Alcohol and death certification: a survey of current practice and attitudes

Official estimates of all deaths from cirrhosis in England and Wales are lower than those in other countries with similar levels of alcohol consumption.[1] In addition, officially recorded death rates from alcoholic cirrhosis appear lower than rates suggested by results of clinical surveys.[2] These discrepancies are thought to be due largely to underrecording on death certificates, perhaps to avoid the statutory obligation (waived in July 1984) to report deaths related to alcohol to the coroner.[3] We have examined the recording of alcohol abuse on death certificates and factors influencing such recording.

Methods and results

All 125 death certificates signed by the 43 preregistration housemen at Middlesex and University College Hospitals over a three month period were examined. Those with a diagnosis in which alcohol may have been aetiologically important (n=40) were selected for review of the case notes (table). Thirty eight case notes were available and were reviewed (*a*) to establish the accuracy of certification with special reference to alcohol and (*b*) to assess the adequacy of the history of alcohol consumption recorded.

Diagnoses on death certificates selected for review of case notes

Diagnosis	No of cases
Cirrhosis of the liver (two patients certified as alcoholic)	4
Carcinoma of the tongue	2
Carcinoma of the oesophagus	2
Carcinoma of the stomach	5
Carcinoma of the pancreas	5
Cardiovascular diseases	17
Cerebrovascular diseases	5

The word alcoholic appeared on only two certificates; these were two of the four patients with a diagnosis of cirrhosis of the liver. All four, however, had established alcoholic liver disease. Two other patients, both with carcinoma of the tongue, had an alcohol intake of more than 100 units a week. Eight case notes were excluded from the study because patients had been too ill to give a history or a standardised history sheet had been used. Of the remaining 30 case notes, 17 (57%) gave a quantitative assessment of alcohol consumption, seven gave qualitative comments, and six gave no history.

A self administered questionnaire covering (*a*) possible reasons for not recording alcohol abuse on death certificates and whether such recording was considered important, (*b*) knowledge of the current coroners' rules concerning alcohol related deaths, and (*c*) the frequency of recording alcohol consumption was answered by 38 of the 43 housemen. The same questionnaire was also answered by 35 of 49 general practitioners in the Aylesbury Vale district of Buckinghamshire, who were selected as a comparison group.

Ninety per cent of both housemen and general practitioners (35 and 32, respectively) considered it important to record alcohol abuse on death certificates. Almost half the doctors in both groups admitted, however, that they would record it in only 50% of relevant cases. Among housemen this reluctance was related mainly to a lack of firm evidence of the aetiological importance of alcohol in individual cases, although nine (31%) also believed that the stigma of alcoholism or the risk of distressing relatives might influence their recording. This number rose to 18 (51%) among general practitioners ($\chi^2=4{\cdot}88$, df=1, $p<0{\cdot}05$). Less than 20% in each group were aware of the current coroners' rules. Almost half the housemen were ignorant of such rules, whereas two thirds of general practitioners thought that the old rules still applied. All but one houseman claimed "almost always" to record details of alcohol consumption routinely, whereas only 21 (60%) general practitioners thought they did so ($\chi^2=13{\cdot}38$, df=1, $p<0{\cdot}001$).

Comment

These results confirm a reluctance to record alcohol abuse on death certificates, despite a general acknowledgment of the importance of doing so. Although housemen claimed "almost always" to record alcohol consumption routinely, almost half the case notes contained inadequate information. Nevertheless, this represents a 50% improvement compared with a similar study six years ago.[4] Ignorance about current coroners' rules remains widespread and may influence recording by both general practitioners and housemen. Routine recording of alcohol consumption in case notes and of alcohol abuse on death certificates must be encouraged to enable accurate statistics relating to alcohol to be generated and used in health care planning.

We thank the house officers and the general practitioners who completed the questionnaire.

1 Taylor D. *Alcohol: reducing the harm*. London: Office of Health Economics, 1981.
2 Morgan MY. Alcohol as a cause of liver disease in the United Kingdom. In: Hall P, ed. *Alcoholic liver disease: pathobiology, epidemiology and clinical aspects*. London: Edward Arnold, 1985:208-25.
3 Maxwell JD, Knapman P. Effect of coroners' rules on death certification for alcoholic liver disease. *Br Med J* 1985;291:708.
4 Barrison IG, Viola L, Murray-Lyon IM. Do housemen take an adequate drinking history? *Br Med J* 1980;281:1040.

