Clinical versus laboratory detection of alcohol abuse: the alcohol clinical index

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Abstract

To determine reliable indicators of alcohol abuse a comprehensive set of clinical and laboratory information was acquired from three groups of subjects with a wide range of drinking histories: 131 outpatients with alcohol problems, 131 social drinkers, and 52 patients from family practice. Findings from clinical examination provided greater diagnostic accuracy than laboratory tests for detecting alcohol abuse. Logistic regression analysis produced an overall accuracy of 85-91% for clinical signs, 84-88% for items from the medical history, and 71-83% for laboratory tests in differentiating the three groups. Further analyses showed 17 clinical signs and 13 medical history items that formed a highly diagnostic instrument (alcohol clinical index) that could be used in clinical practice. A probability of alcohol abuse exceeding 0·90 was found if four or more clinical signs or four or more medical history items from the index were present. Despite recent emphasis on the laboratory diagnosis of alcohol abuse simple clinical measures seem to provide better diagnostic accuracy.

Introduction

Although many patients are excessive drinkers, their alcohol problems often pass unrecognised.[1-3] Problem drinkers consult their doctors at a higher rate than average and tend to use diagnostic facilities more often than the general population.[4 5] The failure to identify alcohol problems may be partly due to the doctor's diagnostic approach. In clinical practice doctors tend to concentrate on physical sequelae of alcohol abuse at the expense of recording a drinking history and relevant psychosocial information.[6] This may impede detecting the young problem drinker, who generally has fewer laboratory or clinical signs of diseases associated with alcohol abuse but often exhibits social and psychological problems related to excessive drinking.[7-9]

The assessment of psychosocial factors using interview and questionnaire methods is of major value in detecting alcohol abuse.[10] Biomedical research has also indicated the usefulness of various laboratory tests, such as γ-glutamyltransferase activity and mean corpuscular volume, as markers of excessive drinking.[11-13] Several recent studies have underscored the diagnostic advantage of combining biomedical and psychosocial data.[14-16] Laboratory tests and clinical signs possess more objectivity for diagnosis than self reports of alcohol problems, which may be subject to denial. Laboratory tests, however, possess only moderate sensitivity for detecting alcohol abuse.[13 14 17] Furthermore, the stratification of clinical signs

of alcohol abuse in terms of their diagnostic importance has received little consideration.[11]

Published reports continue to highlight selected findings on clinical examination that may be related to excessive drinking.[11 18] The possibility that clinical or laboratory indicators may be insensitive or redundant for the detection of alcohol abuse has not been explored in sufficient detail.[19] We therefore studied a comprehensive set of 108 potential indicators of alcohol abuse to establish a short list of clinical and laboratory criteria for identifying alcohol abuse.

Methods

We compiled 108 clinical and laboratory tests from an extensive review of relevant studies[10 11] and used them to examine 314 subjects, who comprised three groups with a wide range of drinking histories (outpatients with alcohol problems, social drinkers, and family practice patients). To evaluate the discriminating power of different tests under ideal conditions 131 outpatients with documented alcohol problems were compared with 131 social drinkers. These groups were matched for age (mean age 38 (11 SD) years) and sex (86 men, 45 women). The outpatients with a broad range of alcohol problems were voluntarily seeking help from the Addiction Research Foundation, Toronto. Their mean daily alcohol intake in the previous six months had been 130 (120·8 SD) g ethanol. They had been excessive drinkers—that is, consuming more than 400 g ethanol/week—for a mean of 7·7 (6·7) years and reported a broad range of consequences related to drinking as assessed by the Michigan alcoholism screening test[20]: mean score 26·3 (9·4), where a score of five or more suggested problem drinking. The social drinkers from the Toronto area consumed fewer than seven drinks (95 g ethanol) per week and no more than two drinks (27 g ethanol) per occasion and had low scores on the Michigan alcoholism screening test: mean 0·3 (2·3).

A group of 61 patients from family practice (Toronto area) were used to make the study results more applicable to routine medical practice. Nine subjects were excluded because they reported excessive drinking (more than 60 g on average per day). This was done to provide a comparison group of patients who were not excessive drinkers. There were no differences (p>0·28) in age and sex between the 52 family practice patients and the other two groups (mean age 37 (15); 29 men, 23 women). The daily intake of alcohol by this group was 13·8 (15·1) g ethanol, and their mean score on the Michigan alcoholism screening test was low at 1·4 (3·9).

