psychiatric hospitals (two of whom had been admitted for more than a year). Four had died, the causes of death being ruptured aortic aneurysm, heart attack, suffocation during an epileptic fit, and road traffic accident. In three of these deaths mental illness seemed to be contributory—in two cases because the subject had not sought treatment and in the third (the accident) because the subject was wandering the streets after absconding from the hostel. Finally, three subjects could not be traced; all had suddenly departed leaving their belongings behind but without leaving contact addresses.

Nine of the 48 subjects had been admitted to psychiatric hospitals during the 18 months. At the time of admission all had been hostel residents. Four of these patients had had two or more hospital admissions in the 12 months before the 18 month follow up had begun and five patients had had no admissions in that period. Among the four with two or more admissions, three were still in hospital at follow up and one had returned to his hostel but showed no improvement on the behavioural rating scale. Hospital case notes showed that the mental state of four of the other five patients had improved considerably after treatment. Three of these four subjects also showed improvement on the behavioural rating scale: two had improved general behaviour scores, as reported above, and one had an improved deviant behaviour score, falling from 5 to zero. None of the nine subjects admitted to hospital had been rehoused.

Discussion

Our data show that the general outcome of the 48 subjects was not good. Only 10 patients had been resettled in bedsits, supported accommodation, or family homes, nine had been admitted to psychiatric hospitals, and 16 had had a poor outcome as shown by death (four subjects), disappearance (three), inpatient care exceeding one year (two), sleeping rough (one), or marked deterioration in behaviour (six).

Not only was the rate of rehousing low but accommodation was limited to bedsits owned by private landlords, supported accommodation managed by the hostels, and the family home. Of these three kinds of placement, only supported accommodation seems likely to be of long term benefit. Neither private landlords nor family homes are likely to meet the needs of our subjects. Such mentally disabled people would probably fare best in small group homes or cluster flats supported by local social or psychiatric services. None of our subjects had achieved a placement of this kind. Perhaps the explanation is that once people become homeless they become isolated from mainstream community care. This theory is further supported by the fact that a third of the subjects had a poor general outcome.

The high rate of admission to psychiatric hospitals is consistent with the high level of psychiatric morbidity found in this group of subjects. This high admission rate does not necessarily mean a failure of community care; on the contrary it may indicate success in identifying treatable mental illness. On the other hand, there is cause for concern that none of the subjects admitted to psychiatric hospitals was rehoused.

Overall our findings suggest that the homeless mentally ill should be given increased access to supported accommodation, where their needs could be better met. If conventional agents cannot provide such access the hostel staff should be given adequate funds to do so.

MM was supported by a grant from Oxford Regional Health Authority and subsequently by the Wellcome Trust. We thank the hostel staff, in particular Mike Hall and Steve Curry, for their help.

1 Lowry S. Concern for discharged mentally ill patients. *BMJ* 1989;**298**:209-10.
2 Fischer JP, Breaker WR. Homelessness and mental health: an overview. *International Journal of Mental Health* 1986;**14**:6-41.
3 Timms PW, Fry AH. Homelessness and mental illness. *Health Trends* 1989;**3**:70-1.
4 Priest RG. The homeless person and the psychiatric services: an Edinburgh study. *Br J Psychiatry* 1976;**10**:233-5.
5 Marshall M. Collected and neglected: are Oxford hostels for the homeless filling up with disabled psychiatric patients? *BMJ* 1989;**299**:706-9.
6 Leach J. Providing for the destitute. In: Wing JK, Olsen R, eds. *Community care for the mentally disabled*. Oxford: Oxford University Press, 1979.
7 Baker R, Hall JN. REHAB: a new assessment instrument for chronic psychiatric patients. *Schizophr Bull* 1988;**14**:97-111.

(Accepted 11 November 1991)

Alcohol consumption and its relation to cardiovascular risk factors in British women

G Razay, K W Heaton, C H Bolton, A O Hughes

University Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HW
G Razay, MRCP, *research fellow*
K W Heaton, FRCP, *reader in medicine*
C H Bolton, PHD, *lecturer in biochemistry*

University Department of Epidemiology and Public Health Medicine, University of Bristol, Bristol BS8 2PR
A O Hughes, MSC, *senior lecturer in medical statistics*

Correspondence to:
Dr Razay.

