, at least in women, may support previous findings that a carotid bruit is associated with atherosclerosis in most subjects as indicated by their coronary risk factors being significantly higher compared to those without carotid bruit.

5. FINAL REMARKS AND IMPLICATIONS

Cardiac arrhythmia is a common finding in the general population. Therefore, further clinical investigations are warranted only when serious concomitant symptoms are present. The association between palpitations, arrhythmia and lifestyle including smoking, coffee consumption and psychological symptoms should be underlined. The depressed, coffee-drinking patient with palpitations should be reassured and not medicalized. A follow-up study of the subjects with palpitations in our study might give risk estimates of the predictive power of this symptom.

It seems worthwhile to remember that women probably have about the same prevalence of silent ischaemia as men, but the prognostic significance may be different according to sex, and must be left for further studies.

The present findings suggest that single lead ECG is a feasible method in screenings which may improve the prediction of coronary heart disease in populations. Further investigation of the present data set, in other populations and in women is warranted, with special emphasis on the predictive value of other variables including arrhythmia.

There seems to be a trend in the industrialized countries for a growing percentage of the population to adopt a sedentary lifestyle (111). The amount and intensity of physical activity probably only needs to be modest for the beneficial effects on health to be obtained. When it comes to primary prevention, there is enough knowledge to justify public health strategies aimed at increasing the activity among all age groups and both sexes. Population studies on changes of coronary risk factors and disease in subjects who change level of physical activity are few (112) and should be performed.

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ERRATA

1. Paper I, Table I: In the second line of the table, the text (Whole population, No known disease, Known disease) has been moved two columns too far to the right.
2. Paper IV, Methods: 316 men is written instead of 313 men. In paper V the same error is repeated, in addition to 297 instead of 294 men.

Appendix 1

Questionnaire 1, The Tromsø survey 1986-87

HELSEUNDERSØKELSEN I TROMSØ

(Gjelder bare den person som brevet er adressert til.)

Helseundersøkelsen kommer nå til Deres distrikt.

Tid og sted for frammøte vil De finne nedenfor.

De finner en orientering om undersøkelsen i den vedlagte brosjyren.

Vi ber Dem vennligst fylle ut spørreskjemaet på baksiden og ta med dette til undersøkelsen.

Vi ber Dem eventuelt melde fra om fravær på den vedlagte fraværsmeldingen.

Med hilsen

KOMMUNEHELSETJENESTEN I TROMSØ
FYLKESLEGEN I TROMS UNIVERSITETET I TROMSØ
STATENS HELSEUNDERSØKELSER

Født dato | Personnr. | Kommune | Kretsnr.

Møtested | Kjønn | Første bokstav i etternavn | Dag og dato | Klokkeslett

HØYDE	VEKT	ANM 70	M	P	Ø	KODE 75	AVVIK	ARM	MAN	APP.NR.	TSM 82

MÅLING 1		MÅLING 2		MÅLING 3	
MAR 65	S 68	MAR 91	S 94	MAR 97	S 100
HR 103	D 106	HR 109	D 112	HR 115	D 118

A FAMILIE

	JA	NEI	VET IKKE
Har en eller flere av foreldre eller søsken hatt hjerteinfarkt (sår på hjertet) eller angina pectoris (hjertekrampe)? 12			

B EGEN SYKDOM

	JA	NEI
Har De, eller har De hatt:		
Hjerteinfarkt? 13		
Angina pectoris (hjertekrampe)? 14		
Hjerneslag? 15		
Sukkersyke? 16		
Er De under behandling for:		
Høyt blodtrykk? 17		
Bruker De:		
Nitroglycerin? 18		

C SYMPTOMER

	JA	NEI	
Får De smerter eller ubehag i brystet når De:			
Går i bakker, trapper eller fort på flat mark? 19			
Går i vanlig takt på flat mark? 20			
Dersom De får smerter eller vondt i brystet ved gange, pleier De da:			
Stoppe? 21			1
Saktne farten?			2
Fortsette i samme takt?			3
Dersom De stopper eller saktner farten, går da smertene bort:			
Etter mindre enn 10 minutter? 22			1
Etter mer enn 10 minutter?			2
Har De vanligvis:	JA	NEI	
Hoste om morgenen? 23			
Oppspytt fra brystet om morgenen? 24			

D MOSJON

Bevegelse og kroppslig aktivitet i Deres fritid. Dersom aktiviteten varierer mye, f.eks. mellom sommer og vinter, så ta ett gjennomsnitt. Spørsmålet gjelder bare det siste året.

Sett kryss i den ruten som passer best.

Leser, ser på fjernsyn eller annen stillesittende beskjeftigelse? 25	1
Spaserer, sykler eller beveger Dem på annen måte minst 4 timer i uken? ..... (Her skal De også regne med gang eller sykling til arbeidsstedet, søndagsturer m.m.)	2
Driver mosjonsidrett, tyngre hagearbeid e.l.? .. (Merk at aktiviteten skal vare i minst 4 timer i uken.)	3
Trener hardt eller driver konkurranseidrett regelmessig og flere ganger i uken?	4

E SALT/FETT

Hvor ofte bruker De salt kjøtt eller salt fisk til middag?

Sett kryss i den ruten som passer best.

Aldri eller sjeldnere enn en gang i måneden 26	1
Inntil en gang i uken	2
Inntil to ganger i uken	3
Mer enn to ganger i uken	4

Hvor ofte pleier De å strø ekstra salt på middagsmaten?

Sett kryss i den ruten som passer best.

Sjelden eller aldri 27	1
Av og til eller ofte	2
Alltid eller nesten alltid	3

Hva slags margarin eller smør bruker De vanligvis på brødet?

Sett kryss i den ruten som passer best.

Bruker ikke smør eller margarin på brød 28	1
Smør	2
Hard margarin	3
Myk (Soft) margarin	4
Smør/margarin blanding	5

Hva slags fett blir vanligvis brukt til matlaging i husholdningen Deres?

Sett kryss i den ruten som passer best.

Smør eller hard margarin 29	1
Myk (Soft) margarin eller olje	2
Smør/margarin blanding	3

F RØYKING

	JA	NEI	
Røyker De daglig for tida? 30			
Dersom svaret er «JA», svar da på dette:			
Røyker De sigaretter daglig? ..... 31 (håndrullede eller fabrikkfremstilte)			
Dersom De ikke røyker sigaretter nå, svar da på dette:			
Har De røykt sigaretter daglig tidligere? 32			
Dersom De svarte «JA», hvor lenge er det da siden De sluttet?			
Mindre enn 3 måneder? 33			1
3 måneder – 1 år?			2
1–5 år?			3
Mer enn 5 år?			4
Skal besvares av de som røyker nå eller som har røykt tidligere:			
Hvor mange år til sammen har De røykt daglig? 34			År
Hvor mange sigaretter røyker eller røykte De daglig? Gi opp antallet sigaretter daglig ..... 36 (håndrullede + fabrikkframstilte)			Sigaretter
Røyker De noe annet enn sigaretter daglig?			
Sigarer eller serutter/sigarillos? 40			
Pipe? 41			
Dersom De røyker pipe, hvor mange pakker tobakk (50 gram) bruker De i pipen på en uke?			
Gi opp gjennomsnittlig tall på pakker i uken 42			Tobakkspk.

G KAFFE

Hvor mange kopper kaffe drikker De vanligvis hver dag?

Sett kryss i den ruten som passer best.

Drikker ikke kaffe, eller mindre enn en kopp 45	1
1 – 4 kopper	2
5 – 8 kopper	3
9 eller flere kopper	4

Hva slags kaffe drikker De vanligvis hver dag?

	JA	NEI
Kokekaffe 46		
Filterkaffe 47		
Pulverkaffe 48		
Koffeinfri kaffe 49		
Drikker ikke kaffe 50		

H ARBEID

	JA	NEI
Har De i de siste 12 månedene fått arbeidsledighetstrygd? 51		
Er De for tiden sykemeldt, eller får De attføringspenger? 52		
Har De full eller delvis uførepensjon? 53		
Har De vanligvis skiftarbeid eller nattarbeid 54		

Har De i det siste året hatt:

Sett kryss i den ruten som passer best.

For det meste stillesittende arbeid? ..... 55 (f.eks. skrivebordsarb., urmakerarb., montering)	1
Arbeide som krever at De går mye? ..... (f.eks. ekspeditørarb., lett industriarb., undervisn.)	2
Arbeide der De går og løfter mye? ..... (f.eks. postbud, tyngre industriarb., bygningsarb.)	3
Tungt kroppsarbeid? ..... (f.eks. skogsarb., tungt jordbruksarb., tungt bygningarb.)	4

	JA	NEI
Er husmorarbeid hovedyrket Deres? 56		

I ETTERUNDERSØKELSE

	JA	NEI
Har noen i husstanden Deres (utenom Dem selv) vært innkalt til nærmere undersøkelse hos lege etter den siste hjerte-karundersøkelsen? 57		

Dersom denne helseundersøkelsen viser at De bør undersøkes nærmere: Hvilken almenpraktiserende lege ønsker De da å bli henvist til?

Skriv navnet på legen her →

..... 58

Ingen spesiell lege .....

Ikke skriv her

61

Appendix 2

Questionnaire 2, The Tromsø survey 1986-87

pp-2.

45

Tilleggsspørsmål til Helseundersøkelsen i Tromsø 1986-87.

Hjerte-karsykdommene, som Hjerte-karundersøkelsene i 1974 og 1979–80 spesielt tok opp, er en mangeartet sykdomsgruppe med tildels dårlig kjente årsaksforhold. I Tromsø vil vi derfor forsøke å få en mer fullstendig kartlegging av forhold som kan være av betydning for sykdommens forløp, f.eks. kosthold, psykisk press *«stress»*, sosiale forhold og sykdomsforekomst blant slektninger. En slik kartlegging er også viktig for å finne fram til sykdomsskapende forhold for kreftsykdommene, som er en sykdomsgruppe vi også vil prøve å bekjempe i årene som kommer.

Sammen med innkallingen fikk De et spørreskjema som De leverte ved undersøkelsen. Dette spørreskjema kartlegger helseforholdene bedre og inkluderer spørsmål om noen forskjellige sykdommer og fysiske/psykiske plager. Spesielt er det tatt med spørsmål vedrørende svangerskap, fødsel og menstruasjon.

Dessuten er vi interessert i å få oversikt over hvordan folk bruker helsetjenesten, for å få kunnskap om hvordan helsetjenesten kan bedres.

Vi håper De vil være brydd med å fylle ut også dette skjemaet, og sende det tilbake til Tromsø Helseråd i den utleverte konvolutt. Alle opplysninger i forbindelse med Helseundersøkelsen vil bli behandlet strengt konfidensielt. Har De noen kommentarer til undersøkelsen kan De skrive dem i kommentarfeltet på siste side.

Med hilsen

Tromsø Helseråd Fagområdet medisin

HELSETILSTAND

Hvordan er Deres helsetilstand?
Sett kryss i den ruten der «Ja» passer best.

		Ja
Meget dårlig	12	☐ 1
Dårlig		☐ 2
Hverken god eller dårlig, middels		☐ 3
Bra		☐ 4
Utmerket		☐ 5

SYKDOM

Har De, eller har De hatt:
Kryss av «Ja» eller «Nei» for hvert spørsmål.

		Ja	Nei
Hudsykdommen psoriasis	13	☐	☐
Astma	14	☐	☐
Allergisk eksem	15	☐	☐
Høysnue	16	☐	☐
Kronisk bronkitt	17	☐	☐
Sår på magesekken	18	☐	☐
Sår på tolvfingertarmen	19	☐	☐
Blindtarms-operasjon	20	☐	☐
Magesårs-operasjon	21	☐	☐
Leddgikt (kronisk revmatoid artritt)	22	☐	☐
Kreftsykdom	23	☐	☐
Epilepsi (fallesyke)	24	☐	☐
Migrene	25	☐	☐

INFEKSJON

		Antall / Ja	Nei
Hvor mange ganger har De hatt infeksjon slik som forkjølelse, influensa, «ræksjuka» og lignende siste halvår?	26	☐	
Har De hatt slik infeksjon siste 14 dager?	27	☐	☐

SYKDOM HOS FORELDRE OG SØSKEN

Kryss av for de slektningene som har eller har hatt noen av sykdommene:

		Mor	Far	Bror	Søster
Hjerneslag eller hjerneblødning	28	☐	☐	☐	☐
Sukkersyke	32	☐	☐	☐	☐
Leddgikt (revmatoid artritt)	36	☐	☐	☐	☐
Kreft	40	☐	☐	☐	☐
Psoriasis	44	☐	☐	☐	☐
Magesår eller tolvfingertarmsår	48	☐	☐	☐	☐
Astma	52	☐	☐	☐	☐

		Ja	Nei
Kryss av dersom slektningene ikke har eller har hatt noen av disse sykdommene	56	☐	☐

MEDISINER

		Ja	Nei
Har De siste år brukt tabletter, sprøyter eller astmaspray mot astma eller allergi	60	☐	☐

Har De brukt følgende medisiner siste 14 dager?

		Ja	Nei
Smertestillende	61	☐	☐
Febersenkende	62	☐	☐
Eksemsalve	63	☐	☐
Blodtrykksmedisin	64	☐	☐
Hjertemedisin	65	☐	☐
Sovemedisin	66	☐	☐
Nervemedisin	67	☐	☐
Migrenemedisin	68	☐	☐
Medisin mot epilepsi (fallesyke)	69	☐	☐
Annen medisin	70	☐	☐

KONTAKT PGA. EGEN HELSE ELLER SYKDOM

Hvor mange besøk har De hatt siste år på grunn av egen helse eller sykdom?

		Antall besøk
Hos vanlig lege	71	☐
Hos spesialist utenfor sykehuset	72	☐
På legevakta	85	☐
Hos bedriftslege	87	☐
Hos fysioterapeut	89	☐
Hos kiropraktor	81	☐
Hos naturmedisiner (homeopat, soneterapeut o.l.)	83	☐
På sykehusets poliklinikk	85	☐
Antall innleggelser på sykehus siste år	87	☐

KOSTHOLD

Hvor mange brødskiver spiser De vanligvis daglig?
Sett kryss i den ruten der «Ja» passer best

		Ja
Mindre enn 2 skiver	88	☐ 1
2 – 4 skiver		☐ 2
5 – 6 skiver		☐ 3
7 – 12 skiver		☐ 4
13 eller flere skiver		☐ 5

Hva slags melk drikker de vanligvis?
Sett kryss i den ruten der «Ja» passer best.

		Ja
Drikker ikke melk	89	☐ 1
Melk (helmelk), søt, sur		☐ 2
Lettmelk		☐ 3
Skummet melk, søt, sur		☐ 4

Hvor mange glass/kopper melk drikker De vanligvis daglig?

		Ja
Mindre enn ett glass/kopp	90	☐ 1
1 – 2 glass/kopper		☐ 2
3 – 4 glass/kopper		☐ 3
5 eller flere glass/kopper		☐ 4

FISKEMAT

Hvor ofte spiser De torsk/sei eller annen mager fisk til middag eller som pålegg?
Sett kryss i den ruten der «Ja» passer best.

		Ja
Sjeldnere enn en gang i uken	91	☐ 1
1 gang i uken		☐ 2
2 ganger i uken		☐ 3
3 eller flere ganger i uken		☐ 4

Hvor ofte spiser De fet fisk som sild, kveite, uer, makrell, laks, ørret til middag eller som pålegg?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjeldnere enn en gang i uken	92	☐ 1
1 gang i uken		☐ 2
2 ganger i uken		☐ 3
3 eller flere ganger i uken		☐ 4

Bruker De tran regelmessig?
Sett kryss i ruten der «Ja» passer best.