Each subject underwent a standardised physical examination that focused on signs associated with excessive drinking.[11] The four doctors conducting the examination were not directly associated with the project, although they were aware of the drinking status of subjects. The 42 clinical signs were elicited in a systematic format and grouped according to the organ or system affected (table I). A self administered medical history questionnaire was developed to include a comprehensive assessment of symptoms associated with alcohol abuse.[11] The 44 items were grouped in table II according to nine clinical domains, and their empirical clustering was verified using item analysis procedures. A comprehensive range of 22 laboratory tests (table III) was performed on a sample of venous blood. The alcohol markers section in table III included tests that are commonly related to excessive drinking.[13-15] Other tests in table III were grouped according to haematology, urea, and electrolytes (sequential multiple analysis 6), or routine chemistry (sequential multiple analysis 12) for comparison with previous investigations of these series.[12] The Michigan alcoholism screening test was administered and alcohol intake was estimated by a standardised, self administered questionnaire. The drinking history was corroborated by a detailed interview in which lifetime alcohol use was documented.[21]

Clinical and laboratory items were compared between groups using either a χ^2 test for qualitative variables or a t test for quantitative variables. Because of the large number of variables evaluated (42 clinical signs, 22 laboratory tests, 44 medical history items) a stringent measure of significance, p>0·001, was used when interpreting univariate statistical tests in tables I, II, and III.

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The diagnostic value of composite indices of clinical measures was investigated using logistic regression analysis from the biomedical computer programs P series statistical software package.[22] This multivariate procedure estimates the probability of alcohol abuse occurring, conditional on a linear function of clinical measures, through the logistic distribution functional form:

$$\text{probability of alcohol abuse} = \frac{e^{\alpha + \beta x}}{1 + e^{\alpha + \beta x}}$$

where e (approximately 2·718) is the base of the natural logarithms, α is a constant, and βx is the weighted linear combination of measures—for example, neurological signs. A cutpoint on the probabilities was chosen that

scores for medical history items were used as input for logistic regression analyses. A predictor variable was considered useful in table V if its logistic regression coefficient divided by its standard error exceeded 2·0.

Results and comment

The prevalence of physical signs associated with alcohol abuse is given in table I. A broader range of clinical findings was noted in the outpatients with alcohol problems than in either the social drinkers or family practice patients. With respect to vital signs, a significant difference ($p<0·001$) was