BMJ 1992;**304**:80-3

Abstract

***Objective*—To examine the relation between alcohol consumption and risk factors for coronary heart disease in women.**

***Design*—Cross sectional study of a stratified random sample of the population grouped into five categories of habitual alcohol consumption.**

***Setting*—People registered with general practitioners at two large health centres in east Bristol, England.**

***Subjects*—1048 women aged 25-69 years.**

***Main outcome measures*—Fasting plasma concentrations of insulin, total cholesterol, total triglycerides, and high density lipoprotein cholesterol, including its subfractions HDL_2 and HDL_3, and body mass index.**

***Results*—Compared with non-drinkers women consuming a moderate amount of alcohol (1-20 g/day) had lower plasma concentrations of triglycerides, by 0·19 mmol/l (95% confidence interval 0·07 to 0·35); cholesterol, by 0·4 mmol/l (0·19 to 0·61); and insulin, by 1·4 mU/l (0·43 to 1·97) and a lower body mass index, by 1·2 kg/m² (0·43 to 1·97). They also had higher concentrations of high density lipoprotein cholesterol, by 0·09 mmol/l (0·03 to 0·15); HDL_2 cholesterol by 0·05 mmol/l (−0·02 to 0·10) and HDL_3 cholesterol, by 0·06 mmol/l (0·06 to 0·11). All these were independent of body mass index, smoking habits, and taking oral contraceptives.**

***Conclusions*—Moderate alcohol consumption is associated with lower levels of cardiovascular risk factors in women. Insulin may have a central role.**

Introduction

In most epidemiological studies moderate alcohol consumption has been associated with a decreased risk of coronary heart disease.[1-4] Because of the well known inverse relation between high density lipoprotein

cholesterol concentration and coronary heart disease[5,6] it is generally assumed that any protective effect of alcohol is mediated by its wellknown effect in raising high density lipoprotein concentrations.[7-9] However, alcohol increases concentrations of HDL_3[10,11] and, according to some reports, HDL_2 cholesterol.[12,13] A protective effect from HDL_2 cholesterol has been found in most studies of coronary heart disease[14,15] and a protective effect from HDL_3 cholesterol has been found in some.[11,16] The mechanism by which coronary heart disease is reduced in drinkers remains unclear, and a search for factors other than high density lipoprotein subfractions is required.

Prospective studies have shown that raised plasma insulin concentration is associated with increased risk of coronary heart disease,[17-19] but there are no published data on the relation between alcohol consumption and plasma insulin. We examined the relation between alcohol consumption and plasma concentrations of insulin and lipoproteins in a large stratified random sample of British women.

Subjects and methods

We studied 1048 women aged 25-69 who, between October 1987 and March 1989, attended an epidemiological survey in Kingswood, Bristol (70·1% of those invited).[20] There were 304 women aged 25-29, 324 aged 30-39, 199 aged 40-49, 138 aged 50-59, and 83 aged 60-69. We excluded 10 of these women because they had diabetes and one women who was taking a lipid lowering drug. This stratified random sample of the east Bristol population was obtained from the lists of people registered with the 19 general practitioners working in the two main health centres in east Bristol. Women were sent letters signed by their general practitioner asking them to cooperate with a survey on the prevalence of and causes of gall stones, including eating habits but not mentioning alcohol. A few days later a clerk telephoned the woman or, if necessary, a field worker visited her home to offer an appointment at a small local hospital. Most women who refused were approached again after some months. Non-attenders were telephoned or visited again. The size and composition of the sample were dictated by the primary aim of the study, which was to determine the prevalence of gall stones.