		Ja
Nei	93	☐ 1
I mørketida		☐ 2
Hele året		☐ 3

FROKOST

		Ja	Nei
Spiser De vanligvis frokost daglig?	94	☐	☐

MIDDAGSMAT

Hvor ofte spiser De vanligvis kjøtt til middagen?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjeldnere enn en gang i uken	95	☐ 1
1 – 2 ganger i uken		☐ 2
3 – 4 ganger i uken		☐ 3
5 eller flere ganger i uken		☐ 4

Hvor ofte bruker De fett (smør, margarin, remulade, majones og lignende) til eller på middagsmaten?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjeldnere enn en gang i uken	96	☐ 1
1 – 2 ganger i uken		☐ 2
3 – 4 ganger i uken		☐ 3
5 eller flere ganger i uken		☐ 4

		Ja	Nei
Bruker De vanligvis grønnsaker som del av middagsmaten?	97	☐	☐

FRUKT

Hvor ofte spiser De vanligvis frukt?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjeldnere enn en gang i uken	98	☐ 1
Omtrent en gang i uken		☐ 2
2 – 3 ganger i uken		☐ 3
4 – 5 eller flere ganger i uken		☐ 4
Omtrent daglig		☐ 5

ALKOHOL

		Ja	Nei
Er De total avholdsmann/-kvinne		☐	☐

Hvis nei,
– Hvor ofte pleier De å drikke øl?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri, eller noen få ganger i året	100	☐ 1
1 – 2 ganger i måneden		☐ 2
Omtrent 1 gang i uken		☐ 3
2 – 3 ganger i uken		☐ 4
Omtrent hver dag		☐ 5

Hvor ofte pleier De å drikke vin?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri, eller noen få ganger i året	101	☐ 1
1 – 2 ganger i måneden		☐ 2
Omtrent 1 gang i uken		☐ 3
2 – 3 ganger i uken		☐ 4
Omtrent hver dag		☐ 5

– Hvor ofte pleier De å drikke brennevin?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri, eller noen få ganger i året	102	☐ 1
1 – 2 ganger i måneden		☐ 2
Omtrent 1 gang i uken		☐ 3
2 – 3 ganger i uken		☐ 4
Omtrent hver dag		☐ 5

Omtrent hvor ofte har De i løpet av siste år drukket alkohol tilsvarende minst 5 halvflasker øl, en helflaske vin eller ¼ flaske brennevin?
Sett kryss i ruten der «Ja» passer best.

		Ja
Ikke siste år	103	☐ 1
Noen få ganger		☐ 2
1 – 2 ganger i måneden		☐ 3
3 eller flere ganger i uken		☐ 4

FYSISK AKTIVITET

Hvor ofte utfører De fysisk aktivitet av minst 20 minutters varighet og som fører til at De blir svett eller andpusten?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjelden eller aldri	104	☐ 1
Ukentlig		☐ 2
Flere ganger i uka		☐ 3
Daglig		☐ 4

Dersom De vanligvis utfører slik aktivitet minst en gang i uka, hvor mye tid bruker De ukentlig til slik aktivitet?
Sett kryss i ruten der «Ja» passer best.

		Ja
Mindre enn 30 minutter i uka	105	☐ 1
Mellom 30 minutter og 1 time i uka		☐ 2
Mellom 1 og 2 timer i uka		☐ 3
Mer enn 2 timer i uka		☐ 4

VANE- OG KOSTENDRINGER

Har De endret Deres vaner/kosthold i løpet av de siste 5 år når det gjelder:(Sett kryss for hvert spørsmål)

		Bruker nå		
		mer	som før	mindre
Fett i kosten	106	☐	☐	☐
Soyamargarin eller matoljer	107	☐	☐	☐
Skummet melk eller lettmelk	108	☐	☐	☐
Kaffe-forbruk	109	☐	☐	☐
Alkohol-forbruk	110	☐	☐	☐
Fysisk aktivitet	111	☐	☐	☐

EKTESKAPS-/SAMBO-FORHOLD

		Ja	Nei
Er De gift eller samboende	112	☐	☐

Hvor gammel var De da De første gang giftet Dem eller innledet et samboerforhold? ... 113 ☐ år

HUSSTAND

		Antall
Hvor mange personer bor det i deres husstand?	115	☐

		Ja	Nei
Er noen i Deres husstand 10 år eller yngre?	117	☐	☐
Trenger noen i Deres husstand spesielt tilsyn/pleie – *utenom barna?*	118	☐	☐

SKOLEGANG

Hvor mange års skolegang har De (ta også med folkeskole og ungdomsskole)? ... 119 ☐ år

ARBEID

Har De hatt lønnet arbeid hele siste år?
Sett kryss i ruten der «Ja» passer best.

		Ja
Fulltidsarbeid	121	☐ 1
Deltidsarbeid		☐ 2
Ikke lønnet arbeid		☐ 3

Hvor stor del av det daglige arbeid i hjemmet gjør De vanligvis selv?
Sett kryss i ruten der «Ja» passer best.

Alt eller nesten alt	122	☐ 1
Minst halvparten		☐ 2
Mer enn en fjerdedel		☐ 3
Mindre enn en fjerdedel		☐ 4

RYGG- OG LEDDPLAGER

		Ja	Nei
Har De i løpet av siste år vært plaget av smerter i ryggen som har vart lenger enn 4 uker?	123	☐	☐
Hvis ja, bedrer ryggsmertene seg dersom De beveger Dem?	124	☐	☐
Har De vært plaget av stivhet i ryggen om morgenen som varte lenger enn 30 minutter?	125	☐	☐

Har De i løpet av siste 3 år vært plaget av smerter i noen av de følgende ledd i mer enn 3 måneder?

		Ja	Nei
Kneleddene	126	☐	☐
Albueleddene	127	☐	☐
De innerste fingerleddene	128	☐	☐
Andre ledd	129	☐	☐

		Ja	Nei
Hvis ja, merket De stivhet i leddene om morgenen av mer enn 30 minutters varighet	130	☐	☐

PLAGER I HODE, NAKKE OG SKULDRE

Hvor ofte er De plaget av hodepine?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjelden eller aldri	131	☐ 1
En eller flere ganger i måneden		☐ 2
En eller flere ganger i uken		☐ 3
Daglig		☐ 4

Hvor ofte er De plaget av smerter i nakke eller skuldre?
Sett kryss i ruten der «Ja» passer best.

		Ja
Sjelden eller aldri	132	☐ 1
En eller flere ganger i måneden		☐ 2
En eller flere ganger i uken		☐ 3
Daglig		☐ 4

Reduserer plagene i hodet, nakken eller skuldrene Deres arbeidsevne?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri, eller i ubetydelig grad	133	☐ 1
I noen grad		☐ 2
I betydelig grad		☐ 3
Klarer ikke vanlig arbeid		☐ 4

		Ja	Nei
Har De noen gang fått røntgenundersøkt ryggen, nakken og/eller skuldre	134	☐	☐

SØVNLØSHET/BEVISSTLØSHET

		Ja	Nei
Hender det at De er plaget av søvnløshet	135	☐	☐

Hvis ja, når på året er De mest plaget?
Sett kryss i ruten der «Ja» passer best.

		Ja
Ingen spesiell tid	136	☐ 1
Særlig mørketiden		☐ 2
Særlig i midnattsoltiden		☐ 3
Særlig høst og vår		☐ 4

		Ja	Nei
Har De gjennom hele siste år vært plaget av søvnighet slik at det går ut over arbeidsevnen?	137	☐	☐
Har De siste år hatt anfall med plutselig tap av bevissthet?	138	☐	☐
Har De merket anfall med plutselig endring i pulsen eller hjerterytmen siste år	139	☐	☐

REAKSJONER PÅ PROBLEMER

Hvis De får store personlige problemer, regner De da med å få hjelp og støtte fra ektefelle, samboer eller familie? ..... 140 — Ja ☐ Nei ☐

Har De i lengere tid følt behov for å oppsøke noen på grunn av personlige problem siste år, uten at De har tatt slik kontakt? ..... 141 — Ja ☐ Nei ☐

Har De i de siste 14 dager følt Dem ute av stand til å takle Deres vanskeligheter?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri eller sjelden	142	☐ 1
Av og til		☐ 2
Ofte		☐ 3
Nesten hele tida		☐ 4

Har De i de siste 14 dager følt Dem ulykkelig og nedtrykt (deprimert)?
Sett kryss i ruten der «Ja» passer best.

		Ja
Aldri eller sjelden	143	☐ 1
Av og til		☐ 2
Ofte		☐ 3
Nesten hele tida		☐ 4

Hender det ofte at De føler Dem ensom?
Sett kryss i ruten der «Ja» passer best.

		Ja
Meget ofte	144	☐ 1
Av og til		☐ 2
Aldri eller nesten aldri		☐ 3

RESTEN AV SKJEMAET BESVARES BARE AV KVINNER

MENSTRUASJON

Hvor gammel var De da De fikk menstruasjon første gang? ..... 145 — ☐ år

Når begynte Deres siste menstruasjon? .. 147 — dag / mnd. / år

Hvor mange dager er/var det vanligvis fra menstruasjonens 1. blødningsdag til neste menstruasjons 1. blødningsdag (= tiden mellom to menstruasjoners begynnelse)? . 153 — ☐ dager

Pleier/pleide menstruasjonen å være regelmessig ..... 155 — Ja ☐ Nei ☐

Bruker De vanligvis smertestillende tabletter under menstruasjonen? ..... 156 — Ja ☐ Nei ☐

PLAGER FØR MENSTRUASJON

Har De før menstruasjon noen av disse plagene:

– Er De nedtrykt (deprimert) eller irritabel?
Sett kryss i ruten der «Ja» passer best.

		Ja
Ubetydelig	157	☐ 1
Merkbart		☐ 2
Plagsomt		☐ 3

– Har De smertefulle bryst?
Sett kryss i ruten der «Ja» passer best.

		Ja
Ubetydelig	158	☐ 1
Merkbart		☐ 2
Plagsomt		☐ 3

– Har De hovne hender/føtter, vektøkning, eller følelse av å «ese ut»?
Sett kryss i ruten der «Ja» passer best.

		Ja
Ubetydelig	159	☐ 1
Merkbart		☐ 2
Plagsomt		☐ 3

Forsvinner plagene når menstruasjonen kommer? ..... 160 — Ja ☐ Nei ☐

Bruker De mot slike plager:
– vanndrivende tabletter? ..... 161 — Ja ☐ Nei ☐
– andre medisiner? ..... 162 — ☐ ☐

SVANGERSKAP

Hvor mange barn har De født? ..... 163 — Antall ☐

Hvor gammel var De første gang De var gravid? ..... 164 — ☐ år

PREVENSJON

Bruker eller har De brukt P-piller eller spiral? ..... 166 — Ja ☐ Nei ☐

Hvis ja, hvor mange år har De tilsammen brukt:
P-piller? ..... 167 — ☐ år
Spiral? ..... 169 — ☐ år

Hvor gammel var De da De begynte med:
P-piller? ..... 171 — ☐ år
Spiral? ..... 173 — ☐ år

Hvis De har sluttet med P-piller, uteble da menstruasjonen i mer enn 6 måneder uten at De var gravid? ..... 175 — Ja ☐ Nei ☐

Har de måttet slutte med P-piller fordi De fikk høyt blodtrykk? ..... 176 — Ja ☐ Nei ☐

KREFTPRØVE

Hvor mange ganger har De fått tatt kreftprøve (celleprøve) fra livmorhalsen siste 3 år? ... 177 — Antall prøver ☐

Hvor mange år siden er det siden siste prøve? ..... 178 — ☐ år

Deres kommentarer: 179

Takk for hjelpen! Husk å postlegge skjemaet idag!

Tromsøundersøkelsen 1986-7

Appendix 3

Questionnaire, The ECG monitoring study

Navnelapp

1. MEDISINER

Ja Nei

Bruker du noen faste medisiner? . . ☐☐
Eventuelt hvilke:

_____

_____

2. BESVIMELSE

Ja Nei

Har du siste år hatt anfall med plutselig og fullstendig tap av bevissthet? . . ☐☐
Hvis "Ja": Vennligst svar på de neste spørsmålene:

Omtrent hvor mange slike anfall har du hatt siste år? . . . . . Antall ☐

Hvor lenge varte bevisstløsheten ved det lengste anfallet?
Kryss av ett alternativ:

Under 1 minutt . . . . . ☐
1-5 minutter . . . . . ☐
Over 5 minutter . . . . . ☐

Hadde du følelse av hjertebank eller uregelmessig puls før eller etter anfallet? . . . . . Ja Nei ☐☐

I hvilken kroppstilling kom anfallet?
Kryss av ett alternativ:

Liggende eller sittende. . . . . ☐
Stående . . . . . ☐
Idet du reiste deg . . . . . ☐

Var anfallet ledsaget av følgende symptomer:
Kryss av for hvert symptom.

Ja Nei

Tung pust . . . . . ☐☐
Brystsmerter . . . . . ☐☐
Angst . . . . . ☐☐
Kvalme . . . . . ☐☐
Svette . . . . . ☐☐
Ufrivillig vannlating . . . . . ☐☐

3. PULSENDRING

Ja Nei

Har du hatt anfall med plutselig endring i pulsen eller hjerterytmen siste år? . . . . . ☐☐

Hvis "Ja": Vennligst svar på de neste spørsmålene:

Omtrent hvor mange slike anfall har du hatt siste år? . . . . . Antall ☐

Omtrent hvor rask var pulsen?
Kryss av ett alternativ:

Under 50 . . . . . ☐
50-100 . . . . . ☐
100-150 . . . . . ☐
150 og mer . . . . . ☐

Hvordan var hjerteslagene?
Kryss av for hvert spørsmål.

Ja Nei

Raskere enn normalt . . . . . ☐☐
Langsommere enn normalt . . . . . ☐☐
Uregelmessige . . . . . ☐☐
Normale men harde . . . . . ☐☐
Normale med ekstraslag i form av at hjertet gjør et ekstra hopp eller tar en pause . . . . . ☐☐

Var anfallet ledsaget av følgende symptomer?
Kryss av for hvert symptom.

Ja Nei

Uvelhet, kvalme . . . . . ☐☐
Svimmelhet . . . . . ☐☐
Kraftig vannlating . . . . . ☐☐
Tung pust . . . . . ☐☐
Ubehag i brystet, men ikke smerter . . . . . ☐☐
Brystsmerter . . . . . ☐☐
Besvimelse . . . . . ☐☐
Angst . . . . . ☐☐
Svette . . . . . ☐☐
Stikninger rundt munn og/eller i hender . . . . . ☐☐

Spesielle forhold før anfallet.
Kryss av for hvert spørsmål.

Ja Nei

Sterk fysisk anstrengelse . . . . . ☐☐
Drukket mye kaffe . . . . . ☐☐
Røkt mye . . . . . ☐☐
Drukket mye alkohol . . . . . ☐☐
Sovet lite . . . . . ☐☐
Var nedtrykt/deprimert . . . . . ☐☐

Kommentarer (bruk baksiden ved behov):

_____

_____

_____

Takk for hjelpen. Ta med skjemaet på kontrollen!

75 %

margin 10

Navnelapp

1. MEDISINER

Ja Nei

Bruker du noen faste medisiner? . . ☐☐

Eventuelt hvilke:

_____

_____

2. BESVIMELSE

Ja Nei

Har du siste år hatt anfall med plutselig og fullstendig tap av bevissthet? . . ☐☐

Hvis "Ja": Vennligst svar på de neste spørsmålene:

Omtrent hvor mange slike anfall har du hatt siste år? . . . . . Antall ☐

Hvor lenge varte bevisstløsheten ved det lengste anfallet?
Kryss av ett alternativ:

- *Under 1 minutt* . . . . . ☐
- *1-5 minutter* . . . . . ☐
- *Over 5 minutter* . . . . . ☐

Hadde du følelse av hjertebank eller uregelmessig puls før eller etter anfallet? . . . . . Ja Nei ☐☐

I hvilken kroppstilling kom anfallet?
Kryss av ett alternativ:

- *Liggende eller sittende* . . . . . ☐
- *Stående* . . . . . ☐
- *Idet du reiste deg* . . . . . ☐

Var anfallet ledsaget av følgende symptomer:
Kryss av for hvert symptom.

Ja Nei

- *Tung pust* . . . . . ☐☐
- *Brystsmerter* . . . . . ☐☐
- *Angst* . . . . . ☐☐
- *Kvalme* . . . . . ☐☐
- *Svette* . . . . . ☐☐
- *Ufrivillig vannlating* . . . . . ☐☐

3. PULSENDRING

Ja Nei

Har du hatt anfall med plutselig endring i pulsen eller hjerterytmen siste år? . . . . . ☐☐

Hvis "Ja": Vennligst svar på de neste spørsmålene:

Omtrent hvor mange slike anfall har du hatt siste år? . . . . . Antall ☐

Omtrent hvor rask var pulsen?
Kryss av ett alternativ:

- *Under 50* . . . . . ☐
- *50-100* . . . . . ☐
- *100-150* . . . . . ☐
- *150 og mer* . . . . . ☐

Hvordan var hjerteslagene?
Kryss av for hvert spørsmål.

Ja Nei

- *Raskere enn normalt* . . . . . ☐☐
- *Langsommere enn normalt* . . . . . ☐☐
- *Uregelmessige* . . . . . ☐☐
- *Normale men harde* . . . . . ☐☐
- *Normale med ekstraslag i form av at hjertet gjør et ekstra hopp eller tar en pause* . . . . . ☐☐

Var anfallet ledsaget av følgende symptomer?
Kryss av for hvert symptom.