TABLE I—*Clinical signs*

	Social drinkers (n=131)	Alcohol outpatients (n=131)	Family practice patients (n=52)
	Mean (SD)		
Vital signs:			
Peak expiratory flow rate (l/min)	440·0 (124·9)	348·5 (115·7)	Not measured
Systolic blood pressure (mm Hg)	127·9 (17·3)	130·7 (18·7)	123·4 (14·5)
Diastolic blood pressure (mm Hg)	77·4 (13·6)	82·0 (13·5)	74·8 (10·6)
Pulse rate (beats/min)	73·6 (12·3)	76·3 (10·9)	73·5 (6·5)
Body mass: weight (g)/height ^{2}(cm)	2·49 (0·4)	2·40 (0·4)	2·49 (0·4)
Neurological signs:			
*Hand tremor†	1·3 (0·6)	1·8 (0·7)	1·0 (0·0)
*Tandem gait†	1·1 (0·3)	1·4 (0·7)	1·0 (0·1)
*Deep knee bend†	1·3 (0·6)	1·7 (0·7)	1·0 (0·3)
Reflexes (biceps, knees, ankles)‡	1·5 (0·4)	1·6 (0·3)	1·0 (0·1)
Muscle wasting (arms, legs)†	1·0 (0·1)	1·1 (0·4)	1·0 (0·0)
Limb tenderness†	1·0 (0·2)	1·0 (0·2)	1·0 (0·1)
Glove and stocking anaesthesia†	1·0 (0·2)	1·1 (0·3)	1·0 (0·0)
Liver and abdomen signs:			
*Spider naevi (>5)	4	42	3
*Collateral circulation†	1·1 (0·3)	1·4 (0·5)	1·0 (0·0)
Liver tenderness†	1·0 (0·1)	1·2 (0·5)	1·1 (0·2)
*Gynaecomastia	7%	29%	2%
*Abdomen tenderness†	1·1 (0·3)	1·3 (0·4)	1·1 (0·3)
Liver enlargement§	0·2 (0·8)	1·8 (2·8)	2·7 (4·9)
Spleen enlargement§	0·0 (0·2)	0·1 (0·6)	0·3 (1·7)
Peripheral oedema†	1·0 (0·0)	1·04 (0·2)	1·06 (0·3)
Ascites†	1·0 (0·0)	1·05 (0·2)	1·0 (0·0)
	No (%) present		
Face inspection:			
*Rhinophyma	7 (5)	31 (24)	1 (2)
*Facial erythema	17 (13)	46 (35)	3 (6)
Periorbital oedema	1 (1)	12 (9)	1 (2)
Engorged vessels in conjunctivae	24 (18)	38 (29)	2 (4)
Arcus senilis	16 (12)	20 (15)	0
Oral inspection:			
*Coated tongue	9 (7)	63 (48)	3 (6)
*Oedema of soft palate	24 (18)	71 (54)	3 (6)
Poor oral hygiene	7 (5)	30 (23)	4 (7)
Parotid swelling	5 (4)	20 (15)	2 (4)
Fetor of alcohol	1 (1)	8 (6)	3 (6)
Hand inspection:			
*Nicotine stains	4 (3)	72 (55)	5 (10)
*Palmar erythema	10 (8)	37 (28)	3 (6)
Bitten nails	20 (15)	31 (24)	6 (11)
White nails	29 (22)	42 (32)	2 (4)
Finger clubbing	5 (4)	12 (9)	1 (2)
Dupuytren's contracture	3 (2)	1 (1)	1 (2)
Bruises, scars, and burns:			
*Bruises/abrasions	12 (9)	69 (53)	3 (6)
*Scars (secondary to trauma)	41 (31)	96 (73)	10 (19)
*Cigarette burns	0	35 (27)	1 (2)
*Tattoos	7 (5)	35 (27)	4 (7)
Seborrhoea/dermatitis	35 (27)	29 (22)	1 (2)

*p<0·001 for distinguishing social drinkers from alcohol outpatients, as well as alcohol outpatients from family practice patients (*t* test or χ^2).
†Graded (1=normal, 2=mild, 3=moderate, 4=severe).
‡Graded (1=normal, 2=abnormal); index score is the average for left and right biceps, knees, and ankles.
§Measured in cm below costal margin.

maximised the accuracy of classification for distinguishing patients with alcohol problems from those without (social drinkers or family practice patients). Diagnostic sensitivity, specificity, and predictive power were determined according to standard procedures (see table IV).[23]

A separate logistic regression analysis was conducted within each group of laboratory tests in table III to allow comparisons with previous studies that have examined these tests. The clinical signs and medical history items were grouped by clinical domains (tables I and II), and each group was scored by adding individual results. Selected groups of clinical signs (vital signs, neurological signs, and liver or abdomen signs) were converted into normal deviate scores (mean 0, SD 1) before addition in order to put them into the same metric. The seven summary scores for clinical signs and nine summary

evident between the peak expiratory flow rate of social drinkers and that of outpatients with alcohol problems, and the outpatients had a higher diastolic blood pressure than either the social drinkers or family practice patients ($p<0·01$). Several neurological findings (hand tremor, tandem gait, and deep knee bend) consistently differentiated the outpatients from either social drinkers or family practice patients. Signs of liver disease were also important diagnostic factors (table I). Inspection of the face, mouth, and hands showed several items that clearly distinguished outpatients with alcohol problems from the other two groups. Also clinical signs of past or present injury (bruises, scars, or burns) were common among those who abused alcohol.

The outpatients with alcohol problems had many varied symptoms

associated with alcohol abuse (table II). More than half of the symptoms listed in table II separated the outpatients with alcohol problems from both the social drinkers and family practice patients; alcohol withdrawal symptoms, anxiety, and depression in particular. The laboratory test results are given in table III. Both γ-glutamyltransferase and mean corpuscular volume significantly differentiated (p<0·001) the outpatients with alcohol problems from the social drinkers and family practice patients. On routine blood chemistry, albumin and aspartate aminotransferase activity differentiated excessive from normal drinkers, and the outpatients had a lower mean blood urea nitrogen value than the other subjects.

signs achieved an overall accuracy of 91% for diagnosis, with good sensitivity (93%) and positive predictive value (95%). The most useful diagnostic findings on examination included neurological signs, face and mouth inspection, signs of injury, and liver-abdomen signs. The medical history and routine chemistry had good diagnostic sensitivity (90% and 93%, respectively). Lower specificity was expected because of the greater likelihood of abnormal clinical findings unrelated to excessive drinking among the family practice patients.