(Accepted 9 April 1987)

Academic Department of Psychiatry, University College and Middlesex School of Medicine, London W1N 8AA
GARY BELL, BA, MRCPSYCH, lecturer

Department of Psychiatry, Wexham Park Hospital, Slough SL2 4HL
ANNE CREMONA, MRCPSYCH, consultant

Correspondence to: Dr Bell.

Doctors have no time for alcohol screening

Despite the incidence of alcohol related problems in patients in general hospitals[1] most of those who drink to excess are undetected because doctors fail to take accurate histories of alcohol intake.[2] Evidence suggests that early identification of problem drinkers improves outcome and that brief interventions are beneficial and cost effective.[3] We report our efforts to encourage junior medical staff to incorporate a few brief screening questions on alcohol intake into the medical histories they take from patients.

Patients, methods, and results

Doctors at York District Hospital were encouraged to administer brief alcohol screening questionnaires to patients admitted to nine medical and orthopaedic wards. The questionnaire incorporated items on regular consumption and binge drinking and included modified CAGE questions[4] (copies available on request). Standard units of alcohol were used to measure consumption, one unit equalling half a pint of beer, lager, or cider; a single measure of spirits; a glass of wine; or a small glass of sherry. "Safe levels" were those recommended by the Health Education Council.[5] Staff knew that some of the patients identified as at risk would be offered education about alcohol, the potential benefits of which would be evaluated subsequently.

In the first nine months of the study 32% of all patients admitted who were aged 16 and over were screened for alcohol problems (n=1606). Of those screened, 90 (18%) of the 514 men admitted to the medical wards and 74 (20%) of the 367 men admitted to the orthopaedic wards were "positive" for alcohol related problems. The corresponding figures for women were 12/330 (4%) and 8/395 (2%), respectively. A sample of every fifth admission screened by a researcher (NR) confirmed these results.

There was no screening bias with regard to age or sex although there was with regard to specialty: 39% of patients admitted to the orthopaedic wards were screened compared with 28% of patients admitted to the medical wards. This difference is probably explained by the nature of the admissions, patients in orthopaedic wards generally having non-urgent conditions and therefore being easier to screen than patients on acute medical wards, where there may have been more pressure of time. This impression is confirmed by the poor screening rate in the only acute orthopaedic ward.

Comment

An accurate medical history is probably the doctor's best diagnostic tool. Brief screening questionnaires are more sensitive in detecting alcohol problems than laboratory tests and are quick and relatively cheap.[3] The alcohol screening questionnaire suggested that 18-20% of the men admitted were problem drinkers. The doctors regarded the questionnaire as easy to administer, taking only one or two minutes to complete. Most believed that screening for alcohol problems was worth while and that the questionnaire facilitated this, although the questionnaire was administered to only a third of the patients admitted during the study period. The main reasons given for failure to screen were the patient's condition and pressure of time.

Some doctors screened most of the patients they admitted while others screened few or none at all. Although individual doctors were working on several wards, some of which were regarded as busier than others, their screening rates remained fairly constant, suggesting that the individual doctor's attitude to alcohol problems is an important factor in their

detection. This study showed that questioning patients about their drinking habits was not perceived by doctors as a priority. Many patients who drink to excess therefore remain unidentified and a major reducible cause of disease and death is ignored.

We thank the staff of York District Hospital for their cooperation in this work. The study was supported by a grant from the Health Promotion Research Trust; the opinions expressed are ours, not those of the trustees.

1 Lloyd G, Chick J, Crombie E, Anderson S. Problem drinkers in medical wards: consumption patterns and the disabilities in newly identified male cases. *Br J Addict* 1986;**81**:789-95.
2 Barrison IG, Viola L, Murray-Lyon IM. Do housemen take an adequate drinking history? *Br Med J* 1980;**281**:1040.
3 Babor TF, Ritson EB, Hodgson RJ. Alcohol related problems in the primary health care setting: a review of early intervention strategies. *Br J Addict* 1986;**81**:23-46.
4 Mayfield D, McLeod G, Hall P. The CAGE questionnaire: validation of a new alcoholism screening instrument. *Am J Psychiatry* 1974;**131**:1121-3.
5 Health Education Council. *That's the limit*. London: Health Education Council, 1986.