The women were invited to attend a morning or evening clinic after fasting for at least five hours. Height and weight were measured in indoor clothing without shoes, from which body mass index was calculated in kg/m^2. We asked about history of heart disease and diabetes mellitus, and about drugs taken, including cholesterol lowering drugs and oral contraceptives. A dietary questionnaire was given for self completion at home, but the method of completion was explained at the clinic. The questionnaire included questions about beer, wine, sherry or vermouth, spirits, and liqueurs, and women were asked to ring a number or letter corresponding to the number of times a week that a drink in each category was consumed. Women were asked how much they usually consumed on one occasion in terms of pints or glasses. Spirits were divided into singles or doubles. The average daily consumption of alcohol was calculated on the assumption that half a pint of beer or lager contains 9 g of alcohol, a glass of wine, 10 g, and a measure of spirits, 8 g. Moderate drinking was defined as 1-20 g/day and 21-30 g/day as borderline, and heavy drinking as >30 g/day. The questionnaire also asked women whether they were current smokers, ex-smokers, or non-smokers of tobacco, but in this analysis they were simply classified as current smokers or non-smokers.

We obtained venous blood from 1016 (98%) of women (from 518 in the morning and from 498 between 17 30 and 20 00). Plasma was separated and analysed for glucose by an automated glucose oxidase method and for insulin by radioimmunoassay.[21] Sodium azide as a preservative was added to blood on arrival at the lipoprotein laboratory. Lipoproteins, including high density lipoproteins 2 and 3, were separated within 24 hours by a combination of precipitation and microultracentrifugation.[22] Plasma lipids and lipoprotein fractions were stored at −20 °C until analysed. Cholesterol and triglycerides were measured in plasma and lipoprotein fractions by enzymatic procedures (Boehringer-Mannheim). The concentration of HDL_2 cholesterol was obtained by estimating the difference between HDL and HDL_3 cholesterol concentrations. Lipid analyses were carried out in large batches to minimise interassay variation. Analyses of high density lipoprotein subfraction were possible in only the last 698 women (66%).

Mean fasting insulin concentration was consistently lower in the evening than the morning by about 1 mU/l. Therefore all the evening values were adjusted upwards by 1 mU/l.

All statistical calculations were performed using the Minitab program.[23] Analysis of variance or *t* test was used to compare groups, and adjustment for confounding factors was performed by analysis of covariance. Plasma triglyceride and insulin values were skewed, so logarithmic values were used in all statistical analyses. In the case of plasma triglycerides the log distribution was normal but for insulin the laboratory cut off level of 3·0 mU/l was still skewed at this part of the distribution, although using the log value improved the normality of the remaining observations.

Results

Of the 1048 women, 966 (92%) returned their questionnaires and, of these, 771 admitted consuming alcoholic drinks (table I). Most women drank moderately, but 116 admitted to drinking more than the recommended maximum of two units (20 g) daily. The proportion who drank fell progressively with age.

Table II shows the mean values for each variable studied and table III the mean values at five levels of alcohol consumption. Women taking oral contraceptives (n=168) were excluded from these analyses because these drugs affect plasma lipid concentrations. In moderate drinkers (1-20 g/day) compared

TABLE I—*Alcohol consumption in women of different ages*

Alcohol consumption (g/day)	No (%) of women aged: 25-29 (n=268)	30-39 (n=297)	40-49 (n=187)	50-59 (n=134)	60-69 (n=80)	All ages (n=966)
0	42 (16)	55 (19)	32 (17)	40 (30)	26 (33)	195 (20)
1-10	137 (51)	165 (56)	100 (53)	66 (49)	40 (50)	508 (53)
11-20	48 (18)	43 (14)	29 (16)	19 (14)	8 (10)	147 (15)
21-30	23 (9)	19 (6)	19 (10)	4 (3)	4 (5)	69 (7)
>30	18 (7)	15 (5)	7 (4)	5 (4)	2 (3)	47 (5)

TABLE II—*Average values for each of the variables studied in the entire population*

	No of subjects with data available	Mean (SD) value
Total cholesterol (mmol/l)	1005	5·15 (1·18)
Total high density lipoprotein cholesterol (mmol/l)	1004	1·36 (0·33)
HDL_2 cholesterol (mmol/l)	698	0·51 (0·28)
HDL_3 cholesterol (mmol/l)	698	0·88 (0·24)
Triglyceride (mmol/l)	993	1·01 (0·23-7·29)*
Body mass index (kg/m^2)	1048	24·6 (4·4)
Insulin (mU/l)	1004	5·0 (3·0-30·0)*
Alcohol consumption (g/day)	966	4·3 (0·0-107·9)*

*Median (range) given because data were skewed.

with non-drinkers plasma triglyceride concentration was 0·19 mmol/l (95% confidence interval 0·07 to 0·35) lower, cholesterol concentration 0·4 mmol/l (0·19 to 0·61) lower, insulin concentration 1·4 mU/l (0·43 to 1·97) lower, and body mass index 1·2 kg/m^2 (0·43 to 1·97) lower; conversely, total high density lipoprotein cholesterol concentration was 0·09 mmol/l (0·03 to 0·15) higher, HDL_2 cholesterol 0·05 mmol/l (−0·02 to 0·10) higher, and HDL_3 cholesterol 0·06 mmol/l (0·06 to 0·11) higher.