The most diagnostic clinical signs and items from the medical history are listed in table VI. This brief list was compiled by identifying which clinical

TABLE II—*Medical history items. Values are numbers (and percentages) positive for each item*

	Social drinkers (n=131)	Alcohol outpatients (n=131)	Family practice patients (n=52)
Trauma history (since 18th birthday):			
*Injured in an assault or fight	8 (6)	64 (49)	7 (13)
Injured in a road traffic accident	17 (13)	41 (31)	10 (19)
Head injury	22 (17)	67 (51)	14 (26)
*Injury after drinking	4 (3)	75 (57)	7 (13)
Fractures or dislocations	29 (32)	71 (54)	17 (32)
Anxiety or depression:			
*Often anxious	39 (30)	88 (67)	19 (37)
*Often depressed	18 (14)	81 (62)	14 (27)
Often has temper outbursts	16 (12)	55 (42)	16 (29)
*Recurrent insomnia	17 (13)	73 (56)	5 (10)
*Contemplated suicide	52 (4)	45 (34)	5 (9)
*Attempted suicide	3 (2)	31 (24)	2 (4)
Gastrointestinal symptoms (frequent):			
Heartburn—indigestion	21 (16)	45 (34)	10 (19)
Difficulty in swallowing	3 (2)	20 (15)	1 (2)
Regurgitation	4 (3)	13 (10)	3 (6)
Acid	8 (6)	30 (23)	8 (15)
Diarrhoea	7 (5)	38 (29)	5 (10)
*Wakes up feeling thirsty	25 (19)	86 (66)	16 (31)
Unpleasant taste in mouth	33 (25)	88 (67)	24 (47)
*Dry coated tongue	9 (7)	56 (43)	8 (15)
*Vomits from drinking	14 (11)	92 (70)	13 (25)
Morning symptoms (often):			
Retches or vomits	1 (1)	22 (17)	1 (2)
*Feels sick	3 (2)	35 (27)	1 (2)
Does not eat breakfast	31 (24)	69 (53)	31 (60)
*Loses appetite	10 (8)	63 (48)	8 (16)
Feels tired	39 (30)	86 (66)	21 (40)
Psychoperceptual withdrawal:			
*Hallucinations	10 (8)	56 (43)	3 (6)
*Memory loss after drinking	13 (10)	11 (84)	7 (13)
*Blackouts after drinking	24 (18)	12 (93)	14 (26)
*Unable to concentrate	18 (14)	71 (54)	10 (19)
*Mental confusion	9 (7)	58 (44)	12 (9)
*Bad at remembering recent events	7 (5)	54 (41)	5 (4)
Psychophysical withdrawal:			
*Delirium tremens	0	42 (32)	0
*Hands shake in morning	1 (1)	42 (32)	1 (2)
*Troubled by frightening dreams	7 (5)	45 (34)	6 (12)
*Headaches	8 (6)	38 (29)	4 (8)
Feels cold or shivers	12 (9)	42 (32)	10 (19)
*Hands often tremble	3 (2)	48 (37)	0
Neuropathy symptoms (one month or more):			
Numbness or tingling in legs	7 (5)	14 (11)	1 (2)
Notable loss of strength in arms or legs	5 (4)	18 (14)	5 (10)
Sexual problems:			
*Experienced sexual problems	10 (8)	37 (28)	4 (8)
Loss of sexual drive	21 (16)	34 (26)	7 (13)
Has had venereal disease	13 (10)	28 (21)	4 (8)
Respiratory symptoms (three months):			
*Coughs most days	12 (9)	50 (38)	5 (10)
*Brings up phlegm	12 (9)	55 (42)	7 (14)

*p<0·001 for distinguishing social drinkers from alcohol outpatients, as well as alcohol outpatients from family practice patients (*t* test or χ²).

