which results in accumulation of intracellular fat. The liver fat in Reye's syndrome is presumably caused by a temporary dysfunction in mitochondrial β -oxidation. It seems reasonable to assume that cases of sudden infant death with such fatty changes also have abnormalities of fatty acid β -oxidation, whether congenital or acquired.

Implicit in the currently accepted definition of the sudden infant death syndrome is that necropsy must be thorough to exclude adequate causes of death.²⁰ Our findings indicate that all such investigations must include histological examination of the liver, and those livers showing diffuse panlobular fatty change should be further subjected to thorough histochemical and biochemical analysis of the state of mitochondrial enzymes. Conversely, the diagnosis of Reye's syndrome cannot be confirmed by the characteristic liver fatty infiltration without tests for mitochondrial enzyme activity (table II).

TABLE II—Histochemical and biochemical activity of mitochondrial enzymes in Reye's syndrome, medium chain acyl-coenzyme A dehydrogenase deficiency, and sudden infant death syndrome, showing diffuse fatty change of liver

Enzyme activities	Reye's syndrome	Medium chain acyl-coenzyme A dehydrogenase deficiency	Fatty change in sudden infant death syndrome*
Cytochrome oxidase	↓	Normal	Normal
Succinate dehydrogenase	↓ ↓	Normal	Normal
Glutamate dehydrogenase	↓ ↓	Normal	?
Short chain acyl-coenzyme A dehydrogenase	presumed ↓ ↓	↓ or normal	?
Medium chain acyl-coenzyme A dehydrogenase	presumed ↓ ↓	↓ ↓	?
Long chain acyl-coenzyme A dehydrogenase	presumed ↓ ↓	↓ or normal	?

*Excluding two cases of medium chain acyl-coenzyme A dehydrogenase deficiency.

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Changes in incidence and prognosis of ischaemic heart disease in Finland: a record linkage study of data on death certificates and hospital records for 1972 and 1981

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Abstract

The components of the decline in mortality from ischaemic heart disease in Finland were studied by analysing the changes in incidence and prognosis between 1972 and 1981. Using personal identification numbers, hospital discharge records and death certificates were linked for all men and women aged 40-64. During this period mortality decreased 15.9% in men and 23.5% in women, incidence 14.2% in men and 19.3% in women, being greatest among 40-49 year olds living in urban areas, and case fatality

7.3% in men and 10.3% in women, owing primarily to a decrease in patients dying of ischaemic heart disease without being admitted to hospital; survival was also better among patients admitted to hospital.

Factors explaining these changes remain unknown because data on risk factors and factors influencing prognosis are limited and largely ecological.

Introduction

The epidemic of ischaemic heart disease in industrialised countries has been waning in some countries while waxing in others over the past 10-15 years.¹⁻⁷ Ischaemic heart disease mortality has been declining in Finland since 1969-70 by a yearly 2%.⁸⁻⁹ This decline may be due either to factors improving the prognosis of incident cases or to a decline in incidence of the disease. Because data on changes in incidence of ischaemic heart disease world wide are few we examined data on admission to hospital for and mortality from ischaemic heart disease in Finland for 1972 and 1981 to assess relative changes in mortality, survival, and incidence.

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Subjects and methods

We based our study on a computer file of the death certificates of everybody aged 40-64 dying of ischaemic heart disease (International Classification of Diseases (ICD) codes 410-414) in Finland in 1972 or 1981 and a computer file of records of all hospital discharges during 1972 and 1981 for the same age group.¹⁰ We also used a computer file of all death certificates for 1973 and 1982 to calculate one year survival rates. The hospital records of all patients with an acute myocardial infarction or chronic coronary heart disease (ICD codes 410 and 412) as the principal diagnosis were included. Hospital discharge data were gathered from all general hospitals.¹¹ Incidence of ischaemic heart disease (first admission to hospital for or death from) was calculated by combining the death certificates and hospital records for the year under consideration. Altogether 3284 men and 768 women in the age group (age at first ischaemic heart disease event in 1972 or 1981) 40-64 years died from ischaemic heart disease during 1972. The corresponding figures for 1981 were 2818 men and 577 women.

The reference population was based on the 1972 and 1981 mid-year populations broken down by sex, five year age groups, and urban versus rural residence. People living abroad were excluded from the reference population when calculating rates of admission to hospital. Analysis was carried out by computing direct standardised rates on incidence, mortality, and one year (365 days) case fatality using as the standard population the combined 1972 and 1981 populations.