TABLE III—*Mean lipid and insulin concentrations according to daily alcohol consumption**

	Alcohol consumption (g/day)					
	0	1-10	11-20	21-30	>30	p Value†
Total triglycerides (mmol/l)	1·31	1·13	1·08	1·08	1·33	0·001
Total cholesterol (mmol/l)	5·6	5·2	5·2	5·0	5·3	0·001
High density lipoprotein (mmol/l)	1·30	1·38	1·43	1·47	1·48	0·001
HDL_2 cholesterol (mmol/l)	0·48	0·53	0·50	0·52	0·54	NS
HDL_3 cholesterol (mmol/l)	0·84	0·87	0·94	0·99	1·00	0·001
Insulin (mU/l)	6·7	5·4	4·8	5·1	5·9	0·001

*Women taking oral contraceptives (n=168) excluded.
†One way analysis of variance.

In heavy drinkers (>30 g/day) compared with moderate drinkers plasma triglyceride concentration was 0·21 mmol/l (0·05 to 0·47) higher, and HDL_3 cholesterol 0·11 mmol/l (0·01 to 0·22) higher. None of the other differences was significant.

The relation between alcohol consumption and total high density lipoprotein cholesterol and HDL_3 cholesterol concentrations was linear, whereas the relation with triglycerides and insulin concentrations gave a U shaped curve. This was confirmed by a highly significant F value if a quadratic term was introduced into the analysis of variance. The form of the relation between alcohol consumption, plasma insulin concentration, and lipid concentrations remained after controlling for body mass index, smoking habits, and the use of oral contraceptives (by one way analysis of covariance).

Most of the women (734) were non-smokers. The prevalence of smoking did not vary with alcohol consumption. A greater proportion of the heavy drinkers (17, 36%) smoked than non-drinkers (49, 25%), but this was not significant. Compared with non-smokers women who currently smoked had a raised plasma triglyceride concentration, by 0·19 mmol/l (0·06 to 0·28), a decreased total high density lipoprotein cholesterol concentration, by 0·12 mmol/l (0·06 to 0·18), and a decreased HDL_3 cholesterol concentration by 0·06 mmol/l (0·01 to 0·11).

Discussion

Our data agree with previous surveys in showing that in the population (in our case women) alcohol consumption is associated with higher concentrations of total high density lipoprotein cholesterol and both its subfractions and lower total cholesterol concentrations[10-13 24] and that smoking is associated with higher triglyceride and lower total high density lipoprotein cholesterol and HDL_3 cholesterol concentrations.[7]

Our study, like all studies dealing with alcohol, is subject to possible biases such as underreporting and inaccuracy and the tendency of problem drinkers not to participate in surveys. However, these biases should not invalidate the findings. The non-response rate of almost 30% raises the question of potential bias in a community based study.[25] However, alcohol was not mentioned to subjects until they attended for the survey, which makes it unlikely that drinking behaviour and non-attendance were connected.

The U shaped relation between alcohol consumption and plasma concentrations of triglycerides and insulin seems to be a new finding and might help to explain the U shaped relation between alcohol consumption and cardiovascular mortality in women.[26] The reduction of mortality in moderate drinkers could be explained by the lower levels of plasma triglyceride, cholesterol, insulin, and body mass index and by the higher concentrations of total high density lipoprotein cholesterol and HDL_3 cholesterol, all of which favour a reduced incidence of ischaemic heart disease.