Table V summarises results from the logistic regression analyses. In comparing outpatients and social drinkers, we found that the highest overall accuracy of 88% was achieved using items in the patient's history. The clinical domains that were most powerful discriminators included: psychoperceptual withdrawal, psychophysical withdrawal, indicators of trauma, and gastrointestinal complaints (table V). Selected clinical signs also provided useful diagnostic information with an overall accuracy of 85%. These signs included inspection of hands and mouth, signs of past or recent injuries, and findings on abdominal examination. Laboratory tests were less powerful than the medical history and clinical signs in distinguishing the outpatients with alcohol problems from social drinkers (71-76% overall accuracy). The alcohol laboratory markers had good specificity (90%) for ruling out cases, but relatively lower sensitivity (71%) for detecting alcohol abuse.

Table V also summarises the results of comparing alcohol outpatients and family practice patients on the clinical and laboratory measures. The clinical

domains were the most powerful discriminators in the logistic regression analyses. Subsequently, items were selected within these clinical domains that significantly differentiated (p<0·001) alcohol outpatients from both social drinkers and family practice patients (tables I and II). The likelihood that a given number of positive findings would be expected in heavy drinkers was computed, based on the odds ratio.[24] The probability of alcohol abuse in figures 1 and 2 rose steeply with an increase in abnormal clinical findings. With a cut off point of four or more clinical signs (fig 1) or four or more medical history items (fig 2) the probability of alcohol abuse exceeded 0·90.

Further results on diagnostic accuracy are given in table VII. The clinical signs and medical history items had positive predictive values exceeding 90%, which supported their use for diagnosis since few false positive decisions were being made. A low false positive rate is important in clinical practice to avoid errors in identifying normal drinkers as alcohol abusers. These clinical items, however, had lower negative predictive values, which meant that certain cases of alcohol abuse would have been undetected (false

negatives). When the clinical signs and medical history items were combined in the alcohol clinical index (table VII) this diagnostic rule improved sensitivity (89%) and negative predictive value (88-92%) and maintained high specificity (88-92%) and positive predictive value (88-97%). Overall, 88-90% accuracy was achieved by this simple diagnostic rule (clinical signs four or more or medical history four or more).

Discussion

In recent years considerable efforts have been directed at the evaluation of laboratory tests as indicators of excessive drinking

and alcohol abuse,[25] at the expense of considering clinical information.[10 11] Laboratory tests, such as γ-glutamyltransferase and mean corpuscular volume, tend to have specificity for ruling out cases but only moderate sensitivity (30% to 40%) for detecting cases of alcohol abuse in ambulatory populations. Various attempts have been made to improve diagnostic accuracy by combining laboratory tests to form an index. Ryback *et al* have found that a composite index based on commonly ordered laboratory tests (haematology, sequential multiple analysis 6, sequential multiple analysis 12 series) can increase diagnostic accuracy.[12] Other investigations have improved diagnostic accuracy by combining laboratory tests and