Ja Nei

- *Uvelhet, kvalme* . . . . . ☐☐
- *Svimmelhet* . . . . . ☐☐
- *Kraftig vannlating* . . . . . ☐☐
- *Tung pust* . . . . . ☐☐
- *Ubehag i brystet, men ikke smerter* . . . . . ☐☐
- *Brystsmerter* . . . . . ☐☐
- *Besvimelse* . . . . . ☐☐
- *Angst* . . . . . ☐☐
- *Svette* . . . . . ☐☐
- *Stikninger rundt munn og/eller i hender* . . . . . ☐☐

Spesielle forhold før anfallet.
Kryss av for hvert spørsmål.

Ja Nei

- *Sterk fysisk anstrengelse* . . . . ☐☐
- *Drukket mye kaffe* . . . . . ☐☐
- *Røkt mye* . . . . . ☐☐
- *Drukket mye alkohol* . . . . . ☐☐
- *Sovet lite* . . . . . ☐☐
- *Var nedtrykt/deprimert* . . . . . ☐☐

Kommentarer (bruk baksiden ved behov):

_____

_____

_____

Takk for hjelpen. Ta med skjemaet på kontrollen!

Appendix 4

Statistical methods

Tables 1-15: The exercise test study

Tables 16-21: The ECG monitoring study

Table 22: The Tromsø survey

Statistical methods

In testing risk factor differences according to different variables presented in this appendix (attendance, arrhythmia, ECG etc), analyses of covariance was performed, using the PROC GLM procedure of the SAS statistical package (113). The analyses were performed with attendance, arrhythmia, ECG etc as dependent variables and age (and sometimes sex) as the covariate. The procedure PROC REG was used for the multiple linear regression analysis of peak expiratory flow.

The exercise test study

Table 1. Population size and response rate according to sex and age. Tromsø 1986-7.

	Men		Women	
	Invited	Response	Invited	Response
Age	n	n (%)	n	n (%)
20-29	44	41 (93.2)	59	51 (86.4)
30-39	86	82 (95.4)	115	98 (85.2)
40-49	92	87 (94.6)	107	96 (89.7)
50-59	73	67 (91.8)	69	67 (97.1)
60-61	18	17 (94.4)		
Total	313	294 (93.9)	350	312 (89.1)

Table 2. Causes for non-attendance among 313 men and 350 women. Tromsø 1986-7.

Cause	Men	Women
	n	n
Temporarily not in Tromsø	2	1
Unknown disease	0	1
Did not have time	0	3
Was not interested	2	4
Moved from Tromsø	0	1
Other reasons	1	4
Non-responders to invitation	14	24
Total	19	38

Table 3. Reasons for interruption of exercise test before 250 W*. Tromsø 1986-7.

	Men (n=293)	Women (n=312)
	n	n
Interrupted test	87	293
Angina pectoris	3	0
ST depression	2	0
Dyspnoea	37	99
General fatigue	36	159
Leg pain	28	64
Other reasons	4	6

* More than one reason is possible for each person

Table 4. ECG criteria for abnormality. Tromsø 1986-7.

Resting ECG code	Criteria for abnormality
Myocardial infarction	Abnormal Q wave (width >=0.04 s and/or depth >25 % of the following R wave provided R>=5mm) except in lead aVF, V1-2, III, aVL.
Possible infarction	R wave reduction but abnormal Q waves.
ST segment and T wave changes	Flattening of T wave V3-6, I, II or negative T wave I, II, III, aVF, V2-6 or ST segment depression >=0.5 mm at 0.08 s after J point.
Supraventricular arrhythmia	Extrasystoles, fibrillation, flutter.
Ventricular arrhythmia	Extrasystoles, tachycardia, fibrillation.
Block	SA, AV, bundle branch block
Left ventricular hypertrophy	V1-5 >50 mm + ST segment depression >=0.5 mm + negative T wave V5-6.
Possible left ventricular hypertrophy	V1-5 >=40mm.
Exercise ECG code	**Criteria for abnormality**
Ischaemia	ST segment depression >=1 mm at 0.08 s after J point.
Possible ischaemia	ST segment depression >=0.5 mm-1 mm or great variation in ST segment depression.
ST segment and T wave changes, supraventricular and ventricular arrhythmias and block as for resting ECG.	

Table 5. Coronary risk factors (mean (SD)) in attenders and non-attenders. Tromsø 1986-7.

	Men		Women	
	Attendance		Attendance	
	Yes (n=294)	No (n=19)	Yes (n=312)	No (n=38)
Age (years)	42.0 (10.9)	42.8 (10.9)	40.3 (9.2)	36.6 (9.2)*
Age adjusted risk factors				
Total cholesterol (mmol/l)	5.9 (1.2)	5.9 (1.1)	5.8 (1.3)	6.1 (1.1)
HDL cholesterol (mmol/l)	1.4 (0.4)	1.4 (0.4)	1.7 (0.4)	1.6 (0.4)
Triglycerides (mmol/l)	1.6 (1.0)	1.6 (0.9)	1.1 (0.6)	1.2 (0.6)
Relative weight (kg/m^2)	24.7 (2.7)	23.8 (2.8)	23.1 (3.2)	23.2 (3.1)
Sys. blood pressure (mmHg)	130.9 (15.6)	130.1 (15.2)	120.2 (16.5)	124.2 (15.1)
Dia. blood pressure (mmHg)	77.5 (10.0)	76.5 (9.9)	72.8 (9.3)	74.0 (9.3)
Heart rate (beats/min)	71.8 (12.0)	70.6 (12.0)	76.3 (12.5)	78.6 (12.5)
Daily smoking (%)	44.2	63.1	43.8	59.0
Mod./hard leisure act. (%)	27.6	32.2	8.8	4.4
Moderate/hard work act. (%)	25.9	16.3	18.1	10.0

* $p<0.02$

Table 6. Some variables registered at the survey and exercise test. Tromsø 1986-7.

	Systolic blood pressure (mmHg)		Previous angina pectoris (yes)		Previous myocardial infarction (yes)	
	Men Mean(SD)	Women Mean(SD)	Men N	Women N	Men N	Women N
Survey	130.8 (15.6)	120.2 (16.5)	6	1	8	1
Exercise test	139.4 (16.7)	129.2 (17.3)	8	1	8	1

Table 7. Questionnaire response and electrocardiographic signs at the exercise test for myocardial infarction and angina pectoris. Tromsø 1986-7.

	Men (N=293) N (%)	Women (N=308) N (%)
Myocardial infarction (MI)		
Questionnaire (Q=yes)	8 (2.7)	1 (0.3)
ECG MI and Q=yes	5 (1.7)	0
Angina pectoris (AP)		
Questionnaire (Q=yes)	8 (2.7)	1 (0.3)
AP during test and Q=yes	3 (1.0)	0
AP during test and Q=yes and ECG ischaemia	2 (0.7)	0

Table 8. Electrocardiographic diagnoses*. Tromsø 1986-7.

	Men (n=293) n	Women (n=312) n
Resting ECG		
Normal	264	304
Myocardial infarction	6	0
ST or T wave changes	4	3
Possible myocardial infarction	4	0
Ventricular arrhythmia	3	3
Block	5	1
Left ventricular hypertrophy	2	0
Possible left ventricular hypertrophy	9	1
Exercise ECG		
Normal	254	273
Ischaemia	10	11
Possible ischaemia	11	9
ST or T wave changes	9	10
Ventricular arrhythmia	7	7
Supraventricular arrhythmia	0	1
Block	4	1

* More than one diagnosis is possible for each person

Table 9. Coronary risk factors (mean (SD)) according to resting ECG. Tromsø 1986-7.

	Men Normal resting ECG		Women Normal resting ECG	
	Yes (n=264)	No (n=29)	Yes (n=304)	No (n=8)
Age (years)	41.4 (10.7)	47.6 (10.7)**	40.2 (9.2)	44.4 (9.3)
Age adjusted risk factors				
Total cholesterol (mmol/l)	5.8 (1.1)	6.4 (1.1)**	5.8 (1.1)	6.3 (1.1)*
HDL cholesterol (mmol/l)	1.4 (0.3)	1.2 (0.4)	1.7 (0.4)	1.6 (0.4)
Triglycerides (mmol/l)	1.6 (1.0)	1.6 (1.0)	1.1 (0.5)	1.4 (0.5)
Relative weight (kg/m^2)	24.8 (3.3)	24.7 (2.7)	23.2 (3.5)	23.3 (2.8)
Sys. blood pressure (mmHg)	130.6 (10.6)	132.7 (15.6)	120.2 (15.2)	118.2 (15.2)
Dia. blood pressure (mmHg)	77.5 (10.1)	78.1 (10.1)	73.1 (9.2)	71.5 (9.3)
Heart rate (beats/min)	71.7 (12.0)	71.9 (12.2)	76.3 (12.6)	76.5 (12.5)
Daily smoking (%)	44.8	47.7	44.3	27.2
Mod./hard leisure act. (%)	27.1	32.7	8.9	0
Moderate/hard work act. (%)	25.4	31.2	17.4	38.8
Physical fitness (W)	230.5 (34.5)	207.3 (34.9)***	160.9 (38.4)	166.1 (38.2)
Peak expiratory flow (l/min)	600.3(132.4)	464.1(136.4)#	445.3 (97.1)	391.1(104.0)

* p<0.05, ** p<0.01, *** p<0.001, # p<0.0001

Table 10. Age adjusted resting and exercise (100 W) blood pressure according to ECG findings. Tromsø 1986-7.

	Men Normal resting ECG		Women Normal resting ECG	
	Yes (n=264)	No (n=29)	Yes (n=304)	No (n=8)
Resting blood pressure (mmHg)				
Systolic	138.7 (15.8)	146.2 (16.0)*	129.2 (15.4)	127.2 (15.5)
Diastolic	89.0 (10.7)	94.4 (10.9)*	82.7 (9.8)	80.0 (9.9)
Exercise blood pressure (mmHg)				
Systolic	166.4 (19.3)	169.4 (19.5)	158.8 (19.9)	168.8 (19.5)
Diastolic	94.0 (11.8)	96.6 (12.0)	91.4 (10.8)	95.8 (10.6)
	Normal exercise ECG		Normal exercise ECG	
	Yes (n=254)	No (n=39)	Yes (n=273)	No (n=35)
Resting blood pressure (mmHg)				
Systolic	138.6 (15.8)	144.9 (16.4)*	128.4 (15.4)	135.4 (15.8)*
Diastolic	89.5 (11.0)	89.9 (11.3)	82.4 (9.9)	84.6 (10.2)
Exercise blood pressure (mmHg)				
Systolic	165.9 (19.3)	171.8 (20.0)	157.9 (19.7)	168.2 (20.0)**
Diastolic	94.0 (12.0)	96.1 (12.4)	90.9 (10.7)	96.4 (10.9)**

* p<0.05, ** p<0.01

Table 11. Age adjusted resting and max exercise heart rate according to ECG findings. Tromsø 1986-7.

	Men Normal resting ECG Yes (n=264)	 No (n=29)	Women Normal resting ECG Yes (n=304)	 No (n=8)
Heart rate				
Resting	65.3 (11.7)	67.2 (11.9)	69.3 (12.0)	69.0 (12.1)
Max exercise	168.9 (19.0)	160.5 (19.2)*	171.7 (17.8)	170.0 (17.6)
	Normal exercise ECG Yes (n=254)	 No (n=39)	Normal exercise ECG Yes (n=273)	 No (n=35)
Heart rate				
Resting	65.8 (11.8)	63.1 (12.1)	69.5 (12.1)	67.7 (12.4)
Max exercise	167.9 (19.3)	168.8 (20.0)	171.0 (17.5)	177.1 (18.2)

* $p<0.05$

Table 12. Coronary risk factors (mean (SD)) according to exercise ECG. Tromsø 1986-7.

	Men Normal exercise ECG Yes (n=254)	 No (n=39)	Women Normal exercise ECG Yes (n=273)	 No (n=35)
Age (years)	40.7 (10.2)	50.5 (10.3)**	39.4 (8.9)	47.5 (8.9)**
Age adjusted risk factors				
Total cholesterol (mmol/l)	5.9 (1.1)	6.1 (1.2)	5.8 (1.2)	6.0 (1.1)
HDL cholesterol (mmol/l)	1.4 (0.3)	1.4 (0.4)	1.7 (0.3)	1.7 (0.4)
Triglycerides (mmol/ll)	1.6 (1.0)	1.5 (1.0)	1.1 (0.5)	1.2 (0.5)
Relative weight (kg/m^2)	24.8 (3.2)	24.4 (3.1)	23.2 (3.3)	23.3 (3.0)
Sys. blood pressure (mmHg)	130.0 (15.3)	136.3 (15.9)*	119.6 (15.2)	124.9 (15.7)
Dia. blood pressure (mmHg)	77.3 (10.0)	79.2 (10.4)	72.8 (9.3)	74.4 (9.7)
Heart rate (beats/min)	72.0 (12.1)	70.2 (12.6)	76.5 (12.6)	74.9 (13.0)
Daily smoking (%)	44.2	50.8	46.2	25.4*
Mod./hard leisure act. (%)	28.5	22.4	9.7	1.5
Moderate/hard work act. (%)	25.5	28.6	16.9	22.4
Physical fitness (W)	228.9 (35.2)	223.8 (36.6)	161.1 (38.3)	160.6 (39.5)
Peak expiratory flow (l/min)	593.9(137.9)	554.1(145.13)	449.5 (84.6)	452.5 (87.3)

* $p<0.05$, ** $p<0.0001$

Table 13. Multiple regression analysis of peak expiratory flow with several risk factors as independent variables. Tromsø 1986-7.

	Men (n=280)		Women (n=302)	
Peak expiratory flow mean	599.57		446.09	
R^2	24.9%		15.1%	
Variables	B	p	B	p
Age	-1.954	0.0001	-1.332	0.0023
Cholesterol	6.211	ns	-4.240	ns
HDL cholesterol	-5.317	ns	7.724	ns
Triglycerides	-4.486	ns	1.099	ns
Systolic BP	0.005	ns	0.514	ns
Relative weight	-14.683	ns	7.847	ns
Heart rate	-0.250	ns	-0.209	ns
Physical fitness	17.391	0.0001	12.454	0.0001
Leisure activity	-0.104	ns	-6.502	ns
Smoking	24.741	0.0057	-9.707	ns

Table 14. Coronary risk factors (mean (SD)) according to carotid bruit. Tromsø 1986-7.

	Men Carotid bruit Yes (n=4)	No (n=289)	Women Carotid bruit Yes (n=3)	No (n=309)
Age (years)	46.0 (9.3)	41.9 (10.9)	52.0 (9.3)	40.2 (9.3)*
Age adjusted risk factors				
Total cholesterol (mmol/l)	6.2 (1.1)	5.9 (1.1)	7.2 (1.1)	5.8 (1.1)*
HDL cholesterol (mmol/l)	1.4 (0.4)	1.4 (0.4)	1.6 (0.4)	1.7 (0.4)
Triglycerides (mmol/l)	1.3 (1.0)	1.6 (1.0)	1.4 (0.5)	1.1 (0.5)
Relative weight (kg/m^2)	23.2 (2.8)	24.8 (2.9)	26.7 (2.9)	23.1 (2.8)*
Sys. blood pressure (mmHg)	121.2 (15.4)	131.0 (15.4)	136.3 (15.2)	120.0 (15.1)
Dia. blood pressure (mmHg)	69.2 (10.0)	77.7 (10.0)	78.6 (9.3)	73.0 (9.3)
Heart rate (beats/min)	74.2 (12.1)	72.7 (12.1)	76.3 (12.6)	76.3 (12.5)
Daily smoking (%)	50.0	45.0	66.7	43.8
Mod./hard leisure act. (%)	25.0	27.6	3.2	8.7
Moderate/hard work act. (%)	25.0	25.9	3.2	18.1
Physical fitness (W)	214.3 (35.1)	228.4 (35.0)	160.2 (38.4)	161.0 (38.3)

* $p<0.05$

Table 15. Age adjusted heart rate and blood pressure during fitness study according to carotid bruit. Tromsø 1986-7.

	Men		Women	
	Carotid bruit Yes (n=4)	No (n=289)	Carotid bruit Yes (n=3)	No (n=309)
Heart rate				
Resting	59.5 (11.7)	65.6 (11.7)	66.4 (12.1)	69.3 (12.0)
Max exercise	168.4 (19.1)	168.0 (19.0)	179.1 (17.7)	171.6 (17.6)
Resting blood pressure (mmHg)				
Systolic	148.5 (15.9)	139.3 (15.8)	153.1 (15.3)	128.9 (15.3)**
Diastolic	84.9 (10.8)	89.6 (10.9)	95.8 (9.8)	82.5 (9.7)*
Exercise blood pressure (mmHg)				
Systolic	180.6 (19.0)	166.5 (19.2)	201.2 (19.2)	158.7 (19.3)***
Diastolic	88.4 (11.8)	94.4 (11.9)	110.3 (10.6)	91.3 (10.5)**

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

The ECG monitoring study

Table 16. Population size and response rate according to sex and age. Tromsø 1990-2.