(Accepted 15 April 1987)

Centre for Health Economics, University of York, York YO1 5DD
N ROWLAND, BA, DIPCOUNS, research fellow
A MAYNARD, BA, BPHIL, professor of economics

Booth Park Hospital, York
A BEVERIDGE, MRCPSYCH, MPHIL, senior registrar in psychiatry
P KENNEDY, MD, FRCPSYCH, consultant psychiatrist
W WINTERSGILL, FFCM, MRCGP, community physician

District Hospital, York YO3 7HE
W STONE, FRCP, consultant physician

Correspondence to: Ms Rowland.

Successful treatment of infertility due to polycystic ovary disease using a combination of luteinising hormone releasing hormone agonist and low dosage menotrophin

Infertility and early loss of pregnancy in women with polycystic ovary disease may be caused by excessively high concentrations of luteinising hormone during follicular development.[1] Different regimens have been suggested to improve the poor outlook for these patients,[2] including the use of "pure" follicle stimulating hormone (Metrodin)[3] and suppression of endogenous luteinising hormone production by treatment with a luteinising hormone releasing hormone agonist before stimulation of the ovaries.[4] Luteinising hormone releasing hormone agonists, given continuously rather than cyclically, reduce gonadotrophin concentrations to prepubertal values. We describe the successful treatment of a patient with polycystic ovary disease and primary infertility of five years duration using a "block and replace" approach.

Case report

A woman started taking the contraceptive pill at the age of 18 because of irregular menstrual cycles and menorrhagia and stopped taking it at the age of 23 because she wished to become pregnant. Her cycles were irregular for 18 months, and a gynaecological opinion was sought. Examination showed that she was slim and non-hirsute. Investigations showed a testosterone concentration of 6·0 nmol/l (normal range in women <2·5 nmol/l), and luteinising hormone was raised throughout the menstrual cycle (37, 45, >50, 24, >50 IU/l); Prolactin concentration was normal, but serial mid-luteal progesterone was <3 nmol/l, confirming non-ovulatory cycles. Clomiphene citrate 50 mg taken on days 2-6 induced regular cycles, but persistently low progesterone values showed that they were non-ovulatory.

Ultrasound examination showed the classic appearance of polycystic ovaries, which were confirmed histologically. Laparoscopy and injection of dye showed that the uterus and fallopian tubes were normal. Clomiphene citrate 100 mg taken on days 2-6 induced an increase in oestradiol concentration to 830 pmol/l with a dominant follicle 17 mm in diameter; however, mid-luteal progesterone concentration remained poor at 15 nmol/l, despite intramuscular injection of human chorionic gonadotrophin 10 000 IU. Treatment with menotrophin (Pergonal), as directed by the manufacturers, was started, but after three ampoules (225 IU follicle stimulating hormone and 225 IU luteinising hormone) on days 3, 5, and 7 oestradiol concentration rose to 2760 nmol/l. Ultrasound examination confirmed moderate hyperstimulation with three to four follicles in each ovary.

After the follicles had regressed a low dosage regimen of menotrophin was introduced using one ampoule (75 IU follicle stimulating hormone and 75 IU luteinising hormone) daily and increasing by half an ampoule each week until a dominant follicle developed. On day 14 there was a single follicle 22 mm in diameter, and oestradiol concentration was 2340 pmol/l. Human chorionic gonadotrophin 5000 IU was given intramuscularly to provide a surge in luteinising hormone, and seven days later the progesterone concentration was 68 nmol/l, suggesting ovulation. Ultrasound examination, however, showed hyperstimulation, with eight cysts 20-30 mm in diameter on the right ovary and four cysts on the left ovary. A similar regimen in the next cycle resulted in a single follicle 18 mm in diameter on day 14; this time no human chorionic gonadotrophin was given. Mid-luteal progesterone concentration was 11 nmol/l, and the follicle regressed. Thus low dose menotrophin treatment induced normal follicular development but no spontaneous ovulation, and human chorionic gonadotrophin induced hyperstimulation.