TABLE III—*Laboratory test results. Values are means (SD in parentheses)*

Test and reference ranges	Social drinkers (n=131)	Alcohol outpatients (n=131)	Family practice patients (n=52)
Alcohol markers:			
Blood alcohol concentration (mmol/l)	0	9·4 (45·7)	0
*γ-Glutamyltransferase (11-51 U/l men) (7-33 U/l women)	22·0 (20·0)	49·7 (66·6)	24·7 (16·0)
High density lipoprotein (0·8-1·8 mmol/l)	1·4 (0·3)	1·5 (0·3)	1·4 (0·3)
*Mean corpuscular volume (76-96 fl)	88·5 (7·3)	95·2 (6·8)	90·7 (4·5)
Haematology (including mean corpuscular volume):			
Red blood cell count (4·0-6·2×10¹²/l men) (4·0-5·5×10¹²/l women)	4·8 (0·5)	4·8 (0·8)	4·8 (0·4)
White blood cell count (5·0-10·0×10⁹/l)	6·2 (1·7)	7·2 (1·8)	6·8 (2·0)
Haemoglobin concentration (130-180 g/l men) (120-160 g/l women)	144·1 (12·9)	149·2 (17·4)	146·9 (15·2)
Packed cell volume (0·40-0·54 l men) (0·37-0·47 l women)	0·43 (0·07)	0·45 (0·04)	0·44 (0·04)
Platelet count (150-500×10⁹/l)	249·3 (59·0)	239·8 (66·7)	252·3 (84·2)
Urea and electrolytes:			
*Urea nitrogen (3·0-6·4 mmol/l)	5·6 (1·6)	14·3 (1·3)	5·3 (1·6)
Chloride (98-109 mmol/l)	103·2 (2·6)	102·3 (2·9)	102·4 (3·2)
Carbon dioxide (24-32 mmol/l)	27·8 (2·3)	27·9 (2·1)	28·5 (2·4)
Potassium (3·5-5·0 mmol/l)	4·4 (3·3)	4·4 (0·4)	4·5 (0·4)
Sodium (135-150 mmol/l)	140·1 (1·8)	139·7 (2·1)	140·1 (2·3)
Routine chemistry			
Alkaline phosphatase (56-244 U/l)	164·2 (48·8)	206·2 (64·4)	180·7 (46·6)
Total protein (64-80 g/l)	73 (5·0)	75 (5·0)	74 (4·0)
*Albumin (40-50 g/l)	46 (4·0)	48 (3·0)	46 (3·0)
Total bilirubin (2-18 μmol/l)	8·5 (6·8)	6·8 (3·4)	8·5 (3·4)
*Aspartate aminotransferase (8-30 U/l)	21·0 (7·9)	32·5 (38·0)	20·7 (6·5)
Alanine aminotransferase (6-30 U/l)	18·3 (8·7)	33·0 (48·9)	20·1 (8·6)
Urate (172-494 μmol/l)	303 (83)	309 (83)	321 (89)
Cholesterol (3·6-7·0 mmol/l)	5·4 (1·0)	6·0 (1·1)	5·2 (1·0)

*p<0·001 (*t* test) for distinguishing social drinkers from alcohol outpatients, as well as alcohol outpatients from family practice patients.

Conversion: SI to traditional units—Urea nitrogen: 1 mmol/l=2·75 mg/100 ml. Urate: 1 mmol/l=16·81 mg/100 ml. Cholesterol: 1 mmol/l=38·67 mg/100 ml. Bilirubin: 1 μmol/l=0·06 mg/100 ml.

TABLE IV—*Formulas for sensitivity and specificity*

	Group	
	Alcohol outpatient	Normal drinker
Predicted result:		
Alcohol abuse	a	b
Normal	c	d

Sensitivity=a/(a+c), specificity=d/(b+d), positive predictive value=a/(a+b), negative predictive value=d/(c+d), overall accuracy=(a+d)/(a+b+c+d).

questionnaire data.[14 16] For example, an index based on three laboratory tests (γ-glutamyl-transferase, mean corpuscular volume, and high density lipoprotein) and a brief history of trauma achieved a diagnostic sensitivity exceeding 80% while maintaining 90% specificity.[14]

There is a paucity of research that has attempted to use simple clinical information for detecting alcohol abuse.[7 8] Our data from clinical examination provided better diagnostic accuracy than laboratory tests (table V), which are invasive and provide delayed information. We found a set of clinical signs and items in the

TABLE V—*Diagnostic value of clinical and laboratory data using logistic regression analysis*

	Sensitivity %	Specificity %	Positive predictive value %	Negative predictive value %	Overall accuracy %
Distinguishing alcohol outpatients from social drinkers:					
Clinical signs	73	94	91	81	85
Medical history	92	84	85	91	88
Laboratory tests:					
Haematology	84	71	78	77	78
Urea and electrolytes	68	74	73	71	72
Routine chemistry	82	70	71	79	76
Alcohol markers: γ-glutamyltransferase, mean corpuscular volume, high density lipoprotein, blood alcohol concentration	71	90	79	89	71
Distinguishing alcohol outpatients from family practice patients:					
Clinical signs	93	87	95	83	91
Medical history	90	69	88	73	84
Laboratory tests:					
Haematology	92	37	76	69	74
Urea and electrolytes	98	16	74	80	74
Routine chemistry	93	58	84	78	83
Alcohol markers: γ-glutamyltransferase, mean corpuscular volume, high density lipoprotein, blood alcohol concentration	84	50	76	63	72

TABLE VI—*Alcohol clinical index*

Clinical signs*	Medical history items*
Hand tremor	Inability to concentrate
Tandem gait	Troubled by mental confusion
Deep knee bend	Difficulty in remembering recent events
Spider naevi (>5)	Hallucinations
Collateral circulation	Hands shake in morning
Gynaecomastia	Troubled by frightening dreams
Abdominal tenderness	Wake up with a headache
Rhinophyma	Hands often tremble
Facial erythema	Injured in an assault or fight
Coated tongue	Wake up feeling thirsty
Oedema of soft palate	Dry coated tongue
Nicotine stains	Cough on most days
Palmar erythema	Bring up phlegm
Bruises or abrasions	
Scars (secondary to trauma)	
Cigarette burns	
Tattoos	

*Definitions and instructions for using the alcohol clinical index may be obtained by writing to the first author (HAS).

