small, and a larger series of patients is needed to estimate the size and persistence of the excess more precisely. It will be interesting to determine from such a series if there is also an excess of soft tissue sarcomas and squamous cell cancer of the skin, which are also increased in incidence in renal transplant patients.

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Alcohol consumption and premature death in middle-aged men

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Summary and conclusions

All the men living in Malmö born in 1926-9 were invited for a screening examination which included an assessment of alcohol consumption and measurement of gamma-glutamyltransferase (GGT) activity. They were followed for up to four years (median 2) and their mortality assessed. Sixty-two deaths occurred, 41 (0·9%) among the 4571 men who attended the screening investigation and 21 (1·3%) among the 1609 who did not respond to the invitation. Evidence of alcohol abuse or an alcohol-related cause of death was present in 25 (61%) of the deaths among the attenders and 13 (62%) of those among the non-responders. GGT values at the screening investigation were significantly increased in 19 (46%) of those who died, but established risk factors, such as cholesterol and triglyceride concentrations and blood pressure, had little predictive value.

Measurement of GGT provided an objective index of alcohol consumption, though the full clinical importance of a raised value needs further assessment. The finding that heavy alcohol consumption was the single most important factor associated with premature death in these middle-aged men has important implications for prevention.

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Introduction

Though alcohol is recognised to be a major contributory factor in disease and premature death among middle-aged men, the medical profession still tends to underestimate the importance of alcohol in the range of disease.[1] There have been demands for a "public health strategy" to identify and prevent alcohol abuse through screening[2 3] and for powerful political and economic measures to curb consumption. An important prerequisite for any of these measures is increased knowledge of the medical complications of alcohol consumption.

We established a screening clinic to try to identify and prevent alcohol abuse, and we report here a study on the short-term mortality of middle-aged men who were invited for screening.

Subjects and methods

Screening—A section of preventive medicine was established in autumn 1974 within the medical clinic at Malmö General Hospital. Total male birth-year cohorts in Malmö were invited to a screening investigation, where they underwent measurements of height and weight, pulse rate, blood pressure (standing and recumbent and before and after 10 minutes' rest), and skinfold thickness; simple pulmonary function tests; electrocardiography; and several fasting laboratory estimates, including γ-glutamyltransferase (GGT). All laboratory analyses were performed according to standard methods in the hospital's department of clinical chemistry. GGT was analysed according to the recommendation of the Scandinavian Enzyme Committee.[4] The usual laboratory upper limit of normal for GGT was 1·0 μkat/l, but in our screening clinic we used 1·44 μkat/l as a cut-off limit for further investigation. Attenders were also asked to fill in a questionnaire on their alcohol intake. Findings at the screening investigation were checked one or two weeks later and if confirmed the patient was invited for further investigation and treatment at special outpatient clinics within the department of preventive medicine. These included clinics for blood pressure, lipids, "borderline diabetes," and raised GGT values.[4]

Study of population—Screening started in November 1974 with men born in 1926. Subsequent birth-year cohorts of men were examined consecutively in the following years together with women and a few younger birth-year cohorts. At the end of 1978 all men living in Malmö who were born in 1926-9 had been invited for screening—a total of 6180. Of these, 4571 (74%) responded. All the men were aged 48 or 49 years at the screening investigation, and they were followed-up for up to four years (median two years).

Mortality—Information on deaths occurring after the invitation to screening in these birth-year cohorts was obtained from a register at the department of pathology, Malmö General Hospital. This register covers all deaths in Malmö, including those where necropsy is not performed. There were 41 (0·9%) deaths among the 4571 men who attended for screening and 21 (1·3%) among the 1609 who did not. The necropsy rate was 90% (36/41) among attenders and 100% among the non-responders.