The relation between alcohol and plasma insulin concentration is not clear. Studies of insulin release mediated by glucose have produced conflicting results, showing both an inhibiting[27] and a potentiating effect.[28] When obese rats prone to atheroma were exposed to long term high alcohol intake, fasting insulin concentrations were lower and pancreatic β cell hyperplasia was reduced,[29] suggesting that sensitivity to insulin was increased. Our data suggest that, in women at least, moderate alcohol consumption increases insulin sensitivity but high consumption decreases it. Others have suggested that high alcohol consumption might damage hepatic insulin receptors.[30]

The U shaped relation with plasma triglyceride concentration deserves comment. Alcohol consumption is associated with a rise in lipoprotein lipase activity, which is responsible for the peripheral breakdown of plasma triglycerides,[13] but in heavy drinkers plasma triglyceride concentration rises due to increased triglyceride synthesis mediated by the action of insulin on the liver.[31] Alcohol reduces hepatic lipase activity, which is responsible for the removal of high density lipoprotein from the circulation, and this may explain why in people with moderate alcohol consumption high density lipoprotein concentration increases.

The reduction in plasma total cholesterol concentration with moderate alcohol consumption may have resulted from reduced hepatic cholesterol synthesis caused by lower plasma insulin concentration as insulin has a key role in hepatic synthesis of cholesterol through stimulation of hydroxymethylglutaryl coenzyme A reductase, the rate limiting enzyme.[32] We suggest, therefore, that insulin has a central role in explaining the links between alcohol consumption and the risk of coronary heart disease.

Against the possible cardiac benefits of one or two drinks a day must be balanced the increases in subarachnoid haemorrhage[1] and breast cancer,[33] which have been reported with moderate alcohol consumption, and of course the risk of moderate drinking becoming heavy with its many risks to body, mind, and social function.

We thank Pauline Emmett and Fiona Braddon for their help and advice in the statistical analysis; the chemical pathology laboratory at Bristol Royal Infirmary for the glucose and insulin assays; and Janice Taberner and Lee Downs, who carried out the lipid assays. Many others helped to make this study possible.[20]

1 Stampfer MJ, Colditz GA, Willet WC, Speizer FE, Hennekens CH. A prospective study of moderate alcohol consumption and the risk of coronary disease and stroke in women. *N Engl J Med* 1988;**319**:267-73.
2 Gordon T, Kannel WB. Drinking habits and cardiovascular disease. The Framingham study. *Am Heart J* 1983;**105**:667-73.
3 Jackson R, Scragg R, Beaglehole R. Alcohol consumption and risk of coronary heart disease. *BMJ* 1991;**303**:211-5.
4 Marmot MG, Rose G, Shipley MJ, Thomas BJ. Alcohol and mortality: a U-shaped curve. *Lancet* 1981;i:580-3.
5 Jacobs DR, Mebane IL, Bangdiwala SJ, Criquit MH, Tyroler HA. High density lipoprotein cholesterol as a predictor of cardiovascular disease mortality in men and women. The follow up study of the lipid research clinics prevalence study. *Am J Epidemiol* 1990;**131**:32-45.
6 Miller GJ, Miller NE. Plasma HDL concentration and development of ischaemic heart disease. *Lancet* 1975;i:16-9.
7 Haffner S, Appelbaum-Bowden D, Wahl PW, Hoover JJ, Warnick GR, Albers JJ, *et al*. Epidemiological correlates of high density lipoprotein subfractions, apolipoproteins AI, AII, and D and lecithin cholesterol acetyltransferase. Effects of smoking, alcohol and adiposity. *Arteriosclerosis* 1985;5:169-77.