Recording of personal identification numbers was not complete on all hospital records. The proportion of missing or incorrect identification numbers was 10.9% in 1972 and 5.7% in 1981 among 40-64 year olds, varying by sex and by province from 3.3% to 15.4%. This source of error was taken into account by calculating adjusted case fatality figures under the assumption that the survival of the patients with incomplete identification number was similar to that of patients with complete data. The adjustment was performed in each sex, five year age group, and province combination. Place of residence was missing from 0.1% of death certificates and 1.2% of hospital records. The urban or rural state of communities was coded and analysed according to data for 1981.

Results

Tables I and II show our findings. Changes in incidence, case fatality, and mortality varied greatly by age and sex. We also analysed absolute change (%) in one year case fatality among patients admitted to hospital for ischaemic heart disease (table III).

Discussion

Computer linkage of death certificates and hospital records became possible for the first time in 1972, when the personal identification number was included in both data sets. The reliability of recordings of ischaemic heart disease as the cause of death on death certificates was satisfactory for epidemiological purposes.^{12,13} The reliability of ischaemic heart disease as a hospital discharge diagnosis has been studied systematically in Finland and found to be satisfactory for epidemiological purposes.¹³ In a sample of hospitals about 15% of discharge records on ischaemic heart disease were missing.¹³ The probable decrease in the proportion of missing records during 1972-81 would offset the real decline in incidence. The results for changes in incidence were probably therefore a slight underestimate. Date of birth was nearly always recorded but not the personal identification number (10.9% missing in 1972, 5.7% in 1978). The effect of this source of error was adjusted for as described above. On the other hand, changes in diagnostic criteria for ischaemic heart disease in hospital records or changes in incidence of unrecognised myocardial infarctions¹⁴ could not be assessed.

The causes of the decrease in ischaemic heart disease mortality have been debated,^{1-4,8,15-18} but definite evidence has not been found. Possible explanations have been linked to changes in diet, physical activity, and smoking as well as in medical care (improved intensive care, new antiarrhythmic drugs, and better control of hypertensive patients). In Finland the decrease in mortality seems to be associated with both decreased incidence and better survival.

TABLE I—Relative change (%) in incidence, case fatality, and mortality for ischaemic heart disease (IHD) in Finland in 1972 and 1981 among men and women aged 40-64 years

Age (years)	Incidence		Case fatality (first year)		Mortality		No of deaths due to IHD in 1981	
	Men	Women	Men	Women	Men	Women	Men	Women
40-44	-33.7	-40.4	-22.2	-33.1	-49.1	-63.7	120	9
45-49	-33.5	-42.3	+0.6	+40.5	-29.1	-8.9	274	23
50-54	-16.3	-26.5	-9.9	-7.3	-25.5	-33.1	548	73
55-59	-3.1	-23.3	-4.3	-11.3	-10.3	-28.7	861	143
60-64	-2.0	-6.4	-4.1	-12.1	-5.5	-16.6	1015	329
Age adjusted total	-14.2	-19.3	-7.3	-10.3	-15.9	-23.5	2818	577

TABLE II—Relative change (%) in incidence, case fatality, and mortality for ischaemic heart disease in 1972 and 1981 in Finland among men and women aged 40-64 years. (Absolute figures in 1981 in parentheses)

	Men	Women
<i>Incidence*</i>		
Urban areas	-19.1 (1270)	-21.9 (296)
Rural areas	-8.5 (1338)	-16.4 (314)
Whole country	-14.2 (1303)	-19.3 (305)
<i>Case fatality (first year)†</i>		
Urban areas	-5.6 (33.4)	-9.6 (21.6)
Rural areas	-8.9 (34.9)	-11.3 (23.6)
Whole country	-7.3 (34.1)	-10.3 (22.6)
<i>Mortality*</i>		
Urban areas	-18.6 (470)	-23.8 (75)
Rural areas	-12.9 (485)	-23.3 (85)
Whole country	-15.9 (477)	-23.5 (80)

*Number/100 000 people/year.

†Proportion of cases leading to death in first year (%).