Alcoholism is impractical for routine use in clinical practice,[18] and our study reinforces a previous finding that many of the council's criteria are redundant for the diagnosis of alcohol abuse.[19] A clinical examination based on the Le Go grid has been widely used in France for the early detection and medical diagnosis of alcoholism.[26] Only limited data, however, are available on the diagnostic validity of the Le Go method.[27] The combination of psychosocial and medical data can improve diagnostic accuracy over use of questionnaire methods alone.[78] Our study indicates that certain clinical findings can be used to complement psychosocial or laboratory data for improving the identification of alcohol abuse.

Further study of the alcohol clinical index is encouraged in different populations and clinical settings. (A form giving details of how to administer the index is available from HAS.) Our outpatients with alcohol problems averaged under 40 years of age (roughly two thirds between 27 and 49) and had a wide range of drinking histories and alcohol related problems. With older patients a reduction in diagnostic specificity might be observed because of

TABLE VII—*Diagnostic value of alcohol clinical index*

|	Sensitivity %	Specificity %	Positive predictive value %	Negative predictive value %	Overall accuracy %
Distinguishing alcohol outpatients from social drinkers:					
Clinical signs (cutpoint ≥4)	75	93	92	79	84
Medical history (cutpoint ≥4)	69	94	92	75	82
Clinical signs ≥4 or medical history ≥4	89	88	88	88	88
Distinguishing alcohol outpatients from family practice patients:					
Clinical signs (cutpoint ≥4)	75	99	99	61	81
Medical history (cutpoint ≥4)	69	94	97	56	77
Clinical signs ≥4 or medical history ≥4	89	92	97	92	90
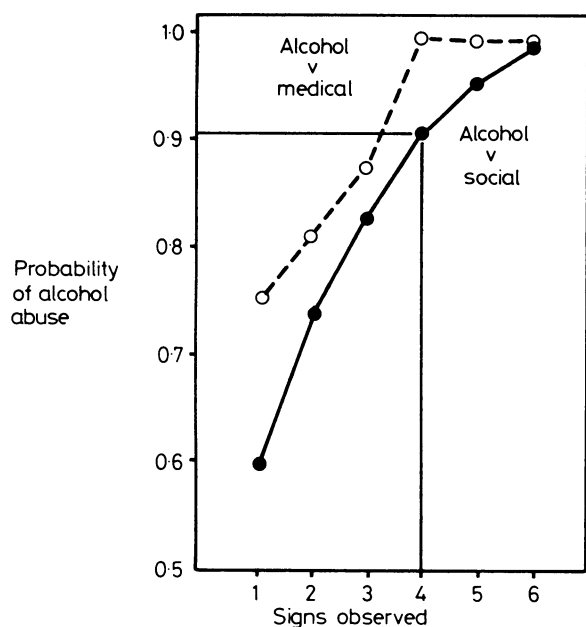
FIG 1—Probability of alcohol abuse estimated at different cutpoints of clinical signs observed on the alcohol clinical index. Outpatients with alcohol problems were compared with social drinkers and with family practice patients.
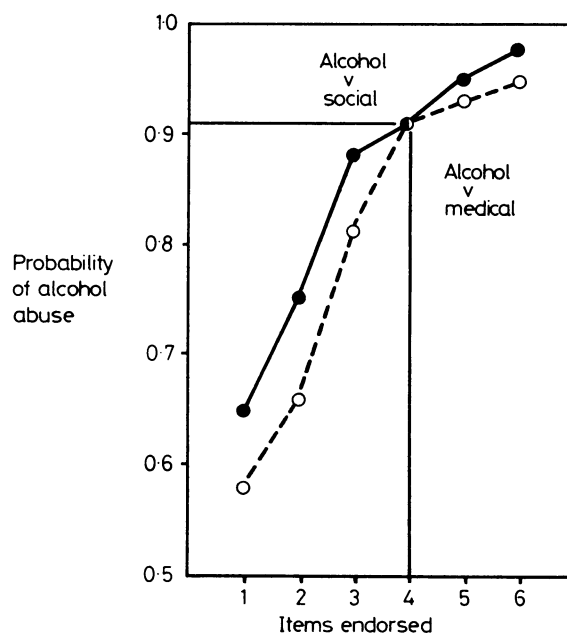