Alcohol consumption—To assess the part that alcohol consumption may have played in each death all accessible information on the deaths was reviewed: protocols from screening death certificates, necropsy reports, police reports, and hospital records, including those of the alcohol clinic of Malmö General Hospital. The men who died were classified as having an alcohol-positive history if this information showed at least one of the following: (*a*) a history of heavy alcohol consumption; (*b*) registration at the alcohol clinic; (*c*) a raised GGT value at the screening investigation which, after interview with one of us (HK), was considered to be due to heavy drinking.[4] Those men who did not fulfill these criteria included a few who had only scanty or inconclusive information. All deaths from medical complications of alcoholism—for example, cirrhosis of the liver, oesophageal varices, and alcohol intoxication—and cases where alcohol intoxication contributed to the death according to necropsy reports were classified as probable alcohol-related deaths. Deaths from conditions that may have been related to alcohol consumption were classified as possible alcohol-related deaths.

Results

Table I shows causes of death, GGT values, and histories of alcohol consumption and smoking in the attenders at the screening clinic. Nineteen men had raised GGT activities and 21 a history of heavy alcohol consumption. These findings are related to the main causes of death in table II: raised GGT values and history of alcohol consumption were particularly prominent among men who committed suicide or died in accidents and those who died of miscellaneous causes (which included cirrhosis of the liver, bleeding oesophageal varices, etc).

Table III shows the causes of death according to the death certificates and a retrospective classification of alcohol consumption among non-responders. Mortality after the date of invitation to screening among non-responders was almost one and a half times that among attenders (1·3% compared with 0·9%). A directly alcohol-related cause of death was found in 10 of the 21 cases and a positive history of heavy alcohol consumption in 13 (62%).

There was a lower incidence of cardiovascular deaths, a higher

TABLE I—*Cause of death, alcohol and smoking history, and other findings at screening investigation in 41 participants who later died*

Case No	Cause of death	GGT (μkat/l)	History of heavy alcohol intake	History of smoking	Other screening findings*	Time between investigation and death (yr)	Alcohol-related death
1	Malignant glioma	0·42	−	+		1	
2	Cardiac hypertrophy, alcohol intoxication	1·93	+	+	BP (diastolic) 105 mm Hg	2	Probable
3	Alcohol and barbiturate intoxication	2·33	+	+		1½	Probable
4	Malignant histiocytosis	2·98	+	+		1½	
5	Pancreas cancer	1·84	+	+		1	Possible
6	Alcohol intoxication	8·14	+	+	Triglyceride 2·8 mmol/l	1	Probable
7	Train accident (engine-driver)	2·42	+	−	Triglyceride 3·5 mmol/l, relative body weight 1·4	1	Possible
8	Ethanol and hexapropymate intoxication	0·82	+	+		½	Probable
9	Cor pulmonale	0·46	−	NA	PF 2·1 l/min, FVC 0·7 l	2 mnth	
10	Suicide (car exhaust)	0·60	−	+		1½	
11	Liver cirrhosis, chronic alcoholism	1·77	+	+	Relative body weight 2·2	2	Probable
12	Bronchial cancer	1·44	−	NA	ESR > 100 mm in 1 h	2½	
13	Alcoholic cardiomyopathy	1·11	+	+		2½	Probable
14	Pancreatitis	3·22	+	+	Hb 114 g/dl, glucose 8·6 mmol/l	2	Probable
15	Diffuse abdominal adenocarcinoma	0·30	−	+		2	
16	Pulmonary embolism	0·74	+	+		1½	Probable
17	Myocardial infarction	1·94	−	+	Hb 108 g/dl, ESR 89 mmol/l	1	
18	Liver cirrhosis	31·7	+	+	Relative body weight 1·4, glucose 7·9 mmol/l	1	Probable
19	Myocardial fibrosis	0·91	+	+		1	
20	Pulmonary embolism	1·58	+	NA	Glucose 7·2 mmol/l	1	Probable
21	Duodenal rupture (traumatic ?)	0·79	+	+		1	Probable
22	Dissecting aortic aneurysm	0·53	−	+	BP 215/135 mm Hg	1 mnth	
23	Myocardial infarction	1·86	+	+		3	
24	Pancreatic neoplasm	0·47	+	+		4	Possible
25	Coronary sclerosis	0·35	−	+		3	
26	Chronic myeloid leukaemia	5·15	−	−	WBC 59 × 10⁹/l	3	
27	Cardiosclerosis, diabetes	1·93	?	NA	Triglyceride 3·1 mmol/l glucose 7·3 mmol/l	3	
28	Malignant melanoma	0·84	−	−		2	
29	Systemic hypertension	1·65	+	+	BP 220/150 mm Hg, relative body weight 1·5; creatinine 438 μmol/l	2½	
30	Bronchial cancer	0·72	−	+		2½	
31	Pancreatic neoplasm	0·79	−	−	Relative body weight 1·4	2½	
32	Cardiosclerosis	0·75	−	+	BP 170/115 mm Hg	3	
33	Myelitis, myocarditis	0·33	−	−		2½	
34	Cerebral haemorrhage	0·60	−	+	BP 190/115 mm Hg, relative body weight 1·6	2	
35	Myocardial infarction	1·33	−	−	BP 155/110 mm Hg	2	
36	Cardiosclerosis	2·59	+	+		2	
37	Cardiosclerosis	0·56	−	−	BP 165/115 mm Hg	1½	
38	Bleeding oesophagal varices	2·96	+	+	BP 175/105 mm Hg, glucose 8·2 mmol/l	2	Probable
39	Bronchopneumonia	3·19	+	+	Triglyceride 3·1 mmol/l, glucose 9·0 mmol/l	1 mnth	Possible
40	Myocardial infarction	0·44	−	+	Operated angina pectoris 1966	1	
41	Myocardial infarction, diabetes	0·56	−	+	Known diabetes with complications	2	