Suppression of the persistently high luteinising hormone concentrations with a luteinising hormone releasing hormone agonist was attempted. Buserelin was given intranasally four times daily (total daily dose 1200 μg), and luteinising hormone, follicle stimulating hormone, and oestradiol concentrations were monitored. The table shows the rise in oestradiol concentration with initial stimulation and subsequent suppression of gonadotrophins and oestradiol. After one ampoule of menotrophin given intramuscularly each day for 10 days oestradiol concentration was 1840 pmol/l and two follicles, 17 and 20 mm in diameter, were seen on ultrasound examination. Human chorionic gonadotrophin 5000 IU was given intramuscularly, but ultrasound examination confirmed failure of ovulation, with cyst formation (42 and 50 mm in diameter) and a progesterone concentration of 24 nmol/l associated with theca cell luteinisation. This regimen was repeated. On day 11 the oestradiol concentration was 1800 pmol/l, and ultrasound examination showed one follicle 18 mm in diameter and three follicles 10-12 mm in diameter. Human chorionic gonadotrophin 7500 IU was given intramuscularly, and seven days later the progesterone concentration was very high (>100 nmol/l), suggesting conception. Buserelin was withdrawn and a singleton pregnancy was confirmed on ultrasound examination. At the time of writing the pregnancy had reached 30 weeks gestation and was progressing normally.

Concentrations of oestradiol, follicle stimulating hormone, and luteinising hormone before and after treatment with buserelin

	Oestradiol (pmol/l)	Follicle stimulating hormone (IU/l)	Luteinising hormone (IU/l)
Before treatment with buserelin	170	5·3	37
After treatment with buserelin:			
1 week	450	2·9	26
2 weeks	880	0·7	7·1
3 weeks	70	0·8	3·2
6 weeks	80	3·2	2·0

Comment

Our patient's treatment was hampered by her tendency to hyperstimulate with pronounced cyst formation, probably related to her persistently raised luteinising hormone concentration. The combination of suppression of endogenous luteinising hormone production and low dose menotrophin treatment resulted in conception in the second cycle of treatment. This regimen, or one using a long acting luteinising hormone releasing hormone analogue,[5] may be helpful in other patients with polycystic ovary disease.

1 Howles CM, Macnamee CM, Edwards RG, Goswamy R, Steptoe PC. Effect of high tonic levels of luteinising hormone on outcome of in-vitro fertilisation. *Lancet* 1986;ii:521-2.
2 Franks S, Adams J, Mason H, Polson D. Ovulatory disorders in women with polycystic ovary syndrome. *Clin Obstet Gynaecol* 1985;**12**:605-32.
3 Ginsburg J, Sallam H, Scammell G, *et al*. Ovulation and pregnancy after pure FSH therapy in sclerocystic ovarian change. In: Ludwig G, Thompson K, eds. *Archives of gynaecology*. Berlin: Springer Verlag, 1986.
4 Fleming R, Haxon MJ, Hamilton MPR, *et al*. Successful treatment of infertile women with oligomenorrhoea using a combination of an LHRH agonist and exogenous gonadotrophins. *Br J Obstet Gynaecol* 1985;**92**:369-73.
5 Thomas EJ, Jenkins J, Lenton EA, Cooke ID. Endocrine effects of goserilin, a new depot luteinising hormone releasing hormone agonist. *Br Med J* 1986;**293**:1407-8.

(Accepted 9 April 1987)

Endocrine Section, Department of Medicine II, Southampton General Hospital, Southampton SO9 4XY
MARY ARMITAGE, BSC, MRCP, senior registrar
TERENCE WILKIN, MD, MRCP, senior lecturer

Department of Radiology, Southampton General Hospital, Southampton SO9 4XY
KEITH DEWBURY, FRCR, MB, consultant radiologist

Correspondence to: Dr Mary Armitage, Medicine II, Level D, South Block, Southampton General Hospital, Southampton SO9 4XY.