FIG 2—Probability of alcohol abuse estimated at different cutpoints of medical history items endorsed on the alcohol clinical index.

medical history (table VI) that could discriminate the outpatients with alcohol problems from social drinkers and family practice patients. This brief alcohol clinical index yields information that is highly diagnostic of alcohol abuse (table VI). These findings underscore the diagnostic value of taking a careful medical history and eliciting a short list of clinical signs.

Several attempts have been made to standardise methods for the diagnosis of alcoholism using physiological and clinical data. The extensive list of criteria prepared by the National Council on

the increased likelihood of some abnormal clinical findings unrelated to alcohol abuse. Accordingly, higher cut off points than four clinical signs or four medical history items may be necessary in an older population for obtaining reasonable diagnostic accuracy. With a predominately younger population—for example, early 20s—that has fewer abnormal clinical findings,[79] lower cut off scores on the alcohol clinical index might be necessary for maintaining diagnostic sensitivity. The present study used matched (by age and sex) samples of ambulatory subjects who were representative of social drinkers as well as outpatients in alcohol

treatment clinics and in general practices. Additional investigations of the alcohol clinical index are planned in other contexts, such as specialist medical wards (for example, gastroenterology and psychiatry) and emergency service departments, where one might expect a higher prevalence of excessive drinking and alcohol related morbidity among patients.

The present study has provided a comprehensive comparison of the relative diagnostic value of clinical and laboratory information for the identification of alcohol abuse. Our findings underscore the value of selected items from the medical history and clinical signs, which can be combined to form an objective index. Previously, incomplete knowledge of the diagnostic power of specific clinical items has prevented firm recommendations about indicators of excessive drinking.[11] Our advice is to administer the alcohol clinical index routinely during clinical examination and to corroborate it by a brief questionnaire on alcohol problems such as the Michigan alcoholism screening test or CAGE (cut down on drinking, annoyed by others criticizing your drinking, guilt feelings over drinking, eye-opener or drink in the morning),[15 20 28] as well as by laboratory tests determining mean corpuscular volume and γ-glutamyltransferase activity.[14] This practical strategy could make significant inroads on identifying drinking problems that often remain undetected in medical wards and general practices.

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Does super efficient starch absorption promote diverticular disease?

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Abstract

The amount of starch escaping absorption in the small intestine was measured in eight patients with symptomatic diverticular disease and eight controls. Unabsorbed starch was calculated from breath hydrogen measurements after a potato meal compared with the hydrogen response to lactulose. The proportion of unabsorbed starch was low in all the patients (mean 3·3%) and was only about a quarter of that in the controls (12·4%; p<0·01).

These findings confirm that unabsorbed starch provides an important quantity of carbohydrate reaching the colon and suggest that super efficient starch absorption, by reducing this provision, may promote the development of diverticular disease.

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Introduction

Recent work has shown that dietary starch is not completely absorbed by the small intestine.[1 2] The degree of malabsorption of starch varies with different food sources.[1 3] Furthermore, one recent study found wide variation in starch malabsorption among people eating the same meal, with between 2% and 20% of the starch escaping absorption in the small intestine.[2]

There is considerable evidence that a low intake of dietary fibre predisposes to diverticular disease.[4-6] Fibre is the main exogenous source of colonic carbohydrate. In view of the possibility that unabsorbed starch may also contribute a sizable quantity of carbohydrate to the colon we hypothesised that people who absorb starch particularly well, such that little reaches their colon, might be more at risk of diverticular disease. We call this better than average starch absorption "super efficient."

Subjects and methods

We studied eight patients with symptomatic diverticular disease. They had presented complaining of lower abdominal pain, with or without altered bowel habit, and had at least 10 colonic diverticula seen on barium enema.