*Recumbent blood pressure (BP) was measured after 10 minutes' rest. Relative body weight = actual weight/ideal weight for age, sex, and height. PF = Peak flow. FVC = Forced vital capacity. ESR = Erythrocyte sedimentation rate. Glucose value was that measured at 120 minutes during glucose tolerance test. WBC = White blood count.

Conversion: SI to traditional units—GGT: 1 μkat/l ≈ 60 iu/l. Triglyceride: 1 mmol/l ≈ 89 mg/100 ml. Glucose: 1 mmol/l ≈ 18 mg/100 ml.

incidence of alcohol-related deaths, and a higher incidence of attendance at the alcoholic clinic among the non-responders than among those who attended the screening clinic (table IV). Nevertheless, over half the attenders had a history of heavy alcohol consumption and about half had raised GGT activities. Altogether 25 (61%) of the attenders had a history of alcohol consumption or raised GGT values, or both.

Table V shows that the premature deaths among attenders were concentrated in those whose GGT values were in the upper quintiles of the distribution of values among the whole population screened. A similar correlation did not exist for other values commonly regarded as risk factors (table V), though there was a slight tendency for those who died to have serum urate values in the upper quintiles; urate concentrations may also correlate with alcohol consumption.[4]

TABLE II—*Causes of death among participants and non-responders according to GGT activities and alcohol history*

	Participants			Non-responders		Total	
	No	No with raised GGT	No with history of heavy alcohol intake	No	No with history of heavy alcohol intake	No	No with history of heavy alcohol intake
Cardiovascular diseases	15	6	6	5	4	20	10
Cerebrovascular diseases	1	0	0	2	1	3	1
Miscellaneous causes ..	10	6	8	9	5	19	13
Malignant neoplasms ..	10	4	3	1	1	11	4
Suicide/accidents ..	5	3	4	4	2	9	6
Total ..	41	19	21	21	13	62	34