8 Belfrage P, Berge B, Hagerstrand J, Nilsson-Ehle P, Tornqvist H, Wiebe T. Alteration of lipid metabolism in healthy volunteers during long term ethanol intake. *Eur J Clin Invest* 1977;7:127-31.
9 Thornton J, Symes C, Heaton K. Moderate alcohol intake reduces bile cholesterol saturation and raises HDL cholesterol. *Lancet* 1983;ii:819-22.
10 Haskell WI, Camargo C, Williams PT, Vranizan KH, Kraus RM, Lindgren FT, *et al*. The effect of cessation and resumption of moderate alcohol intake on serum high density lipoprotein subfractions: a controlled study. *N Engl J Med* 1984;**310**:805-10.
11 Diehl AK, Fuller JM, Mattock MB, Salter AH, el-Gohar R, Keen H. The relationship of high density lipoproteins to alcohol consumption, and other lifestyle factors, and coronary heart disease. *Atherosclerosis* 1988;**69**:145-53.
12 Miller NE, Bolton CH, Hayes TM, Bainton D, Yarnell JWG, Baker IA, *et al*. Associations of alcohol consumption with plasma high density lipoprotein cholesterol and its major subfractions: the Caerphilly and Speedwell collaborative heart disease studies. *J Epidemiol Community Health* 1988;**42**: 220-5.
13 Taskinen MR, Valimaki M, Nikkila EA, Kuusi T, Ehnholm C, Ylikahn R, *et al*. High density lipoprotein subfractions and post heparin plasma lipases in alcoholic men before and after ethanol withdrawal. *Metabolism* 1982;**31**: 1168.
14 Miller NE, Hammett F, Saltissi S, Rao S, Van Zeller H, Coltart J, *et al*. Relation of angiographically defined coronary artery disease to plasma lipoprotein subfractions and apolipoproteins. *BMJ* 1981;**282**:1741-4.
15 Ballantyne FC, Clark RS, Simpson MS, Ballantyne D. High density and low density lipoprotein subfractions in survival of myocardial infarction in control subjects. *Metabolism* 1982;**31**:433-7.
16 Hennekens CM, Buring JE, O'Connor GT, Goldhaber SZ, Stampfer MJ, Breslow J, *et al*. Moderate alcohol consumption and risk of myocardial infarction [abstract]. *Circulation* 1987;**76**(suppl iv):501.
17 Pyorala K. Relationship of glucose tolerance and plasma insulin to the incidence of coronary heart disease. Results from two population studies in Finland. *Diabetes Care* 1979;**2**:131-4.
18 Ducimetiere P, Eschwege E, Papoz L, Richard JL, Claude JR, Rosselin G, *et al*. Relationship of plasma insulin levels to the incidence of myocardial infarction and coronary heart disease mortality in middle aged population. *Diabetologia* 1980;**19**:205-10.
19 Welborn TA, Wearn K. Coronary heart disease incidence and cardiovascular mortality in Busselton with reference to glucose and insulin concentration. *Diabetic Care* 1979;**2**:154-60.
20 Heaton KW, Braddon FEM, Mountford RA, Hughes AO, Emmett PM. Symptomatic and silent gall stones in the community. *Gut* 1991;**32**:316-20.
21 Oettle GJ, Emmett PM, Heaton KW. Glucose and insulin responses to manufactured and whole food snacks. *Am J Clin Nutr* 1987;**45**:86-91.
22 Fehily AM, Yarnell JWG, Bolton CH, Butland BV. Dietary determinants of plasma lipids and lipoproteins, the Caerphilly study. *Eur J Clin Nutr* 1988;**42**:405-13.
23 Minitab. *Reference manual (release 7)*. Pennsylvania: Minitab, 1989.
24 Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, *et al*. Alcohol and blood lipids. *Lancet* 1977;ii:153-5.
25 Evans SJW. Good survey guide. *BMJ* 1991;**302**:302-3.
26 Criqui M, Cowan L, Tyroler A, Bangdirvala S, Heiss G, Wallace RB, *et al*. Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality. *Am J Epidemiol* 1987;**126**: 629-37.
27 Patel DG, Singh SP. Effect of ethanol and its metabolite on glucose mediated insulin release from isolated islets of rats. *Metabolism* 1979;**28**:85-9.
28 Metz R, Berger S, Mako M. Potentiation of the plasma insulin response to glucose by prior administration of alcohol. *Diabetes* 1969;**18**:517-22.
29 Russell JC, Amy RM, Manickavel V, Dolphin PJ. Effect of chronic ethanol consumption in atherosclerotic-prone JCR:LA corpulent rats. *Arteriosclerosis* 1989;**9**:121-8.
30 Ohler G, Bleyl M, Matthes KJ. Hyperinsulinaemia in hepatic steatosis. *Int J Obes* 1982;**6**(suppl 1):137-44.
31 Steiner G. Hypertriglyceridemia and carbohydrate intolerance: interactions and therapeutic implications. *Am J Cardiol* 1986;**57**:27-30G.
32 Dietschy JM, Brown MS. Effect of alterations of the specific activity of the intracellular acetyl CoA pool on apparent rate of hepatic cholesterogenesis. *J Lipid Res* 1974;**15**:508-16.
33 Willett WC, Stampfer MJ, Colditz GA, Rosna BA, Hennekens CH, Speizer FE. Moderate alcohol consumption and the risk of breast cancer. *N Engl J Med* 1987;**316**:1174-80.