	Men		Women	
	Invited	Response	Invited	Response
Age	n	n (%)	n	n (%)
20-29	5	4 (80.0)	7	5 (71.4)
30-39	11	10 (90.9)	10	8 (80.0)
40-49	28	25 (89.3)	23	21 (91.3)
50-59	16	13 (81.3)	10	10 (100.0)
60-61	10	8 (80.0)		
Total	70	60 (85.7)	50	44 (88.0)

Table 17. Causes for non-attendance in 50 women and 70 men. Tromsø 1990-2.

Cause	Men n	Women n
Unknown disease	1	1
Did not have time	0	1
Was not interested	0	1
Moved from Tromsø	3	0
Dead	2	0
Non-responders to invitation	4	3
Total	10	6

Table 18. Coronary risk factors (mean (SD)) in attenders and non-attenders. Tromsø 1990-2.

Attendance	Yes (n=104)	No (n=16)
Age (years)	48.8 (9.9)	48.4 (9.9)
Men (%)	57.7	62.5
Age/sex adjusted risk factors		
Total cholesterol (mmol/l)	6.2 (1.2)	6.1 (1.2)
HDL cholesterol (mmol/l)	1.4 (0.3)	1.5 (0.3)
Triglycerides (mmol/l)	1.6 (0.9)	1.4 (0.9)
Relative weight (kg/m^2)	25.0 (3.2)	23.9 (3.2)
Sys. blood pressure (mmHg)	125.7 (14.7)	125.4 (14.7)
Dia. blood pressure (mmHg)	77.0 (9.2)	70.6 (9.2)*
Heart rate (beats/min)	73.7 (11.9)	71.5 (12.0)
Daily smoking (%)	47.0	69.4
>5 cups of coffee/day (%)	57.7	74.7
Mod./hard leisure act. (%)	12.7	23.9
Moderate/hard work act. (%)	18.3	24.9
Physical fitness (W)	192.9 (41.8)	178.4 (41.2)
Peak expiratory flow (l/min)	511.7(152.0)	447.6(152.4)

* p = 0.0110

Tabell 19. Self-reported characteristics of the arrhythmic episodes. Tromsø 1990-2.

	Men (n=24) n (%)	Women (n=19) n (%)
Number of paroxysms		
1-10	18 (75)	13 (68)
>10	6 (25)	6 (32)
Heart rate		
<50	2 (8)	4 (21)
50-150	11 (46)	8 (42)
>150	11 (46)	7 (37)
Rhythm		
Faster than usual	14 (58)	12 (63)
Slower than usual	2 (8)	0
Irregular	15 (63)	10 (53)
Normal but hard	7 (29)	5 (26)
Concomitant symptoms		
Nausea or dizzines	16 (67)	6 (32)
Dyspnoe	7 (29)	6 (32)
Heart pain	6 (25)	4 (21)
Micturition	2 (8)	1 (5)
Sweating	6 (25)	6 (32)
Anxiety	7 (29)	3 (16)
Preceding conditions		
Physical activity	4 (17)	4 (21)
Coffee	6 (25)	2 (11)
Smoking	0	1 (5)
Alcohol	2 (8)	0
Insomnia	4 (17)	9 (47)
Depression	2 (8)	6 (32)

Table 20. Coronary risk factors (mean (SD)) according to any arrhythmia. Tromsø 1990-2.

	Any arrhythmia Yes (n=82)	No (n=15)
Age (years)	49.0 (9.9)	48.2 (6.2)
Men (%)	61	40
Age/sex adjusted risk factors		
Total cholesterol (mmol/l)	6.1 (1.2)	6.3 (1.2)
HDL cholesterol (mmol/l)	1.5 (0.4)	1.5 (0.3)
Triglycerides (mmol/l)	1.6 (1.0)	1.6 (1.0)
Relative weight (kg/m^2)	25.3 (3.6)	24.3 (3.5)
Sys. blood pressure (mmHg)	125.0 (14.9)	129.1 (15.0)
Dia. blood pressure (mmHg)	77.2 (9.6)	76.7 (9.7)
Heart rate (beats/min)	73.6 (12.5)	76.6 (12.7)
Max. heart rate (beats/min)	135.9 (22.7)	138.7 (22.9)
Daily smoking (%)	44.2	53.8
>5 cups of coffee/day (%)	61.0	31.9*
Mod./hard leisure act. (%)	10.6	24.3
Moderate/hard work act. (%)	16.1	21.9
Physical fitness (w)	188.8 (39.6)	198.4 (39.6)
Peak expiratory flow (l/min)	503.7(141.7)	542.0(141.8)

* p=0.0408

Table 21. Coronary risk factors according to serious* arrhythmia. Tromsø 1990-2.

	Serious arrhythmia Yes (n=13)	No (n=84)
Age (years)	52.5 (9.8)	48.3 (8.7)
Men (%)	62	57
Age/sex adjusted risk factors		
Total cholesterol (mmol/l)	6.4 (1.2)	6.1 (1.2)
HDL cholesterol (mmol/l)	1.4 (0.3)	1.5 (0.4)
Triglycerides (mmol/l)	1.4 (1.0)	1.6 (1.0)
Relative weight (kg/m^2)	25.9 (3.2)	25.0 (3.6)
Sys. blood pressure (mmHg)	122.4 (15.0)	126.2 (14.9)
Dia. blood pressure (mmHg)	74.0 (9.6)	77.6 (9.5)
Heart rate (beats/min)	71.7 (12.7)	74.5 (12.5)
Max. heart rate (beats/min)	142.0 (22.8)	135.4 (22.6)
Daily smoking (%)	45.4	45.8
>5 cups of coffee/day (%)	71.2	54.0
Mod./hard leisure act. (%)	15.1	12.4
Moderate/hard work act. (%)	15.8	17.2
Physical fitness (w)	174.6 (39.1)	192.9 (39.2)
Peak expiratory flow (l/min)	379.0(137.4)	529.3(131.7)**

* Serious arrhythmia includes complex premature ventricular beats (n=9) and >100 ventricular premature beats (n=4)
** p=0.0004

Table 22. Levels of physical activity in leisure time and intensity of physical activity according to sex, and age-adjusted means of some coronary risk factors. Tromsø 1986-7.

	Number		Cholesterol (mmol/l)		HDL cholesterol (mmol/l)		Smoking (%)	
	Men	Women	Men	Women	Men	Women	Men	Women
Physical activity level in leisure								
Sedentary	2387	2425	5.98	5.69	1.34	1.62	60	56
Walking	5167	6736	5.89	5.58	1.35	1.64	48	44
Exercises	2372	939	5.72	5.42	1.39	1.68	37	38
Hard training	457	109	5.47	5.40	1.43	1.70	21	18
p value for trend			0.000	0.000	0.000	0.000	0.000	0.000
Physical activity of 20 minutes' duration								
Never	3516	4351	5.94	5.68	1.34	1.62	56	52
Weekly	3080	3149	5.84	5.50	1.34	1.65	45	41
Several days/week	2175	1313	5.71	5.46	1.41	1.69	32	31
Daily	592	349	5.74	5.72	1.42	1.66	42	47
p value for trend			0.000	0.000	0.000	0.000	0.000	0.000

Paper I

The Tromsø Study: Associations between Self-reported Arrhythmia, Psychological conditions, and Lifestyle

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Løchen M-L. The Tromsø Study: Associations between self-reported arrhythmia, psychological conditions, and lifestyle. Scand J Prim Health Care 1991; 9: 265–70

In a survey, 19222 males and females aged 12–64 answered a question about sudden changes of heart rate or rhythm during the preceding year. The prevalence of reported arrhythmia was 12.4% in males and 17.2% in females.

Multiple logistic regression was performed with self-reported arrhythmia as the dependent variable and psychological, lifestyle, and coronary risk factors as independent variables. The highest odds ratio for reported arrhythmia concerned poor compared with excellent health status; 3.86 in males and 2.98 in females. The relative risk for reported arrhythmia according to frequency of physician consultations was 2.28 in males and 1.70 in females, and odds ratios in both sexes were significant for psychological problems and smoking.

The findings suggest that attention should be paid to the psychological conditions and lifestyle of patients who report irregular heartbeats. Self-reported arrhythmia may be a minor problem from a clinical point of view, but we still do not know its prognostic implications. Further work is necessary to determine the predictive strength of selfreported arrhythmia for morbidity and mortality.

Key words: arrhythmia, epidemiology, palpitations

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Sensations of irregular heartbeats or palpitations are quite common symptoms (1). The majority of those with irregular heartbeats who consult the general practitioner or the specialized health care system probably suffer from benign conditions such as sinus tachycardia, strong and hard heartbeats or nervous hypersensitivity. A small proportion of those who complain probably have true arrhythmias such as ventricular ectopic beats, conduction disease, and paroxysmal atrial fibrillation. These conditions may be associated with increased morbidity and mortality (2).

The present study presents the prevalence of self-reported arrhythmia in a population, and describes its associations with some psychological conditions, lifestyle, and coronary risk factors.

MATERIALS AND METHODS

In 1986–87, 28847 subjects were invited to ta
in a health survey in the municipality of T
Norway. This survey included all males bor
1925 to 1966, all females born from 1930 t
and a sample of those born from 1967 t
Among the invited subjects, 49 were dead, 1
moved out of the area, and 583 were temp
living outside Tromsø during the screening
giving 26615 as the eligible population. 218
jects attended the screening (179 without invi
giving an attendance rate of 81.3% of the
population.

Enclosed with the letter of invitation was
tionnaire covering among others physical ac

Age	Whole population				No known disease				Known	
	N of subjects		% reported arrhythmia		N of subjects		% reported arrhythmia		N of subjects		% repo arrhyth
	M	F	M	F	M	F	M	F	M	F	M
12–19	455	503	3.3	4.6	454	503	3.3	4.6	1	0	0
20–29	2164	2535	9.4	12.3	2146	2524	9.4	12.3	18	11	11.1
30–39	2954	3183	12.2	18.1	2910	3146	12.0	17.9	44	37	29.5
40–49	2300	2260	13.3	21.9	2183	2180	12.8	21.0	117	80	23.9
50–59	1520	1037	16.9	22.2	1254	927	13.4	20.4	266	110	33.5
60–64	311		18.0		224		13.8		87		28.7
Total	9704	9518	12.4	17.2	9171	9280	11.4	16.6	533	238	29.5
p-value for linear trend			0.0000	0.0000			0.0000	0.0000			NS

leisure time, smoking habits, and coffee consumption. The participants were asked to fill in the questionnaire at home and bring it to the examination. This study was to a large extent a copy of the previous population studies in Tromsø, and the methods and details have been described earlier (3, 4).

A second questionnaire to be filled in at home and returned by mail was distributed to those who attended the screening. This included questions about previous diseases, symptoms, psychological and social conditions. Altogether 9704 men and 9518 women completed this questionnaire. The question on "arrhythmia" was: "Have you observed sudden changes in your heart rate or heart rhythm during the preceding year? Yes/No". The analyses were done for the whole population, and separately for those who answered "yes" (n = 771) and "no" (n = 18451) to questions about previous myocardial infarction, angina pectoris, stroke, diabetes mellitus, or antihypertensive treatment, i.e. present and previously known disease, because these diseases probably have a higher prevalence of serious arrhythmias.

Statistical methods.

Differences in self-reported arrhythmia between the age groups were tested for linear trend by using analysis of covariance.

The age-adjusted prevalences of self-reported arrhythmia according to some selected variables from the multivariate analysis were performed by one-way analysis of covariance for each sex separately. The analyses were done with self-reported arrhythmia as the dependent variable, the psychologi lifestyle variables as the grouping variable, year age groups as the covariate in order to for age.

Multiple logistic regression analysis (5) w formed for each sex separately with self-re arrhythmia as dependent variable, and the fol independent variables: age, total cholesterol, cholesterol, triglycerides, systolic blood pr body mass index, resting heart rate, daily s (yes/no), leisure time physical activity (grade 1 to 4 with increasing activity), number of coffee per day (< 1, 1–4, 5–8, > = 9), use of (yes/no), frequency of alcohol intoxication (n few times/year, 1–2 times/month, >= 3 month), use of analgesics, asthma medicine a chotropic drugs during previous two weeks (y frequency of consulting a physician or "alte medicine" during previous year, general sub state of health (very poor, poor, medium, excellent), insomnia (yes, no), mental dep and problems with coping, both during previo weeks (both coded: never, sometimes, often, always), need for contact with someone bec personal problems without having had such (yes/no).

Only significant ($p < 0.05$) independent va in the logistic regression analysis for at least the sexes were kept in the final analysis. The of the analysis are displayed as odd ratios (O 95% confidence intervals of self-reported a mia. The estimated ORs are adjusted for the ence of the other variables in Table III.

	N of subjects*		Self-reported arrhythmia			
	Males	Females	Males		Females	
			%	p	%	p
Insomnia				0.000		0.000
No	6785	5570	9		13	
Yes	2760	3805	19		23	
Mental depression				0.000		0.000
Never	6405	5618	10		13	
Sometimes	1595	2803	18		22	
Often	211	477	31		32	
Almost always	72	128	29		36	
Contact problems				0.000		0.000
No	8795	8108	11		15	
Yes	687	1180	29		32	
Health status				0.000		0.000
Very poor	18	13	33		54	
Poor	246	221	38		47	
Medium	1484	1483	24		33	
Good	5021	4949	11		16	
Excellent	2784	2683	5		7	
Use of analgesics				0.000		0.000
No	7639	5965	11		13	
Yes	1236	2683	18		25	
Intoxication by alcohol				0.000		0.000
Never	1355	3585	12		17	
Few times/year	3966	3383	11		17	
1–2 times/month	3131	1058	12		16	
>=3 times/month	280	41	26		34	
Consumption of coffee				0.012		0.000
<1 cup	1218	1428	9		12	
1–4 cups	3114	3909	11		16	
5–8 cups	3886	3330	12		19	
>=9 cups	1485	848	15		24	
Daily smoking				0.000		0.000
No	5349	5288	10		15	
Yes	4354	4230	15		20	

* Fluctuations reflect missing answers.

Statistical analyses were performed with the SPSSX program (6).

RESULTS

Table I shows the age and sex specific prevalence of self-reported changes of heart rate or rhythm in the whole population, and according to whether the subjects reported disease or not. Arrhythmia was reported by 17.2% of the females and 12.4% of the males in the whole population, which gives a female/male ratio of 1.4. The prevalence in healthy subjects was about the same, whereas reported arrhythmia was more common in subjects with present and previously known disease; 39.1% in females and in males (sex ratio 1.3). The prevalence o reported arrhythmia increased with age in males ($p < 0.0001$) and females ($p < 0.001$) w known disease. In subjects with known previo ease, no age trend was observed in either sex. ever, the number of subjects with known disea rather small.

Table II shows the age-adjusted prevalence reported arrhythmia in the whole population by some psychological and lifestyle variabl lected from the multivariate analysis. Coffe sumption was also included, even though it contribute significantly in the regression anal

psychological and social conditions.

Variable	Males		Females	
	OR	(95% CI)	OR	(95% CI
Age (10 years)	1.20	(1.14–1.27)	1.28	(1.21–1.3
Insomnia (Yes/no)	1.54	(1.34–1.76)	1.37	(1.22–1.5
Depression (always/never)	1.59	(1.19–2.12)	1.41	(1.07–1.8
Coping problems (always/never)	1.54	(1.07–2.23)	1.65	(1.19–2.2
Contact problems (Yes/no)	1.77	(1.44–2.18)	1.56	(1.32–1.8
Health status (Poor/excellent)	3.86	(3.01–4.95)	2.98	(2.37–3.7
Use of analgesics (Yes/no)	1.17	(0.98–1.39)	1.51	(1.34–1.7
Smoking (Yes/no)	1.27	(1.11–1.44)	1.24	(1.11–1.3
Intoxicated by alcohol (>=3 times per month/never)	1.28	(1.05–1.56)	1.16	(0.96–1.4
Physician consultations (10 times per year/never)	2.28	(1.72–3.02)	1.70	(1.34–1.7
Alternative med. consultations (10 times per year/never)	1.13	(0.71–1.78)	1.51	(1.18–1.9

In both sexes, self-reported arrhythmia was strongly associated with sleep disturbance, mental depression, and contact problems. The prevalence of reported arrhythmia in subjects suffering from insomnia was about double that in those not suffering from insomnia. Similar associations were observed for depression and contact problems.

A positive relationship between reported arrhythmia and a "poor" or "very poor" self-rated state of health was observed in both sexes, but the relationship was more prominent in females than in males. Twice as many females as males used analgesics, and reported arrhythmia was positively associated with the drug use. In both sexes the prevalence of arrhythmia increased with rising frequency of intoxication by alcohol. The same was true for the association between reported arrhythmia and daily smoking and coffee consumption. The positive association with coffee consumption was stronger in females than in males.