TABLE III—*Causes of death and history of alcohol consumption in non-responders*

Case No	Cause of death	Time between investigation and death (yr)	Alcohol history	Alcohol related death
1	Pulmonary embolism, pancreatitis	2 mnth	+	Probable
2	Pulmonary embolism	2	−	
3	Cardiac hypertrophy	1	+	Probable
4	Dissecting aortic aneurysm	½	−	
5	Myocardial infarction	½	+	
6	Myeloid leukaemia	1 mnth	−	
7	Burn injury, alcohol intoxication	½	+	Probable
8	Myocardial infarction	1 mnth	+	
9	Cerebral haemorrhage, diabetes mellitus	3	−	
10	Accidental drowning	2	−	
11	Pneumonia, myelopathy	2	−	
12	Hepatic cirrhosis	2	+	Probable
13	Colonic carcinoma	2	+	
14	Hepatic cirrhosis	2½	+	Probable
15	Respiratory insufficiency	2	−	
16	Suicide, depression	2	−	
17	Ventricular ulcer with haemorrhage	1	+	Probable
18	Subdural haematoma	1½	+	,,
19	Myocardial fibrosis, alcohol intoxication	1	+	,,
20	Drowning, alcohol intoxication	½	+	,,
21	Oesophageal varices	½	+	,,

TABLE IV—*Comparison between attenders and non-responders. Results are numbers of subjects*

	Participants	Non-responders	Total
Relative mortality	0·9% (1)	1·3% (1·45)	1%
Raised screening GGT (≥1·4)..	19/41		
Known at alcohol clinic.. ..	11/41	9/21	20/62 (32%)
Alcoholic history	21/41	13/21	34/62 (55%)
Probable alcohol-related death ..	12/41	10/21	22/62 (36%)
Possible alcohol-related death ..	4/41		
Necropsy	36/41	21/21	57/62 (92%)

TABLE V—*Numbers of deaths among participants according to GGT activities, serum cholesterol and triglyceride concentrations, recumbent diastolic blood pressure after 10 minutes' rest, and serum urate values. Quintiles are derived from distribution of values among all participants screened in 1926-9 birth-year cohorts (n=4571)*

GGT (μkat/l)	≤0·4	−0·52	−0·67	−0·96	≥0·97
No of deaths	3	5	4	9	20
Cholesterol (mmol/l) ..	≤5·0	−5·5	−6·1	−6·8	≥6·9
No of deaths	12	11	7	5	6
Triglyceride (mmol/l) ..	≤1·0	−1·25	−1·50	−2·02	≥2·03
No of deaths	13	3	5	10	9
Diastolic blood pressure (mm Hg)	≤75	−85	−90	−95	≥96
No of deaths	6	12	10	2	11
Urate (μmol/l)	≤257	−292	−322	−358	≥359
No of deaths	5	8	3	10	15

Discussion

In this unselected population of Swedish urban men alcohol was the most important single factor associated with death at about the age of 50: an alcohol-positive history was present in over half the men who died. Our study was a short-term prospective study of mortality in a fairly large population, uniform for age and sex, who were invited for screening and followed for up to four years (median two years). Our analysis of alcohol consumption was partly prospective and partly retrospective.

Many studies have confirmed the general impression that alcoholics have increased morbidity and mortality,[1 2 5-8] and such features should also be identified in population studies. Nevertheless, few population studies have been performed because of the difficulties of measuring alcohol consumption. Reliance on self-reported consumption in questionnaires makes findings open to doubt. At our screening investigation we used a modified Michigan alcoholism screening test,[3 4] but we also used a biochemical index of heavy alcohol consumption.

With methods partly comparable to ours Tibblin studied 2992 men living in Gothenberg who were born in 1913.[5-7] He found that of the 273 who died aged 35-55 17% were heavy drinkers or died from alcohol-related conditions.[7] Lannerstad examined 49 deaths that occurred among 802 men in Malmö at the age of 55-60 years; in 27% he found signs of alcohol abuse, defined as registration at the alcohol clinic or a blood alcohol concentration at necropsy of more than 22 mmol/l, or both.[8] The American epidemiological studies in Framingham and at Chicago Western Electric Company also showed an association between alcohol consumption and all causes of death,[9 10] but comparisons are difficult because these populations were selected and the ages were different.