(Accepted 30 October 1991)

Effect of "fast track" admission for acute myocardial infarction on delay to thrombolysis

Alastair C H Pell, Hugh C Miller, Colin E Robertson, Keith A A Fox

Abstract

***Objective*—To evaluate the impact of a fast track triage system for patients with acute myocardial infarction.**

***Design*—Comparison of delays in admission to hospital and in receiving thrombolytic treatment before and after introducing fast track system with delays recorded in 1987-8. Patients fulfilling clinical and electrocardiographic criteria for myocardial infarction were selected for rapid access to the cardiac care team, bypassing evaluation by the medical registrar.**

***Setting*—Major accident and emergency, cardiac and trauma centre.**

***Subjects*—359 patients admitted to the cardiac care unit during 1 February to 31 July 1990 with suspected acute infarction.**

***Main outcome measures*—Accuracy of diagnosis and delay from arrival at hospital to thrombolytic treatment.**

***Results*—248 of the 359 patients had myocardial infarction confirmed, of whom 127 received thrombolytic treatment. The fast track system correctly identified 79 out of 127 (62%) patients who subsequently required thrombolytic treatment. 95% (79/83) of patients treated with thrombolysis after fast track admission had the diagnosis confirmed by electrocardiography and enzyme analysis. The median delay from hospital admission to thrombolytic treatment fell from 93 minutes in 1987-8 to 49 minutes in fast track patients ($p<0{\cdot}001$). Delay in admission to the cardiac care unit was reduced by 47% for fast track patients (median 60 minutes in 1987-8 *v* 32 minutes in 1990, $p<0{\cdot}001$) and by 25% for all patients (60 minutes *v* 45 minutes, $p<0{\cdot}001$).**

***Conclusion*—This fast track system requires no additional staff or equipment, and it halves inhospital delay to thrombolytic treatment without affecting the accuracy of diagnosis among patients requiring thrombolysis.**

Department of Cardiology, Royal Infirmary, Edinburgh EH3 9YW
Alastair C H Pell, MRCP, *research fellow*
Hugh C Miller, FRCP, *consultant cardiologist*
Colin E Robertson, FRCSE, *consultant emergency physician*
Keith A A Fox, FRCP, *professor of cardiology*

Correspondence to:
Professor Fox.

BMJ 1992;**304**:83-7

Introduction

Experimentally and clinically prognosis after acute myocardial infarction is dependent on the duration of myocardial ischaemia.[1-7] Although clinical benefit has been shown with thrombolysis in patients presenting more than six hours after onset of symptoms of myocardial infarction, very early thrombolytic treatment maximises the functional and survival benefits.[8-12] Delay in the hospital constitutes an important and potentially avoidable component of the total delay. Studies in major cardiac centres in the United States and Europe report hospital delays of 50-130 minutes.[2 3 13-15] Practical triage systems for minimising such delay have not yet been established.

An audit of patients admitted to our hospital in 1987-8 found median delays from arrival to thrombolysis of 93 minutes, and the delay in patients with unequivocal electrocardiographic evidence of acute myocardial infarction was no shorter than in patients with suspected myocardial infarction.[16] A relatively small proportion of patients (23% of those with infarction) received thrombolytic treatment. The audit identified several factors that contributed to inhospital delay, including triplication of patient assessment by accident and emergency staff, the duty medical registrar, and the cardiac care team. The triage system was common to all medical emergencies and the requirement for thrombolysis was assessed only after admission to cardiac care unit.

Edinburgh Royal Infirmary is the main accident and emergency and trauma centre for the Lothian region and in 1990, 68 641 new patients were evaluated. A duty medical registrar received all emergency medical referrals and was responsible for their initial evalua-