In Table III the odds ratios and 95% confidence intervals of self-reported arrhythmia according to lifestyle, psychological conditions and social conditions in the whole population are shown.

In both sexes, the highest OR for self-reported arrhythmia concerned poor compared with excellent health status; 3.86 and 2.98 in males and females, respectively. The relative risk for reported arrhythmia according to frequency of physician consultations was higher in males than in females; 2.28 versus 1.70, while the opposite pattern was true for "alternative medicine" consultations; 1.51 in females and 1.13 (non-significant) in males. The relative risk of reported arrhythmia according to contact problems was 1.77 in males and 1.56 in females, relative risk according to coping problems in males and 1.65 in females. Males had a 1.59 for depression and 1.54 for insomnia, c with estimates of 1.41 and 1.37 among fema ORs of self-reported arrhythmia accordin other independent variables were either qui significant in one sex only.

In healthy subjects (data not shown), t ratios of self-reported arrhythmia were a same as in the whole population. In subj known disease (data not shown), self-rep rhythmia was significantly related to a p jective state of health in both sexes, in ad intoxication by alcohol in males.

DISCUSSION

This study supports the clinical impression sensation of irregular heartbeats is a comm rience both in the healthy and in the non population. The prevalence is quite compar another study that reported a frequency of males and 21.6% in females (1). It is no what proportion of the self-reported arrhyth jects has at some stage consulted a doctor b arrhythmia. But the strong association symptoms of arrhythmia and physician cons in both sexes, and even "alternative medici sultations in females, indicates that the quite frequently apply for professional help of their aches, pains and worries which po clude irregular heartbeats.

This survey included only one question

reported arrhythmia. No information was available on the pulse rate or irregularity of the heart activity during the paroxysm, or on concomitant symptoms such as dyspnoea, chest discomfort, polyuria, dizziness, faintness, or syncope. Although the method may seem crude, the study gives the opportunity to assess the magnitude of the problem of selfreported arrhythmia and to evaluate a number of possible determinants.

In subjects with known disease, the highest OR of selfreported arrhythmia was for the state of health. Since the other variables explored (except intoxication by alcohol in males) did not show any significant relation to reported arrhythmia, they will not be discussed further.

A major finding in the study was the strong and consistent association between self-reported arrhythmia and psychological factors (insomnia, depression, coping and contact problems). This may be interpreted in more than one way. First, a true arrhythmia may cause anxiety. Second, psychological problems may be "somatized" as increased sensitivity to the normal activity of the heart. Although the latter mechanism may seem more plausible, both may be operative in the population. In addition, distressed people may be more likely to report sensations of arrhythmia because of excessive health concern.

That self-reported arrhythmia was more prevalent in females is in accordance with other studies indicating that females in general experience more symptoms than males, in particular symptoms related to tension and depression (7).

The associations between self-reported arrhythmia and coffee consumption, cigarette smoking, intoxication by alcohol, and the use of analgesics may be due either to a harmful influence on the heart rhythm of these factors, or to the use of these stimulants to relieve true arrhythmic symptoms. An alternative interpretation might be that the reporters of arrhythmia represent a group of individuals with a nervous constitution, monitoring themselves closely, and likely to take stimulants and drugs. The association between self-reported arrhythmia and coffee consumption is in accordance with previous reports that found strong relations between caffeine consumption (particularly in the form of coffee), and palpitations (1) and ventricular premature beats (8). Caffeine and nicotine have been shown to increase serum concentrations of adrenaline and noradrenaline, and intracellular levels of cyclic adenosine monophosphate were also increased (9, 10). actions are similar to those produced in a response, so that it is conceiveable that this mechanism responsible for the self-reported arr mia in coffee consumers and cigarette smo There is also considerable certainty about the r alcohol in precipitating arrhythmia. Substantia sumption of alcohol may induce ventricular pr ture beats (11).

In the present study self-reported arrhythmi not related to the major risk factors for cor heart disease, except smoking. The strong po association between reported arrhythmia and rette smoking is not surprising because smoking affect heart rate and precipitate arrhythmia. the association may be of some importance in of the known relationship between smoking and den cardiac death (12).

Among the reporters of arrhythmia there is likely a vast majority of benign conditions a minority of serious arrhythmias with malign pr sis. Self-reported arrhythmia may be of mino portance from a clinical point of view, but we s not know the prognostic implications of the tom. Whether inclusion of some questions rhythmia in surveys may improve the predicti those at high risk of premature cardiovascular bidity and mortality, remains to be seen. low-up study of the individuals in this study provide more insight into the predictive streng selfreported arrhythmia.

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Paper II

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Arrhythmias in subjects with and without a history of palpitations: the Tromsø study

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KEY WORDS: Ambulatory electrocardiography, arrhythmia, epidemiology, palpitations, questionnaire.

The study looked at palpitations in relation to the prevalence of arrhythmia, as assessed by 24-h ambulatory electrocardiography (ECG) in a population sample. The subjects were randomly drawn from among those involved in a cardiovascular survey. Forty-three of those who answered 'Yes' and 54 of those who answered 'No' (84% of those eligible) to the following question, participated: 'Have you observed sudden changes in your heart rate or heart rhythm during the preceding year?' In both groups mean age was 49 years and 58% were men.

There was no relationship between recorded arrhythmia and perceived palpitations during monitoring. The prevalence of at least one arrhythmic episode (ventricular or supraventricular arrhythmia or pauses >=1·5 s) was significantly higher in those who had perceived palpitations during the previous year (98%) than in those who had not (74%) (P<0·0014). Through a simple question about palpitations during the preceding year we were able to identify significantly a population with true arrhythmias. However, the question could not be used to define a population entirely without arrhythmia. The high prevalence of arrhythmia in subjects without reported palpitations indicates that it is a normal finding which alone should not demand further clinical investigations.

Introduction

Palpitations may be a terrifying event for the patient and pose diagnostic problems for the physician. Ambulatory electrocardiography (ECG) has been widely used for assessing the prevalence of arrhythmias both in patients with symptoms[1,2], and in apparently healthy subjects[3–6]. To our knowledge, however, subjects with and without palpitations have not previously been compared in a population-based study.

In a previous paper we studied the prevalence of reported palpitations during the preceding year in a large general population screened for cardiovascular disease. The prevalence was 12% in males and 17% in females[7]. We found an association between palpitations and psychological factors, but a lack of relationship with the major coronary risk factors except smoking. In a random sample of the screenees we performed 24 h ambulatory ECG monitoring in subjects with and without reported palpitations. The aim was to examine the relationship between ECG-confirmed arrhythmia and the feeling of palpitations during monitoring and the year preceding the monitoring.

Methods

CARDIOVASCULAR SCREENING

In 1986–87, all men born from 1925 to 1966 and all women born from 1930 to 1966 in the municipality of Tromsø, Northern Norway, were invited to a screening. The methods used are detailed elsewhere[8,9], and only a short description will be given. The design of this study closely resembles previous studies performed in Tromsø[10,11].

A total of 28 847 subjects were invited; 26 615 were eligible, and 21 826 attended (81·3% of the eligible population).

The screening was comprised of a questionnaire, which included the following question on perception of arrhythmias (later called palpitations): 'Have you observed sudden changes in your heart rate or heart rhythm during the preceding year? Yes/No'. The questionnaire was returned by 19 915 subjects (91·2% of the participants), of whom 19 222 completed this question.

ECG MONITORING STUDY

Fifty-eight subjects who answered 'Yes' and 62 who answered 'No' to the question on palpitations were randomly selected for further study. Among the invited subjects, two had died and three had left the municipality, leaving 115 people as the eligible population. Of these, 104 attended the study. Speed artifacts during tape recording occurred in seven subjects; their ECGs could not be analysed, and this left 97 persons (84% of the original sample) for the analysis.

Enclosed with the letter of invitation was a questionnaire on various aspects of self-reported palpitations. The questionnaire was checked for inconsistencies at the examination by an experienced clinician, and a medical history, with special emphasis on the aetiology

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Table 1 Characteristics of monitored participants according to the presence of reported palpitations during the year preceding the study

	Palpitations in previous years	
	Yes	No
Monitored subjects		
Total (% men)	43 (55·8)	54 (59·3)
Age		
Mean (SD)	48·8 (8·7)	49·3 (10·0)
Range	29–65	39–67
Clinical evaluation		
Supraventricular tachycardia, n (%)	5 (11·6)	0
Ventricular premature beats, n (%)	22 (51·2)	0
ECG monitoring		
Hours monitored, mean (SD)	21·3 (4·4)	21·4 (4·8)
Heart rate (beats . min^{-1}), mean (SD)	73·9 (11·5)	73·5 (13·0)

and type of reported sensations of arrhythmia, was taken in order to obtain a clinical diagnosis.

Standard 12-lead ECG was taken, and a three-lead 24 h ambulatory ECG was recorded by a standard recorder (Tracker, Reynolds Medical). The subjects were instructed to follow daily activities, and were requested to write down perceived arrhythmic events during the recording, specifying timing, duration and concomitant symptoms. The tapes were scanned by means of a Pathfinder III (Reynolds Medical) high speed arrhythmia analyser. Each tape was scanned at high speed, and thereafter reviewed at a slower speed to detect all abnormalities. One of us (W.Z.), who had no knowledge of the group allocation, spent approximately 3 h analysing each tape. Prints of all arrhythmias, extremes of heart rate and artifacts were produced for detailed analysis and reviewed by two of us (M.L., W.Z.).

STATISTICAL ANALYSES

The statistical analyses were performed using the chi-square test and t-test procedures available in the SAS statistical package[12]. Concordance of reported palpitations at the screening in 1986–87 and at the study in 1990–92 was evaluated with Kappa statistic and McNemar's test.

Results

SUBJECT CHARACTERISTICS

Table 1 shows that there were no significant differences between those with and without reported palpitations during the year before the ECG monitoring with regard to age, sex, hours monitored, and heart rate. All subjects had normal resting ECGs, except three in each group whose ECGs indicated previous myocardial infarction. The clinical evaluation of those with palpitations during the year before the examination revealed suspected ventricular premature beats in 22 subjects and paroxysmal supraventricular tachycardia in five subjects. The remaining 16 subjects, who reported palpitations during the previous year, were diagnosed as having no arrhythmia, functional palpitations or sinus tachycardia at clinical evaluation. The prevalence of ventricular arrhythmia was not significantly different between the diagnostic groups (data not shown).

Table 2 Arrhythmias (ventricular, supraventricular premature beats or pauses >=1·5 s) during 24 h ECG according to palpitations reported simultaneously during monitoring

	Palpitations during monitoring		
	Yes	No	Total
Arrhythmias in 24 h ECG			
Yes	0	82	82
No	5	10	15
Total	5	92	97

Table 3 Arrhythmias (ventricular, supraventricular premature beats or pauses >=1·5 s) during 24 h ECG according to the presence of reported palpitations during the year preceding the study

	Palpitations in previous year		
	Yes	No	Total
Arrhythmias in 24 h ECG			
Yes	42	40	82
No	1	14	15
Total	43	54	97

P=0·0014
Sensitivity: 51%
Specificity: 93%
Positive predictive value of palpitations: 98%
Negative predictive value of no palpitations: 26%.

PALPITATIONS AND ARRHYTHMIA DURING MONITORING

Five subjects from the 'Yes' group complained of palpitations in the course of the recording, but no arrhythmia was objectively demonstrated at the same time (Table 2). Conversely, no subjects with recorded arrhythmias felt palpitations at the time when the arrhythmias were noted.

HISTORY OF PALPITATIONS AND ARRHYTHMIA DURING MONITORING

The prevalence of at least one arrhythmic episode (ventricular premature beats, supraventricular premature beats or pauses >=1·5 s) during monitoring was significantly higher in the 'Yes' group (98%) than in the 'No' group (74%) (P<0·0014) (Table 3). An age stratified analysis revealed the same trend for those 50 years and older as for those below 50 years.

The sensitivity of self-reported palpitations was 51%, indicating that among those with recorded arrhythmia,

Table 4 Ventricular arrhythmias observed in monitored subjects

	Palpitations in previous year	
	Yes (n=43) n (%)	No (n=54) n (%)
Ventricular premature beats		
0	12 (27·9)	22 (40·7)
1	7 (16·3)	7 (13·0)
2–9	12 (27·9)	12 (22·2)
10–49	5 (11·6)	6 (11·1)
50–99	1 (2·3)	3 (5·6)
100+	6 (14·0)	4 (7·4)
P=0·19 for difference in number of premature beats		
Complex premature ventricular beats*		
Couplets	2 (4·7)	2 (3·7)
Bigeminy	2 (4·7)	1 (1·9)
Multifocal	1 (2·3)	0
Ventricular tachycardia	1 (2·3)	0
Total	6 (14·0)	3 (5·6)
P=0·10 for difference in number of complex beats		

*Each subject is only counted once and classified by the most complex category for this subject.

only 51% reported palpitations during the preceding year. The specificity of the symptom was 93%, indicating that among those without recorded arrhythmia, all subjects except one reported no palpitations. The positive predictive value of reporting palpitations during the previous years was 98%, whereas the negative predictive value was only 26%.

Table 4 shows that there was no significant association between reported palpitations and ventricular arrhythmia during monitoring. No significant age trend with regard to ventricular arrhythmia was found (data not shown). As displayed in Table 5, no significant association between reported palpitations and supraventricular arrhythmia, or pauses exceeding 1·5 s during monitoring, was found either. The analyses finally included only those who gave a similar response to the question on palpitations, both at the initial screening and at the subsequent examination (Yes=34 and No=42), but the results were similar.

AGREEMENT BETWEEN REPORTED PALPITATIONS AT INITIAL SCREENING AND AT THE SUBSEQUENT EXAMINATION

Table 6 shows that 78% of the participants gave the same answer to the question about perceived palpitations on two occasions at least 3 years apart (i.e. related to the year preceding the initial screening and the year preceding this study). A moderately high Kappa was found (0·57). There was no difference between the proportion who shifted from 'Yes' to 'No' and vice versa (P>0·50). An age stratified analysis was also done (data not shown), but the response pattern did not differ.

Table 5 Supraventricular premature beats and pauses observed in monitored subjects

	Palpitations in previous year	
	Yes (n=43) n (%)	No (n=54) n (%)
Supraventricular premature beats		
0	20 (46·5)	33 (61·1)
1–9	17 (39·5)	17 (31·5)
10+	6 (14·0)	4 (7·4)
P=0·15		
Pauses (s)		
0–1·49	24 (55·8)	35 (64·8)
1·50–1·74	13 (30·2)	13 (24·1)
1·75–1·99	5 (11·6)	3 (5·6)
2·0+	1 (2·3)	3 (5·6)
P=0·37		

Discussion

Our results confirm the poor relationship between recorded arrhythmia and perceived palpitations during 24-h ECG monitoring[1,13–15].

This is the first study dealing with the occurrence of arrhythmias, as shown on 24-h ECGs, related to answers to a standardized questionnaire, including a question on palpitations. Previous studies were based on patients and healthy volunteers, such as students or members of a jogging club[3,5,6,16]. The strength of this study is that it originates from a population-based survey with a high attendance rate. The response rate to the question on palpitations was also high, and it is likely that the participants interpreted the question on palpitations in accordance with our intention. We checked all the questionnaires at the clinical examination and found no inconsistencies with regard to this question.

Twenty-four hour ambulatory ECG has a limited ability to identify patients with rare attacks of arrhythmia. The low sensitivity of the question on palpitations is another problem, although it certainly underlines that arrhythmias are very common even in the asymptomatic population. However, the question on palpitations is not a suitable measure of the prevalence of arrhythmia. The high prevalence of arrhythmias, even in subjects without reported palpitations during the previous year (74%), suggests that the question cannot be used to define a

Table 6 Agreement between palpitations reported at the initial screening (1986–87) and in the present study (1990–92)

Palpitations at screening	Palpitations at present study		
	Yes	No	Total
Yes	34	12	46
No	9	42	51
Total	43	54	97

Kappa=0·57.

population without arrhythmia. However, as the high positive predictive value shows (98%), a simple question on palpitations during the preceding year seems to possess a significant ability to identify in a population individuals who really have arrhythmias. In addition, there may be more serious arrhythmias in those with a history of palpitations than in those who reported no palpitations, as suggested by our results. The question may constitute a first step in a process of selecting a population with a serious arrhythmia problem, but the population size should be increased in order to identify rarer attacks. The ability of a positive answer to the question on palpitations to serve as an independent predictor with regard to cardiovascular disease should also be tested.

The rather high agreement observed between the answers to questions on palpitations in the two studies performed 3 years apart indicates consistency in response. The reasons for shifting from one answer to another may either be due to a lapse of memory, or to the new occurrence or else the disappearance of palpitations.