Serum GGT activity has been shown in series of alcoholics[11-13] and in population studies[4 14-17] to be the best single biochemical index of high alcohol consumption. Nevertheless, the clinical significance of raised activities has still not been assessed. Measurement of GGT activity may play a useful part in screening investigations for alcohol abuse, especially in middle-aged men. We have already shown that 75% of those with raised GGT values can be classified as heavy users of alcohol.[4] Our present results illuminate the question of clinical significance by showing that the risk of dying was six to seven times greater in men with GGT values in the highest quintile than in those with values in the lowest quintile. It remains to be seen whether the heavy consumer of alcohol with a normal serum GGT value on screening has a better outlook.

Interestingly, a high serum cholesterol value appeared to be an inverse risk factor for premature death—a finding supported by other reports.[8 18] In a mortality study with a short follow-up such as ours some individuals may have already been ill and in poor nutritional state at the time of the examination, but the relationship between cholesterol concentration and premature death merits further consideration. It is hard to evaluate the role of smoking in our cohorts; 70% of the 41 attenders who died were smokers, and they were concentrated in the subgroup with a history of heavy alcohol consumption. This probably reflects the known association between alcohol consumption and smoking.

An assessment of drinking habits is a relevant aim in population investigations of middle-aged men, and measurements of serum GGT may help if coupled with a clinical evaluation and treatment programme. Although our observations on short-term mortality permit no certain general epidemiological conclusions at this stage, we think that our findings are clear and relevant and carry important implications for prevention.

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Surgical treatment of primary hyperparathyroidism in the elderly

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Summary and conclusions

Twelve patients aged over 70 with primary hyperparathyroidism (persistent hypercalcaemia and raised serum parathyroid hormone concentrations) underwent parathyroidectomy, which was well tolerated by all. After operation serum calcium concentrations returned to normal and the commonest symptoms before operation (muscle weakness, malaise, and mild to severe dementia), although not related in severity to the degree of hypercalcaemia, improved. Mental function was greatly improved.

The findings suggest that primary hyperparathyroidism should be sought in any elderly patient with hypercalcaemia and that more such patients with the diagnosis should be considered for parathyroidectomy irrespective of age.

Introduction

Biochemical screening detects many cases of hyperparathyroidism that would otherwise go unrecognised. Most show no evidence of bone disease or renal disease. The commonest group of patients with hyperparathyroidism presenting at our clinics complain of muscle weakness, tiredness, and general malaise. The next commonest group are patients who appear to be completely asymptomatic but in whom hypercalcaemia is found by chance during investigations for other problems. With the increased recognition of the disease, many elderly patients have been found to have hyperparathyroidism. In such patients the non-specific nature of the symptoms may readily be attributed to "aging," and the decision to operate may be difficult. We report our experience of parathyroidectomy in 12 patients aged 70 or over, which encourages us to recommend operations in symptomatic patients in this age group.

Patients, methods, and results

The table gives the details of the 12 patients. The commonest symptoms were muscle weakness, mental slowing, and decreased mobility. Before operation, three of the patients (cases 2, 7, and 8) were unable to manage at home and were awaiting long-term hospital care, while two others (cases 5 and 11) were able to live at home only because of considerable attention from relatives living in the same house. Another patient (case 6) had been transferred to sheltered accommodation before operation. In all 12 patients the hypercalcaemia was discovered during routine biochemical investigations for assessment of deteriorating physical state or for completely unrelated medical problems. Hyperparathyroidism was diagnosed when persistent hypercalcaemia was present with raised serum parathyroid hormone concentrations. No patient showed radiological evidence of the disease. At operation a single parathyroid adenoma was found in all cases except case 10, in which two adenomas were removed at separate operations. Successful removal of the adenomas was associated with rapid return of the serum calcium concentration to normal. The operation was well tolerated by all the patients, and no serious complications occurred. Only one patient (case 5) developed severe symptomatic hypocalcaemia. Although mild asymptomatic hypocalcaemia often occurred after operation, all patients eventually achieved normocalcaemia without long-term treatment with calcium supplements or vitamin D.

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