TABLE III—Absolute change (%) in fatality due to ischaemic heart disease among all incident cases and among patients admitted to hospital aged 40-64 in 1972 and 1981 by sex. (Absolute percentage of one year fatality in 1981 in parentheses)

	Men	Women
<i>Patients admitted to hospital</i>		
Urban areas	-0.2 (14.2)	+0.6 (8.7)
Rural areas	-0.9 (13.8)	-1.8 (8.6)
Whole country	-0.5 (14.0)	-0.5 (8.7)
<i>All incident cases</i>		
Urban areas	-2.0 (33.4)	-2.3 (21.6)
Rural areas	-3.4 (34.9)	-3.0 (23.6)
Whole country	-2.7 (34.1)	-2.6 (22.6)

Changes in health behaviour should be found among men and women born in 1920-30. Reliable data on such changes in the population are not available for Finland. There are some ecological data on diet and smoking,⁸ but it is difficult to draw conclusions from ecological exposure data to individual end points. Between 1972 and 1981 smoking increased slightly among middle aged women and decreased slightly among men. The proportion of animal fats in the diet has decreased.⁸ Changes in physical activity have not been studied systematically, but physical activity has decreased at work and increased at leisure.

Changes in risk factors for coronary heart disease between 1972 and 1977 have been studied in the North Karelia project in a sample of the population of two counties in eastern Finland, representing about 9% of the Finnish population.^{19,20} This study showed, for 1972 and 1977: a slight decrease in smoking among men and a slight increase among women; a slight decrease in mean serum cholesterol concentration; and a decrease in systolic and diastolic blood pressure, especially among women.

The Public Health Act of 1972 has improved primary health care, particularly in rural areas. Treatment of high blood pressure has improved over the whole country. The age standardised

proportion of drug treatment of hypertension among 45-64 year olds increased during 1972-81 from 3.5% to 14.6% for men and from 7.8% to 17.9% for women as reported by the Social Insurance Institution.^{21 22} The better control of high blood pressure may be associated with the decrease in case fatality.

When causes of changes in incidence are considered the basic question is the change in prevalence and degree of coronary atherosclerosis.^{23 24} In Finland the necropsy rate has been quite high during the past 30 years,¹⁰ but there has been no systematic recording of the prevalence of coronary atherosclerosis. The effect of various factors on changes in incidence of ischaemic heart disease, however, remains open to debate as long as individual data are not available.

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Pulmonary haemorrhage complicating Wegener's granulomatosis and microscopic polyarteritis

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Abstract

The incidence and characteristics of pulmonary haemorrhage in a series of 89 patients with systemic vasculitis were analysed. Pulmonary haemorrhage occurred in 32 of these patients and was associated with haemoptysis in all 32, alveolar shadowing in the chest radiograph in 28, and a significantly raised transfer coefficient in 30. Pulmonary haemorrhage usually resolved with treatment by immunosuppressive drugs but was the cause of death in 11 patients. In contrast with patients with antibasement membrane antibodies there was no correlation between pulmonary haemorrhage and cigarette smoking.

Pulmonary haemorrhage is a cause of serious morbidity in patients with systemic vasculitis.

Introduction

The association of pulmonary haemorrhage with necrotising glomerulonephritis is usually called Goodpasture's syndrome. Many patients with Goodpasture's syndrome have auto-antibodies to glomerular and alveolar basement membrane antigens¹ and the term is sometimes restricted to these. Nevertheless, glomerulonephritis and pulmonary haemorrhage co-exist in other diseases, such as Wegener's granulomatosis,² microscopic polyarteritis,³ systemic lupus erythematosus,⁴ and, rarely, mixed essential cryoglobulinaemia, Henoch-Schönlein purpura, rheumatoid vasculitis, and mixed connective tissue disease.⁵

Descriptions of pulmonary haemorrhage and nephritis in patients without antglomerular basement membrane antibody disease have generally been restricted to individual reports of severe, often fatal cases and the overall prevalence has not been documented reliably. This is partly because of the rarity of the underlying diseases and also because the diagnosis of pulmonary haemorrhage is usually circumstantial. For example, haemoptysis does not differentiate bleeding from the large airways, commonly found in Wegener's granulomatosis, and alveolar haemorrhage; haemoptysis may be trivial⁶; radiological features are not specific and may even be absent⁷; and traditional confirmatory investigations such as lung biopsy, bronchoscopy with lavage, or radioisotope techniques are cumbersome or invasive. The diagnosis has been simplified considerably by the observation that the diffusing capacity for carbon monoxide, especially

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