In accordance with several other studies[15,17–21], ventricular premature beats were the rule rather than the exception in our study population, and few subjects had more complex forms of arrhythmia.

The prevalence of supraventricular ectopic beats was lower than in some previous studies[5,17], although other studies, using ambulatory ECG in healthy subjects, have even shown a higher prevalence of supraventricular than ventricular premature beats[3,6]. This discrepancy may partly be due to the fact that our population consisted of somewhat younger and healthier subjects, and partly due to technical differences.

The prevalence of pauses exceeding 1·50 s was similar to the prevalence reported in a previous study[16].

This study gave us an opportunity to validate the clinical evaluation of an experienced clinician, but this did not improve our capability of identifying those with serious arrhythmia. This shows that the question on palpitations may be sufficient in a population where the aim is to distinguish between those with and those without serious arrhythmias. The results indicate that arrhythmias may be judged more objectively in those reporting palpitations on a questionnaire than from a subsequent clinical evaluation. However, the diagnostic misclassification probably did not have serious consequences for those who were diagnosed as having no arrhythmia, functional palpitations or sinus tachycardia at the clinical evaluation. An exception may be one person in this group who had complex premature ventricular beats.

In conclusion, no relationship between recorded arrhythmia and perceived palpitations during 24 h ECG monitoring was observed in this population. Nonetheless, a simple standardized question with regard to sudden changes in heart rate or rhythm the preceding year seems to be suitable for defining a population where almost everybody has arrhythmia. This study lends support to the view that cardiac arrhythmia, irrespective of subjective symptoms, is a normal finding in a population, and alone should not demand further clinical investigations. Elucidation of the predictive power of simple questions on palpitations with regard to cardiovascular disease will require further prospective studies.

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Paper III

The predictive value of single lead electrocardiogram for myocardial infarction in young and middle-aged men. The Tromsø study.

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Abstract

Objective: To assess the risk of developing myocardial infarction according to QRS duration and T wave amplitude.

Design: A cohort follow-up for 3.9 years after a cardiovascular screening which included electrocardiography of lead I.

Setting: Population based survey in Tromsø, Northern Norway in 1986-87.

Subjects: 6628 men without previous myocardial infarction aged 25-61 years.

Main outcome measure: First occurrence of myocardial infarction or sudden death.

Results: During the 25748 person-years of observation, 82 myocardial infarctions (55 non-fatal and 24 fatal myocardial infarctions, and three sudden deaths) were identified. Risk of myocardial infarction increased with QRS duration and with decreasing T wave amplitude. A proportional hazards model with adjustment for possible confounders, yielded a relative risk of myocardial infarction between QRS duration $\geq$ 120 msec compared with QRS < 80 msec of 3.74 (p for linear trend: 0.015). The multivariate relative risk for T wave amplitude $\geq$ 0.70 mV compared with T amplitude < 0.40 mV was 0.55 (p for linear trend: 0.036). When both QRS duration

and T wave amplitude were included in the multivariate model, T wave amplitude retained its predictive power, while QRS duration became marginally non-significant (p=0.067).

Conclusion: T wave amplitude is an independent predictor of myocardial infarction in men without previous myocardial infarction. Increasing QRS duration clearly indicates higher risk of myocardial infarction. However, when T wave amplitude is included as a covariate, the predictive power of QRS becomes non-significant.

Key words:
coronary heart disease, electrocardiography, epidemiology, myocardial infarction, prospective study, sudden cardiac death

Introduction

In population studies, the electrocardiogram (ECG) has usually been interpreted by means of a categorical classification such as the Minnesota Code (1). However, both in routine diagnostics and in epidemiological use of the ECG, computerized methods gives the possibility to interpret the ECG as continuous variables (2,3).

The relation between QRS duration and myocardial infarction has been addressed previously (4-12), but the results were inconsistent.

T wave inversion has been established as a predictor of myocardial infarction (4,8-10,12).

The purpose of this paper is to assess the risk of developing myocardial infarction according to QRS duration and T wave amplitude. The population available was 6628 free living men without previous myocardial infarction.

Materials and methods

In 1986-87 all men born between 1925 and 1966, all women born between 1930 and 1966, and a sample of those born between 1967 and 1974, were invited to be screened for cardiovascular risk factors in the municipality of Tromsø in northern Norway.

Women were not included in this paper due to few

events of myocardial infarction. The analysis is restricted to men above 24 years at the screening (no myocardial infarction below this age). In this age group 12391 men were invited, 11524 were eligible, and 9182 attended the screening (79.7% of the eligible). Due to computer software problems in the first ten weeks of the screening, only 6769 men were available for the analysis. Attendees with self-reported myocardial infarction were excluded from the analysis (n=130). In addition, 11 subjects were found to have had previous myocardial infarction after linkage to the local hospital register. They were excluded, leaving 6628 men to be included in the analysis.

The study was to a large extent a replication of previous population studies in Tromsø (13,14). The screening methods are described elsewhere (15,16), but a short description is given here.

The screening comprised a questionnaire included in the invitation, and measurements of heart rate and blood pressure (Dinamap Vital Signs Monitor 1846, Critikon Inc., Tampa, Fla.), weight, height and collection of a non-fasting blood sample for determination of total cholesterol, HDL cholesterol and triglycerides. The analyses of the blood samples were done at the Department of Clinical Chemistry,

University Hospital of Tromsø.

Electrodes were fixed on the forehead and on both wrists with the subject sitting. After resting, a stable ECG baseline was required before a 10 seconds recording of lead I was made. The data were sampled (125 samples per second with 8 bits' resolution) with a Hewlett-Packard HP78352A Patient Monitor and transmitted to a microcomputer. A tape with the data was sent to and analyzed by the University Department of Medical Cardiology, Glasgow Royal Infirmary (17). The ECG variables were analyzed according to age and prediction of myocardial infarction.

Both the positive (T+) and the negative (T-) T wave amplitudes were measured. The reference level was the QRS onset. We used the sum of the amplitudes as measurement of the T wave. However, we also analyzed the impact of positive, biphasic and negative T waves as categories.

QRS duration and T wave amplitude were included in the multivariate models both as grouped variables (QRS: <80 msec, 80-89 msec, 90-119 msec, 120+ msec. T: <0.40 mV, 0.40-0.69 mV, 0.70+ mV) and as continuous variables.

The ECGs with a QRS duration exceeding 119 msec were classified after manual readings without

knowledge of events status. The following criteria were used: **I.** Left bundle branch block; a broad QRS duration with an inverted T wave, **II.** Right bundle branch block; a broad QRS complex with a broad S wave with notching of the peak, **III.** Intra-ventricular conduction defect; no features of I or II, **IV.** Wolff-Parkinson-White syndrome; as III, but short PR interval and a delta wave.

A subject was a case when the first event of myocardial infarction occurred or in case of sudden death. The participants were followed up until Dec 31st 1990 with respect to the first myocardial infarction through the only hospital in the municipality and the Death Register at the Central Bureau of Statistics. The mean follow-up period was 3.9 years. During the 25748 person-years of observation, 82 myocardial infarctions (55 non-fatal myocardial infarctions, 24 coronary deaths and three sudden deaths) were identified.

Statistical analysis

Age-adjusted incidence rates were calculated by the direct method (18) using the person-years distribution of the entire cohort as standard.

The proportional hazards regression model was used to adjust for other risk factors. The risk factors

were: Age (years), angina pectoris (yes/no), total and HDL cholesterol (mmol/l), cups of coffee drunk each day (<1, 1-4, 5-8, 9+), daily smoking (yes/no), systolic blood pressure (mmHg), antihypertensive treatment (yes/no) and heart rate (beats/min). The analyses were performed using the SAS statistical package (19,20).

Results

The mean QRS duration decreased slightly with age ($p=0.011$), whereas the prevalence of QRS duration $\geq$120 msec increased ($p=0.02$) (table 1).

The mean T wave amplitude decreased with age ($p<0.0001$) (table 2). There was a highly significant positive age trend for both biphasic and negative T wave amplitude ($p<0.0001$).

The age-adjusted incidence rate of myocardial infarction according to QRS duration increased two-three times from the lowest category (<80 msec) to the highest category (120+ msec) (table 3). Table 4 gives the relative risk of myocardial infarction according to QRS duration. The age-adjusted relative risk with the shortest QRS duration as reference, increased to 3.43 with a significant linear trend ($p=0.013$). In a multivariate model the estimate remained almost unchanged. The significant age-

adjusted relative risk of myocardial infarction for bundle branch block compared to QRS less than 80 msec, decreased to 3.98 in a multivariate model and became non-significant.

The age-adjusted incidence rate of myocardial infarction according to T wave amplitude decreased about three times from the lowest category (<0.40 mV) to the highest category (0.70+ mV) (table 5). As presented in table 6, the age-adjusted relative risk of myocardial infarction decreased to 0.44 for the highest T wave amplitude compared to the lowest T wave amplitude (p=0.002). In a multivariate model the relations were somewhat modified, but the trend remained significant (p=0.036). Compared with positive T wave, the biphasic and negative waves showed nonsignificantly increased relative risks.

Table 7 shows multivariate relative risk estimates of myocardial infarction where QRS duration and T wave amplitude were included as continuous variables. Multivariate relative risk of 10 msec difference of QRS duration was 1.15 (p=0.067). For 0.20 mV difference of T wave amplitude the multivariate relative risk was 0.84 (p=0.018).

Discussion

The results indicate that T wave amplitude is an

independent predictor of myocardial infarction in young and middle aged men without previous myocardial infarction. The finding is supported by the presence of a graded and continuous relationship between T wave amplitude and myocardial infarction. Even when T wave amplitude varied within completely "normal" limits, there was a significant change in relative risk of myocardial infarction. The independent association between T wave amplitude and myocardial infarction was retained in all the analyses.

T wave inversions have been reported as significant predictors of myocardial infarction (4,8-10,12). We also found a somewhat higher risk for myocardial infarction given a negative T wave, but in the multivariate analysis negative T wave came out as non-significant. This indicates that it is more important to analyze the T wave amplitude itself than to study different patterns or categories of the T wave.

QRS duration clearly indicates higher risk of myocardial infarction with broader QRS complexes, although the multivariate model where both T wave amplitude and QRS duration were included yielded QRS duration as a non-significant predictor. This may be due to the distribution of QRS duration with

relatively few subjects with broad QRS complexes and therefore relatively few cases of myocardial infarction in this part of the distribution. Previous papers dealing with the predictive value of QRS duration for myocardial infarction have not adjusted for other ECG variables in the multivariate analyses (4-12).

Some studies have suggested that for subjects without known coronary heart disease, prolongation of QRS duration was not particularly risky (4-6,10-12), while others found an association between QRS width and coronary heart disease (8,9). Our findings indicate that changes of QRS duration within the normal range have prognostic importance. However, the largest step-up in the event rate was found with the emergence of what is commonly regarded as abnormal QRS duration, $\geq$ 120 msec.

The choice of lead I alone for ECG registration was made to facilitate the screening. The QRS and T measurements were based on an average PQRST cycle. This is in contrast to the Minnesota coding (1) which is based on a combination of pattern recognition and measurements according to the majority rule. This means that a coded item has to be present in the majority (but not necessarily all) of the beats in the lead. The Minnesota code thus

intends to reduce the effect of beat to beat variability, while our method utilises all QRS complexes and T waves from the 10 second recording. The Minnesota code is a syntactic classification system (21). It extracts various feature patterns measured and uses a "grammar" to categorize the patterns into a set of classes. There are no Minnesota codes for QRS durations and T wave amplitudes alone. The codes also include special requirements with regard to other amplitudes, durations, ratios or axis in the ECG complex. ECG characteristics treated according to the Minnesota code may not give sufficient information as to their predictive value, because it may be difficult to find a cut-off point under or above which a certain ECG variable is normal or pathological. Treating an ECG measurement as a continuous variable may give a better estimate of its contribution to risk.

In accordance with previous findings, both QRS duration and T wave amplitude decreased with age, but the QRS duration was longer and the T amplitude was higher than previously reported (21). The difference in QRS width may be ascribed to slight differences in computing procedure and equipment. The sitting position during ECG recording and higher body height may partly explain the higher mean T

amplitude in our subjects.

The prevalence of QRS ≥120 msec was somewhat lower than reported in other studies (22-24). One reason may be that we excluded men with previous myocardial infarction which decreases the prevalence of QRS prolongation.

The prevalence of diphasic and negative T waves in the present study compares well with a previous Scandinavian study (8), although others report a considerably lower prevalence (12,22). The discrepancies probably partly reflect coronary risk factor variations between the populations.

The QRS duration increases usually when the ventricular mass or size increases. The increase is due to blocking of specific parts of the conduction system or diffusely decreased conduction. These conditions have been shown to be arrhythmogenic (25), and may account for some of the increased risk associated with QRS duration.

T wave amplitude is a function of the repolarization of the ventricles, and varies with a number of factors including axis change, recording position, weight and height. It is also well established that repolarization is severely influenced by hypertrophy and myocardial ischemia. These factors may be important in relation to the

present findings.

In summary, T wave amplitude is an independent predictor of myocardial infarction in men without previous myocardial infarction. Increasing QRS duration clearly indicates higher risk of myocardial infarction. However, when T wave amplitude is accounted for in the multivariate model, the predictive power of QRS becomes non-significant.

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Table 1.

QRS duration (msec) and prevalence of QRS ≥120 msec according to age.

Age (years)	N	Mean (SD)	QRS ≥120 msec n (%)
25-34	2183	83.8 (13.2)	13 (0.6)
35-44	2221	83.5 (13.5)	11 (0.5)
45-54	1415	83.0 (13.6)	10 (0.7)
55+	809	82.4 (15.2)	11 (1.4)
Total	6628	83.4 (13.6)	45 (0.7)
p values linear trend		0.011	0.020

Table 2.

T amplitude (mV) and prevalence of diphasic and negative T wave according to age.

Age (years)	N	Mean (SD)	Diphasic n	Diphasic (%)	Negative n	Negative (%)
25-34	2183	0.59 (0.31)	139	(6.4)	19	(0.9)
35-44	2221	0.56 (0.30)	206	(9.3)	12	(0.5)
45-54	1415	0.49 (0.28)	187	(13.2)	12	(0.9)
55+	809	0.41 (0.30)	179	(22.1)	22	(2.7)
Total	6628	0.54 (0.30)	711	(10.7)	65	(1.0)
p values linear trend						
		<0.0001		<0.0001		<0.0001

Table 3.

Incidence rate of myocardial infarction according to QRS duration.

QRS	Person-years	CHD n	Incidence rate per 1000 person-years: Crude	Incidence rate per 1000 person-years: Age-adjusted
Duration (msec)				
<80	10145	26	2.56	2.42
80-89	7280	20	2.75	2.86
90-119	8151	34	4.17	3.85
120+	172	2	11.63	6.31
QRS ≥120 msec				
IVCD + WPW*	103	0		
Block**	69	2	28.99	9.28

* IVCD = intraventricular conduction defect (n=25), WPW = Wolff-Parkinson-White (n=1)

** Block = both left (n=5) and right bundle branch block (n=14)

Table 4.

Relative risk of myocardial infarction according to QRS duration.

QRS	Relative risk (95% CI)	
	Age-adjusted	Multivariate* adjusted
Duration (msec)		
<80	1.00 (ref)	1.00 (ref)
80-89	1.19 (0.67-2.13)	1.26 (0.70-2.26)
90-119	1.79 (1.08-2.99)	1.76 (1.05-2.95)
120+	3.43 (0.81-14.47)	3.74 (0.88-15.86)
p values linear trend		
	0.013	0.015
QRS ≥120 msec		
Block**	4.88 (1.19-19.98)	3.98 (0.97-16.31)

* Relative risk estimated by proportional hazards regression with age group, angina pectoris, total- and HDL cholesterol, coffee consumption, systolic blood pressure, antihypertensive treatment, smoking and heart rate as additional covariates

** Block = both left (n=5) and right bundle branch block (n=14)

Table 5.

Incidence rate of myocardial infarction according to T wave amplitude and T wave pattern.

T wave	Person-years	CHD n	Incidence rate per 1000 person-years: Crude	Incidence rate per 1000 person-years: Age-adjusted
Amplitude (mV)				
<0.40	8021	43	5.36	4.41
0.40-0.69	10800	32	2.96	3.02
0.70+	6927	7	1.01	1.39
T wave pattern				
Positive	22759	62	2.72	2.89
Diphasic *	2742	18	6.56	4.59
Negative	248	2	8.06	3.14

* Includes four subjects with flat T wave

Table 6.

Relative risk of myocardial infarction according to T wave amplitude and T wave pattern.

T wave	Relative risk (95% CI)	
	Age-adjusted	Multivariate** adjusted
Amplitude (mV)		
<0.40	1.00 (ref)	1.00 (ref)
0.40-0.69	0.69 (0.44-1.10)	0.78 (0.49-1.24)
0.70+	0.44 (0.20-1.01)	0.55 (0.24-1.26)
p values linear trend		
	0.002	0.036
T wave pattern		
Positive	1.00 (ref)	1.00 (ref)
Diphasic *	1.51 (0.89-2.58)	1.20 (0.70-2.06)
Negative	1.67 (0.41-6.86)	1.24 (0.29-5.27)

* Includes four subjects with flat T wave

** Relative risk estimated by proportional hazards regression with age group, angina pectoris, total- and HDL cholesterol, coffee consumption, systolic blood pressure, antihypertensive treatment, smoking and heart rate as additional covariates

Table 7.

Relative risk of myocardial infarction according to QRS duration and T wave amplitude as continuous variables.

Variable	Relative risk (95% CI)	
	Age-adjusted	Multivariate* adjusted
QRS (10 msec)	1.16 (1.00-1.35)	1.15 (0.99-1.32)
T (0.20 mV)	0.78 (0.68-0.89)	0.84 (0.73-0.97)

* Relative risk estimated by proportional hazards regression with age group, angina pectoris, total- and HDL cholesterol, coffee consumption, systolic blood pressure, antihypertensive treatment, smoking, heart rate and T (for QRS) or QRS (for T) as additional covariates

Paper IV

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The Tromsø study: the prevalence of exercise-induced silent myocardial ischaemia and relation to risk factors for coronary heart disease in an apparently healthy population

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KEY WORDS: Coronary risk factors, exercise test, silent ischaemia, systolic blood pressure.

A random population sample of 294 men and 312 women aged 20–64 years, all apparently healthy, were examined following a screening to determine the prevalence of silent ischaemia and its relations to coronary risk factors. Based on exercise testing, the prevalence of silent ischaemia was 2·5% in men and 3·4% in women. In men, silent ischaemia was positively related to systolic blood pressure ($P<0·001$). The other risk factors did not show any significant associations with silent ischaemia. However, the men with silent ischaemia had a higher coronary heart disease risk score, and a tendency towards more symptoms and signs suggesting a poorer health status than the other men and the women. In the 21 men classified as 'hypertensives', silent ischaemia was more common than in the normotensive men (14% versus 2%, $P<0·001$). No such difference was observed in women. In conclusion, silent ischaemia may be a sign of hypertension and a generally increased risk of coronary heart disease in men, but probably not in the majority of women. This further supports that exercise electrocardiography has no role in screening asymptomatic persons for coronary heart disease, probably with the exception of middle-aged men with an increased risk.

Introduction

The current interest in preventive health care has initiated an increase in screening for coronary heart disease, including exercise testing of both symptomatic and asymptomatic subjects. Controversy still exists as to how a positive exercise test should be approached clinically in asymptomatic subjects.

The prevalence of asymptomatic ST segment depression (silent ischaemia) during exercise testing has been estimated to about 2–4% of the middle-aged male population[1,2]. However, studies that have assessed the prevalence of silent ischaemia in unselected populations, and in females, are scarce. In addition, a better understanding of the associations between silent ischaemia and coronary risk factors may be of relevance for selecting the appropriate clinical approach.

The present study reports the prevalence of exercise-induced silent ischaemia in a population of men and women with no known history of coronary heart disease. The relations between silent ischaemia and risk factors for coronary heart disease are shown, and the possible consequences for risk of coronary heart disease are discussed. The study is unique because the prevalence of silent ischaemia was assessed in a random population sample, and because both sexes were included.

Materials and methods

GENERAL SURVEY

In 1986–87, 28 847 subjects were invited to take part in a health survey in the municipality of Tromsø. This included all men born between 1925 and 1966, and all women born between 1930 and 1966. Among the invited subjects were 49 who had died; 1600 had moved and 583 were temporarily living outside Tromsø during the screening period, leaving 26 615 as the eligible population. Some 21 826 subjects attended the screening (179 without invitation), giving an attendance rate of 81·3% of the eligible population. Enclosed with the letter of invitation was a questionnaire which covered (amongst other things) the following aspects: previous cardiovascular disease, any symptoms suggestive of angina pectoris and smoking habits. The participants were asked to fill in the questionnaire at home and return it at the examination. This questionnaire was almost identical to those used in the previous studies in Tromsø[3,4]. The examination comprised checking of the questionnaire for inconsistency, measurements of weight, height and blood pressure and the collection of venous non-fasting blood samples for measurement of blood lipids. The laboratory analyses were performed at the Institute of Medical Biology, University of Tromsø.

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SPECIAL SURVEY

The present study constitutes a sub-sample of those attending the screening born between 1925 and 1966. Invitation was sent by mail 2–6 weeks after the initial screening to 316 men and 350 women, who were randomly selected from the screened population. Some 294 men and 312 women attended the survey, giving attendance rates of 93% and 89% in men and women respectively.

The examinations were performed from October 1986 to June 1987. One man and four women were unable to perform the exercise test because of musculoskeletal system disabilities. In the analysis, eight men and

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one woman with previous myocardial infarction were excluded.

A graded sub-maximal or maximal bicycle exercise test with a pedalling frequency of 60 min^{-1} was performed. The initial work load was set at 25 W, with increments of 25 W for every minute up to a maximum of 250 W (i.e. 10 min). Physical fitness was defined as the maximum workload (W) performed. The ECG was monitored on an oscilloscope. The exercise ECG (CH leads, V4, V5 and V6) was recorded on paper at 50 mm . s^{-1} before and immediately after the exercise test with the subject sitting on the bicycle. The exercise test was interrupted prematurely when specific symptoms or signs made this necessary. The ECG was read without knowledge of the person's identity by two physicians. When disagreement occurred about the interpretation of the ECGs, a consensus was reached.

The exercise ECG was defined as ischaemia when there was a depression of at least 1 mm of a horizontal or downsloping ST-segment 80 ms after the J point. The degree of ST-segment depression was measured in mm. Silent ischaemia was defined as an ischaemic response without any cardiac pain.

STATISTICAL ANALYSIS

Age adjustment of the crude rates of silent ischaemia was performed according to the direct method. The men and women respectively, in the whole survey were used as the standard population for each sex. In testing risk factor differences according to silent ischaemia, analysis of covariance was used. Multiple logistic regression analysis was performed for each sex separately with silent ischaemia as the dependent variable. The following were used as independent variables: age, total cholesterol, HDL cholesterol, triglycerides, systolic blood pressure, body mass index, daily smoking, resting heart rate and antihypertensive drug treatment. Regression coefficients between ST depression (mm) and systolic/diastolic blood pressure were estimated using simple linear regression analysis. The prevalence of silent ischaemia was compared in hypertensives and normotensives for each sex separately with analysis of covariance. The SAS (Statistical Analysis System)[5] was used in the statistical analyses.

Results

Table 1 presents the distribution of silent ischaemia according to age and sex. The age-adjusted prevalence of silent ischaemia was 3·4% in women and 2·5% in men. The difference between sexes was not significant. In both sexes, the prevalence of silent ischaemia increased with age, except for a decrease in the oldest age groups. The age trends were, however, not significant. The mean age of men without silent ischaemia was 41·5 years, compared to 51 years for men with silent ischaemia. The corresponding ages in women were 40·1 years and 45·3 years. The age differences between those with and without silent ischaemia were not significant in either sex. Two men in the oldest age group felt cardiac pain which was interpreted as angina pectoris, in addition to ischaemia, during the exercise test. They are excluded from the analyses presented here, but this exclusion did not change the main findings described.

Table 1 Prevalence of silent ischaemia

Age (years)	Men			Women		
	N	n	(%)	N	n	(%)
20–39	123	1	(0·8)	148	2	(1·4)
40–44	50	1	(2·0)	51	2	(3·9)
45–49	34	1	(2·9)	44	2	(4·5)
50–54	37	1	(2·7)	48	5	(10·4)
55–59	24	3	(12·5)	16	0	(—)
60–64	17	1	(5·9)			
Crude rates						
20–59	268	7	(2·6)	307	11	(3·6)
20–64	285	8	(2·8)			
Age-adjusted rates (Mean ± SE)						
20–59	2·5 ± 1·9			3·4 ± 2·0		

Coronary risk factors and some other attributes in subjects with and without silent ischaemia are shown in Table 2. Men with silent ischaemia had a 17·7 mmHg higher systolic blood pressure compared to those with a normal ECG ($P<0·001$), and antihypertensive drug treatment was significantly more prevalent in the men with silent ischaemia (21% versus 4%). There were no other significant differences between subjects with and without silent ischaemia. However, there was a tendency for the men with silent ischaemia to have more clinical symptoms and signs than the other men. The males with silent ischaemia had a higher prevalence of morning cough and morning expectorate, a higher incidence of CHD in close relatives, lower physical fitness and a greater prevalence of low physical activity in leisure time. In addition, the men with silent ischaemia had a higher CHD risk score. The risk score is based on total cholesterol, blood pressure, smoking and sex, and has been used in previous Norwegian population studies[6]. As the distribution of the score was highly positively skewed, the logarithmic transformation was used in the analysis. This tendency almost disappeared in females, and no significant differences were found.

The multiple logistic regression analysis confirmed the univariate analyses (Table 3). The relative risk of silent ischaemia according to systolic blood pressure in men was 1·63 (95% CI 1·08–2·45). This means that the relative risk of silent ischaemia increased 1·63 times per 10 mmHg of increase in systolic blood pressure. The RRs of silent ischaemia according to the other independent variables were not significant.

Table 4 shows that there was a stronger relation between degree of ST segment depression (expressed as mm ST depression) and blood pressure in men ($P=0·05$ for diastolic blood pressure) than in women.

Twenty-nine men were classified as 'hypertensives', which includes 21 men with systolic blood pressure of more than 160 mmHg and/or diastolic blood pressure

Table 2 Age-adjusted coronary risk factors and other selected attributes in subjects with and without silent ischaemia (SI)

Risk factor	Men		Women	
	Normal n = 277	SI n = 8	Normal n = 296	SI n = 11
Cholesterol (mmol . l^{-1})	5·85	6·07	5·82	5·96
HDL cholesterol (mmol . l^{-1})	1·41	1·28	1·68	1·60
Triglycerides (mmol . l^{-1})	1·56	1·52	1·09	1·27
Systolic BP (mmHg)	130·2	147·9†	120·1	120·4
Diastolic BP (mmHg)	77·2	81·5	72·9	74·6
Smoking (%)	44·0	37·0	45·0	21·0
Ex-smoking (%)	28·0	31·0	24·0	26·0
Log CHD risk score	1·25	1·48	0·46	0·54
Body mass index (kg . m^{-2})	2·47	2·58	2·31	2·35
Heart rate (beats . min^{-1})	71·8	70·6	76·4	74·6
Fitness (W)	231·0	217·0	161·0	159·0
Low physical activity (%)	21·0	48·0	26·0	20·0
CHD in close relative (%)	48·0	89·0	47·0	58·0
Antihypertensive treatment (%)	4·0	21·0*	2·0	8·0
Morning cough (%)	12·0	23·0	12·0	8·0
Morning expectorate (%)	9·0	23·0	7·0	8·0

*$P < 0·05$, †$P < 0·001$.

Table 3 Logistic regression coefficients (B) with standard errors of the mean (SEM) of silent ischaemia on coronary risk factors

Risk factor	B (men)	SEM	B (women)	SEM
Cholesterol (1 mmol . l^{-1})	−0·0341	0·32	0·0693	0·31
HDL cholesterol (0·2 mmol . l^{-1})	−1·9957	1·45	−0·4723	0·97
Triglycerides (0·2 mmol . l^{-1})	−0·1312	0·48	0·3141	0·61
Systolic BP (10 mmHg)	0·0541*	0·02	−0·0135	0·02
Body mass index (1 kg . m^{-2})	−0·2826	1·48	−0·3337	1·17
Smoking (yes/no)	−0·6814	0·85	−1·2509	0·83
Heart rate (10 beats . min^{-1})	−0·0092	0·04	−0·0052	0·03
Antihypertensive treatment (yes/no)	0·4168	1·16	1·1845	1·42
Age (10 years)	0·0768	0·05	0·0537	0·05

*$P < 0·02$

Table 4 Regression coefficients (B) between ST depression (mm) and blood pressure (BP)

	B (men)	B (women)
Systolic BP	0·0320	0·0051
Diastolic BP	0·0450*	−0·0002

*$P = 0·05$

Table 5 Prevalence of silent ischaemia in 'hypertensives' (SBP > 160 mmHg and/or DBP > 95 mmHg and/or taking antihypertensive drugs) and normotensives

	Silent ischaemia in hypertensives			Silent ischaemia in normotensives			P
	N	n	(%)	N	n	(%)	
Men	29	4	(13·8)	256	4	(1·6)	< 0·001
Women	19	2	(10·5)	288	9	(3·1)	ns

greater than 95 mmHg, and eight men taking antihypertensive drugs, but with blood pressure below the mentioned limits. Among these men, four (13·8%) had silent ischaemia compared to four (1·6%) from among the 256 normotensive men ($P < 0·001$) (Table 5). Nineteen women were classified as 'hypertensives' (15 had increased blood pressure and four were taking antihypertensive drugs). The difference in silent ischaemia between women with and without hypertension (10·5% and 3·1%) was not significant.

Discussion

This study represents a random population sample. This is in contrast to most other studies of silent

ischaemia which recruited the study participants from among volunteers or clinical or other selected populations.

The study shows a tendency towards more unfavourable coronary risk factors in men with silent ischaemia compared to those without, even though the difference was significant only for systolic blood pressure. The strong positive association between systolic blood pressure and silent ischaemia, but not with other conventional risk factors, has also been observed by others[7,8]. In addition, the positive correlation between degree of ST depression and blood pressure in men is noteworthy, and in accordance with a previous study showing a particularly marked increase in CHD risk in men when ST depression exceeds 2 mm[9]. The lack of similar significant associations in females might be due to random variation. A more likely interpretation is that silent ischaemia represents different conditions in the two sexes. Silent ischaemia may be a sign of hypertension and a generally increased risk of CHD in men, while silent ischaemia in women does not necessarily represent a latent, clinically significant disease. The lower prevalence of smokers among women with compared to women without silent ischaemia, may again support the impression that the women with silent ischaemia were in a good state of health.

In summary, this study indicates that in the absence of other conventional risk factors, diagnosing silent ischaemia offers no apparent benefit to risk assessment in asymptomatic, healthy women. In men, silent ischaemia may represent pathology. The tendency towards more atherogenic risk factors in men with silent ischaemia indicates that the best approach may be to diagnose and modify risk factors that are alterable. However, certain sub-groups such as middle-aged men with two or more risk factors for coronary heart disease, are at risk of presenting initially with myocardial infarction or sudden death[10], and the exercise ECG screening of such persons is probably advised.

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Paper V

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The Tromsø study: physical fitness, self reported physical activity, and their relationship to other coronary risk factors

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Abstract

***Study objective*—The aim was to investigate the associations between physical fitness, leisure physical activity, and coronary risk factors.**

***Design*—This was a cross sectional study of a random sample of men and women, following a population survey.**

***Setting*—The municipality of Tromsø, Norway in 1986–1987.**

***Participants*—All men born 1925–1966 and all women born 1930–1966 were invited to the survey; 21 826 subjects attended (81% of the eligible population): of these, 297 men and 312 women, randomly selected, attended the present study (attendance rates 94% in men and 89% in women).**

***Measurements and main results*—Fitness was tested by bicycle ergometry. Physical activity was reported on a questionnaire. Multiple regression analysis was performed with fitness and leisure activity as dependent variables, and coronary risk factors as independent variables. Fitness and leisure activity were positively related ($p < 0.05$). Prominent findings for fitness were negative associations with age and smoking ($p < 0.05$), and positive associations with body mass index in both sexes ($p < 0.01$). HDL cholesterol and systolic blood pressure were significant predictors of fitness in men ($p < 0.01$). Smoking emerged as a strong negative predictor for leisure activity in women ($p < 0.01$), and a negative relation between leisure activity and total cholesterol was found in men ($p < 0.01$).**

***Conclusions*—The study indicates that coronary risk factors are more closely linked to physical fitness than to leisure physical activity.**

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The epidemiological evidence linking either objectively measured physical fitness or subjective reports on physical activity to risk of coronary heart disease is abundant but inconclusive.[1–11] One unsolved problem concerns to what extent the cardiovascular risk is related to fitness as such or to the accompanying levels of physical activity. Another is whether the physical fitness factor and the level of physical activity act directly or through concomitant variation in other known coronary risk factors.[12–19] Few epidemiological studies involve simultaneous assessments of objectively measured physical capacity as well as subjective reports on habitual physical activity. The present report aims at studying cross sectionally both the physical performance and the self reported physical activity levels in the same population, their intercorrelations and the associations with other coronary heart disease risk factors.

Methods

In 1986–87, 28 847 subjects were invited to take part in a health survey in the municipality of Tromsø. This included all men born between 1925 and 1966, and all women born between 1930 and 1966. Among the invited subjects, 49 were dead, 1600 had moved, and 583 were temporarily living outside Tromsø during the screening period, giving 26 615 as the eligible population; of these, 21 826 subjects attended the screening (179 without invitation), giving an attendance rate of 81·3% of the eligible population. The screening programme started August 1986 and was finished April 1987. Enclosed with the letter of invitation was a questionnaire covering among others the following aspects: previous cardiovascular disease, physical activity at work and in leisure time, and smoking habits. The participants were asked to fill in the questionnaire at home and return it at the examination. This questionnaire was almost identical to those used in the former studies in Tromsø.[20 21] The examination comprised checking the questionnaire for inconsistency, measurements of weight, height, and blood pressure, and the collection of venous non-fasting blood samples for measurements of blood lipids.

Total cholesterol and triglycerides were analysed in fresh serum by enzymatic colorimetric methods with commercial kits (CHOD-PAP, Boehringer Mannheim, for cholesterol, and GPO-PAP, Boehringer Mannheim, for triglycerides). High density lipoprotein (HDL) cholesterol was determined after precipitating serum with $MgCl_2$. The laboratory analyses were performed by the Division of Clinical Chemistry, University Hospital of Tromsø.

In the questionnaire, physical activity in leisure time was graded from I to IV according to which of the following categories could best describe the participants' level of physical activity: Grade I: reading, watching television, or activities which do not need physical activity; Grade II: walking, cycling, or some other form of physical activity for at least 4 h per week; Grade III: exercises to keep fit, heavy gardening, etc, for at least 4 h per week; Grade IV: hard training or participation in competitive sports, regularly and several times a week. If leisure activity varied between summer

and winter, the person was asked to state the average.

Physical activity at work was graded from I to IV according to the following categories: I = mostly sedentary work, II = work leading to much walking, III = work leading to much walking and lifting, IV = heavy manual labour. The questions on physical activity in this study have been widely used in different Scandinavian studies, since they were first described in 1968, and they have been extensively validated.[22–25]

In the analyses, physical activity categories III and IV were merged both in leisure and at work since there were very few subjects in category IV.

The present study constitutes a random subsample of those attending the screening. Invitation was sent by mail 2–6 weeks after the initial screening to 316 men and 350 women who were randomly selected from the screened population; 297 men and 312 women attended the survey, giving attendance rates of 94% and 89% in men and women, respectively.

Four women and one man were unable to perform the exercise test because of disabilities in the musculoskeletal system. One woman and eight men with previous myocardial infarction were included in the study.

The examinations were performed from October 1986 to June 1987. The examination consisted of a history of previous disease, a 12 lead resting electrocardiogram and an exercise test. Physical working capacity was measured with a graded submaximal or maximal bicycle exercise test with a pedalling frequency of 60/min. The initial work load was set at 25 W, with 25 W increments every minute up to a maximum of 250 W (10 minutes). The tests were interrupted prematurely when symptoms such as angina pectoris, dyspnoea, or exhaustion made this necessary. No complications were noted during the exercise tests. Physical fitness was defined as the maximum work load (watts) performed.

Because of missing data, the number of subjects included in the individual analyses varies.

Table I Levels of physical fitness and moderate/heavy physical activity in leisure and at work according to age.

	Men					*Women*				
		Fitness (watts)		*Activity*			*Fitness (watts)*		*Activity*	
Age (years)	*n*	*Mean*	*(SD)*	*Leisure (%)*	*Work (%)*	*n*	*Mean*	*(SD)*	*Leisure (%)*	*Work (%)*
20–9	41	249·4	(3·9)	41·5	51·2	50	169·0	(41·5)	10·0	26·0
30–9	82	243·3	(16·2)	34·1	25·6	98	164·8	(35·7)	12·2	15·3
40–9	87	238·2	(26·1)	24·1	16·1	95	167·1	(38·6)	8·4	15·8
50–9	66	195·1	(57·6)	21·2	21·2	65	140·4	(35·0)	3·1	16·9
60–1	17	182·4	(53·6)	5·9	35·3					
20–59	276	231·1	(38·7)	29·0	25·4	308	161·0	(38·8)	8·8	17·5
20–61	293	228·2	(41·2)	27·6	25·9					

STATISTICAL ANALYSIS

SAS (Statistical Analysis System)[26] was used in the statistical analyses. Two way analysis of covariance were performed to test for linear trend of fitness according to activity at leisure and at work. To determine the age adjusted coronary risk factors, analysis of covariance was used. Age adjusted correlation coefficients were determined for combinations of physical fitness and leisure physical activity with the coronary risk factors. Multiple linear regression analyses were performed to estimate the partial impact of coronary risk factors as independent variables on the dependent variables physical fitness and physical activity at leisure.

Results

SUBJECT CHARACTERISTICS

Fitness and activity levels of the study population are presented in table I. The mean physical fitness in the age group 20–59 years was higher in men (231 W) than in women (161 W) ($p < 0.0001$). In men, fitness generally decreased with age. In women, the trend was not so clear, as there was a tendency for better fitness in the age group 40–49 years than in the younger age group. However, the age trend was signified for each sex separately ($p < 0.0001$).

High and moderate physical activity in leisure time was considerably more common in men (29%) than in women (9%) ($p < 0.001$). For women, there were no significant differences in physical activity in leisure time between the age groups below 50 years. In men, leisure time physical activity in general decreased with age ($p < 0.05$).

At work, women were less physically active than men (18% versus 25%), and the difference between the sexes was significant ($p < 0.01$). Only in men was a significant difference in physical activity at work present between the age groups, the youngest and the oldest being the most active. However, this finding is based upon small numbers in the oldest age group (17 men).

Conventional coronary risk factors as well as their correlations with fitness and leisure activity are shown in table II. The table indicates that a number of significant relations between the fitness level and the risk factors were present. Important observations were the significant associations between fitness level and total cholesterol ($p < 0.05$), triglycerides ($p < 0.05$), heart rate ($p < 0.001$), and smoking ($p < 0.001$) in both sexes. HDL cholesterol and systolic blood pressure (both $p < 0.001$) in men and body mass index in women ($p < 0.01$) were also significantly related to fitness.

Table II Age adjusted means of coronary risk factors and their correlation with physical fitness and physical activity in leisure.

	Men (n = 292)				*Women (n = 308)*			
			Correlation with				*Correlation with*	
	Mean	*(SD)*	*Fitness r*	*Activity r*	*Mean*	*(SD)*	*Fitness r*	*Activity r*
Cholesterol (mmol/litre)	5·90	(1·24)	−0·142*	−0·183†	5·82	(1·32)	−0·143*	−0·033
HDL cholesterol (mmol/litre)	1·40	(0·36)	0·205‡	0·086	1·68	(0·38)	0·065	0·0235
Triglycerides (mmol/litre)	1·57	(0·95)	−0·206‡	−0·070	1·10	(0·56)	−0·146*	0·002
Body mass index (kg/m^2)	2·47	(0·29)	0·050	−0·098	2·32	(0·30)	0·158†	0·066
Systolic blood pressure (mm Hg)	130·9	(15·6)	−0·213‡	−0·021	120·2	(16·5)	−0·104	0·001
Heart rate (beats/min)	71·8	(12·0)	−0·203‡	−0·102	76·3	(12·5)	−0·185‡	−0·169†
Daily smoking (%)	44·2		−0·296‡	−0·146*	43·8		−0·210‡	−0·218†

* $p < 0.05$; † $p < 0.01$; ‡ $p < 0.001$

Only a small number of risk factors were significantly associated with activity score. Smoking habits showed a significant relation to activity in both sexes ($p < 0.05$), in addition to total cholesterol in men and heart rate in women (both $p < 0.01$).

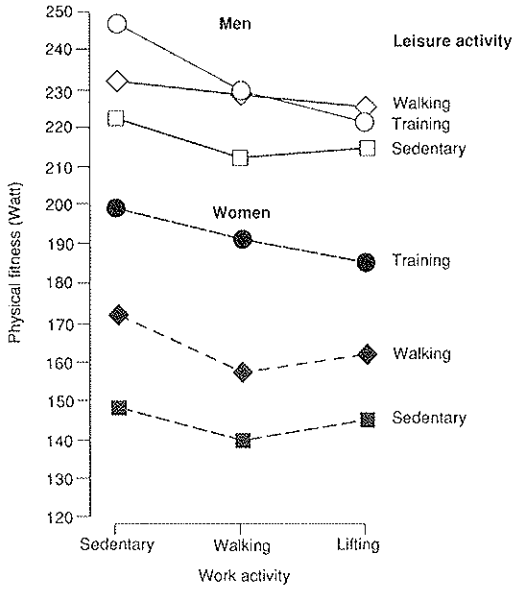
RELATIONSHIPS BETWEEN FITNESS AND ACTIVITY

The age adjusted physical fitness by level of physical activity in leisure time and at work is shown in the figure. For both sexes, fitness increased with activity in leisure time ($p < 0.001$), while it decreased with activity at work ($p < 0.05$).

MULTIPLE REGRESSION ANALYSIS

Tables III and IV give the results of the multiple linear regression analyses with physical fitness and leisure activity, respectively, as the dependent variables. In general, the impression from the univariate analyses was confirmed. For fitness, prominent findings were negative associations with smoking and age and positive associations with physical activity in leisure and at work and body mass index in both sexes. Previous myocardial infarction, HDL cholesterol, and systolic blood pressure were significant predictors of fitness in men. The explained variance varied between 46 and 26% in men and women respectively. Thus a substantial part of the variation in fitness was explained by the other coronary heart disease factors.

For leisure physical activity, smoking and heart rate emerged as having firm negative associations with activity in women. A negative association between physical activity and total cholesterol was observed in men. The explained variance was 12% in men and 16% in women, ie much lower than for fitness.



Age adjusted physical fitness (watts) according to physical activity in leisure time and at work. $p < 0.001$ for linear trend of fitness by leisure activity in both groups; $p < 0.05$ for linear trend of fitness by work activity in both groups.

Table III Multiple regression analysis of physical fitness with several risk factors as independent variables.

	Men (n = 292)		*Women (n = 308)*	
Fitness mean	*228·17*		*161·04*	
R^2	*46·2%*		*25·9%*	
Variables	*B*	*p*	*B*	*p*
Infarction	−44·509	0·0001	−10·135	0·7703
Cholesterol	−1·051	0·5555	−3·462	0·0974
HDL cholesterol	15·344	0·0062	4·175	0·4795
Triglycerides	−3·116	0·1583	−7·904	0·0714
Body mass index	18·973	0·0078	24·268	0·0009
Systolic BP	−0·414	0·0011	−0·193	0·1694
Smoking	−11·909	0·0021	−8·683	0·0384
Heart rate	−0·197	0·2263	−0·242	0·1448
Physical activity				
in leisure	5·336	0·0396	19·428	0·0001
at work	−4·929	0·0104	−5·833	0·0341
Age	−1·916	0·0001	−0·644	0·0157

BP = blood pressure

Table IV Multiple regression analysis of physical activity at leisure with several risk factors as independent variables.

	Men (n = 292)		*Women (n = 308)*	
R^2	*12·1%*		*16·1%*	
Variables	*B*	*P*	*B*	*P*
Infarction	0·463	0·0855	0·268	0·6211
Cholesterol	−0·109	0·0077	0·011	0·7255
HDL cholesterol	−0·058	0·6531	0·003	0·9773
Triglycerides	0·040	0·4264	0·072	0·2976
Body mass index	−0·241	0·1431	−0·081	0·4835
Systolic BP	0·002	0·3994	0·001	0·5678
Smoking	−0·170	0·0574	−0·170	0·0093
Heart rate	−0·004	0·3123	−0·005	0·0367
Physical fitness	0·003	0·0396	0·005	0·0001
Physical activity				
at work	0·071	0·1109	0·014	0·7483
Age	−0·002	0·7096	0·005	0·2435

BP = blood pressure

Discussion

FITNESS-ACTIVITY RELATIONSHIPS

The assessment both of activity and fitness levels has inherent methodological problems. Activity grading is simple to perform in an epidemiological study, but the grading is crude and both over- and underestimation of activity level must occur. The awareness in the population of the importance of activity may lead to biased reporting.

Testing of fitness is more cumbersome, even in the shortened version used in this study. We used 250 W as the maximum load, which was found to be too low in many men. The fitness factor might have turned out to be more important if a true maximum exercise test had been performed in all subjects. However, about 95% of the men exercised to a heart rate exceeding 130 beats/min. Another crucial methodological factor was the choice of bicycle ergometry which measures external work only and gives an advantage to heavy and large subjects.

As expected, highly significant positive correlations were found between fitness and leisure activity (figure). Still, the correlations were rather poor, with age adjusted correlation coefficients of 0·18 in men and 0·39 in women (data not shown). This may indicate that the fitness factor and the activity factor may be regarded as two different dimensions that carry largely independent information, and cannot readily be substituted for each other. This also raises the question regarding which of these factors is most important as a predictor of disease.

The fitness-activity relation was stronger in females than in men, which may partly be explained by the test being truly maximal in most females.

Our results confirm previous studies indicating that leisure time activity is the important physical activity factor in a modern, affluent society.[2 27]

The negative association between activity at work and fitness may partly be explained by the nature of activity at work, which mostly consists of short bursts of activity which does not give rise to sustained heart rate increase or improve aerobic exercise capacity. In addition, physical activity at work may be regarded as a measure of social class rather than as an indication of activity and fitness level.

RISK FACTORS, ACTIVITY AND FITNESS

Most of the individual risk factors were more closely associated with fitness than with activity. When all risk factors were included in the multivariate analysis, they accounted for 26–46% of the variance in objectively measured fitness, while only 12–16% of the variance in self reported activity was explained. This further supports the argument given above that fitness should be regarded as an independent factor which cannot entirely be explained from the reported activity.

The difference between fitness and activity was particularly noteworthy with regard to blood pressure. While no association between blood pressure and activity was observed, blood pressure and fitness were firmly negatively associated in men. Increased exercise capacity may partially be caused by a lower blood pressure. Both in acute experiments[28] and in heart failure[29] it is well established that the impedance to left ventricular ejection is one of the major determinants of stroke volume, and thus physical fitness. It has previously been thought that in the normal individual compensatory factors such as cardiac hypertrophy maintain a constant exercise capacity at high pressure levels. Our present findings may indicate that these compensatory factors are incomplete and that exercise capacity therefore decreases with increasing pressure in normal subjects as well. This result should, however, be interpreted with care, as the relation between fitness and systolic blood pressure was significant in men only, even though women showed the same tendency.

As expected, smoking was negatively related both to fitness and activity. The cause may be twofold; on the one hand smoking may decrease exercise performance; on the other, active persons may be more health concious and thus less likely to smoke.

FITNESS OR ACTIVITY, STRENGTH OR TRAINING?

Activity and fitness are commonly regarded as more or less interchangeable: active people get fit. In this study we found the association to be comparatively weak. It would therefore seem fruitful to recall how activity and fitness are related and how they are not.

One difference between the two is with regard to causative factors. While the activity level is influenced by a host of psychological, social, and environmental factors, fitness may primarily be influenced by constitutional, ie, mostly genetic, factors.[17] Genes linked to fitness may of course also be linked to genes associated with lipids and blood pressure.

In addition to the fact that activity leads to fitness, the opposite may also occur. If you are born with a high physical capacity, you may tend to be more active. The influence on conventional risk factors like lipids, smoking, blood pressure, and obesity is usually thought to take place through the activity factor. Our observation that fitness generally was much more closely linked to the risk factors is therefore remarkable. One explanation may be that fitness was more precisely measured than activity in this study, as discussed above. A more elaborate questionnaire or interview on leisure time physical activity might have given a better estimation and a stronger relation to coronary risk factors than observed. We believe that this is only part of the explanation, and that fitness actually is more closely linked to risk factors. This may be caused by a salutary effect of fitness on risk factors which is not expressed through the activity level. Even more probable is the hypothesis outlined above, that risk factors, in particular smoking and blood pressure, have retroactive effects on fitness, in contrast to activity. Thus fitness may have a bidirectional "benign circle" interrelationship with the risk factors, while the activity effect may be unidirectional.

Conclusion

From this cross sectional study no conclusions can be drawn with regard to the long term effects of activity and fitness on risk of disease or with regard to effect of changes in lifestyle. That must be left to longitudinal studies. Our study confirms the association between fitness and activity, although the strength of the association was weaker than presumed. It also indicates that coronary risk factors are more closely linked to fitness than to activity. The results indicate that fitness should be added as an important variable in addition to activity in future studies on the relationship between exercise habits and disease.

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